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Sequencing and Prediction of Adolescent Soft Drug Initiation: Systematic Review, Quantitative Investigation, and Dual Cross-Validation

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SEQUENCING AND PREDICTION OF ADOLESCENT SOFT DRUG INITIATION: SYSTEMATIC REVIEW, QUANTITATIVE INVESTIGATION, AND DUAL CROSS-VALIDATION

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In providing a comprehensive investigation of alcohol, cigarette, and marijuana initiation among adolescents, the overarching goals of the research were to build upon extant findings and address important literature gaps. A four-fold purpose founded the study: 1) provide a systematic review of the soft drug initiation literature; 2) test a modified version of Kandel's (2002) drug sequencing hypothesis; 3) determine if predictors of soft drug initiation differ in kind or saliency by biological age and drug type; and 4) examine age- and drug-specific determinants of the timing at which initiation occurs. Supplemental attention also was directed at evaluating the utility of Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

Through the quantitative component of the research, nine hypotheses were tested. Cross-sectional data were derived from a rural sample of 6th, 9th, and 12th grade students who completed the 2004 Primary Prevention Awareness, Attitude, and Use Survey (PPAAUS). All of the hypotheses obtained some degree of support; more support was yielded for the specific risk factor hypothesis than the common factor model, and convincing evidence was

obtained for Kandel's drug sequencing hypothesis and Petraitis et al.'s distal-proximal mediation hypothesis.

The findings also supported the bulk of the directional hypotheses and several of the direct and indirect effects propositions put forth in social learning theory, the social development model, and the theory of planned behavior. In contrast, the results called into question some of the direct effects articulated in Hirschi's original statement of social control and underscored some possible limits of the social development model.

In an effort to gauge the validity of the findings, a dual cross-validation scheme was employed. The systematic review cross-validation involved comparing the quantitative results for two major hypotheses to those yielded from the systematic review. Through a further quantitative cross-validation, the hypotheses findings were compared to those derived from a sample of 6th, 9th, and 12th grade students who completed the 2001 PPAAUS. On balance, a relatively strong degree of convergence was obtained. This confluence served to bolster the reliability and validity of the results. Policy and programmatic implications also were indicated.

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For Lucas, Mazzy, Katie, and all other like children.

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CHAPTER 1

INTRODUCTION

In the grocery store, a 5-year-old boy selected wine and asked, *"Is this alcohol?"* The researcher replied, *"Yes,"* and the boy said, *"I want it, and I want some smokes."*

At the check-out counter, a 3-year-old girl identified the cigarettes she was buying. Camels: *"Animal ones for Daddy."* Marlboros: *"Mommy smokes these."*

(Dalton et al., 2005)

Quite perceptive, some children as young as age 3 already have begun to develop an awareness of (and cognitive expectation for) alcohol and cigarette use. In a recent adult role-playing study (Dalton et al., 2005), substantial percentages of preschoolers (2 to 6 years of age) purchased cigarettes (29%) and alcohol (62%) at a toy grocery store in preparation for a make-believe evening with friends. Disconcerting are the findings that about half of these children correctly identified alcohol (58%) and cigarettes (50%) by brand name, with some children more adept at identifying the names of these products than the names of those that are more age-appropriate, such as snacks and cereal. Dalton et al.'s (2005) findings not only speak to the ability that young children have for internalizing and emulating general social cues to which they have been exposed; these researchers also found that compared to children whose parents self-reported cigarette and alcohol abstinence, children whose parents drank on a monthly basis or smoked were 3 and 4 times more likely, respectively, to purchase alcohol and cigarette products.

Given the multiple cultural forces (e.g., media, pharmaceutical corporations, and the alcohol, tobacco, and music industries) that glamorize, celebrate, and encourage drug use, along with the prevalence of soft drug use among adults and the emerging finding that parents constitute the #1 source of alcohol for a considerable number of adolescents (American Medical Association [AMA], 2005), it is not surprising that young children are developing a keen awareness of the central role that these drugs play in the lives of many parents and adults (see, e.g., Cieply, 2007; Dombrink, 1993; Grube, 2004; Jurgensen, 2007; Strasburger, 1995; Wakefield, Flay, Nichter, & Giovino, 2003; Watson, 2005). Although troubling, it also is hardly a surprise that national and statewide epidemiologic drug surveys conducted over the past 15 years suggest as many as 10% of typical 4th graders in the U.S. already have initiated alcohol use (Donovan, 2007). In an effort to understand why cognitive expectations concerning soft drug use may develop in American children by 5 years of age, it is important to place the issue of soft drug use within a historical context.

A Cultural Mainstay

To begin, as the oldest known psychoactive drug in the U.S. and the world, alcohol has been firmly embedded in American culture since its first migration aboard Puritan ships (Inaba & Cohen, 2004). Today, alcohol remains a focal point of American life, from its incorporation into holiday traditions and religious ceremonies, to social gatherings, weekday happy hours, and weekend parties.

The current legal drinking age in the U.S., 21 years, is the highest of any country in the world (Babor et al., 2003). The prohibition of alcohol to minors first began in the mid 1800s with the enactment of various state laws. The push for establishing these laws originated from various puritanical temperance groups in the early 1800s, whose interest was to restrict both adult and youth access to alcohol (Alcohol and Tobacco Tax and Trade Bureau, 2006). Major subsequent curtailments of legal access to alcohol included the enactment of the 1920 National Prohibition Act and the passage of the 18th Amendment to the U.S. Constitution. After state ratification, the federal government repealed Prohibition in 1933 with the passage of the 21st Amendment. Although the federal ban against the manufacturing, transportation, and sale of alcohol was lifted, prohibition laws remained intact in many states. States without these laws allowed for the sale of alcohol to adults, while restricting the sale of alcohol to anyone under the age of 21 years.

After the 26th Amendment was enacted in 1971, and those between the ages of 18 and 21 years were afforded the right to vote, states began lowering minimum drinking ages from 21 years to 20, 19, and 18 years (Inaba & Cohen, 2004). Influenced by federal coercion, states then reversed their legal drinking ages in the 1980s, increasing them back to 21 years (Humfleet, Munoz, Sees, Reus, & Hall, 1999). Decisions to increase the legal drinking age were founded on concerns about the impact of alcohol consumption on youth psychosocial development, along with the strong correlation found between lowered drinking ages and motor vehicle accidents and fatalities (Cook & Tauchen, 1984).

In comparison to alcohol, tobacco use in the U.S. began as early as 1 B.C., when American Indians utilized tobacco leaves in religious rituals and medicinal practice (Borio, 2005). Since 1847, when the first cigarette was sold in the U.S. by a newly founded British company, Phillip Morris, cigarette production and sales have evolved into one of the most profitable businesses ever established in the U.S. (Randall, 2006).

First influenced by the 1964 U.S. Surgeon General's report on the negative health consequences of cigarette smoking, restrictions on cigarette advertising and use have become increasingly stringent in modern times. Today, all tobacco advertising on television and radio is banned; smoking is prohibited on all commercial airline flights; many states restrict smoking in public establishments; and tobacco companies also are mandated to inform the public of the health dangers associated with smoking (Randall, 2006). Although there is no federal law mandating that tobacco products only be sold to persons of a specified minimum age, coercive encouragement by the federal government has aided in the passage of legislation in all 50 states requiring the sale of tobacco products only to persons over the age of 18 (Inaba & Cohen, 2004).

Finally, mankind has been using marijuana for at least 4,000 years (Abadinsky, 2001). Not only has it been used for its euphoric properties and ability to produce a "high," but also for its medicinal properties in countries such as China and India, and in areas of South America, the Middle East, and southern Africa (Abadinsky, 2001). In the U.S., marijuana first was grown and used as a source of fiber during Colonial Times. Over time, Americans began utilizing it to treat various health conditions and illnesses, such as rheumatism and labor pains during childbirth (Joy, Watson, & Benson, 1999).

The criminalization of marijuana has been a cornerstone of federal drug policy, starting with the 1914 Harrison Narcotics Act (Gray, 2001). Through the passage of the Controlled Substances Act (CSA), a subsidiary Act of the Comprehensive Drug Abuse Prevention and Control Act (CDAPCA) of 1970, marijuana was deemed a Schedule I substance (Drug Enforcement Agency [DEA], 2006). In general, Schedule I drugs are viewed as having no medicinal value and being dangerous in terms of the potential for abuse and dependence (DEA, 2006).

In an effort to centralize federal drug enforcement under one agency, the DEA was established in 1973 (Abadinsky, 2001). Since its inception, the DEA has been responsible for the federal enforcement of drug laws and interdiction efforts. The “war on drugs,” which initially began in the 1970s at the direction of President Nixon, was employed in full force from the 1980s, under the Reagan administration, through at least the early 1990s, under the Bush administration. This “war on drugs” and the associated “zero tolerance” approach were geared toward all illicit substances, including marijuana. During this time, mandatory sentences for drug possession were re-introduced, and drug enforcement and interdiction initiatives increased, as did arrests for drug offenses, including marijuana possession (Abadinsky, 2001; Inaba & Cohen, 2004).

Although the “war on drugs” still continues in various capacities today, increasing tolerance of marijuana use has led to amplified public support for its legalization. Although its use remains a federal crime, 13 states have passed one or more medical marijuana laws or have provided for exceptions to existing state laws (Gray, 2001; Pacula, Chriqui, & King, 2004). Several cities also have decriminalized the possession of small amounts of marijuana, including Seattle, San Francisco, and Denver (Osher, 2005).

Overall, while various safeguards regulating (or prohibiting) the use of soft drugs have been established, alcohol, cigarettes, and marijuana remain firmly embedded in American culture, lifestyle, and mindset. The fact that the use of these drugs dates back prior to our nation's founding, and as such is not likely to abate in the future, is not a valid argument, however, for ignoring or encouraging soft drug use among youth. As shown in the next section, the rationale that underlies efforts to prevent (or delay) adolescent use is justified strongly by the associated negative consequences.

A Social and Public Health Problem

A social problem has been characterized as a "situation incompatible with the values of a significant number of people who agree action is needed" (Rubington & Weisberg, 1989, p. 4). In modern times, the impetus for changing a social condition deemed as unacceptable or intolerable typically is founded upon epidemiological assessments of its consequences and scope. Both are key ingredients requisite for elevating a behavior from a minor, tolerable phenomenon to one of major societal concern (Rubington & Weisberg, 1989). Once a condition or behavior is pushed to the forefront of public consciousness, and consensus is reached concerning proposed change, corrective and preventive action typically is taken to narrow the gap between the "real" and "ideal" condition.

Adolescent drug use has been and remains a substantial social and public health problem in the U.S. Like any other major social issue, the importance of treating adolescent soft drug use as a public health priority and directing efforts toward studying and preventing (or delaying) this behavior lie predominantly in its negative consequences and scope.

Consequences

All actions have consequences, either positive or negative. Although there are various psychosocial and medicinal benefits associated with alcohol and marijuana use, arguments for directing efforts at preventing (or at least delaying) adolescent soft drug initiation are founded largely on the plethora of negative health and social consequences associated with drug use.

Psychosocial and Medicinal Benefits

The majority of American alcohol consumers are law-abiding citizens who actively contribute to society, raise healthy families, and are able to control their alcohol use. Like alcohol consumption, cigarette use is a legally proscribed behavior, with individuals 18 years and older free to indulge in a “smoke” if they desire. Nicotine, the active ingredient in cigarettes, is an addictive substance, but one that typically does not interfere with normal day-to-day functioning (Imperato & Mitchell, 1986). Although marijuana use is a federal crime and is illegal in most states, epidemiological data indicate that many adults who smoke marijuana are only occasional users and do not use in a way that would be characterized as abuse or dependence (Johnston, O’Malley, Bachman, & Schulenberg, 2007). These observations underscore the fact that not only do individuals react differently to the psychoactive effects of drugs, but the consequences of soft drug use are not always negative.

With the exception of cigarettes, there are several medicinal and physiological benefits found to be associated with moderate soft drug use. Among other things, moderate alcohol use by adults has been linked with a lower risk of coronary heart disease (Zakhari, 1997) and a reduced risk for dementia in older adults (Orgogozo et al., 1997). Although the medicinal use of marijuana is prohibited at the federal level and in many states, those coping with illnesses (such as AIDS and multiple sclerosis) and those undergoing cancer chemotherapy report an increase in appetite and a reduction in pain and nausea through marijuana use (Joy, Watson, & Benson, 1999). Others have reported physiological benefits, including enhanced feelings of relaxation.

Economic Toll

Despite the psychosocial and medicinal benefits associated with moderate alcohol and marijuana use, from an economic perspective, drug use and abuse has been referred to as “one of the most costly health problems in the U.S.” (Office of National Drug Control Policy [ONDCP], 2004, p. xiii). From all accounts, it appears these costs are not decreasing, but rather have been increasing over time. For instance, the total economic cost of drug abuse over a recent 10-year time period increased almost 70%, from $107.5 billion in 1992 to $180.8 billion in 2002.

Mortality

In addition to economic costs, soft drug use is implicated in an alarmingly high rate of preventable deaths each year in the U.S. For example, as the "leading cause of preventable death" (National Institute on Drug Abuse [NIDA], 2001, p. 3), tobacco is implicated in more annual deaths (445,000 on average) than the combined number of annual deaths attributable to AIDS, suicide, homicide, accidents (such as motor vehicle crashes and fire), alcohol, cocaine, and heroin use (Centers for Disease Control and Prevention, 2002; NIDA, 2001).

In addition, alcohol use is indicted in the annual deaths of more than 130,000 people in the U.S., a sixteen-fold difference compared to the 8,000 total American deaths each year attributed to the use of all illegal drugs. On the adolescent front, 31% of drivers between the ages of 15 and 20 years who died in traffic accidents in 2003 were drinking alcohol either while they were driving or shortly before they got into their motor vehicles (National Highway Traffic Safety Administration, 2004).

Finally, similar to cigarette smoking, there exists no documented case in the U.S. (or the world) of a fatal marijuana overdose. Evidence does suggest that marijuana use may be an indirect cause of death; however, the number of these deaths pales in comparison to those for which alcohol and cigarettes are implicated (ONDCP, 2004). For example, the ONDCP (2004) reports that five people died in 1999 and three in 2000 due to various "mental and behavioral disorders" stemming from the ingestion of tetrahydrocannabinol (THC), the major psychoactive chemical found in marijuana (p. B-12).

Morbidity

Soft drug use also is implicated in a considerable number of preventable illnesses and diseases. Alcoholic liver disease, the 12th leading cause of death in the U.S., shortens the average lifespan between 9 and 22 years (Yoon, Yi, & Hilton, 2005). Moreover, compared to nonsmokers, cigarette smokers are 7 times more likely to develop mouth, lung, and throat cancer, and they are at an increased risk for heart disease and stroke (Blot, 1992; NIDA, 2001).

The number of marijuana-related illnesses also has increased in recent years. Between 1995 and 2002, for example, the number of patients seen by emergency department personnel for marijuana-related illnesses increased 164%, from 45,259 to 119,474 (Office of Applied Studies [OAS], 2003a). Furthermore, 16% of emergency room patients seeking treatment for marijuana-related illnesses in 2002 were between 6 and 17 years of age (OAS, 2003).

Drug Abuse and Dependence

It is important to recognize that drug initiation is a key first step in the progression to more serious forms of drug use, including abuse and dependence. Both simply are not possible without first initiating use (Stein, Newcomb, & Bentler, 1987).

Adolescence, the developmental period between the onset of puberty and the assumption of adult roles and responsibilities, is a time of particular vulnerability to the effects and consequences of drug use. The human brain, which generally reaches maturity when individuals reach their 20s (Giedd, 2004), is still developing throughout the teenage years. Youth who continue psychoactive drug use not only are exposed to toxic chemicals at a time in which the human brain is growing and developing, but they are subsequently exposed for longer periods of time than people who initiate use in adulthood, when the brain is fully developed. Youth who initiate drug use during childhood or early adolescence, and then develop patterned use, also may contend with homeostasis and drug tolerance at a relatively early age. These physiological processes taking place in the developing brain subject adolescents to significant risk for drug dependence (see, e.g., Brown, Tapert, Granholm, & Delis, 2000; Crews, Braun, Hoplight, Switzer, & Knapp, 2000; DeBellis & Clark, 2000).

The goal of preventing (or at least delaying) soft drug initiation among youth has great appeal, particularly given the plethora of research indicting early age of drug initiation as a salient risk factor for future drug use, abuse, dependence, and a variety of conduct problems (see, e.g., Bentler, 1992; Breslau & Peterson, 1996; Grant & Dawson, 1997, 1998; Hinson, Heeren, & Winter, 2006a, 2006b; Newcomb & Bentler, 1988; Robins & Przybeck, 1990). Epidemiological research suggests that the majority (65%) of adolescents and adults admitted to drug treatment facilities for abuse and dependence in 2005 self-reported adolescence as the time period during which they initiated the drug for which they were seeking treatment (OAS, 2006b). Moreover, among youth admitted to drug treatment programs in 2005, 57% of those 15 years and younger, and 65% of those between 15 and 17 years of age, were being treated for marijuana abuse alone.

Delaying the initiation of alcohol use until 21 years of age appears beneficial on both physiological and psychological grounds. Of the estimated 14 million adults in the U.S. who abuse or are dependent on alcohol, 95% are estimated to have initiated prior to 21 years of age (Dawson & Grant, 1998; OAS, 2004a). With the onset of alcohol dependence peaking by 18 years of age (Hingson et al., 2006b; Li, Hewitt, & Grant, 2004), delaying initiation until the legal drinking age potentially could decrease risk for abuse and dependence by as much as 60% (Grant & Dawson, 1997). In contrast, compared to youth who wait until they are in their early 20s to initiate alcohol use, adolescents who initiate by 15 years of age are five times more likely to abuse alcohol or become dependent (Grant & Dawson, 1997).

Similarly, adult smokers who initiate cigarette use during adolescence are less likely to stop smoking than adult smokers who began during adulthood (Wiencke, Thurston, Kelsey, Varkonyi, & Wain, 1999). Unfortunately, roughly 80% of adult smokers self-report the early and mid-adolescent years as the time period in which they initiated smoking, and research suggests that at least 75% of adolescent smokers will continue smoking as adults (CDCP, 2002a; Johnston, Bachman, & O'Malley, 1992).

Other Problem Behaviors

Aside from the increased risk for drug abuse and dependence that early age of soft drug initiation poses, adolescent soft drug initiation also is associated with a decreased likelihood of graduating from college, and an increased likelihood of selling drugs, stealing, and engaging in other delinquent and criminal acts (see, e.g., Babor, Caetano, Casswell, Edward, Giesbrecht, & Graham, 2004; Dembo, Schmeidler, Taylor, & Burgos, 1982; Loeber, 1988). There also are strong correlations between adolescent soft drug use and engagement in a variety of other problem behaviors, including early parenthood, violence, poor school performance, and school truancy and drop-out (see, e.g., Derzon & Lipsey, 1999a; Ellickson, D'Amico, Collins, & Klein, 2005; Ellickson, Tucker, Klein, & Saner, 2004; OAS, 2006a; Penning & Barnes, 1982). Among both adolescent and adult drug users, polydrug use also is quite prevalent. Among young people between the ages of 18 and 25, for example, 31% of daily cigarette smokers engaged in illicit drug use in 2000, compared to 28% of past-month smokers and just 7% of nonsmokers (OAS, 2001a).

One of the major reasons why the health and social consequences of soft drug use are so severe is because alcohol, cigarette, and marijuana use are three of the most prevalent forms of psychoactive drug use among both adults and adolescents. The following discussion directs attention to the scope of this problem.

Epidemiology

Some have argued that the illicit status of a drug has lost its value in modern-day society as both a practical and moral deterrent (Baumrind & Moselle, 1985). It is difficult to counter this argument, given the prevalence of soft drug initiation among youth. Not only do more American adults use alcohol, cigarettes, and marijuana than all other psychoactive drugs combined, but soft drug use also is the most prevalent form of psychoactive drug use among juveniles, despite the fact that such drug use is illegal (OAS, 2001b).

Concerning past month use, for example, data from the 2005 National Survey on Drug Use and Health (NSDUH) indicated that among 12-17 year olds, 17% consumed alcohol, 11% smoked cigarettes, and 7% smoked marijuana within the past 30 days (OAS, 2006a). Rates of lifetime soft drug initiation among youth also are concerning. Findings from the 2006 Monitoring the Future survey suggest that among 12th grade students, as many as 73% initiated alcohol use, nearly 50% initiated cigarette use, and slightly more than 40% initiated marijuana use within their lifetime (Johnston et al., 2007).

Recent epidemiologic rates of soft drug initiation and use among both adult and adolescent populations (see e.g., Johnston, O'Malley, & Bachman, 1996; OAS, 2005; OAS, 2006a; O'Malley, Bachman, & Johnston, 1988) reveals six overall issues of central importance. First, of crucial significance is the long-standing observation that the incidence of soft drug use is an age-graded, curvilinear phenomenon. Specifically, the incidence of drug use during young childhood is low. As youth transition to adolescence, however, incidence rates increase dramatically. By the time adolescents' age into young adulthood, many or most already have initiated soft drugs (OAS, 2006a). Some abstainers in this population initiate during early adulthood, but rates of initiation at this time are considerably lower than during adolescence. As this population continues to age, a very small segment of remaining abstainers initiate use. Levels of soft drug initiation after 26 years of age are extremely low and pale in comparison to levels in adolescence and, to a lesser extent, young adulthood (OAS, 2006a).

Second, the differential prevalence of alcohol, cigarette, and marijuana initiation and use among adolescents mirrors that found among adults. Regardless of age, race, or gender, higher rates of prevalence and incidence are observed for legal and more socially accepted soft drugs (alcohol and cigarettes) than for marijuana, with alcohol constituting the most commonly initiated and used drug of the three (OAS, 2006a).

Third, similar to changes in adult rates of soft drug use, the prevalence and incidence of adolescent soft drug use rise and fall over time, often in response to aggregate-level changes in the perceived risks and benefits that adolescents ascribe to particular soft drugs, along with changes in social acceptance and approval of their use (O'Malley et al., 1988). Throughout the 1990s and over the past decade, the initiation of alcohol and cigarette use among 8^{th}, 10^{th}, and 12^{th} grade students generally has declined, while lifetime marijuana initiation generally increased throughout the 1990s before declining in more recent years (Johnston, O'Malley, Bachman, & Schulenberg, 2006). While these downturns are welcome, current initiation and use levels are still very high compared to those observed in the early 1990s (Johnston et al., 1996).

Fourth, although cross-sectional and descriptive in nature, several different types of epidemiologic data suggest that involvement in soft drug use is fairly stable, sequential, and hierarchical phenomena. The most common form of soft drug initiation is alcohol, followed by combined alcohol and cigarette use, and then combined alcohol, cigarette, and marijuana use (OAS, 2005). Although the descriptive nature of the data preclude definitive statements of temporal ordering from being made, data from the 2004 NSDUH, concerning the average age of alcohol, cigarette, and marijuana initiation (14.8 years of age, 14.9 years of age, 15.1 years of age, respectively), suggest that among adolescents who initiate all three soft drugs, alcohol use appears to have been initiated first by many adolescents, followed by cigarette initiation, and then the initiation of marijuana use (OAS, 2005). Together, these patterns of soft drug initiation suggest that increasing involvement in more serious drug use is a hierarchical phenomenon, with the number of adolescent initiates becoming successively smaller as each additional soft drug is initiated.

Fifth, rates of adolescent soft drug initiation vary along major demographic lines, including gender, race, and age. Since the mid-1980s, a prior gender disparity in adolescent soft drug use has changed, with near gender convergence in marijuana initiation and a reversal in gender disparity for alcohol and cigarette initiation (OAS, 2005). Today, not only is alcohol and cigarette initiation slightly more common among adolescent females than males, but females also are initiating soft drug use at slightly younger ages than their male counterparts (Johnston et al., 2006).

In addition, white adolescents generally have lower use and initiation rates than American Indian youth, but higher use and initiation rates than Hispanic, black, and Asian youth (OAS, 2005). Asian adolescents have the lowest rates, while rates among black and Hispanic youth generally fall in the middle.

Finally, adolescence is a time period marked by radical change in drug-related behavior. In general, 12-13 year olds constitute the smallest proportion of past year soft drug initiates, while 16-17 year olds constitute the largest proportion. Alcohol and cigarette initiation typically occurs in 9^{th} grade, while marijuana initiation is most apt to occur in 10^{th} grade (Johnston et al., 2006). Recent data on the age of soft drug initiation among younger juveniles is particularly concerning. The 2004 NSDUH estimate that among 12-13 year olds who used soft drugs in 2003, roughly 50% initiated alcohol, cigarette, and marijuana use by 12 years of age or younger (OAS, 2005).

Utility of Prevention and Research

While preventing (or delaying) the initiation of soft drug use among adolescents appears to be a formidable challenge, it is a worthwhile endeavor. The utility of preventing or delaying soft drug initiation among adolescents may be argued on three major fronts.

First, the use of psychoactive drugs by people of all ages is an issue that warrants monitoring; however, drug use during adolescence should be approached with considerably more concern. Although indicators of rates of soft drug incidence among youth provide a more positive trend in recent years, the downturn in alcohol, cigarette, and marijuana initiation is not as large as that observed for other delinquent acts (Johnston et al., 2007). Moreover, although a statistically normative behavior, the initiation and use of soft drugs among adolescents carries high human and social costs.

Second, and in a related vein, preventing or delaying soft drug initiation also is appealing given the considerable literature demonstrating the likelihood of early drug initiation as a risk factor for the initiation and use of other drugs, as well as future drug abuse and dependence. With respect to the former concern, Kandel and Yamaguchi's (2002) cross-sectional analysis of 1994-1995 NSDUH data for 18-40 years olds (N = 21,000) is illuminating. The ages of alcohol, cigarette, and marijuana initiation among self-reported heroin users were several years younger than the ages of initiation for these drugs among adults who did not initiate heroin use. For instance, among adults who reported cigarette and marijuana initiation only, the average age of cigarette initiation was 14.6 years, while the average age of cigarette initiation among alcohol and cigarette only initiates was 15.8 years of age. In contrast, heroin users, who almost always reported cigarette initiation, reported first using cigarettes at 12.6 years of age (Kandel & Yamaguchi, 2002).

DiFranza and colleagues' (2007) four-year longitudinal study of early adolescent cigarette use also speaks to the potential for drug abuse and dependence. Among the 1,246 6th grade students interviewed and surveyed, nicotine addiction typically emerged prior to students reaching two cigarettes per day. Among cigarette smokers, 35% self-reported losing control over their use within just 30 days of initiating.

Third, preventing soft drug initiation among adolescents has the capacity to effect positive change in rates of soft drug use prevalence and frequency. For example, MTF trend data for marijuana use among 12th grade students points to the relationship between soft drug initiation and frequency of use (Johnston, O'Malley, & Bachman, 1998). The prevalence of marijuana use peaked in 1978 among high-school seniors, when 60% reported having initiated marijuana use at one point in their lives. Of these youth, 20% reported daily marijuana use. In 1992, at the lowest level of 12th grade marijuana use, 33% reported lifetime marijuana use, and only 1 in 20 of these 12th graders reported daily marijuana use. In 1997, when 45% reported lifetime use, slightly more than 10% were daily users.

As Johnston et al. (1998) point out, while the proportion of 12th grade students who initiated marijuana use had declined almost 50% between 1989 and 1992, the proportion of daily marijuana users among the 12th grade marijuana-using population had declined by 300% during this same time period. In contrast, as the proportion of marijuana initiates among high-school seniors increased by more than 33% between 1992 and 1997, the proportion of daily marijuana users among all seniors doubled in that same time period. These data demonstrate that periods of increased rates of overall marijuana use are associated with concomitant or delayed increases in rates of frequent marijuana use. Therefore, successful efforts to prevent or delay adolescent soft drug initiation may be fruitful in terms of controlling rates of frequent soft drug use.

Taking into consideration that soft drug use is more common among American youth than not, and it has remained a relatively permanent fixture in American culture, mindset, and the extracurricular activities of a sizeable number of youth, some researchers have taken a Hirschian (Hirschi, 1969) stance toward the issue, arguing…"we might well ask why some adolescents abstain, rather why most do not" (Baumrind & Moselle, 1985, p. 44). While this contention does hold some weight, the attendant human and social costs of soft drug use, particularly for that occurring during late childhood and early adolescence, continue to justify further research on the topic and associated prevention efforts.

The current study is grounded in a consequentialist perspective of human behavior, approaching the issue of adolescent soft drug use in a fashion similar to Hobbes' approach to life (see Figure 1). Taking a long-term perspective, the current research centers on examining predictors of soft drug initiation. Given the prevalence of soft drug use and its potential negative consequences, it is vital that continued efforts be directed at understanding the relevant causal factors implicated in initiation. This understanding then can inform and facilitate primary prevention programs designed to prevent or delay adolescent soft drug use.

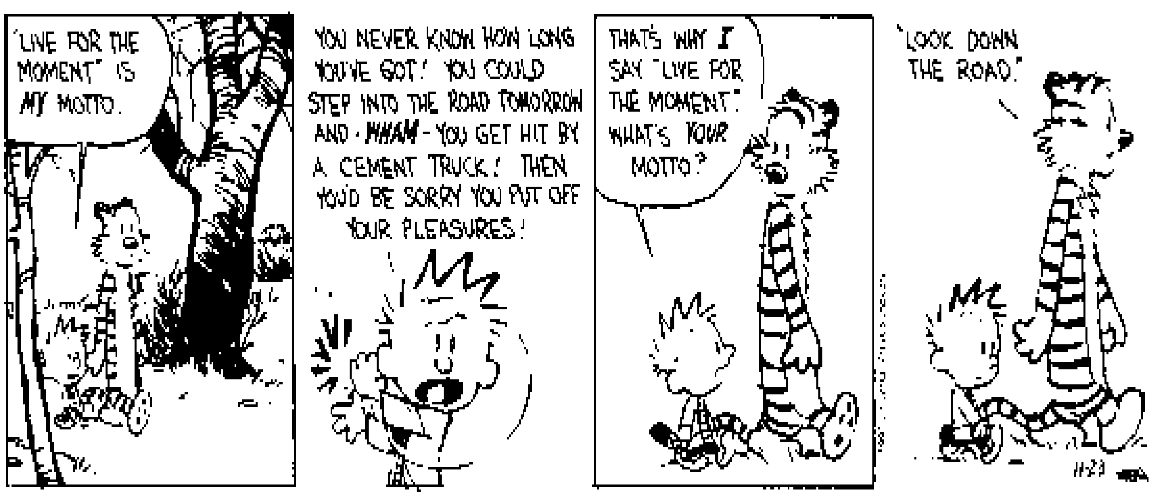

Figure 1. Hobbes gives Calvin some sound advice, by Bill Watterman, 1994. © 1990 Universal Press Syndicate. Reprint permission granted through education provision.

Prevention Science and Attendant Guideposts

Since the mid-1990s, efforts directed at understanding the relevant causal factors implicated in soft drug initiation have fallen under the purview of prevention science. This field of study emerged with an integration of epidemiology, life course development research, and prevention intervention trials (Coie et al., 1993). Founded largely on theoretically-guided risk and protective factor research, prevention science translates basic research into practice in an effort to prevent or condition the development of health-risk disorders and problem behaviors, including adolescent soft drug use. Three major concepts are central to the field of prevention science, as well as the current research. These concepts deal primarily with issues related to risk and protection and adolescent development.

Risk and Protective Factors

The primary guiding premise underlying prevention science is that reducing health-risk or problem behaviors necessitates that malleable precursors or contributory factors be identified and reduced. Reducing these precursory influences, known as risk factors, while bolstering or introducing protective factors that reduce the likelihood that negative outcomes will occur, constitutes the most promising strategy for preventing or delaying the initiation of soft drug use among adolescents (Hawkins, Catalano, & Miller, 1992a).

In general, a risk factor is any precursory influence that increases the probability that a harmful behavior will be initiated, an existing problem condition will be maintained, or a more serious (and negative) state will be experienced

(Coie et al., 1993; Catalano & Hawkins, 1996; Gottfredson, 2001). A more distinctive definition offered by Hawkins and colleagues similarly infers an increased likelihood of developing some type of problem; risk factors are “those characteristics, variables, or hazards, that if present for a given individual, make it more likely that this individual, rather than someone selected from the general population, will develop a disorder” (Pollard, Hawkins, & Arthur, 1999, p. 145).

In contrast to risk factors, protective factors have been conceptualized as “conditions in the individual or environment that mediate or moderate the effects of risk factors or increase resistance to them, and thus inhibit the development of problems even in the face of risk exposure” (Howell & Hawkins, 1998, p. 275). Implicit in this definition is the notion that protective factors can operate in three distinctive ways: as main effects variables, mediating constructs, or moderating variables.

As a main effects variable, the presence of a protective factor may directly increase the likelihood of soft drug abstention, or decrease the likelihood that soft drug initiation will occur (Luthar, 1991; Masten, 1987). A protective factor also may constitute a mediating variable that intervenes or lies between a risk factor and soft drug abstention, reducing a risk factor’s impact on the likelihood that initiation will occur (Baron & Kenny, 1986). Finally, as a moderating variable, a protective factor interacts with a risk factor in determining the effect of risk exposure on the likelihood of drug initiation. When protective factors operate as moderator variables, they specify under what conditions or circumstances a risk factor will have an effect on the likelihood of soft drug initiation (Kirby & Fraser, 1997; Pollard et al., 1999; Waller, Okamoto, Miles, & Hurdle, 2003).

Matrix of Influence

Bronfenbrenner's (1979, 1989) typology of ecological systems provides a conceptual frame of reference for organizing risk and protective factors. This typology assumes that behavior is influenced by social contexts and the intrapersonal attributes (e.g., states and traits) that individuals bring to those settings. An individual never is an entity separate from social systems. Rather, individuals always are a part of some environmental system.

Bronfenbrenner's typology highlights the principle of equifinality[1] and documents the central systems in which adolescents interact. The family, peer group, school, and community are the primary social settings in which children and adolescents participate in activities, assume roles, and engage in interpersonal relationships. Youth bring to these environmental contexts a set of personality and dispositional traits and states that influence how they interact with and perceive their environments. In this respect, individuals may be conceived as an ecological system themselves.

Conceptualized as a matrix of influence that is nested and hierarchical in organization, not only does each system (or domain) have a unique influence on youth, but these domains are interrelated, whereby one system can have an impact on other systems, and multiple influences within various systems can work together in impacting adolescent behavior. This nested, hierarchical

[1] Equifinality is the notion that there are a myriad of pathways to a given problem behavior, not a unique, single causal pathway (Glantz, 1992; Tarter & Mezzich, 1992). Applied to adolescent soft drug use, initiation is rooted in multiple causes, or a variety of different causal chains.

organization of social systems serves as a natural prioritization of social influences. According to ecological theory, risk and protective factors found within the ecological domains more proximal to adolescents (e.g., family and peers) have a stronger influence on adolescent behavior than risk and protective factors that emanate from more distal social systems, such as schools and the larger community (Bronfenbrenner & Ceci, 1994; Resnick & Burt, 1996). Guided by this ecological-developmental perspective, prevention science emphasizes the importance of identifying and examining changes in prominent domains of influence and associated risk and protective factors as youth grow older and social interaction increases.

Purpose of the Research

The information discussed earlier in this chapter suggested three major questions that were worthy of further investigation. These questions established the focus of the current research:

RQ#1: Is involvement in soft drug use a sequential and hierarchical phenomena? If it is, what is the typical sequence of involvement?

RQ#2: What factors predict soft drug initiation and age of initiation among adolescents?

RQ#3: Since the initiation of soft drug use appears to vary, in part, as a function of biological age, what factors predict soft drug initiation and age of initiation for youth at different stages of adolescence?

The present study constitutes a comprehensive assessment of adolescent soft drug initiation. The overarching goals of the research were to reaffirm and build upon extant research findings, address important gaps in the literature, and provide several contributions to the prevention science knowledge base. A four-fold purpose founded the research: (1) provide a comprehensive assessment of the soft drug initiation literature; (2) test a modified version of Kandel's (2002) drug sequencing hypothesis; (3) determine if predictors of soft drug initiation differ in kind or saliency by biological age and drug type; and (4) examine the timing of soft drug initiation by biological age and drug type, in terms of predictors that distinguish early versus later ages of initiation.

The first line of research involved conducting a systematic, comprehensive review of the extant literature on soft drug initiation among adolescents (1970s-2007). Over the past 20 years, comprehensive reviews of the adolescent drug literature have emerged as a vital tool for continually updating this large body of literature and directing attention toward remaining gaps and issues that are empirically ambiguous and worthy of redress. An assessment of extant comprehensive reviews published between the 1970s and the 2000s revealed that the majority of these reviews have not focused on (nor attended to) issues that are empirically important to the field of prevention science today (e.g., drug-specific and age-specific risk factors). The current study addressed this inadequacy by using Research Questions #2 and #3 as a guiding framework for assessing 35 empirical studies on predictors of adolescent soft drug initiation and time to initiation.

The remaining avenues of inquiry were addressed through the examination of data derived from 6th, 9th, and 12th grade students who completed the 2004 Primary Prevention Awareness, Attitude, and Use Survey (PPAAUS). In testing a modified version of Kandel's (2002) drug sequencing hypothesis, adolescent initiation of alcohol, cigarette, and marijuana use was examined in terms of the degree to which each constitute a distinct stage that together captures a latent continuum of drug involvement. In support of Kandel's hypothesis, there is considerable research evidence to suggest that drug involvement constitutes a continuum that is hierarchical in nature, whereby progression along the continuum is experienced by successively smaller numbers of people. Legal drug use typically is initiated prior to marijuana use, while marijuana use typically is initiated prior to hard drug use among those who do progress to harder drug use (Kandel, 1975b). Some empirical ambiguity exists, however, concerning the temporal ordering in alcohol and cigarette initiation (Brook, 1993; Donovan & Jessor, 1983; Huba, Wingard, & Bentler, 1981).

The notion of stages in drug involvement has direct implications for primary prevention initiatives designed to prevent or delay drug initiation. If most youth initiate legal drugs prior to marijuana, the initiation of legal drugs (along with consequent risk factors) may serve as a salient risk factor for marijuana initiation. Prevention programs that direct efforts toward reducing risk factors endemic to legal drug use may indirectly work toward preventing (or delaying) marijuana initiation, since marijuana initiation may be less likely if legal drug use has not been initiated.

The current research also sought to identify predictors of soft drug initiation among adolescents. Within this third area of inquiry, two issues were of particular interest. An attempt was made to determine the degree to which predictors universally predict soft drug use, regardless of biological age or drug type. Specifically, an effort was directed toward determining if predictors are (1) drug-specific, whereby they differ in kind according to drug type, and (2) age-graded, whereby they differ in kind by stage of adolescent development. Two traditional theories (social control theory and social learning theory) and one developmental theory (social development model, SDM) informed this line of inquiry. SDM provided the basis for examining age-graded risk and protective factors.

The purpose of this aspect of the research was to gain some insight into the degree to which equifinality exists and the common factor hypothesis holds for the initiation of different types of soft drugs. Research in this area is mixed with regard to common versus drug-specific risk factors. Some studies have found that various risk factors universally predict all three types of soft drugs, regardless of age, with differences lying primarily in strength of salience (Allen, Donohue, Griffin, Ryan, & Mitchell Turner, 2003). Other studies indicate that risk factors for soft drug initiation are age-specific (Hawkins, Catalano, & Miller, 1992a) and drug-specific (Kandel, 1980b). Results stemming from this line of research also have prevention implications. If risk factors do vary by age or drug type, any effective primary prevention program must be tailored to the age of its audience and the type of drug use the program wishes to prevent or delay.

Finally, soft drug initiation was examined in terms of the timing (i.e., age) at which it occurs. There is considerably less existing research in this area, compared to that which has been conducted on differences between drug initiates and abstainers. By examining soft drug initiation in terms of the age at which it occurs, it is possible to assess if risk factors for the timing of initiation differ in kind or saliency according to drug type or the age group under study.

In an effort to gauge the degree to the validity of quantitative findings, a dual cross-validation scheme was employed. The systematic review cross-validation involved comparing the quantitative results to those generated from a systematic review of the literature on adolescent soft drug initiation. The quantitative cross-validation involved comparing the findings to those obtained from a sample of 6^{th}, 9^{th}, and 12^{th} grade students who completed the 2001 PPAAUS.

Collectively, the following chapters are designed to frame the focus, theoretical lens, and methodological underpinnings of the current research. Chapter 2 begins an examination of etiological explanations for adolescent soft drug use. Three traditional theories (i.e., social control theory, differential association theory, social learning theory) are discussed in terms of their major explanatory constructs, processes, and empirically verified risk and protective factors that are most frequently integrated into developmental theories of adolescent drug use.

Chapter 3 then directs attention to two developmental theories: Kandel's (2002) stage theory and Hawkins and colleagues' (Catalano & Hawkins, 1996) social developmental model. These theories attempt to address one or more of the three research questions posed earlier in this chapter. Extant empirical research is discussed in Chapter 4, including support for Kandel's drug sequencing hypothesis and a general overview of research on predictors of adolescent soft drug initiation.

Chapter 5 presents the methodology underlying the systematic review and describes the first component of the dual cross-validation. Chapter 6 directs attention to the methodology and hypotheses driving the quantitative component of the research and addresses the second element of the dual cross-validation.

Chapters 7-10 present the research findings. Chapter 7 introduces the results of the systematic review, while Chapter 8 details the univariate and bivariate findings. Chapter 9 reveals the multivariate results of the quantitative component of the research, and the dual cross-validation results are presented in Chapter 10. Finally, Chapter 11 discusses the findings and implications of the research, situating results within the larger context of traditional and developmental theory, adolescent drug policy and programming, and prevention science. In closing, the study limits are presented, as are recommendations for further research.

CHAPTER 2

TRADITIONAL ETIOLOGY

The previous chapter provided some conceptual guideposts (e.g., risk, protection, and ecological domains of influence) that found prevention science, risk factor research, and most developmental theories of adolescent drug use. Developmental theories are the products of theoretical syntheses, or the integration of empirically verified risk and protective factors derived from traditional theories of delinquency. Many of the constructs and propositions found in developmental theories are conceptually anchored in the theories of social control (Hirschi, 1969), differential association (Sutherland & Cressey, 1978), and social learning (Akers, 1977), three of the most prominent and empirically supported traditional theories of adolescent delinquency (see, e.g., Costello & Vowell, 1999; Kaplan et al., 1984; Matsueda & Heimer, 1987; Oxford, Harachi, Catalano, & Abbott, 2000; Sampson & Laub, 1990; Voss, 1964).

The current chapter focuses on these three theories in an effort to trace the traditional roots of developmental theory; draw attention to the strengths and inadequacies of traditional explanations; and highlight the importance of viewing adolescent behavior through a developmental lens. Social control theory is discussed first, followed by differential association theory, and then social learning theory. As each respective theory is discussed, attention is paid to major propositions and key explanatory constructs, as well as empirically verified strengths and weaknesses. Where appropriate, issues of particular relevance for the current research are highlighted. In conclusion, the importance of taking a developmental approach to the etiology of adolescent drug use is couched within a summary of the major drawbacks endemic to traditional explanations.

Social Control Theory

In his seminal work, Hirschi (1969) brought the concept of control (specifically social control) to the forefront of criminology. Hirschi's social control theory (a.k.a., social bond theory) is based upon the assumptions of hedonism (i.e., hedonistic calculus), free will, and the notion of value consensus. Hirschi argued that the motivation to commit crime should not be the focus of explanation or study, since people do not vary on this innate drive. Born with an equal dose of self-interest (i.e., hedonism), all individuals weigh the costs and benefits of their actions, and tend toward involvement in behaviors that elicit pleasurable and self-serving consequences, or actions that elicit maximum self-serving benefits. From a risk-protection perspective, deviant motivation may be conceptualized as an invariant and unalterable risk factor. Since all adolescents possess an equal dose of deviant motivation, all are equally at risk for drug initiation. Recognizing that not all people commit deviant acts, Hirschi sought to explain the factors that restrain deviant motivation, thereby decreasing the likelihood of deviant behavior.

Hirschi (1969) posited that law-abiding behavior results from an investment in conformity. In turn, this stake in conventionalism results from the development of a social bond that individuals develop and maintain with agents and institutions of social control. It is through the development of this bond that a sense of moral and individual obligation develops, whereby individuals feel a responsibility for upholding and adhering to the dominant value system of society. As the social bond develops, engaging in deviant behavior becomes a risky endeavor, since doing so may jeopardize existing relationships.

The social bond consists of four elements: attachment, commitment, involvement, and belief (Hirschi, 1969). Attachment constitutes the emotional dimension of the social bond, and refers to the degree of emotional or relational connection between an individual and important others and institutions (e.g., parents, peers, teachers, family, school). Commitment represents the rational dimension of the social bond, and refers to the degree of time and energy invested in prosocial goals (e.g., educational aspirations) and conventional lines of action (e.g., work or education). Involvement, the social dimension of the social bond, refers to the level at which people participate in conventional activities, such as recreation, hobbies, dating, schoolwork, or employment. Belief constitutes the moral dimension of the social bond, and is conceptualized as a general belief in normative standards and values, such as a respect for law and a belief that laws should be obeyed.

Hirschi posited that an inverse relationship exists between the social bond and deviance. When one or more elements of the social bond become weakened, the strength of the social bond weakens, which, in turn, decreases one’s stake in conformity. Once a decrease in conformity occurs, the probability of deviant behavior increases, since deviant motivation no longer is restrained. Hirschi isolated families, peers, and schools as having the most profound impact on the behavior of youth, positing that delinquency (including drug use) is a likely outcome of ineffective ties to these social units. Specifically, drug initiation is likely to occur among adolescents who are weakly attached to parents, peers, and school; not highly committed to education or schooling, nor involved in conventional activities; and who lack a strong sense of respect for and belief in the legitimacy or moral authority of law. While all four elements of the social bond are important, Hirschi prioritizes attachment, particularly attachment to parents, as the most important element of the social bond.

Empirical Validity

Parsimonious, with fairly straightforward and testable propositions, Hirschi's social control theory is among one of the most frequently tested theories within criminology and remains at the forefront of traditional explanations of delinquency. Although technically a theory of conformity that attempts to explain why individuals do not commit deviant acts, the title of Hirschi's (1969) first theoretical statement, *Causes of Delinquency*, inferred that social bonding could be used to explain delinquency as well. Almost all empirical tests have followed suit, explaining delinquency in terms of weak bonding.

Overall, research has provided moderate empirical support for the theory, underscoring the importance of individuals and institutions in which youth most frequently and closely interact: parents, peers, and schools. Weak bonding to school and prosocial parents and peers has been linked to a variety of adolescent deviant behaviors, including early sexual activity, gang membership, delinquency and drug use, school drop-out, and academic problems during adolescence (Catalano, Haggerty, Oesterle, Fleming, & Hawkins, 2004;

Cernkovich & Giordano, 1992; O'Donnell, Hawkins, & Abbott, 1995; Wade & Brannigan, 1998).

Although Hirschi (1969) touted social control theory as a general theory of deviance capable of explaining (in totality) all forms of delinquent behavior among all segments of the adolescent population, a large body of literature has demonstrated that this general explanation should be tempered with several qualifications. Weak prosocial bonding has consistently been shown to explain more variation in minor than serious forms of delinquency; more variation in deviant behavior among lower-class versus middle- or upper-class youth; and more variation in delinquency among white females relative to minority females and males (see, e.g., Agnew 1991a; Akers & Cochran, 1985; Bahr, Marcos, & Maughan, 1995; Cernkovich & Giordano, 1992; Hirschi, 1969; Krohn & Massey, 1980; Marcos & Bahr, 1995; McGee, 1992; O'Donnell et al., 1995; Simons-Morton, Crump, Haynie, & Saylor, 1999; Wiatrowski & Anderson, 1987). In addition, evidence demonstrating that the theory is more powerful in predicting delinquency using cross-sectional versus longitudinal data underscores questions concerning the unidirectional causal ordering that Hirschi postulates (Agnew, 1991a; Akers, 1994).

Empirical tests typically find that weak social bonding explains no more than 45% of the variation in delinquency, drug use, teen pregnancy, school-drop, and violence (see, e.g., Agnew, 1991b; Akers & Cochran, 1985; Cernkovich & Giordano, 1992; Danzinger, 1995; Dull, 1984; Hawkins, Graham, Maguin, Abbott, Hill, & Catalano, 1997; Hirschi, 1969; Krohn & Massey, 1980; Macros & Bahr, 1995; Skinner, Massey, Krohn, & Lauer, 1985; Wade & Brannigan, 1998). Although this amount of explained variance is relatively high according to social science standards (Hansen & Collins, 1994; Junger-Tas & Marshall, 1999), a substantial amount of variation in adolescent antisocial behaviors is left unexplained. When tested against other traditional theories (e.g., strain, differential association, and social learning theories) using the same data, bonding variables typically explain a smaller proportion of variance in delinquency and drug use than peer influence variables (e.g., delinquent peer association, norms, and reinforcement) derived from differential association and social learning theory (see, e.g., Agnew, 1991a; Akers & Cochran, 1985; Bahr et al., 1995; Dembo, Grandon, La Voie, Schmeidler, & Burgos, 1986; Matsueda, 1982; Mears & Field, 2002). Detailed below, several major empirically verified limitations serve as evidence that weak bonding alone fails to provide an adequate explanation of delinquency. This inadequacy has prompted researchers to integrate key bonding constructs with explanatory factors from other traditional theories, particularly differential association and social learning theory.

Direct Effect Propositions

In general, empirical tests indicate that two of the four bond elements, commitment and belief, are directly related to delinquency, and in the expected direction. With few exceptions (Dull, 1984), research typically finds that youth who are committed to conventional institutions and ideals, and who subscribe to the belief that the moral authority of law is legitimate, are less likely to engage in delinquency and drug use (Hindelang, 1973; Krohn & Massey, 1980; Marcos & Bahr, 1995).

Hirschi's (1969) propositions concerning the protective qualities of involvement and attachment have been met with substantial qualification and question (in some instances). Generally, research finds weak support for the protective qualities that Hirschi contends involvement in prosocial activities elicit (Hindelang, 1973; Rankin, 1976; Wiatrowski, Griswold, & Roberts, 1981). In fact, more often than not, research finds counter support, with involvement in prosocial lines of action constituting a risk factor for engagement in minor forms of delinquency and soft drug use (Agnew, 1991a; Browning, Thornberry, & Porter, 1999; Catalano et al., 2004; Cernkovich & Giordano, 1992; Dishion, McCord, & Poulin, 1999; Hawden, 1999; Krohn, Massey, Skinner, & Lauer, 1983; Krosnick & Judd, 1982; Marcos & Bahr, 1995).

In an effort to explain this relationship, researchers point out that Hirschi did not address the intervening processes by which social bonding elements are hypothesized to take effect (Dishion et al., 1999; Hawden, 1999). For instance, Dishion and colleagues (1999) posit that involvement in prosocial activities may increase risk for delinquency because increased involvement brings with it more opportunities to interact with and become attached to deviant peers who can influence adolescent behavior (Dishion et al., 1999). This possibility speaks to one of the major weaknesses of social control theory: Hirschi's proposition concerning attachment.

A considerable body of literature levels a substantial qualification against Hirschi’s (1969) proposition concerning the direct, inhibitory qualities endemic to the attachment element of the social bond. Contrary to Hirschi’s hypothesis, the type of individuals to which youth are attached matters. Attachment to (or association with) delinquent peers (see, e.g., Conger, 1976; Ensminger, Brown, & Kellam, 1982; Elliott et al., 1985; Hindelang, 1973; Krohn & Massey, 1980; Krohn et al., 1983; Marcos & Bahr, 1988) and deviant parents (see, e.g., Allen et al., 2003; Dembo et al., 1986; Jensen & Brownfield, 1983; McDermott, 1984) are salient risk factors for adolescent delinquency and drug use.

In the social control literature, delinquent peer association typically has been interpreted as a proxy measure of delinquent peer attachment, although learning theorists argue that delinquent peer association is not a control theory construct, but a learning theory construct (Akers, 1977). Nonetheless, associating with and being attached to delinquent peers is one of the strongest predictors of delinquency and drug use identified since delinquency research began (see, e.g., Berndt, 1979; Empey & Lubeck, 1971; Forslund & Gustafsen, 1970; Hindelang, 1973; Hirschi, 1969). This risk factor also has been found to be considerably more predictive of delinquency than any single element of social bonding (see, e.g., Agnew, 1993; Dembo et al., 1986; Krohn & Massey, 1980; Matsueda, 1982; Mears & Field, 2002; Skinner et al., 1985). Detailed below in a discussion of social learning theory, the positive association between delinquent peer association and adolescent deviant behavior may be the result of social learning processes.

Hirschi (1969) hypothesizes that a direct relationship exists between each element of the social bond and delinquency. Noted throughout this discussion, research generally supports this straightforward account. A growing body of longitudinal research demonstrates, however, that for many youth, the causal processes outlined by social control theory may only capture a portion of the causal pathway leading to delinquency. Specifically, social bonding also can occur in an interactive and sequential fashion that unfolds over time. For example, research demonstrates that the direct relationship between family bonding and delinquency, risky behavior, and drug use can be partially explained by the mediating qualities of school bonding (Kumpfer & Turner, 1990; Sommers, Fagan, & Baskin, 1994; Wade & Brannigan, 1988; Williams, Ayers, Abbott, Hawkins, & Catalano, 1999).

There also is considerable evidence that variables from other theories can mediate or condition the impact of bonding on delinquency, lending support for the notion that deviant motivation may not be an invariant phenomenon as Hirschi (1969; Gottfredson & Hirschi, 1990) argues. Concerning mediation processes, delinquent peer association (and other peer variables from social learning theory) has been shown to mediate the link between family bonding (e.g., parental attachment) and delinquency (see, e.g., Agnew, 1993; Brown et al., 2005; Krohn & Massey, 1980; Krohn, Skinner, Zielinski, & Naughton, 1989; Kumpfer & Turner, 1990; Marcos, Bahr, & Johnson, 1986; Massey & Krohn, 1986; Wiatrowski et al., 1981). A few longitudinal studies even have demonstrated that delinquent peer association mediates all of the impact of social bonding variables on delinquency (Elliott et al., 1985; Matsueda, 1982).

Concerning moderation processes, weakly bonded youth are more likely to engage in delinquency if they associate with delinquent peers (see, e.g., Agnew, 1993; Akers, 1992; Elliott et al., 1985; Thompson, Smith-DiJulio, & Matthews, 1982; Warr, 1993a, 1993b). Although these findings underscore the importance of prosocial bonding for reducing delinquency, Hirschi's (1969) direct effect propositions infer a straightforward bond-delinquency relationship that is not entirely supported by research.

Reciprocation

Mentioned earlier, cross-sectional tests of social control theory tend to produce more favorable findings than those derived from longitudinal, prospective data (Akers, 1994; Agnew, 1991a). This observation speaks to the possibility that the causal ordering specified by Hirschi (1969) concerning the relationship between low bonding and delinquency may not accurately depict all operable causal processes. Longitudinal and cohort sequential research demonstrate that a reciprocal relationship exists between delinquency and bonding, with weak bonding leading to delinquency, and delinquency in turn leading to further bonding attenuation (see, Blankmeyer, Flannery, & Vazsonyi, 2002; Bryant, Schulenberg, Bachman, O'Malley, & Johnson, 2000; Liska & Reed, 1985). Liska and Reed's (1985) longitudinal analysis, for example, found that delinquency serves as a mediator, whereby delinquent peer attachment increases risk for delinquency, which, in turn, is directly related to decreased school attachment.

A limited number of studies also have found that soft drug use is a better predictor of hard drug use than the attachment and belief elements of the social bond (Marcos & Bahr, 1995; Taub & Skinner, 1990). For example, when soft drug use was entered into Marcos and Bahr's (1995) model predicting hard drug use, the explained variance in hard drug use attributed to social bonding variables decreased, while the total explained variance in hard drug use increased from 8% to 24%. All of these findings underscore that social control theory may better explain the initial occurrence of delinquent behavior than the development (e.g., escalation, de-escalation, or desistance) of a single form of behavior (e.g., involvement in drug use) or delinquent career. As Farrington (1986, 2003, 2005) repeatedly has pointed out, any theoretical explanation of the further entrenchment and escalation of a delinquent career (or form of deviant behavior) must take into account the effect that initial deviance has on the risk factors that contributed to its occurrence. Social control theory fails to provide this developmental account.

Age Differences

Hirschi (1969) did not explicitly address the age-crime relationship, or explain if (and how) uniform-type changes in levels and types of social bonding typically occur as youth transition from childhood to adolescence, or from adolescence to early adulthood (Maddox & Printz, 2003). Developmental research suggests that changes in interpersonal influence typically occur during the adolescent years, whereby the predominant salience of parental influence during childhood declines as peer influence increases (Krosnick & Judd, 1982). Social control research also indicates that levels of bonding differ by age (see, e.g., Agnew, 1985; Johnson, 1984; Krohn et al., 1989; LaGrange & White, 1985; Liska & Reed, 1985; Menard, Elliott, & Wofford, 1993; Rankin, 1980; Simons-Morton et al., 1999), although findings are mixed in identifying specific stages of adolescence that prosocial bonding is most and least salient. An examination of these findings is reserved for Chapter 4.

Of relevance here is that Hirschi (1969) did not specify age-graded changes in bonding. Although social control theory is processual in nature, with changes in social bonding hypothesized to cause changes in behavior, the theory does not specifically link the increase in delinquency and drug use during adolescence to a general decline in social bonding. Moreover, Hirschi did not address the impact that initial delinquency has on levels of bonding at later time periods in adolescence and how this may impact subsequent behavior.

Summary

Hirschi's (1969) social control theory constitutes a significant contribution to understanding delinquency and the social units and individuals most apt to have an influence on adolescent behavior. Although some of Hirschi's propositions warrant important qualification and question, prosocial bonding appears to be a particularly important protective factor for a variety of child and adolescent antisocial behaviors.

The theory's moderate empirical support, coupled with the increased explanatory power yielded when integrated with explanatory constructs from other theories, speak to several related issues. As a standalone theory, social control theory does not properly address the processes by which bonding occurs or changes, or whether a reciprocal relationship exists between delinquency and bonding. In a related vein, research suggests that social control theory provides an important, but partial explanation of delinquency.

Although bonding elements have been shown to have a direct impact on the likelihood of delinquency in straightforward tests of the theory (as Hirschi posited), more comprehensive assessments indicate that the relationship between bonding elements are more complex than Hirschi conceived, with some elements exhibiting mediating and moderating qualities. Moreover, variables from other traditional theories have been found to mediate and moderate the impact of bonding variables on delinquency, and delinquency itself has been found to impact the salience of the social bond. All of these findings lend support for viewing social control theory as a "building block" and a viable theory upon which a more developmentally-explicit, integrated theory is based (Agnew, 1993; Johnson, Marcos & Bahr, 1987; Marcos & Bahr, 1988). Differential association and social learning theory aid in solidifying the importance of this endeavor.

Differential Association Theory

In contrast to the emphasis that control theories of delinquency (e.g., Hirschi's social control theory) place on explaining conformist behavior while treating deviant motivation as a constant, learning theories seek to explain deviant motivation. Learning theorists assume that deviant motivation is not an innate, predispositional characteristic that all individuals possess. Rather, the motivation to commit acts of deviance varies across individuals as a function of differential learning content. One prominent learning theory, Sutherland's (Sutherland & Cressey, 1978) differential association theory, is worth discussing since it not only serves as a distinct explanation of delinquency, but it also constitutes the basis of Akers' (1977) social learning theory.

Differential association theory locates the source of deviance (including drug use) in intimate interpersonal relationships. The theory consists of two primary elements: differential association and definitions. Deviant behavior results from differential exposure to important others (differential association) who espouse the norms, attitudes, and beliefs (definitions) conducive for deviant behavior involvement and the techniques requisite for successfully completing the behavior. Since Sutherland's primary explanatory mechanism is differential exposure to definitions, his nine theoretical propositions can be reduced to one key explanatory postulate: "a person becomes delinquent because of an excess of definitions favorable to law violation over definitions unfavorable to violation of the law" (Sutherland & Cressey, 1978, p. 6).

In explicating four characteristics of relationships (frequency, priority, duration, intensity), Sutherland contended that individuals are more apt to be influenced by the attitudes and norms of those to which they have close, long-term relationships with, and whose opinions and friendships they value. In terms of adolescent drug use, youth are more apt to internalize and be impacted by the drug attitudes and norms of others who are considered close confidants, and to which considerable, long-term, and frequent time is spent. The observation that adolescents spend considerably more time with peers than parents, teachers, and other adults (see, e.g., Kandel & Andrews, 1987; Schulenberg et al., 1997; Simmons & Blyth, 1987; Thornburg, 1982) suggests that adolescents may prioritize the attitudes and norms of peers over others norms and attitudes, with these definitions having an important impact on the behavior of adolescents.

Sutherland did not focus solely on the impact of peer influence, but tests of differential association theory typically have centered on the relationship between adolescent self-reports of personal delinquency and associations with delinquent peers. Mentioned earlier, this relationship has been found to be one of the strongest and most consistently reported findings in the delinquency literature (see, e.g., Akers, Krohn, Lanza-Kaduce, & Radosevich, 1979; Mears & Field, 2002; Tittle, Burke, & Jackson, 1986; Voss, 1964; Warr, 1993a). Research also has found, though, that a deviant attitude is not the only characteristic of a delinquent youth that is transposed onto youth (see Johnson et al., 1987; Warr & Stafford, 1991), lending support for a broader social learning theory.

Social Learning Theory

Social learning theory (Akers, 1977, 1992; Burgess & Akers, 1966) expands upon differential association theory by clarifying the mechanisms involved in the learning process. Unlike Sutherland, who posited that only ideas (e.g., attitudes, norms, and beliefs) are learned from interacting closely with others, Akers explicated that both attitudes and behavior are learned through processes of imitation and differential reinforcement.

Two major assumptions found social learning theory. First, Akers (like Hirschi) assumes that value consensus exists in society, with members agreeing what behaviors should and should not be legally proscribed. Second, Akers (like Sutherland) subscribes to the notion of “tabula rasa” in characterizing human nature. Specifically, Akers assumes that individuals are born with no innate proclivity toward engaging in any type of behavior. Born with a “clean slate,” any tendency to act in a particular fashion is rooted in the nature and content of learned attitudes and norms (Akers, 1977). Hence, in contrast to Hirschi, Akers views deviant motivation as a transferable and variable interpersonal characteristic that is passed on from one person to another. Similar to Sutherland, Akers sought to explain disparity in behavior in terms of this variability in deviant motivation.

Akers reduced Sutherland’s nine theoretical propositions to seven, retaining the concepts of differential association and definitions and integrating Skinner’s (1999) concept of operant conditioning and Bandura’s (1977) construct of imitation. Social learning theory consists of four main explanatory factors: differential association, differential reinforcement, imitation, and definitions (Akers, 1977). Similar to Sutherland, Akers posited that the key to social learning lies in interpersonal interaction, or the degree to which individuals associate with one individual or group over another (differential association). In contrast to Sutherland’s sole focus on differential association and definitions, however, Akers posited that intimate groups and individuals to which an individual differentially associates provide social reinforcements, behavioral models, and definitions necessary for the initiation, continuation, and cessation of behavior.

The Learning Process

As the first step in the social learning process, one imitates the definitions espoused by those to which one differentially associates. Instead of a general construct of definitions postulated by Sutherland (Sutherland & Cressey, 1978), Akers specified two types of definitions: specific and general definitions. Specific definitions constitute attitudes, beliefs, or norms concerning particular types of behavior (e.g., legitimacy of marijuana use), while general definitions constitute one's general moral beliefs (Akers, 1977), a concept synonymous to Hirschi's (1969) social bond element of belief.

The second major step in the social learning process typically is the differential reinforcement of these attitudes and norms by important others. Differential reinforcement refers to actual and expected rewards or punishments (i.e., reinforcements) attached to a particular attitude or behavior (Akers, 1977). Rewards and punishments may be applied by others to which one differentially associates, or they may be anticipated by the individual as a function of past learning experiences and observations of others. Akers specifies that rewards and punishments can be social or non-social in nature. Social reinforcers constitute the rewards and punishments provided by those to which one differentially associates, while nonsocial reinforcers constitute the physiological rewards and punishments (e.g., lack of energy, adrenaline, or drug euphoria) that result from engaging in the given behavior. The initiation of behavior is impacted to a larger degree by social reinforcers, while the continuation or cessation of behavior is influenced by social and nonsocial reinforcement (Akers, 1985). Synonymous with the major tenet underlying rational choice theory (see Akers, 1990), Akers posited that individuals weigh rewards and punishments in deciding whether or not to fully internalize the given behavioral definition (or behavior). If the consequences that stem from holding a particular definition are perceived as more rewarding than punishing, one is more apt to fully internalize the given attitude or norm (Akers, 1985).

Once internalized, attitudes or norms serve as the motivational impetus for imitating the behaviors that coalesce with the internalized definition. As the final step in the basic learning process, behavioral imitation simply involves observing and emulating the behavior that is being modeled. Often the same primary group or individual who initially espoused the internalized definition models the behaviors that are imitated (Akers, 1977).

Reciprocation

Contrary to Hirschi (1969) and Sutherland (Sutherland & Cressey, 1978), Akers hypothesized that reciprocal effects occur; a bidirectional relationship exists between deviant behavior and social learning variables, with causal priority given to differential association. Specifically, after individuals initiate and continue engaging in a given deviant behavior, it is possible that differential associations and resultant friendship patterns may change. Concerning drug use, for example, Akers (1992) contended that both use itself and its associated social consequences “now may begin to have an effect on choice of friends and social setting, therefore, having some feedback effects on differential association” (p. 87). Hence, Akers argues that although differential association serves as the impetus for behavioral involvement, continued behavioral involvement can impact subsequent associations. Akers (1992) further argued this process may be particularly evident among adolescents and adults who become increasingly involved in drug use, since networking with others outside of immediate peer networks may be required in order to maintain a constant drug supply.

Social Learning and Drug Initiation

Applying the social learning process to adolescent drug initiation, the probability of drug abstinence decreases and initiation increases when an adolescent has internalized more positive than negative attitudes and norms espoused by important others concerning the legitimacy of drug use; when the youth is differentially exposed to or interacts with individuals or groups who use drugs relative to individuals who abstain from drug use; and when important others to which the adolescent differentially associates reinforce drug use over abstinence (i.e., more rewards and fewer punishments for drug use than abstinence). Relative to drug initiates, drug abstainers hold more negative than positive attitudes concerning drug use, they are less apt to interact with drug users, and they are provided more negative than positive reinforcements for drug use (Akers, 1992).

Demographic Variation

Akers original formulation of social learning theory in the late 1960s (Burgess & Akers, 1966) did not fully detail why demographic (i.e., age, race, and gender) variation exists in deviant behavior (e.g., drug initiation and use). His theoretical expansion, social structure-social learning theory (Akers, 1998), more appropriately addressed this variation. According to this revision, social learning variables mediate much of the relationship between demographic characteristics and behavior. Hence, age, race, and gender structure the types of associations (and behaviors, definitions, and social reinforcements) to which individuals are apt to be exposed (Akers, 1998; Akers & LaGreca, 1991). When prediction models control for social learning variables in examining the direct relationship between age, race, or gender and delinquency, Akers asserts that these direct relationships will be rendered insignificant.

Concerning biological age differences in behavior, Akers posits that parents, peers, and schools provide youth with the most important interactional contexts in which social learning occurs. Parental models, definitions, and reinforcement are believed to be more important during childhood and early adolescence than during the later adolescent years. In contrast, peer influence is considered most salient during the mid- to late adolescent years, when adolescents spend a great deal of time with peers (Akers & Lee, 1996). As the primary context for drug initiation, peers provide adolescents with the most significant drug models, favorable drug norms, and social reinforcers conducive to drug initiation and use (Akers, 1992).

Empirical Validity

First tested fully in 1979 (Akers et al., 1979), social learning theory is one of the most frequently tested criminological theories in the adolescent drug and delinquency literature (Akers & Jensen, 2006). Both full and partial tests provide moderate to strong support for the major propositions and predictive utility of differential association and reinforcement, imitation, and definitions (see, e.g., Burkett & Warren, 1987; Jessor & Jessor, 1978; Marcos et al., 1986; Orcott, 1987; Sellers & Winfree, 1990; White, Pandina, & LaGrange, 1987; Winfree & Griffiths, 1983). Akers' own empirical tests also are very supportive (see, e.g., Akers & Cochran, 1985; Akers et al., 1979; Akers & LaGreca, 1991; Akers, LaGreca, Cochran, & Sellers, 1989; Akers & Lee, 1996; Hwang & Akers, 2003; Krohn, Lanza-Kaduce, & Akers, 1984). Overall, social learning variables have been shown to explain as much as 68% of the variance in adolescent drug use, 59% of the variation in alcohol use among the elderly, and 54% of the variance in the perceived likelihood of being raped (Akers & Jensen, 2006).

Comparisons of the predictive utility of social learning theory with other traditional theories indicate that social learning variables almost always have stronger net effects than variables from social control, differential association, and strain theories of delinquency (see, e.g., Akers & Cochran, 1985; Benda, 1994; Elliott et al., 1985; Hwang & Akers, 2003; Kandel & Davies, 1992; Matsueda & Heimer, 1987; McGee, 1992; Winfree, Griffiths, & Sellers, 1989). Pratt and Cullen's (2000) recent meta-analysis of self-control research did find, however, that Gottfredson and Hirschi's (1990) construct of low self-control is a more salient predictor of delinquency than two of the most salient social learning variables: delinquent peer association and deviant definitions.

Qualifications

Although social learning theory garners considerable empirical support, it is not without some qualifications. There are four major weaknesses associated with the theory. First, it has been criticized as being logically inadequate (Akers & Jensen, 2006). Since behavioral learning requires a model who provides a reference for norms and behavior, social learning theory does not readily explain new forms of deviant behavior, such as Oxycontin abuse, credit card fraud, identity theft, or Internet child predation (Williams & McShane, 2004).

Second, due to its emphasis on the social learning process, Akers (1977) tends to emphasize proximal risk factors for delinquency while neglecting more distal factors that may be important for explaining the root causes of delinquency. For example, the theory tends to focus explicitly on what occurs after youth become involved with delinquent peers, thereby failing to adequately explain why only some youth associate with deviant peers in the first place (Petraitis, Flay, & Miller, 1995; Williams & McShane, 2004). Although Akers' revised theory (1998) attempts to resolve this issue by positing that demographic characteristics impact delinquency indirectly by structuring the type of institutions and individuals to which people are exposed, this revision provides a more adequate explanation of between-group differences in types of differential association, models, definitions, and differential reinforcement than within-group variation. For example, among early adolescents, why do some youth choose to associate with deviant peers while others avoid this type of association? The answer to this question may lie partly in Hirschi's (1969) notion of a weak social bond.

Third, Akers (1977) characterizes social learning theory as a general theory of crime, capable of explaining all variation in deviant behavior, regardless of the demographic characteristics of the actors. Although some studies have found that learning processes are similar across gender, with little gender disparity in the relationship between learning variables and delinquency and drug use (Bahr et al., 1995; Dembo et al., 1986), other research indicates that social learning variables play a more influential role in male delinquency than female delinquency (Akers, Skinner, Krohn, & Lauer, 1987). Mixed results concerning race differences in the relationship between social learning variables and delinquency also underscore the possibility that social learning theory may not adequately address cultural differences. Some research has found no race differences (Dembo et al., 1986), while other research demonstrates that relative to white youth, black adolescents possess higher rates of delinquent norms and attitudes due to their increased exposure to divorce and broken homes (Matsueda & Heimer, 1987). Social class differences also have been observed, with peer influence factors (e.g., differential association and definitions) more salient among middle-class than lower- or upper-class youth (McGee, 1992).

Social learning theory does, however, appear to provide a fairly adequate explanation of the age-crime curve, with age-graded differences largely accounted for by family socialization, peer associations, and other social learning variables (see, e.g., Conger & Simons, 1995; Krohn et al., 1989; Sampson &

Laub, 1993; Warr, 1993a). For example, Warr’s (1993a) longitudinal study of National Youth Survey data found that when peer influence variables were controlled, the relationship between age and delinquency was reduced to insignificance.

Fourth, empirical tests of integrated theory that combine key constructs from differential association, social control, and social learning theory have disconfirmed some of Akers (1977) social learning hypotheses. For example, Akers hypothesized that a direct relationship exists between prosocial rewards and conforming behavior. Brown and colleagues (2005) used longitudinal data to test this proposition (and others) against those promulgated by Hawkins and colleagues’ (Catalano & Hawkins, 1996) social development model, a developmental theory that integrates key explanatory constructs from differential association, social control, and social learning theory. Brown and associates (2005) found that contrary to Akers (1977) hypothesis, the relationship between prosocial rewards and prosocial behavior was completely mediated by prosocial bonding (operationalized as prosocial attachment and commitment) and prosocial beliefs, with prosocial beliefs exerting a direct impact on prosocial behavior.

This finding underscores two issues. First, as Akers postulated, prosocial rewards do appear to constitute a protective factor for antisocial behavior by reinforcing the continuation of prosocial behavior. However, Brown et al.’s (2005) results suggest that this protective factor is more distal in nature than what social learning theory postulates. Instead of a direct positive relationship between prosocial rewards and conforming behavior, prosocial rewards may contribute to the establishment of a social bond, a relationship not acknowledged by Hirschi (1969) or Akers (1977). Increased attachment and commitment subsequently contribute to the solidification of a prosocial belief system, with this belief system directly related to prosocial behavior. Hence, although Hirschi (1969) and Akers (1977) did not recognize that social bonding and learning may be related, both of their hypotheses concerning a positive relationship between prosocial beliefs (or definitions) and prosocial behavior is supported by Brown et al's (2005) research. Second, and in a related vein, this finding suggests that a more comprehensive and valid explanation of delinquency and drug use may require that key factors from social control and social learning theory be integrated.

Delinquent Peer Association

Across studies, differential association generally is found to be the most salient predictor of deviant or conforming behavior, while imitation is the least salient predictor (see, e.g., Akers, 1992; Akers & Cochran, 1985; Coombs & Landsverk, 1988; Ellickson, Tucker, Klein, & McGuigan, 2001; Lanza-Kaduce et al., 1984; Reifman et al., 1998; Stumphauzer, 1983; Winfree & Bernat, 1998). Of the various individuals with whom youth associate, research indicates that peers exert a significant source of influence. Discussed earlier, one of the most salient predictors of the initiation, escalation, and desistance of adolescent delinquency and drug use is delinquent peer association (see, e.g., Huizinga et al., 1991; Loeber & Stouthamer-Loeber, 1986). Aptly noted by Warr (2002), "Few, if any, empirical regularities in criminology have been documented as often or over as long a period as the association between delinquency and delinquent friends" (p. 40).

Although researchers acknowledge the existence and importance of this relationship, there is debate in the literature concerning why adolescent-peer homophily exists; which of these factors temporally precedes and influences the other; and as a consequence, which theory (social control or social learning theory) and attendant assumption of human nature provides the most adequate explanation for this relationship. Instead of causing adolescent delinquency as Akers (1977) purports, delinquent peer association may be a consequence of adolescent delinquency. The temporal ordering of these variables lies at the heart of the debate, underscoring the importance of utilizing longitudinal data in examining causal pathways.

Traditionally, two major perspectives have attempted to account for the strong correlation between delinquent peer association and adolescent delinquency: the selective association perspective and the socialization perspective (Kandel, 1978b, 1996). The defining difference between these perspectives lies in the causal ordering of these factors. With the advent of more sophisticated longitudinal modeling and empirical tests of reciprocal effects (Farrell, 1994; Liska, Felson, Chamlin, & Baccaglini, 1984), two other explanations for adolescent-peer homophily have emerged in the literature. As extended versions of traditional explanations, these two perspectives simply add the notion of reciprocal effects (Akers, 1992; Thornberry, 1987). These four explanations are briefly outlined below, along with an overview of attendant empirical support.

The selective association perspective is rooted in the social control tradition of explanation. Specifically, delinquency serves as a mediator between weakened social bonds and delinquent peer associations, such that weakened social bonds lead to delinquency, and delinquency (in turn) leads to associations with delinquent peers (Kandel, 1978b). According to this perspective, the strong relationship between adolescent and peer drug use, for example, is primarily the result of selective association (i.e., “flocking”) on the part of adolescent drug users themselves. Adolescent drug users seek out peers to befriend who also engage in drug use, or who are contemplating initiation. Hence, while weakened social bonds serve as the distal (or root) cause of delinquent peer associations, the proximal cause of this association is adolescents’ own drug use (Kandel, 1996). To the extent this phenomenon exists (referred to as “birds of a feather flock together”), the explanatory utility of learning theories is substantially weakened (Fisher & Bauman, 1988; Kandel, 1978b, 1978c, 1996).

In contrast to the selection hypothesis, the socialization perspective is rooted in the differential association/learning tradition. This camp argues that adolescent delinquency is the product of delinquent peer associations (i.e., “feathering”). Although there are differences among researchers in how they conceptualize the role of delinquent beliefs (see Warr & Stafford, 1991), most researchers who subscribe to this perspective contend that associating with delinquent peers leads to adolescent delinquent behavior either directly or indirectly through adolescents delinquent definitions. Regardless of the causal role of delinquent definitions, this perspective argues decisively that the root cause of adolescent delinquency is delinquent peer association.

A third and relatively recent interpretation of peer-adolescent homophily has been termed the interactional perspective (Thornberry, 1987). Instead of the unidirectional relationship between delinquent peer association and adolescent delinquency posited by the socialization and selection perspectives, the interactional perspective combines both perspectives in arguing that a reciprocal, developmental relationship exists between adolescent and peer behavior. According to this perspective, the root cause of delinquency is weak prosocial bonds. It is these weak bonds that free youth to associate with delinquent peers. After the initial learning (or “feathering”) process unfolds and adolescents initiate a given deviant behavior, the selection (or “flocking”) process begins. As the developmental pathway of delinquent behavior unfolds and extends over time, continual “feathering and flocking” occurs.

Discussed earlier, Akers’ (1992) reciprocation hypothesis constitutes the last major perspective concerning delinquent peer-adolescent behavioral homophily. In general, he argues more for the notion that a reciprocal delinquent peer-adolescent relationship begins with peer influence, and pays little attention why youth come to associate with delinquent peers in the first place.

Aside from vocalizing their subscription to the selective association hypothesis, Gottfredson and Hirschi’s (1990) critique of the delinquent peer-adolescent delinquency relationship calls attention to two other possible explanations. In their view, measures of peer delinquency (and drug use) commonly used in survey research simply are “another measure of self-reported delinquency” (Gottfredson & Hirschi, 1990, p. 157). When adolescents are asked to self-report information concerning the behavior of their friends or peer group, Gottfredson and Hirschi contend that behavioral attribution may be operating, whereby adolescents simply self-report information concerning peer delinquency in terms of their own personal delinquency. Hence, convergence in adolescent and peer delinquent behavior may simply be an artifact of the types of measures typically used to capture peer delinquency. However, this attribution hypothesis has been largely disconfirmed by dyad and triad research conducted by Kandel (1978c, 1978d), who obtained drug data from peers themselves.

Gottfredson and Hirschi (1990) also argued that the common cause model offers a plausible explanation for this behavioral convergence. Discussed in Chapter 2, the common cause model refers to the notion that the strong positive correlation between adolescent and peer behavior is due to a common antecedent factor that both deviant youth and peers share, such as low self-control (Gottfredson & Hirschi, 1990).

All four major explanations of peer-adolescent behavioral homophily are supported by research. The selective association hypothesis has garnered substantial empirical support (see, e.g., Bauman & Ennett, 1996; Bauman & Fisher, 1986; Ennett & Bauman, 1994; Fisher & Bauman, 1988; Kandel, 1978a, 1978d, 1996; Wills & Cleary, 1999), as has the peer socialization argument (see, e.g., Akers et al., 1979; Elliott et al., 1985; Krohn, Skinner, Massey, & Akers, 1985; Matsueda, 1982; Minor, 1984; Patterson & Dishion, 1985; Tittle et al., 1986). Moreover, a line of longitudinal research also provides support for the interactional perspective (see, e.g., Agnew, 1991a; Burkett & Warren, 1987; Ginsberg & Greenley, 1978; Kandel, 1978b; Meier, Burkett, & Hickman, 1984; Paternoster, 1988; Thornberry, Lizotte, Krohn, Farnsworth, & Jang, 1994; Warr & Stafford, 1991). Finally, Akers' hypothesis concerning reciprocal effects (i.e., "feathering" and "flocking" that begins with "feathering") also has received support (see, e.g., Akers & Jensen, 2006; Akers & Lee, 1996; Andrews & Kandel, 1979; Sellers & Winfree, 1990; Warr, 1993b).

Although more longitudinal research employing independent measures of peer influence derived from peers is needed (e.g., Kandel, 1978b, 1978c), on balance, research to date suggests that "…'feathering' and 'flocking'…are not mutually exclusive and may instead by part of a unified process" (Warr, 1993b, p. 39). More often than not, peer socialization appears to occur slightly more often and prior to selective association (Warr, 2002). With respect to drug initiation, for example, it is more often the case that adolescents first are introduced to drugs by their peers. After drug use is initiated and use continues, adolescents become more selective in whom they befriend and associate (see, e.g., Akers & Lee, 1996; Sellers & Winfree, 1990; Warr, 1993b). Although this suggests that social learning theory is superior to social control theory in this regard, it is worth noting that social learning theory fails to adequately address why some youth choose to associate with delinquent peers over conforming peers. It appears that any real advancement in this area in terms of understanding root causal mechanisms will require prospective assessment of child relationships beginning in the family, the major social unit that youth are first exposed.

The issue of delinquent peer-adolescent homophily is relevant to the current research in two ways. First, since the cross-sectional nature of the data to be used in the current study precludes a determination of causal ordering between adolescent soft drug initiation and association with drug-using peers, it is not possible to accurately determine the temporal ordering of these variables. Establishing temporal ordering requires longitudinal data. Second, the current research recognizes the possibility that any relationship found between peer and adolescent soft drug initiation may have originated in part from either selective peer association on the part of adolescents themselves, or peer socialization factors. It also is acknowledged that socialization processes may be more salient for the initiation of a given soft drug (e.g., alcohol) that occurs during early adolescence versus the initiation of another soft drug (e.g., marijuana) that may occur during late adolescence. Research on developmental pathways leading to delinquency suggests that selective association appears more operable after a given deviant behavior is initiated, with the selection of more serious delinquent peers occurring prior to further entrenchment and involvement in more serious delinquent behaviors (Elliott et al., 1985; Warr, 2002).

Conclusion

Social control, differential association, and social learning theory are three of the most empirically supported traditional theories of delinquency within the field of criminology. These theories differ more in explaining why factors are related to adolescent drug use and delinquency than identifying what these factors are. For example, although these theories locate causal factors in similar social units, individuals, and groups (e.g., parents, peers, and schools), with prosocial beliefs (or definitions) constituting the greatest conceptual overlap between them (Akers, 1994), they differ in the specification of intervening processes. Social control theory places explanatory emphasis on conforming behavior, since deviant motivation is viewed as a predispositional and invariant trait. The net impact of four protective factors (i.e., attachment, commitment, involvement and belief) constitutes a strong social bond capable of restraining deviant motivation (and delinquency in consequence). From a social control perspective, the proximal cause of soft drug initiation is a weakened social bond. Social bonding elements that have received consistent empirical support include prosocial attachment to others, commitment to conventional lines of action, and a general belief in the moral authority and rule of law.

In contrast, learning theories seek to explain behavior in terms of differential motivation, with nondeviant and deviant motivation viewed as variable and transposable intrapersonal characteristics passed onto others through processes of reinforcement and imitation. As a risk factor and impetus for delinquent behavior, deviant motivation is located in the attitudes and norms permissive of deviant behavior. Both deviant definitions and behavior are transposed onto others through processes of reinforcement and modeling. From a learning perspective, the key causal factor in soft drug initiation is delinquent peer association, a risk factor that can be viewed as both the product of a weakened social bond and the impetus for the internalization and learning of prodrug attitudes, norms, and behavior. Learning constructs that have garnered consistent empirical support include differential association (particularly delinquent peer association) and definitions.

Drawbacks of Traditional Theories

Where relevant, the current chapter pinpointed some aspects of adolescent deviance that traditional theories of delinquency are not able to adequately explain or address. Inadequacies center on issues related to the complexity of interpersonal influences and behavior and the age-crime curve.

Individual-Environment Complexity

In general, traditional theories of delinquency do not adequately address the complexities inherent in the interaction between individuals and their environment. Major complexities include reciprocation between risk and protective factors; causal chains in risk factors; bidirectional relationships between predictors and deviant behavior; developmental considerations; and within-group variation in risk and protective factors for delinquency.

Reciprocation and causal chains. Traditional theories typically infer that explanatory factors do not interact with each other, but are linearly related. Research findings from extended and integrated empirical tests of traditional theories, coupled with developmental research, suggests, however, that causal processes are more complex than were initially conceived by traditional theorists in the 1960s and 1970s. For example, earlier discussion touched upon research findings concerning various risk factors (e.g., delinquent peer association) that have been found to mediate the relationship between bonding variables and delinquency.

In a related vein, research identifying that certain risk factors mediate the relationship between other risk factors and delinquency underscore the need for theory to accommodate risk factor causal chains. Traditional theories typically focus on proximal or distal predictors of delinquency, but rarely both. Proximal factors are those that have a direct and immediate impact on behavior, such as cognitive or affective factors found within the individual (Petraitis et al., 1995). Examples of proximal risk factors for adolescent drug use include willingness or intentions to use drugs; drug-specific attitudes, beliefs, or norms; current decisions and expectancies concerning drug use; and past experiences with drug use. The construct of belief (or definitions) is a good example of a proximal factor that both social learning theory and social control theory share.

While these types of factors are of value for explaining what influences within adolescents themselves place youth at increased or decreased risk for soft drug initiation, proximal risk factors do not explain the “root causes” of drug initiation. They also do not explain what processes lead to the establishment of anti- or prodrug attitudes or beliefs. Although it is important to recognize the significance of adolescents’ general and drug-specific norms or intentions, a comprehensive understanding of the causal pathways that prompt these factors (and ultimately increase risk for drug initiation) require that distal factors also be identified and examined.

Distal risk factors are more removed environmental predictors of antisocial behavior. Petraitis and colleagues (1995) contend that in main effects models, risk factors conceptualized as distal in nature (e.g., school and community-level risk factors) may be less salient predictors of outcomes than more proximal risk factors (e.g., family, peer, and individual-level risk factors), because the effects of distal predictors likely are mediated by the more proximal influences. For example, although weak school attachment may have a direct impact on risk for drug initiation, a more powerful and complete explanation of drug initiation may lie in the impact that weak school attachment has on intentions or willingness to use drugs, which (in turn) may directly increase risk for drug initiation.

Of the three traditional theories discussed, social learning theory provides the most comprehensive explanation of drug use, by identifying both proximal and distal risk factors and explaining how these risk factors are related in a causal pathway. Differential association and differential reinforcement are both distal-type influences, while imitation and definitions constitute more proximal factors. Social control theory primarily locates delinquency in more proximal influences without adequately explaining the processes (and distal factors) involved in shaping and changing the strength of the social bond.

The importance of identifying both proximal and distal influences and salient mediating processes implies that researchers should focus on developing and testing theoretically-guided mediation models. According to Petraitis et al. (1995), however, the direct effects of predictors should be examined first in an effort to identify what risk and protective factors tend to be the most salient. After proximal and distal predictors are identified, mediation models then can be established, with important proximal influences examined for their mediating qualities.

With the exception of social learning theory, traditional theories also generally do not explain the impact of delinquency on explanatory factors and how this reciprocal relationship impacts future social interaction and behavior (Thornberry, 2004). Instead, traditional theories (e.g., social control theory and differential association) tend to posit unidirectional relationships between predictors and problem behaviors. Moreover, although feedback loops from deviant behavior to social learning variables are specified in social learning theory, most empirical tests of the theory tend to examine unidirectional rather than reciprocal relationships (Akers & Jensen, 2006). Extended tests of traditional theories find that parental attachment and school commitment are related reciprocally to delinquency and drug use, and in multiple ways. For example, Thornberry and colleagues' (Thornberry, Lizotte, Krohn, Farnsworth, & Jang, 1991) test of interactional theory found parental attachment and delinquency to be related reciprocally during early adolescence, while a negative unidirectional relationship between the two was found during late adolescence.

Developmental considerations. Although inferred, traditional theories also do not pay adequate attention to many aspects of child and adolescent development. Some of these issues include the relevance of developmental tasks and transitions for youth behavioral development, stability (e.g., trajectories), and change. Hirschi's (1969) social control theory, for example, does not pay adequate attention to the impact that school transitions and corresponding expansions in interpersonal relationships and individual responsibility have on changes in the social bond, and ultimately behavior.

Within-group variation. Prior to the advancement of developmental theory in the 1980s, theory and research tended to take a strict variable-centered approach to explaining delinquency, suggesting that the effects of risk and protective factors work similarly for everyone (Huizinga et al., 1991). Moreover, traditional theories tend to explain behavior in terms of dichotomies, such as drug initiates versus abstainers, or the difference between delinquents and nondelinquents. While this dichotomous conception and study of behavior is important, traditional theories do not take a pattern-centered approach in explaining typological differences among delinquents or drug users themselves (Magnussen & Bergman, 1988, 1990). Youth drug users or delinquents may differ according to deep-seeded, childhood risk and protective factors, the duration and nature of their delinquent or drug-using careers, or the nature and timing of initiation and desistance (Thornberry, 2004).

Developmental research suggests that delinquents and adolescent drug users are not all the same. Various sequences (i.e., causal chains) leading to problem behaviors (and the negative consequences and duration of delinquent and criminal trajectories) differ on important risk factors, many of which are rooted in childhood and early adolescence (see, e.g., Hill, White, Chung, Hawkins, & Catalano, 2000a; Huizinga et al., 1991; Loeber, 1988; Schulenberg et al., 1997).

Importance of Biological Age

In contrast to developmental theories, traditional theories (with the exception of social learning theory) do not take explicit strides in explaining the age-crime curve, one of the most long-standing observations in epidemiological crime and drug research. For instance, with the exception of social learning theory, traditional theories tend to infer that explanatory factors are of equal salience at different stages of youth development. With respect to social control theory, Hirschi (1969) did not explicate if weak parental attachment is a more important facilitator of drug initiation during the high-school years versus late childhood or early adolescence, or if the net effects of the social bond are attenuated by other environmental factors at particular stages of adolescent development (White, Johnson, & Horwitz, 1986).

Traditional theories also fail to adequately address differences in timing of initiation, such as why some youth initiate drug use at earlier (e.g., 8 years of age) versus later ages (e.g., 17 years of age). As Thornberry (2004) aptly notes, if different risk factors underlie late versus early initiation of delinquency (e.g., soft drug initiation) and they are not distinguished theoretically, these different developmental pathways will be confounded in empirical analyses. In turn, this confounding lends to an increased possibility that false positive (or false negative) results will be produced. If translated into practice, artificial results can serve as a significant barrier to preventing delinquent behaviors.

Aptly noted by researchers, this inadequate treatment and explanation of the age-crime curves constitutes one of the major weaknesses of traditional theories. Recognized by LaGrange and White (1985), knowing that delinquency varies by age prompts the “need to recognize the possible limited generalizability of our [traditional] theories to the entire range of adolescence” (p. 20). In highlighting the need to consider the nature of human development and social interaction in any explanation of behavior, Warr (1993a) concurred, noting, “the age gradient of crime is so steep that it requires from any explanation rather profound age-related changes in explanatory variables” (p. 18).

Developmental theories of adolescent antisocial behavior compensate for many of the inadequacies endemic to traditional theories simultaneously integrating and extending key explanatory constructs. The following chapter centers on two developmental theories that together are capable of addressing the three research questions that drive the proposed research.

CHAPTER 3

DEVELOPMENTAL ETIOLOGY

Developmental theories of delinquency and drug use are the products of theoretical syntheses, or the integration of empirically verified risk and protective factors derived from traditional theories of delinquency. Many of the constructs and propositions found in these theories are conceptually anchored in the theories of social control (Hirschi, 1969), differential association (Sutherland & Cressey, 1978), and social learning (Akers, 1977), three of the most empirically supported traditional theories of delinquency.

The previous chapter focused on these three theories in an effort to trace the traditional roots of developmental theory; draw attention to the strengths and inadequacies of traditional explanations; and highlight the importance of viewing adolescent behavior through a developmental lens. Although traditional theories are important explanations of delinquent behavior, they fail to adequately address two major issues: the complexity of relationships between interpersonal influences and behavior, and the age-crime curve.

The purpose of the current chapter is two-fold. First, a brief overview of the developmental paradigm is presented in an effort to highlight how developmental theories compensate for some of the inherent weaknesses in traditional theories of deviance. Second, attention is directed toward two developmental theories, stage theory and the social development model. These theories are discussed in terms of how each seeks to address one or more of the research questions that underlie the current research.

The Developmental Approach

Developmental theories build upon traditional theories of delinquency in three major ways. First, traditional theories of delinquency form the basis of many developmental psychosocial theories, with theoretical integration typically entailing the inclusion of major propositions and key constructs empirically verified as risk and protective factors. Constructs from social control, differential association, and social learning theory have become the most frequently integrated, although assumptional differences between control and learning theories have yet to be adequately resolved (Akers, 1994).

Second, developmental theories take into consideration many developmental issues, including developmental tasks, transitions, and trajectories; developmental changes in risk and protective factors; and the reciprocal nature of risk and protective factors and behavior over time. Although traditional theories (e.g., Akers, 1977; Hirschi, 1969; Sutherland & Cressey, 1978) infer that risk for (or protection from) delinquency constitutes a dynamic process (e.g., learning and bonding as a process), developmental theories bring the notion of time and timing to the forefront.

Most developmental theories attempt to explain how risk, protection, and behavior change over time, and how risk and protection at one period in human development impact behavior and levels of risk and protection at successive periods. Hence, many developmental theories explicate the developmental nature and unfolding of social interaction and behavior, and they address research findings concerning developmental risk factor chains that link minor antisocial behavior to minor delinquency, and minor delinquency to more serious acts (see, e.g., Catalano & Hawkins, 1996; Elliott et al., 1985; Kandel, 2002; Patterson, DeBaryshe, & Ramsey, 1989; Sampson & Laub, 1990, 1993; Thornberry, 1987). Some of the more prominent developmental and life-course theories that attempt to account for various developmental issues (e.g., timing of initiation, developmental changes in risk and protective factors and behavior) are listed in Table 1.

Table 1.

Prominent Integrated Developmental Theories of Adolescent Drug Use

Developmental Theory	Theorist/Year
Domain Model	Huba & Bentler, 1982
Family Interactional Theory	Brook & Brook, 1990
Informal Social Control Theory	Laub & Sampson, 1988; Sampson & Laub, 1990
Interactional Theory	Thornberry, 1987
Adolescent-Limited and Life-Course Persistent Developmental Taxonomy	Moffitt, 1993
Multistage Social Learning Theory	Patterson, DeBaryshe, & Ramsey, 1989 Simons, Conger, & Whitbeck, 1988
Peer Cluster Theory	Oetting & Beauvais, 1986, 1987
Problem Behavior Theory	Jessor, 1992; Jessor & Jessor, 1975
Revised Social Control Theory	Elliott et al., 1985; Elliott et al., 1989
Self-Derogation Theory	Kaplan, 1975, 1980
Social Development Model	Hawkins & Weis, 1985; Catalano & Hawkins, 1996
Social Ecology Model	Kumpfer & Turner, 1990
Stage Theory	Kandel, 1980a, 1985, 2002
Theory of Triadic Influence	Flay & Petraitis, 1994

Third, although both developmental and traditional theories explain between-group differences in behavior (e.g., drug abstainers versus drug initiates), some developmental theories also focus on differences between antisocial youth themselves by identifying and explaining distinct risk factor or behavioral typologies (Magnusson & Bergman, 1990). In this respect, developmental theories are similar to life-course theories in their emphasis on delinquent and criminal careers and how intra- and interpersonal changes in risk and protective factors cumulate over time in impacting stability and change in behavioral trajectories.

In contrast to traditional theories, developmental theorists also generally take explicit strides in attempting to explain why some youth initiate delinquency or drug use at relatively early ages, while others initiate during the later adolescent years. Two examples are the developmental theories established by Moffitt (1993) and Patterson and colleagues (Patterson et al., 1989), both of which characterize “early starters” (or life-course persistent offenders) and “late starters” (or adolescent-limited offenders) as two distinctive types of delinquents who navigate different causal pathways. Age-graded changes in behavior also are the focus of developmental theories, with particular risk factors more salient at one period of development versus another.

As Table 1 clearly demonstrates, there are quite a few prominent developmental theories that may be utilized in explaining adolescent soft drug initiation. All have garnered some degree of empirical support. The following discussion centers explicitly on stage theory (Kandel, 2002) and the social development model (Catalano & Hawkins, 1996). There are three explicit reasons why these two theories (and not others) are discussed in detail: (1) they attempt to address one or more of the research questions posed in Chapter 1[2] (and reiterated at the beginning of Chapter 2); (2) they appear within the research literature frequently, having attained a sizable degree of recognition and prominence among prevention science researchers and criminologists; and (3) both have acquired considerable empirical support. Although these theories attempt to explain different aspects of adolescent drug use, together they capture the nature of adolescent drug initiation in a comprehensive fashion.

There are two other reasons why Kandel's (2002) stage theory is appealing to the current research. Stage theory is able to address Research Question #1 and (to a lesser extent) Research Question #2. Moreover, of the developmental theories established since the 1960s, stage theory is the only theory that explicitly identifies and explains why a common temporal sequencing in polydrug initiation exists, thereby addressing and seeking to explain the developmental nature of drug involvement.

With respect to the social development model (SDM), there are four more specific reasons why this developmental theory is particularly appealing in terms of its applicability to adolescent soft drug initiation. First, the SDM is applicable to the current research due to its ability to address Research Questions #2 and #3.

[2] These three questions include: (1) Is involvement in soft drug use a sequential and hierarchical phenomena? If it is, what is the typical sequence of involvement?; (2) What factors predict soft drug initiation and age of initiation among adolescents?; and (3) Since the initiation of soft drug use appears to vary, in part, as a function of biological age, what factors predict soft drug initiation and age of initiation for youth at different stages of adolescence?

Second, the SDM is a general theory of behavior that incorporates empirically verified risk and protective factors from the traditional theories of delinquency previously discussed: social control, differential association, and social learning theories. Hawkins and colleagues' use these explanatory factors in locating the origin, continuation, and desistance of a wide range of child and adolescent prosocial and antisocial behaviors, including the abstinence and initiation of all drugs.

Third, mentioned earlier, the SDM is one of the most prominent adolescent developmental theories in the field of prevention science. Although a relatively new theory, thus far it has withstood rigorous empirical tests of its key propositions and constructs (see, e.g., Catalano, Park, Harachi, Haggerty, Abbott, & Hawkins, 2005; Hawkins, Guo, Hill, Battin-Pearson, & Abbott, 2001; Huang, Kosterman, Catalano, Hawkins, & Abbott, 2001).

Fourth, the Seattle Social Development Project (and subsidiary programs like Skills, Opportunities, and Recognition [SOAR]), an antisocial behavior prevention program founded on SDM, has been evaluated according to several nationally recognized scientific standards for prevention program effectiveness (e.g., Blueprints for Violence Prevention and the National Registry of Evidence-Based Programs and Practices). The program has been deemed as a "model" (U.S. Department of Health and Human Services, 2001) and "promising" (Center for the Study and Prevention of Violence, 1999) program for youth violence prevention, as well as for the prevention of and reduction in drug initiation and use (Schinke, 2002; U.S. Department of Education, 2002).

Moreover, the social development model founds the basis of Communities that Care ® (Hawkins, Catalano, & Arthur, 2002a), a tested prevention program that falls within the last domain of the prevention science research cycle. This well-known prevention planning system has been used by communities throughout the U.S. (see, e.g., Arthur, Hawkins, Pollard, Catalano, & Baglioni, 2002; Myers & Arter, 2005a, 2005b; Peterson, Hawkins, & Catalano, 1992; Whitlock & Hamilton, 2003), Canada (Buttkus, 2002), Australia (Toumbourou, 2005), the Netherlands (Communities that Care, 2004), and the United Kingdom (Communities that Care, 2006), all in an effort to prevent and reduce drug use and other problem behaviors among youth.

The remainder of this chapter directs attention to the propositions and major constructs underlying stage theory and the social development model, and how each respective theory seeks to address one or more of the research questions that underlie the current research. Due to its relevance for answering Research Question #1, and to a lesser extent, Research Question #2, stage theory is discussed first.

Stage Theory

In Chapter 1, some findings from recent NSDUH rates of adolescent lifetime initiation of single and poly soft drug use were briefly discussed. This assessment yielded several observations concerning the patterning of soft drug initiation, prompting Research Question #1 to be posed. First, 2004 NSDUH data indicate that the most common form of lifetime soft drug initiation is alcohol (12.1%), followed by combined alcohol and cigarettes (6.3%), and combined alcohol, cigarettes, and marijuana (6%). Far more adolescents initiate alcohol (12.1%) use than initiate the sole use of cigarettes (3.4%), the sole use of marijuana (0.4%), or the initiation of any combination of multiple soft drugs. Alcohol and cigarette initiation (6.3%) is more common than the sole initiation of cigarette use (3.4), the sole initiation of marijuana use (0.4%), or other combinations of multiple soft drug initiation.

Second, although alcohol, cigarette, and marijuana initiation is the third most common form of soft drug initiation among adolescents, 2004 NSDUH data suggest that slightly fewer adolescents report the initiation of all three soft drugs (6%) than report the initiation of both alcohol and cigarette use (6.3%). Moreover, far fewer adolescents initiate all three soft drugs (6%) than initiate alcohol use only (12.1%). The initiation of all three soft drugs (6%) is more common, however, than the sole initiation of cigarette use (3.4%), the sole initiation of marijuana use (0.4%), and any other combination of multiple soft drug use (with the exception of alcohol and cigarette initiation).

Third, recent epidemiological drug data suggest that among adolescents who initiate all three soft drugs, alcohol use may have been initiated first by many adolescents, followed by cigarettes, and then marijuana. Specifically, cross-sectional 2005 NSDUH data estimate that among past year initiates, alcohol use was initiated at 14.8 years of age (on average), while cigarette use was initiated at 14.9 years of age, and marijuana use at 15.1 years of age (OAS, 2006a).

These patterns of soft drug initiation suggest that drug involvement appears to be a developmental phenomenon, with legal drugs initiated prior to marijuana. Drug involvement also appears to be hierarchical in the sense that the number of adolescent initiates may become successively smaller as each additional soft drug is initiated. This suggests that the degree of initiation risk varies across soft drugs (or adolescents).

Several prominent social science researchers (Kandel, 2002; Robins, 1979; Thornburg, 1982) support the notion that behavior is a developmental phenomenon. Briefly touched upon in Chapter 2, this perspective is referred to as the simplex perspective (i.e., specific-factor model), and derives from the notion that certain lines of action are developmental in nature, whereby increasing degrees of involvement reflect ordered stages. Transition from one stage of involvement to another stage is associated with unique risk factors. In his work on heroin addiction, for example, Robins (1979) found that different risk factors were associated with different stages of heroin use (e.g., experimental use, regular use, abuse, and addiction).

Denise Kandel's (2002) stage theory can be characterized as one type of simplex model, founded on the premise that involvement with different drugs is not opportunistic, but is ordered and characteristic of a definite pathway from less serious to more serious drug use. Kandel's initial formulation of stage theory was founded on the basis of cross-sectional (Single, Kandel, & Faust, 1974) and longitudinal (Kandel, 1975b) results stemming from research she and her colleagues conducted in examining the drug initiation behaviors of New York high school students. Stage theory, often referred to as gateway theory, was designed to accommodate the finding that although discrete stages in drug involvement exist (e.g., legal drug use prior to illegal drug use), progression along the drug involvement continuum is not deterministic, but instead probabilistic, and largely a function of various drug-specific risk factors.

Kandel (2002) posits that drug involvement is a developmental and hierarchical phenomena. Drug-using careers are conceptualized as developmental in the sense that they unfold over time and tend to follow a common sequence of drug involvement. Constituting a continuum, drug involvement typically begins with less serious, legal drugs. For the successively smaller proportion of individuals who progress along the sequence, more serious and illegal drugs are added to one's drug repertoire. With drug-specific, individual, and social influence risk factors predicting progression, not all individuals are at the same degree of risk for progressing along the sequence. The validity of stage theory rests on meeting two criteria: sequencing of initiation and association in the initiation of drugs.

Sequencing Component

With the increase in adolescent drug research in the 1960s, researchers began studying polydrug use among adolescents. Hamburg (Hamburg, Kraemer, & Jahnke, 1975) and Kandel (Single et al., 1974) were among the first researchers to study the drug histories of young people. The first suggestion of stages in adolescent drug involvement came from analyses of cross-sectional and longitudinal data on patterns of drug involvement among high-school students from New York (Kandel, 1975b; Single et al., 1974). The first study used cross-sectional retrospective lifetime drug initiation data (i.e., 0 = abstention, 1 = initiation for each drug) from 8,206 9^{th}-12^{th} grade students who were surveyed in fall 1971 (Single et al., 1974). Patterns of lifetime use of 14 types of legal and illegal drugs were fit to a Guttman scale and analyzed.

Guttman scaling, initially created to examine the ordering of attitude measures (Guttman, 1944), is well-suited for investigating stages in drug involvement, particularly since this type of scale assumes that the items being examined are unidimensional and cumulative (Mueller, 1986). When fitting lifetime drug initiation data to a Guttman scale, researchers can determine how many and what drugs respondents have initiated. Without drug initiation time data (e.g., age of initiation), however, it is not possible to accurately determine the ordering of drug initiation. The fit of the data implies that respondents at any one stage (or position) on the scale have initiated the drug at that stage and all drugs ranked lower on the scale, but not drugs ranked higher on the scale.

In brief, Kandel and colleagues' (Single et al., 1974) cross-sectional results indicated that patterns of lifetime drug use could be arranged according to a cumulative, unidimensional, and hierarchical sequence of drug involvement. The sequence consisted of four distinct stages in adolescent drug involvement: (Stage 1) beer or wine; (Stage 2) hard liquor or cigarettes; (Stage 3) marijuana; and (Stage 4) other illegal, hard drugs.

Kandel (Single et al., 1974) aptly noted that in order to ensure temporal ordering in the initiation of multiple drugs, prospective longitudinal data are required. Prospective data also decrease response bias (e.g., heaping, recall errors), a plausible threat to the validity of retrospective, cross-sectional data

(Harrison, 1995). Consequently, Kandel (1975b) sought to replicate her cross-sectional findings using longitudinal drug initiation data derived from two cohorts: a representative sample of 5,468 New York 9th-12th grade students, and a representative subsample of 485 12th grade students. Both samples were initially surveyed in fall 1971 (baseline) and followed up 6 months later (Time 1). The senior subsample was followed up a second time (Time 2) after graduation. Instead of examining lifetime initiation drug data (as was done in the previous cross-sectional study), Kandel obtained age of initiation data for 14 types of legal and illegal drugs and fit this data to a modified Guttman scale. The drug sequence generated from the cross-sectional study was replicated in both longitudinal analyses.

Specific longitudinal results (Kandel, 1975b) from the sample of 9th-12th grade students included the following. Concerning baseline drug abstainers, 39% initiated one or more drugs within the six-month follow-up period. Specifically, 36% initiated legal drug use (i.e., alcohol or cigarette use), 2% initiated marijuana use without first initiating one or more legal drugs, and 1% proceeded directly to illegal, hard drug use without initiating legal drugs or marijuana use. It is interesting to note that while only 2% of baseline drug abstainers initiated marijuana use without first initiating legal drug use, 27% of legal drug initiates initiated marijuana use within the follow-up period. Among baseline legal drug initiates, only 4% proceeded directly to hard drug use without first initiating marijuana use. No baseline beer or wine initiates proceeded directly to illegal drug use (including marijuana use) during the follow-up period without first initiating hard liquor or cigarette use. Moreover, roughly 2 to 3 times as many beer and wine initiates proceeded directly to hard liquor use than cigarette use, and more than 50% of those who reported cigarette initiation at baseline proceeded directly to hard liquor during the 6-month follow-up period.

Kandel's (1975b) results also suggest that marijuana initiation may constitute an important stage between legal drug initiation and hard drug initiation. Mentioned earlier, only 1% of drug abstainers and 4% of legal drug initiates proceeded directly to hard drug use within the follow-up period. Within this six-month follow-up period, however, 26% of marijuana initiates proceeded to hard drug use. Although an important drawback of the study was the short-term, six-month follow-up period between baseline assessment and Time 2, the four stages that Kandel identified did not differ by grade-level, gender, race, or parents' education.

Subsequent longitudinal work by Kandel and colleagues (Yamaguchi & Kandel, 1984a, 1984b) involved following the same sample of New York high school students from adolescence to young adulthood over a period of nine years (with multiple waves of assessment). The results of this research founded the basis of further specification and refinement of the initial stage model that Kandel proposed. In general, follow-up analyses confirmed the initial sequence identified when the sample was in high-school, but an additional stage, medically prescribed psychotropic drugs, was identified and added to the extreme end of the sequence, thereby constituting the final stage.

Although this stage was added, researchers commonly view drugs other than alcohol, cigarettes, and marijuana as “other illegal drugs” (or hard drugs), with medically prescribed drugs typically collapsed into the hard drug stage when empirical tests of the sequence are conducted (see, e.g., Donovan & Jessor, 1983; Golub & Johnson, 2001; Hawkins, Hill, Guo, & Battin-Pearson, 2002b). Also, in accommodating the finding that involvement with drugs, for many adolescents, first begins with either cigarette or some type of alcohol use (Kandel, 1975b; Yamaguchi & Kandel, 1984a, 1984b), Kandel combined hard liquor and cigarette initiation with beer and wine initiation in finalizing the first stage of drug involvement: legal drug use.

In sum, the results of Kandel’s early cross-sectional and longitudinal research found the drug sequencing hypothesis outlined in stage theory. Involvement in drug use is characterized as a progressive and hierarchical sequence that begins with legal drugs (alcohol or cigarettes) and proceeds to illegal drugs, beginning with marijuana. The sequence has been recognized by researchers as constituting three discrete stages: (Stage 1) legal drug use; (Stage 2) marijuana use; and (Stage 3) other illegal drug use.

There are two major characteristics of this drug sequence that are worth discussing. First, although Kandel (2002) maintains that the use of certain drugs precedes the use of other classes of drugs, progression in stages of use are probabilistic, not inexorable. Specifically, one’s position at a particular stage in the drug involvement sequence is not indicative of a fully deterministic, causal progression from the drug that has been initiated to the initiation of a drug higher in the sequence. This is in stark contrast to the notion underlying stepping-stone theory, which asserts that all individuals who initiate a given drug will invariably initiate the use of more serious drugs (Grinspoon, 1971; MacCoun & Reuter, 2001).

Kandel (2002) posits that, in the aggregate, the drug sequence is hierarchical in nature, meaning that most individuals do not navigate the full sequence from legal to more serious, illegal drug use. Progression in the sequence is experienced by successively smaller numbers of individuals, such that only a subgroup of individuals at each stage progress to the next stage in the sequence. For example, of those who initiate legal drug use (alcohol or cigarettes), a smaller proportion go on to initiate marijuana use, and of those who initiate marijuana use, an even smaller proportion go on to initiate hard drug use. Kandel maintains that while the initiation of drugs lower in the sequence is necessary, the initiation of a lower-stage drug is in itself an “insufficient condition” for progressing to more serious drug use (Kandel, 2002, p. 4). The stages are “facilitative,” in that the initiation of a lower stage drug increases an adolescents’ risk of initiating a drug higher in the drug sequence (Kandel, 1975b, p. 912). Hence, Kandel is clear in maintaining that progression from one stage to the next higher stage is not deterministic, but probabilistic. The discussion below concerning the prediction component of stage theory speaks to the likelihood of progression.

Second, the sequencing proposition outlined in stage theory differs from stepping-stone theory with respect to the nature of drug involvement. Stepping-stone theory was first advanced in the early 1900s by Prohibitionists intent on banning cigarette and alcohol use (Grinspoon, 1971). Initially, proponents of the theory postulated that cigarette and alcohol use causes hard drug use, although there was virtually no specification concerning how exactly this occurs. With the upsurge in Mexican immigration to the U.S. in the late 1920s and 1930s, coupled with Mexican xenophobia and a reported increase in marijuana use in the U.S. (particularly among Mexicans), stepping-stone theory evolved into a more "scientific" theory grounded in human physiology. Proponents argued that marijuana use causes hard drug use due to the release of chemicals in the brain that cause marijuana users to crave a more intense high (MacCoun & Reuter, 2001). After "stepping up" to the use of hard drugs (e.g., heroin, cocaine, amphetamine), marijuana users stop using marijuana and become hard drug users (Grinspoon, 1971; Towns, 1917; Zimring & Hawkins, 1992).

In contrast to stepping-stone theory, stage theory is founded on the notion that the drug involvement sequence is an additive (or cumulative) phenomena, whereby drug users continue to use drugs lower in the sequence after they initiate drug use at stages higher in the sequence (Kandel, 1975b, 2002). For example, Kandel maintains that legal drug initiates who initiate marijuana use do not stop using alcohol or cigarettes; they simply add marijuana to their drug repertoire. Kandel also hypothesizes that regression in the sequence occurs, with desistance in drug involvement following the same pattern as initiation (e.g., marijuana desistance prior to the desistance of legal drug use).

Association Component

Kandel's (see, e.g., Single et al., 1974; Kandel, 1975b, 1978b; Kandel & Faust, 1975) early research primarily centered on identifying common drug sequences and did not pay particular attention to examining or explaining why the common drug involvement sequence she identified exists. In the late 1970s, however, Kandel began attending to this issue.

Although stage theory is commonly misinterpreted as a contemporary stepping-stone theory (which has resulted in controversy and a certain degree of discount), Kandel (see, e.g., Kandel, 1975b, 1980a, 1982, 2002, 2003) consistently has maintained that the use of one drug does not (in itself) cause the use of other drugs, noting "entry into a particular stage…is not a sufficient prerequisite for entry into the next higher stage" (Kandel, 2002, p. 4). She argues that a time-ordered sequence of initiation may reflect one or more underlying processes, and not necessarily that the specific physiological effects of one drug cause the initiation of another drug in the next stage in the sequence (as proposed by stepping-stone theory). Aside from positing that the initiation of one drug serves as a risk factor for the initiation of a drug higher in the drug sequence, Kandel (2002) also contends that two characteristics of one's drug history are particularly important predictors of progression: age of initiation and frequency of drug use.

Early Age of Drug Initiation

Kandel (2002; Kandel & Logan, 1984) argues that among users of a given drug, those who initiated at a relatively early age are at an increased risk (compared to other users) for both initiating drugs higher in the sequence and abusing or becoming dependent on the drug in question. Hence, not only is the initiation of a given drug itself a risk factor for advancement to more serious drug use, but timing of initiation also is important. Although Kandel does not provide an explicit biological age or age range that constitutes “early,” her proposition is based on findings stemming from research that suggests early drug initiation is that which occurs prior to 15 years of age (see, e.g., Breslau & Peterson, 1996; Grant & Dawson, 1997, 1998; Kandel & Logan, 1984; Kandel & Yamaguchi, 1985, 1993; Robins & Przybeck, 1990; Yamaguchi & Kandel, 1984b).

To illustrate, Kandel and Yamaguchi’s (1985) longitudinal study of a cohort of young adults (previously studied as adolescents) found that after controlling for various individual and environmental risk factors, men who initiated marijuana use at 14 years of age or younger were significantly more likely to have initiated hard drug use by 25 years of age than men who initiated marijuana use during their early adult years. In fact, among men who initiated hard drug use by 25 years of age, only 9% had initiated marijuana use at 21 years of age, while 71% had initiated marijuana use by 14 years of age.

Concerning age of alcohol and cigarette initiation, Kandel and Logan (1984) found that among male legal drug initiates who did not proceed to marijuana or hard drug use by end of the follow-up, 24% had initiated alcohol or cigarette use by 14 years of age, versus 43% who initiated legal drug use at 16 years of age or older. Moreover, among males who initiated marijuana or hard drug use during the follow-up period, 25% had initiated legal drug use by 14 years of age, versus just 6% who had initiated legal drug use at 16 years of age or older. As additional evidence, cross-sectional research indicates that adolescents who initiate powder or crack-cocaine initiate soft drug use two years earlier (on average) than adolescent soft drug initiates who abstain from all forms of cocaine use (Kandel & Yamaguchi, 1993). In addition, research by Robins and Przybeck (1990) found that risk for initiating marijuana use is greatest when alcohol initiation occurs at or prior to 15 years of age.

Frequency of Drug Use

Kandel (2002) also frames the relationship between extent of drug use and risk for drug progression from a probabilistic dose-response perspective, maintaining that as use of a lower stage drug increases in frequency, risk for progression to a higher stage drug increases. Kandel's isolation of drug use frequency as a salient risk factor for progression in the drug sequence derives from a set of longitudinal studies she and her colleagues conducted in the 1980s using data from the cohort of adolescents initially surveyed in the early 1970s (see, e.g., Kandel, Davies, Karus, & Yamaguchi, 1986; Kandel & Logan, 1984; Kandel & Yamaguchi, 1985; Yamaguchi & Kandel, 1984b).

In general, this body of research found that frequent use of alcohol is associated with a significant increase in the likelihood of initiating marijuana, while frequent use of marijuana is associated with an increased risk for initiating hard drugs. For example, Kandel and colleagues (Yamaguchi & Kandel, 1984b) found that among male marijuana users who had not initiated hard drug use within the follow-up period, 75% had used marijuana between 10 and 99 times in their lifetime, while 53% self-reported using marijuana more than 100 times in their lifetime. Kandel also found that among marijuana users in their mid-20s, those who frequently used marijuana were significantly more likely than infrequent users to initiate other illegal drugs within the next 10 years (Kandel & Yamaguchi, 1985).

In reflecting on these (and other) findings, Kandel (2002) posited that although early age of drug initiation may directly increase one's risk of initiating a higher stage drug, it is possible that frequency of drug use may constitute the mechanism (i.e., mediating variable) through which early age of initiation operates in impacting risk for progression. In other words, when one initiates a given drug at a relatively early age (e.g., prior to 15 years), and use of the given drug continues, this longer duration of time (compared to one who initiates at an older age) may increase one's risk for using the drug on a frequent basis. In turn, frequent use may directly increase one's risk for initiating a drug higher in the drug sequence.

Aside from characteristics of drug use (e.g., timing of initiation and frequency of use), Kandel also posits that different environmental and individual-level risk factors are associated with the initiation of different types of drugs. This component of stage theory, often identified separately from Kandel's sequencing hypothesis as adolescent socialization theory (Kandel, 1980a, 1982), derives largely from research Kandel conducted in the late 1970s and 1980s (e.g., Kandel, 1978a; Kandel & Andrews, 1987; Kandel et al., 1978). Unfortunately, this component of stage theory remains less thorough and well-developed than the drug sequencing hypothesis that Kandel poses. In fact, as the following discussion underscores, Kandel (2002) falls short of fully explicating what individual and social influences differentially predict the initiation of different classes of drugs.

Socialization Factors

The adolescent socialization component of Kandel's stage theory primarily emphasizes antisocial parent and peer influences as salient risk factors for the initiation and use of drugs. Kandel (1982, 1985) draws upon Akers' (1977) social learning constructs of differential association, differential reinforcement, imitation, and definitions, along with Hirschi's (1969) social bonding element of commitment, in isolating four broad types of risk factors for adolescent drug use: parental influences, peer influences, adolescents' involvement in antisocial behaviors, and adolescents' prodrug beliefs and norms.

According to Kandel, drug use is one of many adolescent behaviors that result from an interaction between individual characteristics and the competing influence of parents and peers. Although Kandel fails to explicate what these individual characteristics are, she argues that parents and peers are two social agents most influential in shaping adolescent behavior (Kandel, 1982).

Drawing from Akers' (1977) social learning theory, parental and peer influence is posited to occur through two major processes: imitation and social reinforcement. In line with Akers' (1977) proposition concerning behavioral modeling and imitation, imitation occurs when youth model their attitudes, norms, and behaviors after others (e.g., parents and peers) with which they closely interact (i.e., differential association). Kandel hypothesizes that adolescents are more likely to initiate drug use if their parents or peers use drugs.

The second process, social reinforcement, refers to adolescents' internalization of the attitudes, norms, and beliefs (i.e., definitions) to which they are exposed, and the manifestation of these definitions in the form of personal attitudes and behaviors (Kandel, 1982). Adolescents are more apt to internalize attitudes and norms and exhibit corresponding definitions and behaviors if these norms and attitudes are modeled and approved of by others. Similar to Akers' (1977) conceptualization of definitions and the process by which attitudes and norms give way to corresponding behaviors, Kandel (1982) hypothesized that youth who interact with parents or peers who hold prodrug norms or beliefs are at an increased risk for initiating drug use. Youth also are at an increased risk for drug initiation if they hold prodrug norms or engage in delinquent and antisocial acts.

An additional process centers on Hirschi's (1969) bonding element of commitment. Kandel (1982) hypothesized that regardless of parental behavior or norms, a positive and strong parent-child relationship inhibits involvement in drug use (or other deviant behavior) by increasing commitment to conventional lines of action. Negative child-parent interaction, characterized by a lack of parent-child closeness (e.g., attachment), a lack of involvement in adolescents' activities on the part of mothers, and inconsistent (or nonexistent) parental discipline, constitutes the third type of parental risk factor for drug initiation (Kandel, 1980b).

In sum, parents and peers are hypothesized to influence adolescent behavior through modeling (e.g., drug use) and social reinforcement (e.g., prodrug norms). In contrast to the hydraulic (or exclusive) model of parental and peer influence, which is the notion that parental influence only impacts particular types of behavior while peer influence for that given behavior is non-existent (Kandel, 1985, 1989), Kandel advocates a “transitional” model of parent and peer influence (Kandel, 1980b). Both types of influence are important in shaping all types of adolescent behavior and across all stages of adolescence, but the saliency of respective influences differs according to the behavior under consideration.

Similar to other researchers (Thornburg, 1982), Kandel (Kandel & Lesser, 1969, 1973) maintains that parental influence is more important for longer-term and life-long decisions and behaviors (e.g., work, college, and life plans), while peer influence is more salient in influencing more immediate and short-term behavior and lifestyle issues, such as hobbies, extracurricular activities, and drug use. As youth mature and begin to think about and plan long-term goals, peers decline in importance as a socializing agent, while parents increase in importance.

Universal and Drug-Specific Risk Factors

Aside from explicating the two primary processes by which parent and peer influences impact adolescent behavior (e.g., imitation and social reinforcement), Kandel hypothesizes that some risk factors within the four broad types of influences (i.e., parent influences, peer influences, adolescent antisocial behavior, and adolescent prodrug beliefs and norms) are universal, while other risk factors are drug-specific and aid in explaining progression in the drug sequence. Unfortunately, Kandel fails to concretely identify what these universal and drug-specific risk factors are. For instance, although she argues that all drug initiates and users share certain characteristics and attributes (regardless of drug type), since they all are assumed to have begun drug involvement with the same drugs (i.e., legal drugs), Kandel fails to explicate these common risk factors (see Kandel, 1980b, 1982, 1985, 1989, 2002). In calling for further research in this area, Kandel (1982) merely hypothesizes that these respective common risk factors assume differential importance for predicting legal drug initiation versus marijuana or hard drug initiation.

In an effort to offer support for her hypothesis concerning universal and drug-specific risk factors, Kandel relies on findings from her series of longitudinal studies (e.g., Kandel, 1978a; Kandel et al., 1978; Margulies, Kessler, & Kandel, 1977; Paton, Kessler, & Kandel, 1977). In examining predictors of alcohol, marijuana, and hard drug initiation among representative samples of New York youth followed from adolescence to young adulthood, Kandel and colleagues found that some risk factors are universal, while others are drug-specific. In general, the findings suggest that social factors may be more predictive of soft than hard drug initiation, while psychological factors may be more salient predictors of hard than soft drug initiation.

Compared to alcohol abstainers, alcohol initiates were more apt to have engaged in minor delinquent acts, been exposed to drug-using parents and peers, spent more time with peers than parents, and initiated cigarette use. Parental drug use, particularly liquor use, was a more salient predictor of adolescent alcohol initiation than peer drug use (Kandel, Kessler, & Margulies, 1978; Margulies et al., 1977).

Risk factors that distinguished marijuana initiates from marijuana abstainers included adolescent beliefs and norms permissive of marijuana use, involvement with marijuana-using peers, and participation in the same minor delinquent acts predictive of alcohol initiation. While associating with marijuana-using peers was found to be particularly important in predicting marijuana initiation, family domain predictors (e.g., parental drug use and prodrug norms) were not predictive of adolescent marijuana initiation (Kandel et al., 1978). Although not directly related to the current research, Kandel and colleagues (Paton et al., 1977) also found that hard drug initiation is preceded by poor relationships with parents, association with drug-using peers who engage in legal and illegal drug use, depression and psychological distress, and frequent marijuana use.

Alternative Explanation for Sequencing and Association

It is important to note that Kandel's stage notion of drug involvement confronts another theoretical perspective: the common factor hypothesis. Although this model is the theoretical antithesis of stage theory, it also provides an explanation for why a common drug involvement sequence (i.e., legal drug use > marijuana use > hard drug use) may exist.

In general, the common factor perspective refers to the notion that the use of any drug (or participation in any deviant behavior) is an opportunistic response to environmental conditions (e.g., availability of drugs) on the part of individuals who share a common proneness to anti-social or deviant behavior. From this perspective, the time-ordered sequencing of drug involvement that Kandel (2002) hypothesizes is believed to primarily reflect age-graded differences in drug availability and opportunities to engage in types of drug use, not unique parent, peer, or individual-level risk factors. For example, Jessor and Jessor (1980) argued that an identifiable time-ordering in the initiation of multiple drugs merely reflects the order in which opportunities to use soft and hard drugs are presented. Individuals with the highest propensities for deviance are likely to initiate the first drug they are offered, which happens to be alcohol in most cases (Jessor & Jessor, 1980).

Common factor proponents assert it is logical that most adolescents first initiate alcohol or cigarette use, and not marijuana or hard drugs. Since alcohol and cigarettes are legal (for individuals of legal age), more socially accepted, and more widely available than other drugs, the opportunity for initiating legal drugs is greater than that for marijuana or hard drugs. In turn, compared to hard drug use, the greater degree of social acceptability ascribed to marijuana use (see, e.g., Gillmore, Catalano, Morrison, Wells, Iritani, & Hawkins, 1990; Hundleby & Mercer, 1987; Johnston, 1985; OAS, 2004b), coupled with more adults in the general population engaging in marijuana than hard drug use (OAS, 2004b, 2006a), explains why among the class of illegal drugs, marijuana use is initiated first (and by more individuals) than hard drug use.

With respect to Kandel's propositions concerning the utility of early age of drug initiation and frequency of drug use for increasing the risk of progressing in the drug sequence, common factor advocates argue that both risk factors are spuriously related. Specifically, frequency of drug use does not mediate the relationship between early age of drug initiation and risk for initiating other, more serious drugs (as proposed by Kandel). Common factor proponents argue that early age of initiation for one drug, frequent use of that drug, and increased risk for initiating more serious drugs are all highly correlated because these factors are controlled by an underlying propensity toward deviance (see, e.g., Clark, Cornelius, Kirisci, & Tarter, 2005; Elliott et al., 1985; Jessor, 1978; Kaplan, 1975).

Summary

Of the three research questions[3] that found the current study, Kandel's stage theory provides guidance for Research Question #1 and, to a lesser extent, Research Question #2. It does not, however, provide an adequate explanation of age-graded differences in soft drug initiation (Research Question # 3). Moreover, stage theory does a better job of identifying a time-ordered, hierarchical sequence of drug involvement than explaining why this sequence exists.

According to stage theory, involvement in drug use is a developmental phenomena that evolves over time and begins with legal drug use (i.e., alcohol or

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[3] To reiterate, these three questions include: (1) Is involvement in soft drug use a sequential and hierarchical phenomena? If it is, what is the typical sequence of involvement?; (2) What factors predict soft drug initiation and age of initiation among adolescents?; and (3) Since the initiation of soft drug use appears to vary, in part, as a function of biological age, what factors predict soft drug initiation and age of initiation for youth at different stages of adolescence?

cigarettes). Marijuana is the next drug that is initiated, followed by other illegal and hard drugs. This drug involvement sequence is hierarchical in nature, whereby progression in the sequence is experienced by successively smaller numbers of people.

Kandel (2002) isolates two salient risk factors for progressing along the drug involvement sequence: early age of drug initiation and frequency of drug use. Adolescents who initiate legal drug use at a relatively early age (e.g., prior to 15 years) are at an increased risk of initiating marijuana use. Adolescents who use legal drugs on a frequent basis also are at an increased risk for initiating marijuana use (just as youth who initiate and use marijuana are at an increased risk for initiating hard drug use). Kandel (2002) posits that frequent drug use may constitute the mechanism through which early age of initiation increases risk for initiating drugs higher in the drug sequence.

In addition to early age of drug initiation and extent of drug use, Kandel (2002) also hypothesizes that four broad psychosocial domains of influence predict soft drug initiation and increase risk for progressing in the drug sequence. Youth involvement in antisocial or delinquent behaviors is associated with an increased risk for initiating drug use. Youth who interact with parents or peers who use drugs, or who espouse prodrug norms and attitudes, are at an increased risk for internalizing prodrug norms and imitating the drug behaviors they observe others model. Hence, parental and peer prodrug norms and behaviors serve as risk factors for adolescent initiation of drug use.

Kandel's (2002) explication of the differential saliency of parent and peer influences is rather ambiguous and under-developed. Her propositions concerning drug-specific psychosocial risk factors also are not clear and are lacking in many areas. Moreover, Kandel does not adequately address age-graded changes in the saliency of psychosocial influences, or explain whether the ordering of drugs in the drug involvement sequence is the result of increased exposure to drug-using others (particularly peers) and decreased bonding to prosocial others (particularly parents). The social development model, however, compensates for many of these weaknesses and offers a more lucid explanation for age-graded changes in risk and protective factors.

Social Development Model

SDM is a general, developmental theory of behavior that uses a similar process of socialization to explain both prosocial and antisocial behavior (e.g., delinquency, deviance, and drug use). Initially developed in the mid-1980s by J. David Hawkins (Hawkins & Weis, 1985), the theory synthesizes key propositions and constructs from differential association theory (Sutherland & Cressey, 1978), social learning theory (Akers, 1977), and social control theory (Hirschi, 1969) into a unified and comprehensive explanation of how the initiation, continuation, and desistance of prosocial and antisocial behavior occurs. Moreover, the theory addresses Research Questions #2 and #3 of the current research. Although not designed to address Research Question #1, some of the theory's main constructs and assumptions may be used in an attempt to explain why a time-ordered, hierarchical drug sequence exists.

In locating the key proximate causes of all behavior, SDM draws upon and extends social control theory's notion of the social bond. The social learning constructs of imitation and social reinforcement are adopted to explicate how behaviors are initiated, maintained, and terminated. The construct of differential association is used in conceptualizing how prosocial and antisocial behaviors (and respective underlying causal chains) constitute two distinct developmental trajectories (i.e., pathways). With prosocial and antisocial socialization constituting similar processes, the major difference between respective behaviors lies in the nature of agents and institutions of social control to which youth differentially associate and learn beliefs and corresponding behaviors.

In short, the theory hypothesizes that the development of prosocial and antisocial behavior is influenced by the degree of involvement and interaction with prosocial or delinquent peers (differential association); the skills required and the costs and benefits (i.e., rewards) for interaction (social learning); and the extent to which youth subsequently become bonded to prosocial and antisocial social units and institutions (social control). The prosocial trajectory decreases the likelihood of antisocial behavior (e.g., soft drug initiation), thereby increasing the likelihood of prosocial behavior (e.g., soft drug abstention), while the reverse is hypothesized to hold for the antisocial pathway (Catalano & Hawkins, 1996).

In contrast to most traditional theories of delinquency, SDM takes an explicit developmental approach to explaining behavior. Recursive relationships between behavior and risk and protective factors are hypothesized, with these bidirectional relationships occurring both within and between four periods of social development: preschool, elementary school, middle school, and high-school (Catalano & Hawkins, 1996). At each developmental stage, youth develop either prosocial or antisocial behaviors, with behavioral change located predominantly in age-specific changes in the relative saliency of risk and protective factors. These risk and protective factors emanate from the social units and institutions in which youth interact and are exposed: family, peers, school, and the community. Youths' personal characteristics and the nature of parental measures of informal control also are important for shaping the nature of social bonds and the beliefs and behaviors learned by youth.

Key Explanatory Factor

The key explanatory feature of SDM lies in the causal priority ascribed to the social bond. Similar to Hirschi's (1969) conception of the social bond, Hawkins (Catalano & Hawkins, 1996) asserts that prosocial bonding serves as a key informal control mechanism, whereby antisocial behavior is inhibited through a "stake in conformity" that the social bond produces. This "stake in conformity" influences the perceived costs and benefits of engaging in self-serving behavior (e.g., drug use), with the potential jeopardization of existing prosocial relationships and investments perceived as a heavy risk and a potential cost of engaging in the given behavior.

Hawkins' (Catalano & Hawkins, 1996) conceptualization of the social bond differs from Hirschi's (1969) conceptualization in two major ways. First, in contrast to Hirschi's (1969) assertion that the bond consists of four elements (i.e., attachment, commitment, involvement, belief), Hawkins (Catalano & Hawkins,

1996) argues that attachment to others (and institutions) and commitment to lines of action (together) shape one's belief system. From this perspective, attachment and commitment together constitute a conduit through which agents of control may transpose a belief system onto youth. If youth predominantly associate with prosocial others, then prosocial bonding will occur, with prosocial beliefs (and consequent behavior) the product of this bond. In a similar vein, if youth predominantly associate with antisocial others, then antisocial bonding will occur, with antisocial beliefs transposed onto youth and internalized (resulting in antisocial behavior). Both processes serve similar functions, to informally control youth. Similar to Hirschi (1969), Hawkins (Catalano & Hawkins, 1996) further conceptualizes prosocial attachment, commitment, and belief as protective factors that inhibit antisocial behavior. As an extension of social control theory, however, SDM conceptualizes antisocial attachment, commitment, and beliefs as risk factors for antisocial behavior.

Second, SDM also differs from social control theory in terms of how involvement, the fourth element of Hirschi's (1969) social bond, is conceptualized and viewed as operating. Contrary to Hirschi's (1969) argument that involvement in prosocial activities works conjointly with the other three bonding elements to inhibit deviant behavior, a key underlying assumption of SDM is that involvement serves as a precursory mechanism requisite for establishing attachment and commitment (and ultimately a belief system).

The nature of attachment and commitment (prosocial or antisocial) is largely contingent on the types of activities and behaviors youth involve themselves in. If youth become involved in prosocial activities, opportunities for prosocial attachment and commitment present themselves. On the other hand, if youth become involved in antisocial activities (e.g., minor deviant acts, like cheating on homework), opportunities for antisocial attachment and commitment occur. Hence, the involvement construct in SDM is quite important; it constitutes a “fork in the road” in terms of the nature of subsequent bonding, beliefs, and ultimately behavior.

Development of the Social Bond

Discussed in Chapter 3, one major weakness of social control theory is the fact that it does not explain the underlying process by which the social bond is formed, sustained, or weakened. SDM compensates for this drawback by specifying three factors as the causative mechanisms responsible for the development and maintenance of the social bond (Catalano & Hawkins, 1996). Specifically, the level of bonding to a particular social unit (e.g., family) or institution (e.g., school) reflects: (1) opportunities for involvement in activities (Hirschi’s involvement element); (2) skills requisite for participating and interacting in the social unit or institution; and (3) recognition (e.g., rewards) for involvement that is provided by the corresponding social institution or agent (Catalano & Hawkins, 1996).

Opportunities for involvement constitute the potential that youth have for interacting with others in activities. Skills for involvement refer to various skills (e.g., social, intellectual, or athletic) that enable youth to establish and maintain social relationships and complete tasks and activities that correspond with given social units or institutions (e.g., academic skills requisite for succeeding in school). Finally, recognition for involvement constitutes Akers' (1977) construct of positive reinforcement, which essentially consists of rewards for engaging in particular behaviors.

In order for youth to develop bonds to prosocial others and institutions, they need to be provided with opportunities to acquire prosocial skills, opportunities to be involved in prosocial activities, and rewards, recognition, and other types of social support when they become involved in prosocial behavior. Together these factors facilitate socialization by aiding in the establishment of the social bond. Without providing these things, youth will be weakly bonded to prosocial units and institutions.

The following discussion directs attention to the causal mechanisms that underlie each behavioral trajectory and the associated paths of SDM. The general model explains the socialization processes hypothesized to occur across developmental periods. Discussion then turns to four sub-models that were developed to explain the relative saliency of social agents and institutions within four periods of social development. Finally, although not explicitly designed to address Research Question #1, some of SDM's key propositions are applied to Kandel's (2002) stage theory in an effort to provide a clearer explanation concerning why a time-ordered and hierarchical sequence of drug involvement may exist.

General Model

Both prosocial and antisocial trajectories are conditioned by three major types of exogenous factors (distal factors) that structure the social development process. These distal factors include individual constitutional factors, position in the social structure, and external constraints (Catalano & Hawkins, 1996). Constitutional factors constitute stable individual states and traits (e.g., personality characteristics) that have been found through empirical research to be predictive of antisocial behavior. Examples of these risk factors include low self-control, impulsivity, aggressiveness, hyperactivity, difficult temperament, and early onset depression. Similar to Akers' (1998) conceptualization, position in the social structure constitutes demographic characteristics, including age, race, gender, and socioeconomic status (SES).

External constraints refer to both formal and informal social controls that impact the degree of positive reinforcement that adolescents perceive for involvement in a given behavior. Examples of external constraints include formal laws; parental discipline and child-rearing style (e.g., parental monitoring, clarity and content of familial rules and parental expectations for youth conduct); quality of the parent-child relationship; and norms and beliefs of important others, including peers, parents, and other adults (Catalano & Hawkins, 1996).

These three types of exogenous factors are hypothesized to structure the nature and content of social development processes, such that when opportunities, skills, social bonding, and learning processes are taken into account (i.e., controlled for in prediction models), the effects of these factors on behavior (antisocial or prosocial) are rendered insignificant. Hence, Hawkins (Catalano & Hawkins, 1996) hypothesizes that these factors constitute distal predictors of behavior capable of being completely mediated by more proximate socialization factors.

Prosocial Trajectory

The key explanatory constructs that lead to prosocial behavior include: (1) opportunities for prosocial involvement; (2) prosocial involvement; (3) skills for interaction and involvement; (4) prosocial rewards; (5) prosocial bonding (i.e., prosocial attachment and commitment); and (6) belief in the moral order (Catalano & Hawkins, 1996). Together, each of these protective factors is linked to form a protective factor chain that unfolds over time both within and across periods of social development.

Position in the social structure (i.e., age, race, gender, SES) and constitutional factors structure the types of (and extent to which) prosocial opportunities are available. In turn, prosocial opportunities provide youth with the ability to involve themselves in small (but important) prosocial lines of action (e.g., helping mom clean the house, or helping dad wash the car). Involvement in prosocial behaviors is responded to with positive reinforcement.

Four factors impact the receipt of prosocial rewards: (1) the degree of prosocial involvement; (2) the extent to which youth possess the requisite skills for interaction; (3) external constraints; and (4) constitutional factors (Catalano & Hawkins, 1996). Once youth are rewarded for their prosocial involvement, bonding to conventional institutions and others (in the form of attachment and commitment) begins to take root.

Prosocial attachment and commitment, in turn, facilitate in youth a belief in conventional norms and the moral authority of law. With prosocial beliefs and norms internalized, the initiation or continuation of prosocial behavior (e.g., soft drug abstention) occurs. This initiation or continuation of prosocial behavior is made possible by youth imitating the corresponding behaviors that have been modeled by the prosocial individuals or groups to which they are attached. Hence, similar to Akers' (1977) proposition, prosocial norms and beliefs constitute the key proximate cause of prosocial behavior, with these norms internalized prior to the activation of the corresponding behavior.

Antisocial Trajectory

Similar to social learning theory (Akers, 1977), Hawkins (Catalano & Hawkins, 1996) assumes that deviant peers (particularly drug-using peers) constitute the primary socializing agent implicated most often in the initiation of soft drug use. In contrast to social learning theory, however, SDM focuses on the causal processes underlying the relationship between delinquent peer association and adolescent antisocial behavior. Specifically, the theory locates the origin of this relationship in weak bonding to prosocial others (e.g., parents and peers) and institutions (e.g., school). Weak conventional bonding constitutes a salient risk factor for association with (and eventual bonding to) delinquent peers (Catalano & Hawkins, 1996).

Hawkins (Catalano & Hawkins, 1996) further hypothesizes that three different etiological pathways lead to antisocial behavior, with each respective path representing slightly different causal processes that culminate with a unique proximal risk factor for antisocial behavior. Within the context of drug use, soft drug initiation is hypothesized to be directly caused by: (Path 1) perceived rewards for involvement in drug use; (Path 2) attachment and commitment to drug-using others and antisocial institutions; and (Path 3) the internalization of antisocial beliefs or norms.

On the first path, key explanatory factors include: (1) antisocial opportunities; (2) antisocial involvement; (3) skills for interaction; and (4) antisocial rewards. Similar to how exogenous factors condition prosocial development processes, position in the social structure and individual constitutional factors have a direct impact on the types of antisocial opportunities that are available. In turn, antisocial opportunities lead to involvement in non-drug antisocial behaviors (e.g., minor non-drug delinquent acts). After these antisocial behaviors are initiated, they are rewarded by those with whom youth interact (particularly deviant peers). Two factors impact the degree to which minor antisocial behaviors are rewarded. External constraints constitute a protective factor that condition the degree to which youth are rewarded for antisocial behavior. Skills for antisocial interaction also have a direct, positive impact on the degree to which the given antisocial behavior is rewarded. For example, a youth who is skillful in graffiti art may receive more positive reinforcement from peers for this illegal behavior, as compared to a youth who is less skillful.

Once antisocial behavior is rewarded, Hawkins (Catalano & Hawkins, 1996) hypothesizes that this reward structure has a direct, positive impact on the likelihood of soft drug initiation. The reason for this direct relationship is because the perceived rewards stemming from initiating these drugs outweighs the perceived costs. Since youth traversing this path (and all other antisocial paths) are not strongly bonded to prosocial others and institutions, there is little to jeopardize in terms of investments in prosocial relationships and lines of action. If youth perceive there is a low risk associated with getting caught using drugs, this low perceived risk also can contribute to the decision to initiate soft drug use.

On the second path, key explanatory factors (in order of causal importance) include: (1) antisocial opportunities; (2) antisocial involvement; (3) skills for interaction; (4) antisocial rewards; and (5) antisocial bonding. The same causal process (i.e., risk factor chain) outlined in the first pathway operates in this pathway. The only difference here is that instead of a direct relationship between antisocial rewards and soft drug initiation, Hawkins (Catalano & Hawkins, 1996) hypothesizes that rewards lead to increased attachment to deviant and drug-using others (particularly peers), as well as commitment to deviant behavior.

This antisocial bonding, in turn, constitutes a salient risk factor for soft drug initiation (and other deviant behaviors). On this path, these types of antisocial attachments and commitments are hypothesized to constitute the motivational impetus for initiating soft drug use. Commitment to antisocial behavior develops from previous positive experiences with engaging in other, more minor forms of deviant behavior. The more rewards, rather than costs, that are produced from past involvement in these deviant behaviors, the more committed youth will become to initiating more serious forms of deviant behavior, such as soft drug use (Catalano & Hawkins, 1996).

Finally, key explanatory factors found on the third antisocial path include all of the factors found in the first and second path, plus an additional risk factor that is the proximal cause of soft drug initiation: antisocial beliefs or norms. The same causal process that operates in the second pathway also operates in this pathway, although antisocial bonding (antisocial attachment and commitment) has a direct positive impact on antisocial beliefs or norms in this pathway (Catalano & Hawkins, 1996). In turn, antisocial beliefs (e.g., prodrug norms) constitute a salient risk factor for soft drug initiation.

In contrast to the full mediating capacity of prosocial beliefs (on the prosocial trajectory), Hawkins (Catalano & Hawkins, 1996) hypothesizes that antisocial beliefs (or norms) only partly mediate the impact of antisocial bonding and learning variables (and interaction and skill constructs) on soft drug initiation. This hypothesized partial mediation accommodates the direct impact that antisocial bonding and rewards is posited to exert.

Age-Graded Sub-Models

Social control theory (Hirschi, 1969) did not provide clear propositions concerning age-graded changes in social bonding. In attempting to address Research Question #3, SDM compensates for this weakness by positing that the influence of parents, peers, and schools is relative and dependent upon the stage of adolescent social development (and attendant maturational processes and dominant social contexts). Specifically, these age-defined periods of development provide different opportunities for social involvement. In turn, these varying opportunities eventually lead to age-graded differences in social bonding (e.g., differences in terms of kind and saliency), with behavioral differences the resultant outcome of these varying levels and types of bonding.

Hawkins (Catalano & Hawkins, 1996) articulated four sub-models (preschool, elementary school, middle school, and high-school) that constitute distinct, but interrelated, periods of psychosocial development. Risk and protective factors and resultant outcomes are hypothesized to be reciprocally related across developmental periods through “cross-path links.” These cross-path links bind levels and types of social bonding, social learning, and behavioral outcomes in one developmental period to types of socialization processes at the beginning of the next developmental period (Catalano & Hawkins, 1996).

Specifically, on the prosocial trajectory, three social control and learning constructs in a given developmental period are hypothesized to have a direct positive impact on perceived opportunities for involvement and interaction in the next period of social development: (1) belief in the moral order; (2) attachment and commitment to prosocial others and institutions; and (3) perceived rewards for prosocial interaction and involvement (Catalano & Hawkins, 1996).

On the antisocial trajectory, four constructs in a given developmental period are hypothesized to have a direct positive impact on perceived opportunities for problem behavior and antisocial interactions in the next period: (1) antisocial behavior itself; (2) antisocial beliefs or norms; (3) attachment and commitment to antisocial others and institutions; and (4) perceived rewards for problem behavior and interaction with deviant others (Catalano & Hawkins, 1996). Antisocial behavior in one developmental period also is hypothesized to have a direct negative impact on perceived opportunities for prosocial involvement and interaction in the next developmental period.

Preschool

Although not directly relevant for the current research, some aspects of the preschool period of social development are worth discussing. During this socialization period, constitutional factors (e.g., temperament, neurocognitive deficits) are hypothesized to play an important role in hindering the development of cognitive skills. Opportunities for prosocial involvement are confined to social contexts involving the family, the most important socializing unit during this period of development, and child care providers (Catalano & Hawkins, 1996). Parental monitoring and discipline during the preschool period is hypothesized to decrease the likelihood that children during early childhood will perceive that rewards exist for engaging in antisocial problem behaviors.

Elementary School

Hawkins' (Catalano & Hawkins, 1996) theoretical model for the elementary school period is geared explicitly toward explaining the initiation of delinquency and drug use (p. 174). Arguably, the initiation of these behaviors between 1st and 6th grades constitutes a period considered "early." Not only does epidemiological evidence document that most youth initiate drug use during mid-adolescence (Johnston et al., 2007; OAS, 2006a), but empirical research (discussed in

Chapter 1 and earlier in this chapter) further suggests that drug initiation prior to 15 years of age constitutes a time period that may be considered "early" due to the risk that such initiation has for drug abuse, dependence, and use of other drugs (see, e.g., Breslau & Peterson, 1996; Grant & Dawson, 1998; Kandel & Yamaguchi, 1985; Robins & Przybeck, 1990). Due to Hawkins' focus on drug initiation during this time period, salient risk factors during elementary school may be considered predictive of early initiation of soft drug use.

During the elementary school period, schools join the family as important agents of social control and learning. Specific socializing agents include parents, teachers, and classroom peers. Although parents and teachers remain the most important agents of socialization during elementary school, Hawkins (Catalano & Hawkins, 1996) contends that peer interaction begins to increase toward the end of this school period.

Aside from the continued importance of family management and child-rearing practices, classroom management constitutes an important external constraint. Time spent on homework and other academic tasks also are hypothesized to greatly aid in enhancing cognitive and academic skills during this time period. Hawkins (Catalano & Hawkins, 1996) argues it is important that children at this age be provided opportunities to be involved in academic and non-academic activities at school, as well as opportunities to interact with classroom peers. These opportunities constitute protective factors that increase the likelihood that involvement in school will increase. Perceived rewards during this time period primarily center upon school grades and perceived peer support.

On the antisocial path, interaction with antisocial others generally is confined to family members, school personnel, and (to a lesser extent) peers, all of whom may be drug-involved or who engage in deviant behavior. Salient risk factors for soft drug initiation include antisocial bonding, conduct or behavioral problems (e.g., aggression), perceived peer rejection, and poor school performance. Perceived peer rejection and poor school performance at this age also are hypothesized to predict drug use in later periods of social development.

In terms of initiation timing, Hawkins (Catalano & Hawkins, 1996) hypothesizes that early drug initiation has a direct positive impact on perceived opportunities for interacting with drug-using others, and a direct negative impact on perceived opportunities for prosocial interaction and involvement. Hence, early drug initiation (e.g., during the elementary school years) impacts subsequent socialization processes (e.g., nature of social bonding and learning) in both current and subsequent periods of development, with the strength of antisocial bonding increasing (and prosocial bonding decreasing). Early drug initiation also is hypothesized to increase risk for engagement in more serious forms of antisocial behavior in later periods of development (Catalano & Hawkins, 1996).

Middle School

Although Hawkins acknowledges that drug initiation typically occurs during the middle school years, this particular sub-model is directed primarily at explaining the “diversity and frequency of drug use and delinquent behavior” among those who already have initiated these lines of action (Catalano &

Hawkins, 1996, p. 176). During the middle school years, three socialization units exert important influences: parents, school, and peers. By the end of middle school, peers trump other agents of socialization as the most salient socializing force.

Peer association (and consequent bonding and learning influences) is hypothesized to increase significantly during middle school. Compared to the preschool and elementary school periods of development, transition to middle school typically brings with it a considerable change in school context and peer interaction. Most elementary school children attend one central school building that consists of small classrooms. All classes are held in one classroom where students of similar age spend considerable time. Compared to later school years, there is little movement throughout school during the elementary school years, and the student-teacher ratio is relatively small. Upon transitioning to middle school, classes become larger, thereby increasing the student-teacher ratio; students many times must move from one class to another; personal belongings are kept in lockers instead of in classrooms; and interpersonal interaction expands to include progressively larger numbers of diverse peers of varying ages, teachers, and school administrators (Catalano & Hawkins, 1996).

This change in school context, coupled with an increase in interpersonal interaction and an increased desire for autonomy typical at this stage of adolescent development, work in concert to increase the importance that youth place on peer relationships. Hawkins (Catalano & Hawkins, 1996) hypothesizes that peer bonding increases in importance (as compared to earlier periods), with the nature of behavioral impact (i.e., antisocial or prosocial behavior) largely contingent upon the predominant influence (positive or negative) that peer networks exert. Both the behavior and norms of peers are salient predictors of adolescent behavior.

On the prosocial path, prosocial others include family members, teachers, and peers who abstain from drug use and deviant behavior. On the antisocial path, bonding to antisocial peers (drug-users or delinquents) constitutes a salient risk factor for drug use. Perceived rewards for antisocial involvement also include drug-specific rewards during this period, particularly perceptions of legal punishments for drug use and the nonsocial physiological rewards that stem from drug use itself (which Akers also touches upon). Those who already have initiated drug use base their perceptions of rewards upon past drug experiences, while drug abstainers contemplating drug initiation are hypothesized to form evaluations of drug effects based upon the drug-related rewards they observe others receive from use (Catalano & Hawkins, 1996).

High-School

Hawkins argues that not only have many drug initiates began use prior to entering high-school, but many risk and protective factors for drug use have taken root prior to the high-school years (Catalano & Hawkins, 1996). For example, Hawkins postulates that poor parental management and school performance constitute salient risk factors for drug use that emerge in earlier periods of development (e.g., preschool and elementary school).

In taking these observations and assumptions into account, Hawkins (Catalano & Hawkins, 1996) further characterizes the high-school period of social development as a time period in which salient risk and protective factors include those that aid in the continuation of prosocial behavior (e.g., continued drug abstention) and the progression of antisocial behavior from milder to more acute forms. Specifically, this sub-model seeks to explain drug abuse and serious delinquency, with both of these behaviors characterized by their respective frequency, persistence, and associated negative consequences.

During the high-school period, family, school, and peers remain important socializing forces, although community-level influences emerge as important in shaping late adolescent behavior (Catalano & Hawkins, 1996). Of these social units and institutions, peers and the community are hypothesized to exert the strongest influences (with peer influences trumping community influences), since it is during high-school that youth typically take advantage of increased opportunities to be involved in peer- and community-related activities (e.g., work, driving, sports, and social gatherings).

Compared to previous developmental periods, youth experience considerably less parental supervision while becoming increasingly involved in activities independent from parents and other adults. Prosocial family and school bonds are viewed as important, however, in inhibiting drug use and delinquency among those who have yet to initiate (Catalano & Hawkins, 1996).

On the prosocial path, adolescent drug abstainers are characterized as youth who predominantly associate with and are bonded to drug abstainers, particularly peers. In contrast, adolescents traversing the antisocial pathway predominantly associate with drug-using others, particularly peers.

Hawkins (Catalano & Hawkins, 1996) asserts that there are two distinct types of high-school drug users: experimental and regular drug users. Experimental drug users are those who use drugs on an irregular basis, in an effort to enhance autonomy. These youth were not exposed to (or did not exhibit) high levels of earlier risk factors. As these youth navigate through high-school, the social and nonsocial rewards associated with drug use wane, thereby serving as the motivational impetus to either decrease drug use or desist altogether by the end of high-school (Catalano & Hawkins, 1996).

In contrast, regular drug users in high-school are hypothesized to have been exposed to higher levels of early risk factors than experimental drug users. Compared to experimental drug users, regular drug users also perceive fewer rewards for prosocial involvement and interaction, and their perception of the social and nonsocial rewards associated with drug use does not wane over time as they navigate through high-school (Catalano & Hawkins, 1996). Relative to experimental users, regular drug users are at an increased risk for drug abuse.

Sequencing of Polydrug Initiation

SDM was not explicitly designed to address Research Question #1, although it does use several explanatory factors in explaining continuity, progression, and diversity in antisocial behavior. Hence, it is possible to use some of the theory's basic assumptions and key constructs to explain why involvement in drug use may constitute a series of ordered stages that are hierarchical and progressive in nature.

Discussed earlier in this chapter, Kandel's (2002) drug sequencing hypothesis outlines three discrete stages of drug involvement. These stages constitute a drug involvement continuum that begins with less serious drugs (legal for adults) and ends with more serious, illegal drugs. Legal drug use (i.e., alcohol and cigarettes) constitutes Stage 1, marijuana use Stage 2, and other illegal drug use Stage 3. Progression along this drug sequence is hypothesized to be hierarchical in nature, with successively fewer adolescents initiating drugs higher in the sequence. Two aspects of stage theory need to be verified or explained: the existence of time-ordered stages in drug use and the hierarchical nature of progression in the drug sequence.

Existence of Time-Ordered Stages

In an effort to explain why Kandel's hypothesized sequence of drug involvement may exist, it is reasonable to assume that Hawkins (Catalano & Hawkins, 1996) would support an interpretation based upon age-graded differences in drug availability and opportunity. In short, youth are more or less vulnerable to initiating certain drugs at different periods of adolescence because social context and interaction drive and define when most youth will first encounter people who can provide opportunities for different types of drug use.

SDM argues that social context and interaction are age-dependent factors that structure the opportunities that are available for engaging in various lines of action, including various types of drug use (Catalano & Hawkins, 1996).

Consistent with SDM's underlying assumption of hedonism, and the contention that all adolescents are at some level of risk for engaging in antisocial behavior (Catalano & Hawkins, 1996), when opportunities increase to engage in a particular type of drug use, there is an increased likelihood that one will act on the opportunity.

Discussed earlier in this chapter (when the common cause hypothesis was addressed), roughly 40 years of epidemiological drug research indicates that legal drugs are more widely available and used by more people in American society than illegal drugs, while marijuana is the most widely available and used of all illegal drugs (OAS, 2006a). Not only are alcohol and cigarettes more apt to be found in the homes of youth whose parents drink and smoke, these drugs also are more likely to be found in youth's homes than marijuana or other illegal drugs (Johnson, Bryant, Collins, Noe, Strader, & Berbaum, 1998). In a similar vein, due to the increased use of marijuana versus other illegal drugs, youth may have increased access to marijuana within their home than these other illegal drugs.

Outside of the home, SDM hypothesizes that as youth mature and their interaction in society becomes more autonomous, they increasingly are exposed to diverse types of people, including people who use legal and illegal drugs (Catalano & Hawkins, 1996). Since alcohol and cigarettes are legal drugs for adults (and are the most widely used and available drugs), youth are more apt to encounter people who use these drugs (as opposed to illegal drugs).

Consequently, there may be more opportunities to use these drugs than illegal drugs once networks of social interaction expand.

Hierarchy and progression. Compared to Kandel (2002), Hawkins (Catalano & Hawkins, 1996) articulates more clearly (although indirectly) what environmental and drug-related risk factors may explain progression along the drug involvement sequence that Kandel (1975b) proposes. Based upon the major propositions and constructs outlined in SDM, it is reasonable to assume that Hawkins would advocate the application of a modified common factor model to the drug progression component of Kandel's (2002) stage theory.

In short, progressive and hierarchical involvement in drug use may be located in the consequent social control and learning processes that stem from initial drug use. As the manifestation of an underlying causal chain of constitutional and environmental risk factors, early involvement in drug use (i.e., early age of drug initiation) may constitute a unique risk factor for progression to more serious types of drug use (i.e., progression in the drug sequence).

First, the recursive relationships within developmental periods that SDM articulates, as well as the cross-paths hypothesized to link antisocial behavior in one period of social development with antisocial behavior in another, both can be used to explain the association between the initiation of multiple drugs. Recursive relationships between constructs within a given developmental period would result in the initiation of more serious drug use within a few years (at most) of first initiating a less serious drug. In contrast, constructs that constitute cross-path links binding one developmental period to another would culminate in the initiation of a more serious drug several years (or more) after first initiating a less serious drug.

Hawkins (Catalano & Hawkins, 1996) acknowledges the notion of behavioral continuity and progression in recognizing that for some individuals, antisocial behavior constitutes a trajectory that unfolds over time. Minor acts are hypothesized to occur prior to (and be linked with) more serious future acts (Catalano & Hawkins, 1996). This is particularly evident with Hawkins' emphasis on "involvement in antisocial behavior" as an explanatory construct and risk factor. Discussed earlier, this construct refers to involvement in a specific antisocial behavior measured and predicted in the prior period of social development. This construct is distinct from and serves as a salient risk factor for the particular antisocial behavior being predicted in the current developmental period (Catalano & Hawkins, 1996, p. 167).

The notion that antisocial behavior constitutes a risk factor for more serious types of antisocial behaviors in later developmental periods is found in the four constructs that constitute cross-path links. Discussed earlier, these four constructs include: (1) antisocial behavior itself; (2) antisocial beliefs or norms; (3) attachment and commitment to antisocial others and institutions; and (4) perceived rewards for problem behavior and interaction with deviant others (Catalano & Hawkins, 1996).

Antisocial behavior (e.g., legal drug initiation) is hypothesized to impact perceived opportunities for prosocial and antisocial involvement and interaction, constructs that shape the nature and types of subsequent social control and learning processes (Catalano & Hawkins, 1996). Consistent with SDM, more serious involvement in drug use (e.g., marijuana initiation) can occur in one of three ways. First, positive experiences (i.e., rewards) stemming from initial involvement in legal drug use may serve as a sufficient motivational impetus for initiating a more serious type of drug.

Second, if these rewards do not constitute a sufficient motivational impetus, Hawkins (Catalano & Hawkins, 1996) hypothesizes that these rewards can further attenuate the strength of any existing prosocial bonds, while also increasing the strength of antisocial bonding (which already was established prior to first involvement in drug use). According to SDM, the bolstering of antisocial bonding can be sufficient for initiating a more serious type of antisocial behavior, such as the initiation of marijuana use.

Finally, if enhanced antisocial bonding is not sufficient in directly leading to the initiation of a more serious drug, Hawkins (Catalano & Hawkins, 1996) posits that this increase in antisocial bonding can lead to an increase in antisocial beliefs (e.g., prodrug beliefs), with these beliefs directly related to the initiation of more serious forms of antisocial behavior (e.g., more serious types of drugs). In the end, youth who have initiated all three soft drugs may have considerably lower levels of prosocial bonding than drug abstainers or legal drug initiates. In a related vein, soft drug initiates may have considerably higher levels of antisocial bonding than similar drug abstainers or legal drug initiates.

Another aspect of SDM may be applied to explain the hierarchical nature of progressive drug involvement that Kandel (2002) proposes. Youth who become involved in drug use at early ages (e.g., prior to 15 years) may be at a considerably higher risk (than their counterparts) for progressing in the hypothesized drug sequence. Hence, aside from the impact that initial involvement in drug use may have on subsequent levels of prosocial and antisocial bonding, timing of initiation (and attendant risk factors for such) also may explain, in part, progression from minor to more serious types of drug use.

Not only does Kandel (2002) posit that early age of drug initiation constitutes a salient risk factor for the initiation of more serious drugs, but Hawkins also provides a similar hypothesis. According to SDM, early age of antisocial behavior constitutes a salient risk factor for the continuity of, diversity in, and progression of this behavior (Catalano & Hawkins, 1996). Within the context of drug use, it is reasonable to assume that diversity in antisocial behavior refers to involvement with various types of drugs. Since not all youth who initiate drug use begin involvement prior to 15 years of age (or during elementary or middle school), those who do may be at an increased risk for both continued involvement with the drugs they first began using and the initiation of more serious drugs that constitute higher-ordered stages in Kandel's (2002) drug sequence.

It appears reasonable to assume that Hawkins (Catalano & Hawkins, 1996) would support the hypothesis that early drug initiates (compared to their counterparts) may possess individual constitutional risk factors that place them at increased risk for antisocial behavior (e.g., drug use), antisocial behavior that is initiated earlier than their counterparts, and progression in this behavior from minor to more serious forms (e.g., legal to illegal drug use). Discussed earlier, SDM postulates that constitutional risk factors (e.g., neurocognitive deficits, low self-control, impulsivity) hinder both the development of prosocial skills requisite for successfully completing tasks and opportunities for prosocial involvement. Hawkins also hypothesizes that these constitutional factors impact how rewards stemming from antisocial behavior are perceived (Catalano & Hawkins, 1996).

Youth who possess one or more constitutional risk factors may perceive involvement in antisocial behavior as more rewarding than prosocial behavior (relative to their counterparts), particularly if these youth are impulsive or possess low self-control. In turn, these youth may involve themselves in less prosocial activities then their counterparts This may lead to weak prosocial bonding and an increase in antisocial bonding. In addition, these youth may be more weakly bonded to prosocial others than their counterparts simply due to their constitutional make-up and the negative reactions they may perceive or actually receive from prosocial others who frown upon their stable personality characteristics.

In sum, constitutional factors may interact with socialization factors in providing both an avenue and the motivation to engage in soft drug use. Exaggerated perceptions of antisocial rewards stemming from non-drug antisocial behaviors committed in previous developmental periods may serve as a salient risk factor (coupled with other socialization risk factors) for early initiation of drug use. Consistent with SDM, once a soft drug is initiated, prosocial bonds are further attenuated, antisocial bonds are bolstered, and perceptions of rewards stemming from drug use also become enhanced (Catalano & Hawkins, 1996). This process may unfold over time, predisposing early drug initiates to progression in Kandel's (2002) hypothesized drug sequence.

Summary

In attempting to address Research Questions #2 and #3 (concerning universal and age-graded predictors of soft drug initiation), SDM draws upon differential association (Sutherland & Cressey, 1978); all four elements of Hirschi's (1969) social bond; and the social learning (Akers, 1977) constructs of differential reinforcement and imitation. Touched upon throughout this discussion, there are three key features of SDM that distinguish it from traditional and other developmental theories of delinquency.

First, concerning social control theory, SDM acknowledges and incorporates empirical research findings concerning the reciprocal relationship between social bonding and behavior, an obvious improvement over Hirschi's (1969) initial theoretical statement. SDM also attempts to account for the establishment of the social bond by explaining how it is established, sustained, and weakened, an explanation not found in social control theory. As well, it incorporates empirical research on the salient relationship between delinquent peer association and adolescent problem behaviors by explicating that bonding can be prosocial or antisocial in nature.

Second, similar to social learning theory, SDM articulates both risk and protective factors and pathways leading to both conforming and antisocial behavior. Contrary to social learning theory, however, SDM incorporates key constructs from social control theory in acknowledging the causal importance of attachment, commitment, and belief.

Third, SDM takes into account several developmental issues, including age-graded tasks, skills, and behavioral trajectories. Moreover, SDM takes into account the developmental nature of individual-environment interaction in specifying age-graded changes in the type and nature of influential agents and units of social control. With the inclusion of paths that constitute recursive relationships, the theory also explains transitions from one developmental period to the next in explicating how past behavior and socialization processes impact future behavior. This is helpful for explaining behavioral trajectories that constitute progression from minor to more acute or serious forms of antisocial behavior.

Aside from attempting to address Research Question #2, SDM also applies to Research Question #3 (i.e., adolescent stage-specific predictors of soft drug initiation). According to SDM, child and adolescent socialization (i.e., social bonding and learning processes) occurs primarily within the confines of families, schools, and peer groups. The influence of these socializing agents and institutions is relative and largely dependent upon biological age. Across developmental periods, deviant peers constitute the socializing agent most often implicated in the initiation of soft drug use among youth.

Although parents constitute the most salient socializing agent during elementary school, teachers and classroom peers also are important socialization forces. Aside from risk factors for soft drug initiation that are endemic to processes of socialization (i.e., antisocial bonding, beliefs, rewards, skills, and involvement), soft drug initiation during elementary school also is predicted by poor school performance and perceived peer rejection. These risk factors remain salient predictors of antisocial behavior in later developmental periods (e.g., increase in frequency or diversity of drug use).

During the middle school period, parents remain an important socializing agent, but peers emerge as increasingly important, particularly prior to entering high-school. Although parental bonding remains important during the high-school years, peers constitute the most salient socializing force of all social units. Community-level influences also increase during this time period, particularly among older adolescents who become increasing embedded in the larger society through extracurricular activities and increased autonomy.

Although portions of SDM may be tested with cross-sectional data, a full assessment of the theory's empirical validity requires the use of prospective, longitudinal data. The notion of time is a fundamental feature of the theory, with temporal ordering in the structure and operation of risk and protective factors largely dependent on various lapses in time that span one or more periods of social development (e.g., childhood to adolescence).

Conclusion

Both traditional and developmental explanations of adolescent drug use have a strong psychosocial orientation. The three traditional theories discussed in Chapter 2, as well as the two developmental theories discussed in this chapter, all view parents, peers, and schools as important agents of socialization.

Although stage theory (Kandel, 2002) and SDM (Catalano & Hawkins, 1996) approach the explanation of adolescent soft drug initiation from different perspectives, both draw upon aspects of social control theory (Hirschi, 1969), differential association theory (Sutherland & Cressey, 1978), and social learning theory (Akers, 1977).

Kandel's (2002) stage theory attempts to address Research Question #1 (i.e., existence of drug involvement sequence and why it exists), although it does offer propositions that may be used to address Research Question #2 (predictors of soft drug initiation). According to Kandel, drug use is a developmental phenomena that constitutes three discrete stages. Involvement begins with the initiation of a legal drug (i.e., alcohol or cigarettes) and proceeds to illegal drug use, beginning with marijuana. This drug sequence is hypothesized to be hierarchical in nature, with progression experienced by successively smaller numbers of people.

Kandel (2002) isolates two salient risk factors for progression in the drug sequence: early age of drug initiation and frequency of drug use. Relative to youth who initiate legal drug use at older ages and who use these drugs less frequently, adolescents who initiate legal drug use at relatively early ages (e.g., prior to 15 years) or who use legal drugs on a frequent basis are at an increased risk for initiating marijuana use (and eventually other illegal drug use).

Kandel (2002) also isolates four major types of psychosocial risk factors for drug initiation: peer influences, parental influences, involvement in deviant behavior, and prodrug norms and beliefs. Parental and peer influences are particularly important determinants of soft drug initiation, with parental and peer drug use, and parental and peer pro-drug norms, all constituting salient risk factors for drug initiation.

The adolescent socialization component of Kandel's (2002) stage theory is not well developed. In fact, it is rather ambiguous in terms of explaining what risk factors differentially predict involvement in one type of drug over another (and progression in the drug sequence). The theory also does not clearly explain whether progression in the drug involvement sequence is the result (in part) of age-graded changes in the saliency of psychosocial influences. Kandel (2002) urges adolescent drug researchers to identify drug-specific risk factors through empirical research.

SDM (Catalano & Hawkins, 1996) makes explicit attempts to address Research Questions #2 (i.e., predictors of soft drug initiation) and #3 (i.e., age-graded predictors of soft drug initiation). Taking both stage theory and SDM together, four common theoretical expectations emerge. Compared to their adolescent counterparts, the likelihood of adolescent soft drug initiation is greater among youth who: (1) possess beliefs, attitudes, or norms tolerant of deviance or drug use; (2) engage in other problem behaviors (e.g., delinquency or the use of other drugs not under consideration); (3) interact with drug-using parents or parents who condone drug use; and (4) interact with drug-using peers or peers who condone drug use.

In addition, SDM hypothesizes that the likelihood of adolescent soft drug initiation is greater among youth who: (1) are more strongly attached to antisocial than prosocial agents and institutions of socialization; (2) are more strongly committed to antisocial than prosocial lines of action; (3) perceive more opportunities for involvement in antisocial than prosocial activities; and (4) perceive more rewards for engaging in drug use than abstaining from drug use.

SDM articulates that soft drug initiation is most often the result of antisocial peer influence. Peers are less important socializing agents in elementary school than in middle or high-school, while parental influence plays a salient role in the socialization of elementary school children. During the middle school years, parental influence wanes as peer influence increases. By the high-school period, peers constitute the strongest socializing agent, although community-level influences also are important.

SDM was not explicitly designed to address Research Question #1. Nonetheless, since the theory does explain behavioral continuity, progression, and diversity, it is possible to use some of the theory's basic assumptions and key constructs to explain why involvement in drug use may constitute a series of ordered stages that are hierarchical and progressive in nature. It is reasonable to assume that Hawkins (Catalano & Hawkins, 1996) would support an interpretation based upon age-graded differences in drug availability and opportunity. In short, youth are more or less vulnerable to initiating certain drugs at different periods of adolescence because social context and interaction drive and define when most youth will first encounter people who can provide opportunities for different types of drug use (Catalano & Hawkins, 1996). Since legal drugs are more available and are more widely used in society than illegal drugs, there are more opportunities to begin drug use with legal drugs.

It also is reasonable to assume that Hawkins would advocate the application of a modified common factor model to the drug progression component of Kandel's (2002) stage theory. In short, progressive and hierarchical involvement in drug use may be located in the resulting social control and learning processes that stem from initial drug use. As the manifestation of an underlying causal chain of constitutional and environmental risk factors, early involvement in drug use (i.e., early age of drug initiation) may constitute a unique risk factor for progression to more serious types of drug use.

With the discussion of stage theory and SDM complete, the following chapter directs attention to extant empirical research in an effort to provide further context for the methodology and hypotheses driving the current research. Empirical support for Kandel's (2002) drug sequencing hypothesis is presented, along with a brief overview of observed risk and protective factors for soft drug initiation.

CHAPTER 4

SEQUENCING AND PREDICTION RESEARCH

Two developmental theories of adolescent drug use were discussed in the previous chapter: stage theory (Kandel, 2002) and SDM (Catalano & Hawkins, 1996). Stage theory speaks to Research Question #1 of the current research by offering a drug sequencing hypothesis. In contrast, SDM is a well-established theory that can be used to address Research Questions #2 and #3.

The current chapter takes the following form. Empirical support for Kandel's drug sequencing hypothesis first is addressed. A broad overview of risk and protective factors for adolescent soft drug initiation then is provided. Where relevant, the empirical validity of SDM and the traditional theories covered in Chapter 2 are folded into this discussion. Attention then turns to the major gaps in the extant literature and lines of inquiry that are worthy of redress.

Sequencing Research

The observation that involvement in drug use is a relatively time-ordered, sequential, and hierarchical phenomenon was first recognized by Kandel and colleagues in a set of studies they conducted in the mid-1970s (Kandel, 1975b; Kandel & Faust, 1975; Single et al., 1974). Discussed in Chapter 3, the findings from this set of studies founded the basis of Kandel's drug sequencing hypothesis, one of two major components of stage theory. This drug sequence consists of three discrete stages: legal drug use (Stage 1), marijuana use (Stage 2), and other illegal drug use (Stage 3). The initiation of at least one drug that is legal for adults (alcohol or cigarettes) precedes the initiation of marijuana use. In turn, the initiation of marijuana use precedes the initiation of all other illegal drugs (i.e., hard drugs). Kandel posits that this sequence is hierarchical in nature, such that not all individuals progress through the entire sequence.

In short, there is a great deal of empirical support for Kandel's drug sequencing hypothesis. This sequence has been empirically confirmed across age, race, and geographic lines; at different times in history (1970s-2000); with the use of longitudinal, prospective data and cross-sectional, retrospective data; and with different analytic techniques. Although more research is needed, the sequence appears fairly valid in terms of its applicability to the drug involvement behavior of adolescents from Japan (Wada, 1999), Israel and France (Adler & Kandel, 1983), and Sweden and Finland (Kaprio, Hammar, Koskenvuo, Floderus-Myrhed, Langinvainio, & Sarna, 1982).

Numerous bibliographic scans and a search of several computer databases identified 30 U.S. studies that tested Kandel's sequencing hypothesis. These studies are summarized in Table 2 in terms of respective study designs, samples, drug sequences identified, degree of support for Kandel's drug sequencing hypothesis, and analytic techniques used. Roughly 85% of the studies (26/30) found full empirical support for Kandel's drug sequencing hypothesis. The following discussion characterizes the type of research that has been conducted. After this discussion, an explication of the results is provided, with attention paid to demographic differences in sequencing and the role of cigarette initiation in Kandel's proposed sequence of drug involvement.

Table 2.

Empirical Tests of Kandel's Drug Sequencing Hypothesis

Author/year	Design [a]	N [b]	Age/Grade at Measurement	Sample Residence [c]	Observed Sequence [d]	Empirical Support	Analytic Technique [e]
Andrews et al. (1991)	L	a. 756 b. 639	a. 10-16 yrs b. 11-17 yrs	U	A>C>M>O	Yes	Guttman
Baumrind (1990)	L	136	14 yrs	U	A>M>C>O	No	Guttman
Brook (1993)	C	1,132	7th-10th	U	A/C>M>O	Yes	Guttman
Brook et al. (1983)	C	932	9th-10th	U	A/C>M>O	Yes	Guttman
Brook et al. (1986)	C	356	13-18 yrs	U/R	A/C>M>O	Yes	Guttman
Costello et al. (1999)	L	1,420	10-16 yrs	R	A>C>M	Yes	Descriptives
Donovan & Jessor (1983)	C	a. 9,658 b. 3,958	a. 7th-12th b.10th-12th	U/R	NPA>M>PA>O	Yes	Guttman
Ellickson et al. (1992)	L	4,145	7th-10th	U	A>C>M	Yes	Guttman
Federman et al. (1997)	L	985	11/13/15 yrs	R	A>C>M	Yes	Descriptives
Fisher et al. (1987)	C	106	College undergrads	U	A>M>C>O (males) A>M>O (females)	Mixed	Guttman

(Table 2 continues)

(Table 2 continued)

Author/year	Design [a]	N [b]	Age/Grade at Measurement	Sample Residence [c]	Observed Sequence [d]	Empirical Support	Analytic Technique [e]
Fleming et al. (1989)	L	1,007	7th-11th	U	A/C>M>O	Yes	Guttman
Gfroerer et al. (2002)	C	99,752	12+ yrs	U/R	A/C>M	Yes	Descriptives
Golub et al. (2000)	L	892	15/18 yrs	U	A/C>M>O	Yes	Descriptives
Golub & Johnson (2001)	C	100,282	26+ yrs	U/R	A/C>M>O	Yes	Transition Analysis
Golub & Johnson (2002)	C	a. 87,915 b. 3,700 c. 441 d. 7,713	a-c. 12+ yrs d. 18+ yrs	a. U/R b-d. U	A/C>M>O	Yes	Transition Analysis
Gould et al. (1977)	C	1,094	10th-12th	U	A>M>O	Yes	Guttman
Guerra et al. (2000)	C	8,550	9th- 12th	U/R	A/C>M/O	Mixed	Descriptives
Hawkins et al. (2002b)	L	808	18 yrs	U	A>C>M>O	Yes	Latent Transition Analysis
Huba et al. (1981)	C	1,634	7th-9th	U	A>M>O	Yes	Guttman
Kandel (1975b)	L	6,453	9th-12th	U/R	A>C>M	Yes	Guttman
Kandel & Yamaguchi (1993)	C	1,108	12th	U/R	A>M>C>O (males) A&C>M>O (females)	Mixed	Log Linear Guttman

(Table 2 continues)

(Table 2 continued)

Author/year	Design [a]	N [b]	Age/Grade at Measurement	Sample Residence [c]	Observed Sequence [d]	Empirical Support	Analytic Technique [e]
Kandel & Yamaguchi (2002)	C	21,000	18-40 yrs	U/R	A/C>M>O	Yes	Log Linear Guttman
Kandel et al. (1992)	L	1,160	34-35 yrs	U/R	A>M>O (males) A/C>M>O (females)	Yes	Modified Guttman
Martin et al. (1996)	C	176	13-19 yrs	U	A>M>O	Yes	Descriptives
Mills & Noyes (1984)	C	a. 2,036 b. 2,414	8th/10th/12th	U/R	A/C>M>O	Yes	Guttman
Single et al. (1974)	C	8,206	9th-12th	U/R	A/C>M>O	Yes	Guttman
Welte & Barnes (1985)	C	27,335	7th-12th	U/R	A>M>O	Yes	Guttman
White et al. (1986)	C	1,381	12/15/18 yrs	U/R	A>M>O	Yes	Guttman
Yamaguchi & Kandel (1984a)	L	1,325	25 yrs	U/R	A>M>O (males) A/C>M>O (females)	Yes	Modified Guttman
Yu & Williford (1992)	C	3,000	16-24 yrs	U	A>C>M	Yes	Guttman

[a] C = cross-sectional studies that used retrospective data; L = longitudinal studies that used prospective data
[b] N = sample size
[c] U = urban; R = rural
[d] A = alcohol initiation; C = cigarette initiation; M = marijuana initiation; O = initiation of other illegal drugs; NPA = non-problematic alcohol use; PA = problematic alcohol use; / = or; & = required initiation; > = cumulative and hierarchical progression in drug initiation from 1+ drugs to another.
[e] Guttman = Guttman scalogram analysis; Descriptives = Categorical percentages.

Study Designs

Roughly 80% of the research testing Kandel's drug sequencing hypothesis used retrospective, cross-sectional data (n = 25). Of the 11 longitudinal studies conducted, six utilized drug initiation data collected during one wave of assessment (Baumrind, 1990; Costello et al., 1999; Federman et al., 1997; Fleming et al., 1989; Kandel et al., 1992; Yamaguchi & Kandel, 1984a). In terms of examining the sequencing of drug initiation, these six studies technically can be characterized as cross-sectional in nature. The remaining five longitudinal studies used prospective data (Andrews et al., 1991; Ellickson et al., 1992; Golub et al., 2000; Hawkins et al., 2002b; Kandel, 1975b). Utilizing prospective drug data to examine drug sequences constitutes the optimal approach for ensuring that temporal ordering in polydrug initiation is established. This is important because Kandel's drug sequencing hypothesis postulates that time transpires between the initiation of different stages of drug use. By following adolescents over time and assessing prospectively what drugs are initiated and when (e.g., at what biological age, during what calendar year and month), temporal ordering in drug sequencing can be ensured more adequately.

The second best approach to ensuring that temporal ordering in the sequencing of polydrug initiation is established is to use some type of retrospective drug data that is based upon a measure of time. Studies employing this method ask respondents to recall the biological age (or calendar year) that each type of drug was initiated. Although retrospective self-report data concerning the age at which drugs were initiated is subject to several major validity threats (e.g., forward telescoping, recall decay, and heaping), it is a viable method for establishing temporal ordering in the initiation of multiple drugs (Kandel, 2002). Of the 25 studies that utilized cross-sectional data, 10 studies collected retrospective age at drug initiation data and used this data in identifying drug sequences (Costello et al., 1999; Golub & Johnson, 2001, 2002; Gould et al., 1977; Guerra et al., 2000; Kandel & Yamaguchi, 1993, 2002; Kandel et al., 1992; Martin et al., 1996; Yamaguchi & Kandel, 1984a).

Drug Data

Of the 30 U.S. drug sequencing studies examined, 20 studies provided information concerning the historical period (or calendar year) in which drug data were collected. Listed in Table 3, most of these studies collected drug initiation data during the 1970s and 1980s for cohorts of youth born in the 1960s and 1970s (and adults born earlier). Of the six studies that were published in the 2000s, only one (Gfroerer et al., 2002) utilized data collected in the 21st century. Gfroerer and colleagues examined data from marijuana users who participated in the 2000 NSDUH survey. Their investigation focused on identifying predictors of marijuana initiation and using these risk factors to predict time to marijuana initiation. Unfortunately, they did not provide a direct test of Kandel's drug sequencing hypothesis. Their examination of drug sequences was restricted to marijuana users who initiated marijuana use within the past year. Moreover, only categorical percentages were calculated that detailed what percentage of the sample initiated alcohol, cigarettes, and combinations of all three soft drugs.

Hence, the only known published study that used data collected in the 21st century did not provide a direct test of Kandel's drug sequencing hypothesis.

All 30 studies employed measures of lifetime drug initiation (or ever-use), with the exception of three (Brook et al., 1983; Mills & Noyes, 1984; Yu & Williford, 1982). Although these studies found support for Kandel's drug sequencing hypothesis, measures of "current" drug use were utilized, not measures of lifetime drug initiation.

Table 3.

Historical Time Periods in which Drug Sequencing Data were Drawn

Time Period	Drug Sequencing Studies		
1970s: (n = 6)	▪ Baumrind (1990) ▪ Donovan/Jessor (1983)	▪ Gould et al. (1977) ▪ Kandel (1975b)	▪ Mills/Noyes (1984) ▪ Single et al. (1974)
1980s: (n = 11)	▪ Fisher et al. (1987) ▪ Fleming et al. (1989) ▪ Golub/Johnson (2001) ▪ Golub/Johnson (2002)	▪ Golub et al. (2000) ▪ Hawkins et al. (2002b) ▪ Kandel/Yamaguchi (1993) ▪ Mills/Noyes (1984)	▪ Welte/Barnes (1985) ▪ Yamaguchi/Kandel (1984a) ▪ Yu/Williford (1992)
1990s: (n = 7)	▪ Gfroerer et al. (2002) ▪ Golub/Johnson (2001) ▪ Golub/Johnson (2002)	▪ Guerra et al. (2000) ▪ Hawkins et al. (2002b)	▪ Kandel/Yamaguchi (2002) ▪ Kandel et al. (1992)
2000s: (n = 1)	▪ Gfroerer et al. (2002)		

Note. A few studies collected data from multiple time periods.

Analytic Techniques

Documented in Table 2, over two-thirds of the studies (n = 21) used some form of Guttman scalogram analysis in identifying sequences of drug involvement. The majority of these studies (n = 17) utilized traditional Guttman techniques (Guttman, 1944; Menzel, 1953). The second most common analytic strategy employed was the calculation of categorical percentages, or the percentage of a sample that self-reported initiating each type of drug. Due to the popularity of using Guttman scaling for identifying drug initiation sequences, and the fact that the following discussion outlining results relies heavily on empirical findings stemming from traditional Guttman analysis, it is important to orient readers to this type of analytic technique. The following discussion provides a brief synopsis of Guttman scaling. More indepth explanation of this analytic technique is reserved for Chapter 6 (Quantitative Methods).

Guttman Scalogram Analysis

Guttman scaling is well-suited for analyzing the ordering of different types of drugs, since essential properties of a Guttman scale are unidimensionality and cumulation (Guttman, 1944). In terms of drug involvement, a Guttman scale assumes that if an individual initiated a given drug, that individual also has initiated all drugs lower in the scale, but no drugs higher in the scale. Hence, a positive score on a higher ranking drug infers a positive score on all lower ranking drugs, but the reverse does not hold. For example, Kandel contends that the initiation of alcohol or cigarette use precedes the initiation of marijuana use, while the initiation of marijuana use precedes the initiation of other illegal drug use. If the drug sequence to be tested is alcohol>cigarettes>marijuana>hard drug use (with > signifying cumulative and hierarchical progression in drug involvement), it is assumed that an individual who has initiated cigarette use also has initiated alcohol use, but not marijuana or any other illegal drug located higher in the scale.

When Guttman scalogram analysis is employed, typically an a priori sequence is tested (e.g., Kandel's stage sequence) by fitting the prespecified sequence to existing drug data. Once data are fit to this a priori sequence, other sequences sometimes are tested to determine which drug sequence provides the optimal fit. An optimal fit is determined conjointly by assessing two coefficients: the coefficient of reproducibility (CR) and the coefficient of scalability (CS). These indices will be discussed in Chapter 6 (Quantitative Methods). For purposes of understanding the following discussion of Guttman scaling results, it is important to note that a valid Guttman scale is one in which there are few deviations (or errors) from the a priori sequence. If drug involvement does fit a valid Guttman scale, the CR will be .90 or higher (0-1.0 range), while the CS will be .60 or higher (0-1.0 range) (Guttman, 1944; Menzel, 1953). When a Guttman scale obtains these coefficients, involvement in drug use can be assumed to follow a unidimensional and cumulative pattern.

A Guttman scale that provides optimal fit *implies*, but does not prove, temporal ordering in drug use. Hence, the presence of an ordered drug involvement scale does not necessarily reflect the temporal sequence of progression through drug use (Single et al., 1974). The ordinal appearance of a Guttman scale is based upon prevalence estimates, with involvement in each drug type coded dichotomously (0 = abstention, 1 = initiation). While a well fit Guttman scale signifies that an individual responding affirmatively to initiating a drug positioned at Stage 3 is likely to have responded positively to initiating drugs that constitute Stages 1 and 2, it is not necessarily the case that this individual initiated the Stage 1 drug prior to the Stage 2 drug, and the Stage 2 drug prior to the Stage 3 drug.

To establish temporal ordering, researchers using cross-sectional data must incorporate some type of time measure (e.g., age at drug initiation) into Guttman analysis. Of the 21 studies that employed some type of Guttman scalogram analysis, seven utilized data concerning age at drug initiation when identifying the most common drug sequence (Andrews et al., 1991; Ellickson et al., 1992; Gould et al., 1977; Kandel, 1975b; Kandel & Yamaguchi, 1993; Kandel et al., 1992; Yamaguchi & Kandel, 1984). Among these latter studies, three utilized prospective, longitudinal data, so temporal ordering in drug use between baseline and follow-up periods was accounted for (Andrews et al., 1991; Ellickson et al., 1992; Kandel, 1975b). With this foundational information in mind, much of the following discussion of results centers upon coefficients of reproducibility and scalability obtained through Guttman scalogram analysis.

Counter Support

One empirical study (Baumrind, 1990) does not support Kandel's drug sequencing hypothesis. Baumrind examined retrospective lifetime drug data derived from 136 white males and females from Berkeley and Oakland, California. Data were gathered in 1978 and 1979 during Time 3 of an on-going longitudinal study when respondents were 14 years of age. Guttman scaling was used, although temporal ordering in drug initiation was not assessed above and beyond that inferred by the Guttman scalogram analysis. With a CR of .86 and CS of .61 (minimally acceptable), the best fitting drug sequence for the total sample began with alcohol initiation, followed by marijuana initiation, followed by cigarette initiation, and ending with the initiation of other illegal drugs.

This sequence obviously counters Kandel's sequencing hypothesis, which posits that cigarette initiation precedes marijuana initiation. Roughly 65% of the sample self-reported marijuana initiation, compared to 45% who reported cigarette initiation. A closer examination of the data revealed that among marijuana initiates, 37% reported not initiating cigarette use. Baumrind points out that the results concerning the low initiation of cigarette use relative to marijuana initiation may have been due to an antismoking campaign that was launched in one of the cities in which the sample was drawn. This program was directed toward high-school students in the late 1970s, the same time period in which data were collected. Although Baumrind did not attempt to assess the degree to which rates of lifetime cigarette initiation may have been an artifact of this drug prevention initiative, it is possible that lower lifetime rates of cigarette initiation relative to marijuana initiation may be rooted in this antismoking campaign. Moreover, as the discussion below highlights, there is a considerable amount of evidence that suggests cigarette initiation typically occurs prior to marijuana initiation.

Full Support

Outlined in Table 2, 26 empirical studies found full support for Kandel's drug sequencing hypothesis. Across these studies, less than 2% of the total samples initiated marijuana or hard drugs prior to the initiation of legal drug use. Among the proportion of samples that became involved with drug use, the majority first initiated a legal drug (alcohol or cigarettes). Among legal drug initiates, a smaller proportion went on to initiate marijuana use. Across studies, very few respondents self-reported initiating hard drug use prior to marijuana use. Among legal drug initiates who initiated marijuana use, a small proportion initiated other illegal drugs, such as powder and crack-cocaine, heroin, or amphetamines (Golub & Johnson, 2001; Huba et al., 1981; Kandel, 1975b; Kandel et al., 1992).

Of the 17 studies that utilized traditional Guttman scalogram analysis in assessing the validity of Kandel's proposed drug sequence, 8 reported coefficients of reproducibility and scalability for total samples (see Table 4). With the exception of Fleming et al. (1989), drug involvement sequences identified using drug data from total samples produced reproducibility coefficients that either met or exceeded the .90 standard of acceptability (range = .90-.98). The coefficients yielded by Fleming et al. (1989) were well below standards of acceptability (CR = .75, CS = .51). Aside from this study, scalability coefficients produced by all the other studies listed in Table 4 exceeded the .60 standard of acceptability (range = .75-.98). Fleming and colleagues used cross-sectional data derived from Time 2 (1981-1982) of an on-going longitudinal study they conducted on roughly 1,000 7th-11th grade students from Milwaukee, Wisconsin. Although the optimal sequence they found (A/C>M>O) supports Kandel's sequencing hypothesis, both coefficients did not meet minimal acceptability standards. Data were derived from face-to-face interviews, which may have hindered the elicitation of full (or truthful) disclosure of drug involvement.

Table 4.

Empirical Support using Guttman Scaling: Coefficients of Reproducibility and Scalability

	Legal Drug(s)>Marijuana>Other Illegal Drugs	
Author/year	Coefficient of Reproducibility (CR)	Coefficient of Scalability (CS)
Total Sample		
Andrews et al. (1991)	.96	.88
Brook et al. (1983)	.90	---
Fleming et al. (1989)	.75	.51
Gould et al. (1977)	.96	.80
Huba et al. (1981)	.97	.84
Single et al. (1974)	.98	.82
Welte/Barnes (1985)	---	.75
White et al. (1986)	---	.98

--- not reported.

Age Differences

Two studies made an explicit effort to examine age differences in drug sequencing (Hawkins et al., 2002b; Welte & Barnes, 1985). Both studies found that Kandel's hypothesized drug sequence applies to adolescents regardless of age, although age differences in the extent to which youth progress in the drug sequence were found to exist. Younger adolescents tend to initiate a smaller number of drugs than older adolescents, and drug involvement by young adolescents tends to be confined to legal drugs (Stage 1 of Kandel's proposed drug sequence). The results from Hawkins et al.'s (2002b) longitudinal, prospective study are illuminating in this regard.

Hawkins and colleagues examined drug sequences at three discrete school periods: elementary school (10-12 years of age), middle school (13-14 years of age), and high-school (15-18 years of age). They also examined sequence transitions between two school periods: from elementary to middle school and from middle to high-school. Latent transition analysis was used to model static sequences at these discrete periods in time, as well as changes in sequences over time. Drawn from multiple waves of survey assessment, lifetime drug data were derived from 808 adolescent participants in the Seattle Social Development Project who were initially assessed in 1985 during elementary school (as 5th grade students).

Hawkins and colleagues found that as youth mature, the number of youth initiating any drug increases faster between middle and high-school than between elementary and middle school. Among elementary school students who were drug abstainers, 19% began drug involvement with alcohol use by middle-school. Roughly 19% of middle school students who were drug abstainers also began drug involvement with alcohol use by the time they reached high-school.

Age differences in the proportion of youth initiating other drugs emerged with cigarette initiation, and was most pronounced for marijuana initiation. Among alcohol initiates, 19% initiated cigarette use between middle and high-school, compared to 16% who initiated cigarette use between elementary and middle school. Among alcohol and cigarette initiates, marijuana initiation tended to occur at a higher rate between middle and high-school than between elementary and middle school. Roughly 14% of alcohol and cigarette initiates first used marijuana between elementary and middle school, while 36% initiated marijuana use between middle and high-school. Finally, a larger proportion of soft drug initiates began hard drug use between middle and high-school (46%) than between elementary and middle school (36%).

Hawkins and colleagues results speak to two issues concerning drug sequencing among adolescents. First, the drug initiation sequences of younger adolescents generally are shorter than those of older adolescents. A smaller proportion of youth initiate drug use during early than mid- or late adolescence, and drug involvement for most youth begins with alcohol or cigarettes (Stage 1 of Kandel's drug sequence). Second, relative to the transition between elementary and middle school, the transition between middle school (13-14 years of age in this study) and high-school (15-18 years of age) appears to be a particularly important time period for the initiation of cigarette and marijuana use.

Race Differences

Four cross-sectional studies examined race differences in the sequencing of drug involvement (Brook, 1993; Guerra et al., 2000; Kandel & Yamaguchi, 2002; Welte & Barnes, 1985). Regardless of race, all 4 studies found the most common drug sequence to be that proposed by Kandel, with either alcohol or cigarettes initiated prior to marijuana, and marijuana initiated prior to other illegal drugs. While this sequence was found to be the most common, these studies found that racial variability does exist in the degree to which this sequence is adhered and the extent to which individuals of different races progress along the sequence.

The three studies that examined differences between the drug sequencing of white respondents versus minority respondents all found that drug use appears more progressive among white individuals. Similar to findings from national drug epidemiological surveys, a larger proportion of white respondents reported initiating legal drugs, marijuana, and other illegal drugs than minority respondents. Moreover, a larger proportion of white versus minority respondents followed Kandel's proposed drug sequence.

Mixed Support

Of the 30 drug sequencing studies examined, three provided mixed support for Kandel's drug sequencing hypothesis. Two of these studies provided qualified support contingent upon respondents' gender (Fisher et al., 1987; Kandel & Yamaguchi, 1993), while mixed support from the third study (Guerra et al., 2000) is rooted in inherent limitations associated with the broad drug categorizations that were employed.

Gender Differences

Concerning qualified support contingent upon gender, Fisher et al.'s (1987) cross-sectional examination of retrospective drug data from 106 UCLA undergraduates found that Kandel's drug sequence applied to undergraduate females, but not male counterparts. For females, the best fitting sequence began with alcohol, followed by marijuana, and ended with other illegal drugs. Both scale coefficients were acceptable (CR = .95; CS = .75). When cigarette initiation was entered either as a discrete stage, or with alcohol (in constituting one stage of legal drug use), Fisher et al. reported that too many errors occurred, deeming the sequence noted above as the optimal sequence for female undergraduate students. No reproducibility or scalability coefficients were provided for the sequence in which cigarette initiation was loaded on the drug sequence, so it is not possible to assess reproducibility or scalability differences in the fit of these different sequences. For undergraduate males, Fisher et al.'s best fitting sequence did not support Kandel's drug sequencing hypothesis. With a CR of .93, and a CS of .67 (barely acceptable), the optimal sequence for males began with alcohol initiation, followed by marijuana initiation, then cigarette initiation, and finally the initiation of hard drugs.

There were several notable limitations associated with Fisher et al.'s (1987) data, however, which together cast some doubt on the validity of their findings. Major limitations include the fact that temporal ordering was not established (i.e., no time or age of initiation data utilized) and the sample size was quite low. Only 64 males and 42 females participated in the study. Other supplementary study weaknesses include the fact that no information was provided in the published report concerning the time period in which data were collected (e.g., 1970s). The age and racial composition of the sample also was not discussed.

Welte and Barnes (1985) cross-sectional examination of lifetime drug data from roughly 27,000 7th-12th grade students yielded results that conflict with those of Fisher et al. (1987). Regardless of age, race, or gender, youth were found to follow a definite pattern of progression that began with alcohol initiation, followed by marijuana initiation, and finally the initiation of hard drug use. Roughly 22,000 youth reported alcohol initiation, compared to 4,600 who reported the initiation of hard drugs. Moreover, only 40 adolescents (0.1%) reported hard drug initiation without ever using alcohol. Cigarette initiation was found to more of a necessary stage for females than males. Specifically, the following drug initiation sequence yielded a higher scalability coefficient for females than males (CS = .78, .69, respectively): alcohol>cigarettes>marijuana>pills>hard drugs. Hence, contrary to Fisher et al.'s (1987) results, Welte and Barnes (1985) results suggest that cigarette initiation is an important component of female drug sequencing, with results suggesting that cigarette initiation may play a larger role in drug sequencing among females than males.

The second study that yielded gender-specific support for Kandel's drug sequence was a cross-sectional study conducted by Kandel and Yamaguchi (1993). Similar to Welte and Barnes (1985) study, Kandel and Yamaguchi also found that among females, cigarette initiation tends to occur prior to marijuana initiation, although different results were obtained for drug sequencing among male respondents. Log-linear Guttman scaling was employed in examining retrospective age data for first use of alcohol, cigarettes, marijuana, powder cocaine, and crack-cocaine. The sample consisted of 1,108 12th grade students from New York.

Kandel and Yamaguchi (1993) obtained the following optimal drug initiation sequence for females: alcohol and cigarettes>marijuana>powder cocaine>crack-cocaine. As is evident, the typical sequence for females involved both the initiation of alcohol *and* cigarette use prior to marijuana initiation. For 12th grade males, the following drug initiation sequence was found to be optimal: alcohol>marijuana>cigarettes>powder cocaine>crack-cocaine. For the majority of the males, cigarette initiation followed marijuana initiation, but occurred prior to cocaine initiation. Hence, the major gender difference in drug sequencing found in Kandel and Yamaguchi's study was the positioning of cigarette initiation in the Guttman scale, with female high-school seniors tending to initiate cigarette use prior to marijuana initiation, while 12th grade male students typically initiated cigarette use after initiating marijuana use.

The only noticeable limitation associated with Kandel and Yamaguchi's (1993) study, which may have impacted sequencing results, is the manner in which drug initiation ties were handled. Ties occur when respondents provide the same biological age for the initiation of two or more drugs. Kandel and Yamaguchi broke these ties by categorizing respondents data for drug ties according to the drug sequence hypothesized a priori. For example, for a 12th grade female who reported alcohol and marijuana initiation as having occurred during the same biological year, Kandel and Yamaguchi classified this female as having initiated alcohol prior to marijuana, since this is sequence matches Kandel's sequencing hypothesis. Although no detailed information was provided concerning the number of ties in the data, it is possible that gender differences in the sequencing of drug involvement may be an artifact, in part, of the approach taken in treating the data.

Legal-Illegal Dichotomy

The last study that provided mixed results was conducted by Guerra et al. (2000). Discussed earlier, Guerra and colleagues examined cross-sectional, retrospective age data for the initiation of legal (alcohol, cigarettes) and illegal (marijuana, cocaine, and heroin) drugs. Roughly 160 drug initiation sequences were identified and collapsed into six categories of drug patterns: (1) abstention; (2) legal drug initiation only; (3) illegal drug initiation only; (4) legal followed by illegal drug initiation; (5) illegal followed by legal drug initiation; and (6) legal and illegal drugs initiated at the same time. Youth without age data for drug initiation were dropped from analyses and all sequences were examined in terms of weighted percentages.

In general, the results provide qualified support for Kandel's drug sequencing hypothesis. The most common sequence was the initiation of one or more legal drugs, with 39.4% of the sample falling into this category. The second most common pattern was the initiation of legal drugs followed by illegal drugs (30.9%). Only 1.8% of respondents reported initiating an illegal drug prior to a legal drug, and 0.3% reported the sole initiation of an illegal drug. Although Kandel's hypothesis generally is supported, with legal drugs more commonly initiated prior to illegal drugs, Guerra and colleagues did not examine the sequencing of individual types of drugs (e.g., alcohol, cigarettes, marijuana), so it is possible that hard drugs may have been initiated prior to marijuana. Although this is unlikely given the considerable amount of empirical evidence that demonstrates otherwise, their results can only provide limited support for Kandel's sequencing hypothesis.

Importance of Cigarette Initiation

The previous discussion concerning gender differences in the role of cigarette initiation underscores the empirical ambiguity surrounding the importance of cigarette initiation in drug sequencing. This uncertainty appears rooted in the different ways in which cigarette initiation has been examined and the conflicting results that have emerged. Kandel (2002) posits that the initiation of at least one legal drug, alcohol or cigarettes (or both), constitutes Stage 1 of the drug sequence, with this stage preceding marijuana initiation. Of the 30 studies examined, three studies did not examine the role of cigarette initiation at all (Donovan & Jessor, 1983; Martin et al., 1996; White et al., 1986), while findings from 19 studies that did examine the role of cigarette initiation in drug sequences of total samples have resulted in three differing conclusions. The following discussion takes these results up in turn.

Cigarettes as a Stage 1 Drug

Ten studies provided a direct test of Kandel's proposition that cigarettes constitute a Stage 1 drug (Brook, 1993; Brook et al., 1983; Fleming et al., 1989; Golub et al., 2000; Golub & Johnson, 2001, 2002; Gfroerer et al., 2002; Guerra et al., 2000; Mills & Noyes, 1984; Single et al., 1974). Instead of examining cigarette initiation as a stage discrete from alcohol initiation, these researchers classified individuals as Stage 1 initiates (legal drug initiation) if they reported initiating alcohol, cigarettes (or both). All 10 studies found support for Kandel's proposition. With the exception of the study conducted by Fleming et al. (1989), whose study limitations were discussed earlier, all studies that employed Guttman scaling and treated cigarette initiation as part of Stage 1 on the drug sequence produced coefficients that met minimal standards of acceptability (CR = .90-.98; CS = .76-.86).

Sequencing without Cigarette Initiation

In contrast to the support for Kandel's proposition that cigarette initiation constitutes a Stage 1 drug, two studies found that the optimal fitting sequence is one that excludes cigarette initiation altogether (Huba et al., 1981; Gould et al., 1977). Without scaling cigarette initiation, Huba et al.'s (1981) cross-sectional study found that the following Guttman scale of drug initiation produced optimal reproducibility (CR = .97) and scalability (CS = .84): alcohol>marijuana>hard drugs. Gould et al. (1977) also found that cigarette initiation did not scale well when placed with alcohol initiation (in Stage 1) on a Guttman scale (CR = .93; CS = .65). Their cross-sectional study examined lifetime drug data from 1,094 10th-12th grade, urban students from public and private schools in New Haven, Connecticut. The same Guttman scale ordering as Huba et al. (1981) produced optimal coefficients for reproducibility (CR = .96) and scalability (CS = .80).

Cigarette Initiation as a Discrete Stage

In contrast to Gould et al.'s (1977) and Huba et al.'s (1981) results, seven studies found that cigarette initiation loads on a Guttman scale as a discrete stage in itself that follows alcohol initiation, but occurs prior to marijuana initiation (Andrews et al., 1991; Costello et al., 1999; Ellickson et al., 1992; Federman et al., 1997; Hawkins et al., 2002b; Kandel, 1975b; Yu & Williford, 1992). Four of these seven studies constitute four of the five longitudinal studies that used prospective drug data.

Some of the more convincing evidence that cigarette initiation typically occurs after alcohol initiation, but prior to marijuana initiation, comes from the longitudinal, prospective study conducted by Hawkins et al. (2002b). Discussed earlier, Hawkins and colleagues examined drug sequences at three school periods (elementary school, middle school, and high-school) and examined sequence transitions from elementary to middle school, and from middle to high-school. Data were derived from a sample of 808 individuals assessed three times: 10-12 years of age (5^{th} grade in elementary school), 13-14 years of age (middle school), and 15-18 years of age (high-school). Latent transition analysis was used to model sequences at these discrete periods in time, as well as transitions over time.

At each of the three school periods, Hawkins and colleagues found that less than 2% of the sample initiated marijuana or hard drug use without first having initiated alcohol or cigarette use. Also, across all stages of adolescence, a smaller proportion of youth initiated cigarette use only (5% elementary, 3% middle school, 2% high-school) than initiated alcohol use only (26% elementary, 26% middle school, 15% high-school). The most common drug status among elementary and middle school students was alcohol initiation only (26% each), while the second and third most common drug initiation sequences were alcohol and cigarette initiation (15% elementary, 22% middle school) and alcohol, cigarette, and marijuana initiation (6% elementary, 10% middle school). In contrast, the most common drug sequence among high-school students was alcohol, cigarette, marijuana, and hard drug initiation (25%), followed by alcohol, cigarette, and marijuana initiation (20%). The third and fourth most common sequences were alcohol and cigarette initiation (18%) and alcohol initiation only (15%).

Concerning transitions in drug sequences and statuses over time (elementary to middle school and middle to high-school), Hawkins et al. (2002b) found that the most common drug to be initiated was alcohol (19% in both transition periods), followed by cigarettes (16% from elementary to middle school, 19% from middle to high-school). Having initiated both alcohol and cigarette use, students were most likely to have initiated marijuana (14% from elementary to middle school, 36% from middle to high-school).

Results from these four, prospective, longitudinal studies, as well as supplemental evidence from studies examining transitions between the initiation of legal drugs and marijuana suggest that alcohol initiation occurs prior to cigarette initiation for most adolescents, while the initiation of these two soft drugs occurs prior to marijuana initiation among those adolescents who initiate marijuana use. Due to the fact that findings from two studies counter this, and 10 studies did not examine cigarette initiation separately from alcohol initiation, more research is needed in this area.

Summary

According to Kandel (2002), involvement in drug use is a sequential, cumulative, and hierarchical phenomena that begins with the initiation of one or more legal drugs. Stage 2 constitutes marijuana initiation, while Stage 3 constitutes other illegal drugs. At the aggregate-level, not all individuals who initiate legal drug use go on to initiate marijuana use, and of the small proportion that do initiate marijuana use, an even smaller proportion go on to initiate hard drug use. An indepth assessment of 30 studies conducted in the U.S. revealed that this drug sequence yields considerable empirical support, and with few exceptions. Kandel’s drug sequence has been empirically confirmed across age, race, and geographic lines; at different times in history (1970s-2000); with the use of both prospective and retrospective data; and with different analytic techniques.

Variability in Kandel’s drug sequence appears rooted in factors related to race and gender. Not only do white individuals self-report higher rates of soft and hard drug initiation to begin with, but white drug users tend to follow Kandel’s drug sequence to a larger degree than black or Hispanic drug users. Studies examining gender differences in drug sequencing suggest that cigarette initiation may play a more prominent role in drug sequencing among females than males.

There is some empirical ambiguity concerning the role of cigarette initiation in Kandel’s drug sequence. Of the 30 studies assessed, 10 did not examine cigarette initiation separately from alcohol initiation, three studies did not examine the role of cigarette initiation at all, and findings from two studies suggest that cigarette initiation does not constitute a stage (whole or in part) in Kandel's drug sequence. However, four longitudinal, prospective studies suggest that alcohol initiation occurs prior to cigarette initiation for most adolescents who initiate legal drug use, while the initiation of these two soft drugs occurs prior to marijuana initiation among those adolescents who initiate marijuana use. Hence, these prospective studies suggest that cigarette initiation does play an important role in drug sequencing. Since ambiguity remains in this area, and none of the studies examined utilized data collected in the 21st century, examining the role of cigarette initiation in Kandel's drug sequence appears to be an area ripe for investigation.

Another issue that emerged from this assessment is the lack of research that utilized some type of time data (e.g., age at drug initiation) when examining drug sequences. Clearly, a proper assessment of drug sequences requires that some type of time data be incorporated into analyses, since the element of time clearly is evident in Kandel's sequencing hypothesis. Only five longitudinal studies examined utilized prospective drug data and most cross-sectional studies did not incorporate an element of time into analyses of drug sequencing. Cross-sectional research employing Guttman scalogram analysis, the typical analytic technique used in examining drug sequences, should incorporate a measure of time into the examination of drug sequences, since the temporal ordering inferred by a Guttman scale only implies, but does not necessarily prove that temporal ordering in the initiation of multiple drugs exists (Kandel, 1980a).

Prediction Research

Considerable effort has been directed at identifying risk and protective factors for soft drug initiation among youth. Most prediction research has been atheoretical, however, with full tests of theories (e.g., SDM, social control theory, and social learning theory) conducted far less often than empirical assessments of single theoretical constructs. The remainder of this chapter directs attention to predictors that have been empirically verified through both longitudinal and cross-sectional research. Where relevant, empirical support for SDM, social control theory, and social learning theory is highlighted.

This overview is designed to highlight the more prominent and empirically supported determinants of soft drug initiation (and age of initiation), identify relevant gaps in the literature, and ground the research hypotheses that will be tested in the current study. Due to the fact that far more research has examined risk factors than protective factors, this review centers predominantly on the former. Attention first is directed toward major risk and protective factors that have been identified through research where age effects were controlled. A brief overview of age-varying predictors is reserved for the discussion of literature gaps.

Addressed in the following chapter, one aspect of the current research involves cross-validating findings from the quantitative component with those stemming from a systematic, comprehensive assessment of the extant literature on predictors of adolescent soft drug initiation. In an effort to maintain the integrity of this cross-validation, the following discussion approaches the prediction literature, by drawing upon findings from extant comprehensive reviews of the literature. In order to contextualize a research consensus, applicable results from select individual studies are presented.

Major Predictors

Community Domain

The systematic examination of community-related risk factors for soft drug initiation is still in its infancy. In fact, only 10 of the 284 findings in Petraitis et al.'s (1998) review of predictors of marijuana use concerned community-related factors. Of the limited research in this area, there is a general convergence in findings concerning several risk factors (see Table 5).

Table 5.

Community Domain Risk Factors and Empirical Support

Theoretical Construct	Risk Factor	Extant Literature Reviews
Social learning constructs:	Drug use by non-parental adults; prodrug norms of non-parental adults	Conrad et al. (1992) DiFranza et al. (2006) Petraitis et al. (1998)
Community characteristics:	Perceived availability of drugs; Perceived easy access to drugs; Prodrug attitudes/messages; Exposure to media prodrug messages	Charlesworth & Glantz (2005) DiFranza et al., (2006) Hastings et al. (2005) Wakefield et al. (2003)

Soft drug availability (objective or perceived), community norms conducive to drug use, prodrug norms held by non-parental adults, exposure to prodrug media messages and advertising, non-parental adult drug use, and youth perceptions of the ease with which soft drugs may be obtained all have been shown to increase risk for alcohol (Donovan, 2004), cigarette (Charlesworth & Glantz, 2005; DiFranza et al., 2006), and marijuana initiation (Petraitis et al., 1998). These community-level factors generally have been shown to exert small to moderate risk effects. The most salient risk factors include perceived soft drug availability and perceived ease in obtaining these drugs (Robinson, Klesges, Zbikowski, & Galser, 1997). Perceptions of drug use by non-parental adults also is an important predictor. In Petraitis et al.'s (1998) review, for example, one study (Jessor et al., 1980) found that the odds of using marijuana were five times greater among adolescents who believed 25%-50% of non-parental adults smoked marijuana, compared to youth who believed less than 25% of non-parental adults smoked marijuana.

Protective factors. There is a paucity of research on protective factors found at the community-level of social life (Meschke & Patterson, 2003). Limited research suggests that strong bonding to teachers, ministers, prosocial neighbors, and institutions (e.g., community groups and religious organizations), as well as social support from these sources, reduces the likelihood of soft drug initiation among both children and adolescents (Werner, 1994; Werner & Smith, 1982).

School Domain

School-related risk factors predominantly fall within the purview of SDM and social control theory. Of the multiple ways in which schools can influence adolescent behavior, school bonding is of preeminent importance (see Table 6). School failure (i.e., poor grades), low commitment and attachment to education or school, little aspiration for post high-school education, chronic absenteeism, frequent truancy, and negative perceptions of coursework relevance all have been implicated as school-related bonding variables that increase risk for soft drug initiation (see, e.g., Derzon & Lipsey, 1999a, 1999c; Fleming, Kellam, & Brown, 1982; Kandel, 1980b).

Table 6.

School Domain Risk Factors and Empirical Support

Theoretical Construct	Risk Factor	Extant Literature Reviews
School bonding constructs:	Weak school attachment; Weak school commitment; Low school involvement; Weak educational aspirations	Conrad et al. (1992) Derzon & Lipsey (1999a) Derzon & Lipsey (1999c)
School behavior constructs:	Poor school performance; Truancy	

Of these predictors, academic performance, which emerges in importance during the early elementary school years, has consistently been found to be a major risk factor (Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Donnermeyer, 1992). One study found that the odds of initiating marijuana use were almost five times greater among adolescents earning D's or F's in school, compared to youth earning A's or B's (Epstein, Botvin, Diaz, Toth, & Schinke, 1995). Although research is scant, a few studies also have found that school teachers who espouse tolerant norms concerning marijuana use elevate youth risk for marijuana initiation (Petraitis et al., 1998).

Protective factors. In support of SDM and social control theory, numerous school-related protective factors have been found to decrease risk for soft drug initiation. Compared to soft drug initiates, most soft drug abstainers can be characterized as youth who: 1) are more attached and committed to school; 2) earn higher grades in school; 3) spend more time in extracurricular, school-related activities; and 4) devote more time to homework and studying (Catalano et al., 2004; Cernokovich & Giordano, 1992; Jenson, 2004). In support of SDM, research also has found that praise and prosocial support from teachers decreases risk for soft drug initiation among aggressive youth (O'Donnell et al., 1995).

Family Domain

Families, particularly parents, play an important role in shaping the behavior of their children. Research has shown that parents influence their children's drug behavior as role models, whose drug-related behaviors and norms are imitated, and as informal agents of social control, who set and reinforce behavioral standards and nurture the parent-child relationship (see Table 7).

Table 7.

Family Domain Risk Factors and Empirical Support

Theoretical Construct	Risk Factor	Extant Literature Reviews
Family social learning constructs:	Parental drug use; Parental prodrug norms	Conrad et al. (1992) Derzon & Lipsey (1999a) Derzon & Lipsey (1999c) Donovan (2004) Glynn (1981)
Family bonding and relationship constructs:	Attachment to deviant parents; Low prosocial family involvement; Poor adolescent-parent relationship; Family conflict; Poor supervision	

In support of social control theory and SDM, weak prosocial family bonding (in the form of weak attachment, commitment, and involvement) increases risk for soft drug initiation (Browning et al., 1999; Krohn et al., 1983). Poor family management practices, including inconsistent supervision and discipline (Dishion, Capaldi, Spracklen, & Li, 1995), family or parental conflict (Guo, Hill, Hawkins, Catalano, & Abbott, 2002), and offers from parents to smoke cigarettes (Conrad et al., 1992) also are predictive.

Parents also influence their children by serving as behavioral models, and by shaping and reinforcing norms, attitudes, and behavior. Although it is unclear whether nature or nurture is the predominant operating mechanism, research indicates that youth whose parents use soft drugs or engage in serious alcohol or other drug use are at an elevated risk for soft drug initiation, as compared to their adolescent counterparts (see, e.g., Biederman, Faraone, Monuteaux, & Feighner, 2000; Hill, Shen, Lowers, & Locke, 2002b). One study (Li, Pentz, & Chou, 2002a) found that compared to youth who reported that their parents did not use marijuana, adolescents who reported parental marijuana use were twice as likely to initiate all three soft drugs. Of the family-related risk factors, parental drug use and prodrug norms are the strongest precursors of soft drug initiation. In studies that control for age effects, parental prodrug norms typically is a moderately strong predictor of initiation, while parental drug use and the quality of the parent-child relationship both tend to exert smaller effects (Biddle, Bank, & Marlin, 1980; Glynn, 1981). Hence, parents predominantly influence youth soft drug initiation through the norms and behavioral standards they espouse and establish.

Protective factors. Numerous familial protective factors have been found to reduce the likelihood of that one or more soft drugs are initiated. Some of these major factors include positive reinforcement of prosocial behavior, communication of antidrug norms, strong bonding to prosocial parents, low parental conflict, and parental supervision and consistent discipline (see, e.g., Guo et al., 2002; Reifman et al., 1998; Vakalahi, 2001; Werner, 1994).

Peer Domain

Adolescence constitutes the only time in the lifespan that individuals interact so intensively and extensively with same-age peers (Berndt, 1979; Warr, 1993b). The empirical research is unequivocal about the risk effects of peer influence factors on soft drug initiation (Donovan, 2004). Peer influence primarily takes two forms: behavioral modeling and reinforcement of behavior via normative standards (Bauman & Ennett, 1996). In support of SDM and social learning theory, peers increase risk for soft drug initiation by modeling the use of soft drugs (which youth subsequently emulate) and by providing positive reinforcement for use (see Table 8). The quality of the peer-adolescent relationship (i.e., level and type of bonding) and peer-provided opportunities for behavioral involvement also appear important, although these lines of inquiry are less well-developed.

The most consistent and replicable finding in longitudinal and cross-sectional drug research is the strong relationship between adolescent alcohol, cigarette, and marijuana initiation and peer drug use, either perceived by adolescents or reported by peers themselves (Conrad et al., 1992; Derzon & Lipsey, 1999c; Donovan, 2004; Kandel, 1980b). In fact, after age and multiple explanatory factors are controlled, peer drug use remains one of the strongest predictors of soft drug initiation, with risk effects typically stronger than peer prodrug norms and parental influences (see, e.g., Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Donovan, 2004). Although peer prodrug norms constitute an important risk factor, the impact of peers on soft drug initiation primarily is exerted through the modeling of drug use (see, e.g., Duncan, Tildesley, Duncan, & Hops, 1995; Kandel, 1985; Petraitis et al., 1998).

Table 8.

Peer Domain Risk Factors and Empirical Support

Theoretical Construct	Risk Factor	Extant Literature Reviews
Peer bonding constructs:	Attachment to deviant/drug-using peers; Involvement with deviant/drug-using peers	Bauman & Ennett (1996) Conrad et al. (1992) Derzon & Lipsey (1999a) Donovan & Lipsey (1999c)
Peer social learning constructs:	Peer drug use; Peer prodrug norms	Donovan (2004) Glynn (1981)
Availability construct:	Peer-provided opportunities for soft drug use	

Protective factors. Most research on adolescent soft drug initiation has examined peer-related risk factors for initiation, not protective factors (Derzon & Lipsey, 1999a, 1999c; Donovan, 2004). Limited research does suggest, however, that attachment and involvement with peer drug abstainers reduces risk for soft drug initiation (Jenson, 2004; Johnson, 1986; Ried, Martinson, & Weaver, 1987).

Individual Domain

Comprehensive reviews have identified a constellation of risk factors for soft drug initiation that are found within adolescents themselves (see Table 9). Of the various risk factors identified in this domain, the strongest predictors include prodrug norms, positive expectations about drug use, intentions to use, prior initiation and use of other drugs, prior deviant behavior, and biological age.

Table 9.

Individual Domain Risk Factors and Empirical Support

Influences	Risk Factor	Extant Literature Reviews
Constitutional factors:	Impulsivity; sensation-seeking; alienation; rebelliousness; depression; low self-esteem	Conrad et al. (1992) Derzon & Lipsey (1999a) Derzon & Lipsey (1999c) Donovan (2004) Swadi (1999)
Norms and intentions:	Prodrug norms, intentions, expectations; Antisocial norms	
Behaviors:	Prior drug initiation; non-drug use problem behaviors; early initiation of drug use or delinquent behavior; low involvement in prosocial activities	
Demographic characteristics:	Gender; race; age	

Constitutional factors. Personality states predictive of initiation include low self-esteem, depression, anxiety, hopelessness, and stress (Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Kandel, 1980b). Concerning traits, adolescent initiates, compared to soft drug abstainers, have been found to be more rebellious, impulsive, and sociable; they are more apt to engage in risky behavior and be more sensation-oriented; and they are more likely to have feelings of alienation during childhood (see, e.g., Conrad et al., 1992; Donovan, 2004).

Norms and intentions. Three specific types of adolescent norms have been found to increase risk for soft drug initiation: drug-specific norms conducive to use of the corresponding drug, lax attitudes toward drug use and deviant behavior in general, and general antisocial beliefs or norms (Derzon & Lipsey, 1999a, 1999c). Drug-specific norms constitute one factor, in particular, that consistently distinguishes soft drug abstainers from initiates (see, e.g., Clayton, 1992; Derzon & Lipsey, 1999a, 1999c; Petraitis et al., 1998; Wills et al., 1996). One study (Epstein et al., 1995), in particular, found that adolescent marijuana abstainers who held tolerant views of marijuana use were almost four times more likely to subsequently initiate marijuana use, compared to marijuana abstainers who held negative views concerning marijuana use. Research also shows that any tolerance of drug use is associated with an increased risk of involvement with soft drugs (Conrad et al., 1992; Derzon & Lipsey, 1999a), although drug-specific norms tend to be stronger predictors of the initiation of corresponding drugs (Donnermeyer, 1992).

Other soft drug initiation. Alcohol, cigarette, and marijuana initiation have been treated as exogenous variables in examinations of their respective impacts on the initiation of other soft drugs. Results from both direct and indirect tests of stage theory consistently have found that for all three soft drugs, prior initiation of one is highly predictive of the initiation of another, even after controlling for multiple explanatory factors (see, e.g., Conrad et al., 1992; D'Amico & McCarthy, 2006; Derzon & Lipsey, 1999a; Duncan, Duncan, & Hops, 1998; Ellickson et al., 1992; Swadi, 1999). In fact, prior initiation of other soft drug use is among the strongest risk factors yet identified (Swadi, 1999). Interestingly, although risk for soft drug initiation covaries with age, studies controlling for age effects generally find that the significant association between the initiation of one soft drug and another remains (Yamaguchi & Kandel, 1984b). Two major findings and two related issues are of importance.

First, compared to alcohol initiation, prior initiation of cigarette use is a stronger predictor of marijuana initiation, a finding that supports many of the soft drug sequencing tests discussed earlier in this chapter. Findings from several studies are illustrative. After controlling for age effects and numerous other factors, Romer (2003) found that alcohol only initiates were 6.5 times more likely than alcohol abstainers to initiate marijuana use by Time 2, while cigarette only initiates were almost 11 times more likely to initiate marijuana use than cigarette abstainers. In examining 1999 and 2000 NSDUH data, Gfroerer et al. (2002) also found that prior initiation of alcohol use or cigarette use signaled an elevated risk for marijuana initiation. Binary logit results indicated that compared to cigarette abstainers, cigarette only initiates were 12 times more likely to initiate marijuana use. In contrast, alcohol only initiates were 5.6 times more likely than alcohol abstainers to initiate marijuana use. Finally, D'Amico and McCarthy (2006) found that the odds of marijuana initiation among cigarette only initiates was 117% greater than those for cigarette abstainers, while prior alcohol initiation was not predictive of marijuana initiation. Results from Derzon and Lipsey's (1999a) meta-analysis of 64 longitudinal studies on adolescent cigarette initiation also supports these basic findings.

Second, both interaction and mediating effects have been observed for the exogenous variables of prior alcohol and cigarette initiation. Compared to the risk effects associated with alcohol only initiation and cigarette only initiation, the conjoined risk for marijuana initiation attributed to alcohol and cigarette initiation demonstrates a multiplicative effect (Yu & Williford, 1992). After controlling for age effects, studies also have found that cigarette initiation reduces the relationship between alcohol initiation and marijuana initiation, although alcohol initiation remains a significant predictor (see, e.g., Andrews et al., 1991; D'Amico & McCarthy, 2006; Duncan et al., 1998; Ellickson et al., 1992).

Two other issues related to this line of research also are important. In support of stage theory and SDM, both early age of initiation and frequency of use constitute two major characteristics of soft drug behavior that increase risk for the initiation of other soft drugs, particularly marijuana use (see, e.g., Conrad et al., 1992; Donnermeyer, 1993; Golub & Johnson, 2001; Kandel & Logan, 1984). For instance, Yu and Williford's (1992) cross-sectional study of 3,000 16-24 year olds found that alcohol initiation during early adolescence and frequent use of alcohol increased risk for cigarette and marijuana initiation, although the risk effects were stronger for cigarette than marijuana initiation. As well, both prospective and retrospective research support Kandel's (2002) contention that the higher the frequency of drug use, the earlier the age of initiation and the greater the likelihood of initiating other soft drugs (Adler & Kandel, 1983; Killen et al., 1997).

Together, these findings underscore the need for prevention researchers to consider the age at which soft drug initiation occurs, as well as the frequency with which initiates use these drugs. Since frequency of use may explain, in part, the association between the initiation of one drug and that of another, the effects of this attribute should be held constant so that psychosocial differences between soft drug initiates and abstainers can be properly examined in predicting initiation (Clayton & Ritter, 1985; Kandel, 1982). It also appears worthwhile to assess the ages at which youth initiate soft drugs, so it can be determined if early age of initiation explains, in part, why some adolescents initiate all three soft drugs as opposed to just one or two.

Nondrug deviant behavior. A variety of antisocial and delinquent activities increase risk for soft drug initiation, including early and persistent aggressive and defiant behavior, cheating on schoolwork, shoplifting, and fighting (Brook & Brook, 1990; Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Donovan, 2004). Moreover, research supports SDM in finding that childhood conduct problems distinguish between those youth whose alcohol and marijuana use does and does not progress to abuse and continue into adulthood (Robins, 1980).

Protective factors. Major protective factors found within the individual domain include the exhibition of social and problem-solving skills, assertiveness, and a strong sense of self-efficacy, all of which support SDM (see, e.g., Fraser, 1996; Meschke & Patterson, 2003; Rutter, 1985). As well, youth who are involved in religious activities, functions, and services, also are more apt to abstain from soft drug use, a finding that supports both SDM and social control theory (Donovan, 2004; Meschke & Patterson, 2003; Vakalahi, 2001).

Demographic characteristics. Briefly discussed in Chapter 1, gender, race, and age constitute nonmalleable predictors of alcohol, cigarette, and marijuana initiation. Biological age has consistently been shown to be the strongest predictor of the three (Kandel, 2002). Males are more apt to initiate soft drug use than females, as are white youth (versus minorities) and middle adolescent youth (compared to early and late adolescents). Both the processes involved in bringing about risk for and protection against drug initiation, and the impact of risk and protective factors on drug initiation, are partly contingent on these characteristics. In fact, few risk and protective factors have been found to be universal in the sense that they apply equally across gender, race, and age lines. The following discussion directs attention to major gender and race differences in levels and types of risk factors. Age differences are discussed at the end of the chapter.

Although adolescent males and females differ on levels and types of some risk factors, there are surprisingly few gender differences. Some research suggests that male and female adolescents develop different expectancies of alcohol use, with males more apt to perceive positive consequences from use than females (Donovan, 2004). Low self-esteem also appears to be a stronger risk factor for female cigarette and alcohol initiation than male initiation of these drugs (Khoury, 1998). Compared to males, females also appear to be more vulnerable to parental than peer influences, while males are more apt to be impacted by peer drug behavior and norms (Biddle et al., 1980).

Systematic research on race differences in risk and protective factors is in its infancy. Similar to gender, however, evidence to date suggests that researchers should either examine race as a moderating variable, or it should be controlled for in etiological research (Conrad et al., 1992). Compared to black adolescents, white youth tend to be at an increased risk for alcohol and cigarette initiation. In contrast, black youth have been found to be at an increased risk for marijuana initiation, even after controls are in place (Hawkins et al., 2001). Peer drug use and prodrug norms are more powerful predictors of soft drug initiation, particularly alcohol, among white than minority youth. In contrast, black youth appear to be more influenced by parental drug behaviors and norms (Biddle et al., 1980). Compared to other racial groups, low self-esteem and delinquency are stronger risk factors for soft drug initiation among Hispanic youth, while depression is more strongly linked to white adolescent soft drug initiation (Vega, Zimmerman, Warheit, Apospori, & Gil, 1993).

Prioritizing Predictors

Due to differences in the magnitude of individual predictors, the five ecological domains of influence do not exert the same level of risk and protective effects on soft drug initiation. Research incorporating measures from all ecological domains of influence in predicting soft drug initiation (while controlling for age) have found that community and school-level risk and protective factors typically exert the weakest risk effects (Donnermeyer, 1992; Petraitis et al., 1998). For instance, one literature review found that the proportion of variation in soft drug initiation explained by school-related risk factors typically ranges between 2.5% and 3% (Dewey, 1999).

The peer and individual domain consistently exert the strongest effects, although the magnitude of their effects typically varies by biological age and drug type (Kandel & Andrews, 1987; Kandel, Kiros, Schaffran, & Hu, 2004). Risk effects attributed to family domain predictors typically are smaller in comparison to peer and individual domain effects (see, e.g., Conrad et al., 1992; Donovan, 2004; Glynn, 1981). In fact, Derzon and Lipsey's (1999a) meta-analysis of 17 predictors of cigarette initiation found that peer drug use ranked #2 in predictive power, while parental drug use ranked #11. In one longitudinal study (Pierce, Choi, Gilpin, Farkas, & Merritt, 1996) the risk effects for cigarette initiation attributed to peer smoking (60% risk effect) was more than two times the size of that attributed to parental smoking (25% risk effect).

Gaps in the Literature

Although progress has been made, there is much to be learned in the way of psychosocial factors and underlying processes that contribute to an increased risk for soft drug initiation. In fact, a broad review of the prediction literature revealed several glaring gaps that are worthy of redress, particularly if further advancements in prevention science are to be made. Outlined in Table 10, adolescent drug researchers have called for an increase in research directed at identifying: 1) predictors of soft drug initiation among rural youth; 2) predictors of initiation from all five ecological domains of influence; 3) determinants of age of soft drug initiation, particularly early age of initiation; 4) drug-specific risk factors for alcohol, cigarette, and marijuana initiation; and 5) the importance that community drug norms, the mass media, and school teachers have for influencing the soft drug initiation behavior of youth.

Table 10.

Major Calls for Future Research

Research Calls	Source
▪ Main effects of risk factors for initiation	Farrington/Coid (2003)
▪ More research on protective factors	Donovan (2004); Petraitis et al. (1998)
▪ Theoretically-derived hypotheses	Conrad et al. (1992); Donovan (2004); Petraitis et al. (1998)
▪ More analyses identifying mediating relationships	Donovan (2004); Petraitis et al. (1995)
▪ Predictors of initiation among rural adolescents	Botvin (1999); Burt et al. (2000); Donnermeyer (1992); Ellickson et al. (1992); Farrington/Coid (2003); Robertson et al. (1997); Spoth/Redmond (1996)
▪ Age-graded predictors of initiation from all five ecological domains, particularly parent and peer influences among early adolescents	Chassin (1984); Donovan (2004); Glynn (1981); Petraitis et al. (1998); Wills et al. (1996); USDHHS (2007)
▪ Drug-specific risk factors for initiation	Flay & Petraitis (1991); Kandel (2002)
▪ Predictors of age of initiation, particularly early age of initiation	Chassin (1984); Donovan (2004); Petraitis et al. (1998); Hansen et al. (1995); Hawkins et al. (2002b)
▪ Impact of community norms, mass media, parents, school teachers, non-parental adults	Allen et al. (2003); Donovan (2004); Petraitis et al. (1998); Wakefield et al. (2003); Wills et al. (1996)

In addition to these research calls, other promulgations have been made in terms of specific methodological issues or lines of inquiry that require further study. For example, Petraitis et al.'s (1998) comprehensive review brought to the forefront the need for researchers to take a more systematic approach to prediction in grounding research in theoretically-derived hypotheses. Calls for more research on the direct effects of risk and protective factors also have been made (Farrington & Coid, 2003), along with further research on mediating relationships (Donovan, 2004). The following discussion directs attention to some of the more obvious gaps in the literature, provides some insight into current research consensus, and highlights the types of findings that may be obtained by future studies designed to address these issues.

Age-Graded and Drug-Specific Predictors

Discussed in Chapter 3, the common factor and specific risk factor hypotheses have been put forth in an attempt to explain the hierarchical and temporal ordering of drug involvement to which Kandel (2002) initially directed attention. In advocating the specific risk factor hypothesis, Kandel attempted to explain this sequence (i.e., legal drugs>marijuana>hard drugs) by arguing that progression is located in unique risk factors that predict the initiation of different classes of drugs. Although she acknowledged that age effects and frequency of use may partly explain initial and progressive involvement, Kandel failed to articulate what unique risk factors to which progression in the sequence may be attributed.

In contrast, advocates of the common factor hypothesis argue that Kandel's (2002) sequence of drug involvement is an artifact of age-graded differences in the saliency of universal psychosocial and structural risk factors that predict the initiation of all drugs (Bachman et al., 1981; Jessor, 1992; Jessor

& Jessor, 1978; Kaplan et al., 1982). Legal drugs typically are initiated first because they are more widely available and youth have more opportunities for involvement. In contrast, marijuana is less available than alcohol or cigarettes, although it is more available than other illegal, harder drugs. Hence, common factor proponents maintain that the same risk factors that predispose youth to initiate legal drug use also prompt marijuana initiation (Jessor & Jessor, 1978; Kaplan et al., 1982). Adolescents who progress to marijuana use simply exhibit (or have been exposed to) higher levels of risk factors than youth who stop at legal drug use and do not initiate marijuana (or hard drug use). In addition, while normative initiation is explained in terms of environmental-age interactions, common factor proponents argue that early and more frequent involvement in drug use tends to constitute the manifestation of one or more stable, latent individual attributes (Jessor, 1992).

From an empirical standpoint, support for the prediction component of stage theory may be found in findings that attribute the initiation of different drugs to unique (or drug-specific) risk factors (Kandel, 2002). In contrast, support for the common factor hypothesis lies in research that demonstrates the universality of risk factors for the initiation of all three soft drugs (Jessor, 1992). On balance, not much research has explicitly tested either hypothesis. Although full tests require longitudinal data (where progression in drug involvement may be examined as youth mature), cross-sectional research can provide partial assessments of the validity of these hypotheses. For instance, cross-sectional researchers can compare risk factors for soft drug initiation by drug type, as well as within and across stages of adolescence, in examining whether predictors tend to differ in kind or magnitude.

Very few cross-sectional studies have simultaneously compared risk factors by drug type and biological age. In fact, more often than not, either composite measures of soft drug initiation have been used (Chassin, 1984); results have not been compared by drug type when examining the age-varying nature of predictors (Allen et al., 2003; Glynn, 1981); or comparisons have made across soft drugs, but age effects have been controlled (see Allen et al., 2003; Glynn, 1981; Roosa, 2000; Vitaro, Wanner, Brendgen, Gosselin, & Gendreau, 2004). In an effort to provide some insight into the findings that future cross-sectional research may expect to obtain when examining predictors of soft drug initiation by drug type and age, what follows is a brief synopsis of what is currently known about age-graded and drug-specific predictors of soft drug initiation.

Age-graded nature of predictors. It is not surprising that biological age is one of the strongest predictors of initiation (Barnes & Welte, 1986). As youth mature, and the environment in which they interact expands, the impact that factors have for protecting or placing them at risk for soft drug initiation also changes (Schulenberg & Maggs, 2001). Since risk and protective factors exert different effects on initiation at different points in adolescence, prevention researchers who do not examine the interactive impact of age on relationships run the risk of committing Type I or II errors for different age groups contained within total samples (Huba & Bentler, 1980).

Research results concerning the age-graded nature of risk and protective factors is complex. In general, however, research has found that many of the major risk factors discussed earlier change in saliency as youth mature. For example, evidence suggests that community, individual, and school domain risk factors are age-graded. In providing indirect support for Hawkins' (Catalano & Hawkins, 1996) contention that community-level risk factors are more important for older than younger youth, Werner (1994) found that sources of social support found in the community (e.g., neighbors and clergy) decreased the risk for soft and hard drug initiation among older adolescent abstainers, while this type of protection had relatively little impact on the soft drug initiation behavior among early and mid-adolescents. Individual domain risk factors also appear to be more predictive of early age (i.e., prior to 15 years) of alcohol and marijuana initiation than the initiation of these drugs during mid- or late adolescence (Kandel & Andrews, 1987).

On the school front, low commitment to education not only is a stronger determinant of soft drug initiation among 7th and 8th grade abstainers than 9th and 10th soft drug abstainers (Donnermeyer, 1992), but poor school performance appears to be a marginally significant predictor of soft drug initiation among 12th grade soft drug abstainers (Halfors, Vevea, Iritani, Cho, Khatapoush, & Saxe, 2002). Truancy also has been shown to be a more salient risk factor for soft drug initiation among early adolescents than mid- or late adolescents (Dewey, 1999).

The most sweeping age-graded changes in the saliency of risk factors center on the effects of parent and peer modeling and normative standards. In general, peer influence increases in magnitude as youth navigate through adolescence (Allen et al., 2003; Glynn, 1981). By the time adolescents reach mid-adolescence, peers play a more important role than parents in shaping drug use norms and behavior (Beal, Ausiello, & Perrin, 2001; Donovan, 2004; Li, Barrera, Hops, & Fisher, 2002). In fact, the literature is clear in identifying mid-adolescence as the time period in which the impact of peers on soft drug initiation is stronger than the impact of parents, particularly in terms of peer drug use (see, e.g., Donovan, 2004; Urberg, Cheng, & Shyu, 1991). Sometime after 10^{th} grade, however, peer influence declines in importance (Berndt, 1979; Sutherland & Shepard, 2001). Although not articulated by SDM, available evidence suggests that peer drug use remains a stronger predictor of soft drug initiation during late adolescence than parental drug use or prodrug norms (Allen et al., 2003; Margulies et al., 1977).

In contrast to peer influence, the influence of parents on soft drug initiation takes on a curvilinear pattern when plotted across adolescence. The impact of parental influence on soft drug initiation is strongest during early adolescence (Biddle et al., 1980), weakest during mid-adolescence, and possibly as strong during late as opposed to early adolescence (Huba & Bentler, 1980; Kandel & Wu, 1995).

Drug-specific nature of predictors. On balance, the bulk of the extant research has found that predictors of soft drug initiation do not differ in kind by drug type, as Kandel (2002) proposes. With few exceptions, most of the major risk factors discussed earlier have been shown to universally predict the initiation of all three soft drugs. Differences lie mainly in the magnitude of effects, findings that are reinforced by those observed for the age-graded nature of predictors.

Unique risk factors that have been documented primarily center on the drug-specific norms, intentions, and perceptions of drug availability held by adolescents themselves, as well as the drug-specific norms espoused by parents and peers. For example, adolescents' pro-marijuana norms have been found to predict marijuana initiation, but not alcohol or cigarette initiation (Hawkins et al., 2002b; Kandel et al., 1978; Kosterman et al., 2000). Several studies have found that antisocial behavior is significantly associated with alcohol and marijuana initiation, but not cigarette initiation (Dishion et al., 1999; Kandel et al., 1978).

Again, although these studies have found that some risk factors may be drug-specific, the bulk of extant research supports the common factor model, with the strength of predictors varying by drug type (and age). For instance, peer drug use and prodrug norms have been found to be more predictive of marijuana initiation than alcohol initiation or cigarette initiation (Allen et al., 2003; Kandel, 1982; Swadi, 1999). In contrast, parents are more influential in shaping adolescent attitudes and behaviors related to legal drugs (particularly alcohol) than marijuana (Allen et al., 2003; Glynn, 1981). Two other major findings from Derzon and Lipsey's (1999a, 1999c) meta-analyses on predictors of marijuana and cigarette initiation are illustrative in this regard.

First, in their meta-analysis of 67 longitudinal and cross-sectional studies on predictors of marijuana initiation, Derzon and Lipsey (1999c) found that peer and family domain factors were less predictive of marijuana initiation than individual domain risk factors, including prior alcohol use, illegal drug use, cigarette use, and prodrug norms and intentions. For example, while the average effect size for peer drug use, parental drug use, and antisocial parents and peers each equated to roughly .25, the average effect sizes for prior alcohol use and prodrug attitudes and intentions were.35 and .30, respectively. Longitudinal results from other research (Hawkins et al., 2002b) also support these basic findings. Although Derzon and Lipsey (1999c) did not examine whether these results held for age-graded subsamples of youth, their results do suggest that when future research controls for age effects, individual domain predictors should explain the largest proportion of variance (on average) in marijuana initiation than that attributed to other ecological domains.

Second, Derzon and Lipsey (1999a) came to a slightly different conclusion in their meta-analysis of 64 cross-sectional and longitudinal studies on predictors of cigarette initiation. In comparing 1,261 effect sizes for a variety of risk factors from all five ecological domains, they found that peer drug use (.40) was the strongest predictor of adolescent cigarette initiation, followed by prior alcohol use (.35). Adolescent norms conducive to cigarette use and intentions to use cigarettes also exerted slightly greater effects (on average) than almost all family domain predictors. Hence, future research can expect to find that the peer domain exerts stronger effects on cigarette initiation than other domains of influence.

Predicting Age of Initiation

Research consistently underscores the prognostic value that age of drug initiation has for distinguishing adolescents who are at low and high risk for regular and patterned drug use (see, e.g., Adler & Kandel, 1983; Dawson, 2000), as well as drug abuse and dependence (see, e.g., Grant & Dawson, 1997; Hingson et al., 2006a; Robins & Przybeck, 1990). The age at which researchers deem initiation as “early” varies in the literature, and tends to differ depending on the type of drug being discussed. Nonetheless, early age of soft drug initiation typically is viewed as that occurring prior to 15 years of age (Robins, 1980), while early initiation of hard drug use generally is viewed as that which occurs prior to 16 years of age (Dishion et al., 1995).

Given the negative consequences associated with early involvement in soft drug use, it is surprising that little research has examined the timing of soft drug initiation in terms of risk factors that predict initiation at earlier versus later ages (Donovan, 2004; Swadi, 1999; Wills, Resko, Ainette, & Mendoza, 2004). An explicit examination of predictors of early soft drug initiation appears warranted, particularly since available research suggests that risk factors for initiation during childhood and early adolescence differ in salience from those that predict soft drug initiation during later periods of adolescent development.

In general, limited research suggests that adolescents who initiate soft drug use during mid- or late adolescence traverse different developmental trajectories of drug involvement than youth who initiate earlier (Gruber, DiClemente, Anderson, & Lodico, 1996; Loeber, 1988, 1990). Although many of the environmental and interpersonal risk factors that predict initiation during mid- and late adolescence also predict initiation during early adolescence, early soft drug initiates have been found to have been exposed to (or exhibit) more extreme levels of risk factors and lower levels of protection during childhood and early adolescence, particularly constitutional factors (Wills et al., 2004). For example, earlier ages of alcohol, cigarette, and marijuana initiation have been linked to higher levels of negative emotional states and traits during childhood and early adolescence, including persistent conduct disorder, rebelliousness, and difficult temperament (Conrad et al., 1992; Collins et al., 1987; Donovan, 2004; Leukefeld et al., 1998; Swadi, 1999). Some researchers also have found that levels of these personality attributes do not change after initiation has occurred (Wills et al., 2004; Zucker, 1994). On the other hand, late adolescent initiates have been found to have lower levels of risk and higher levels of protection than early and mid-adolescent initiates, with increases in risk observed either during mid-adolescence or contemporaneously during late adolescence.

Concerning risk factors found within the peer, family, and school domains, earlier ages of alcohol initiation have been shown in longitudinal research to be differentially associated with high initial levels of parental drug use, parental prodrug norms, and family conflict; low initial levels of attachment to parents, parental monitoring, and school attachment and commitment; frequent truancy and poor school grades during elementary school; and close and early interaction with drug-using peers (see, e.g., Bailey & Hubbard, 1990;

Cernokovich & Giordano, 1992; Dielman, Butchart, Shope, & Miller, 1991; Dishion & Loeber, 1985; Donovan, 2004; Dishion et al., 1995; Swadi, 1999).

In sum, much more research is needed in this area, including studies that examine risk factors for initiation during early and late adolescence (Chassin, 1984). There also is a paucity of research on age-graded differences in protective factors that serve to delay age of initiation (Donovan, 2004). Moreover, since limited knowledge concerning the drug-specific nature of predictors of age of initiation makes it difficult to draw any tentative conclusions along these lines, future research should address this gap from a more exploratory standpoint, at least until findings can facilitate research expectations.

Mediating Processes

As discussed in Chapter 2, Petraitis et al. (1995) hypothesized that proximal influences tend to mediate the impact of distal influences on adolescent outcomes, including soft drug initiation and use. Although advances have been made in recent years, on balance, much more research has examined the direct effects of risk and protective factors than has investigated the mediating role that risk and protective factors have for conditioning relationships between predictors and soft drug initiation (Petraitis et al., 1998). Even less research has determined if (and how) mediating processes vary by biological age and drug type (Dishion et al., 1999; Wills et al., 1996). Of the research that has been conducted, two distinct lines provide support for Petraitis et al.'s (1995) contention: comparisons of the differential saliency of ecological domains (and associated predictors) and systematic investigations of mediating processes. The former line was discussed earlier.

Direct support for distal-proximal mediation comes from explicit assessments of the mediating capacity of proximal variables. Of the research that has been conducted, there is some convergence concerning the mediating capacity of several proximal risk factors: adolescents' prodrug norms and intentions, peer drug modeling, and peer prodrug reinforcement. In support of SDM, adolescent prodrug norms and intentions have been found to partially mediate the relationship between soft drug initiation and numerous distal risk factors, including poor school performance, low school commitment, parental attachment, the quality of the parent-child relationship, peer relationship variables, and the drug use behaviors of parents, peers, and other adults (see, e.g., Bauman & Ennett, 1996; Biddle et al., 1980; Conrad et al., 1992; Derzon & Lipsey, 1999c; Dewey, 1999).

Drug-using peers also may be a partial mediator, capable of reducing the relationship between family risk factors and soft drug initiation. For instance, research supports SDM in finding that parents influence their children's friendships by having a direct input on the peer selection process (Donovan, 2004; Engels & Bot, 2006; Mounts, 2004), and by shaping the peer selection process through their parenting and discipline practices and standards, as well as family norms (Ary, Duncan, Duncan, & Hops, 1999; Barrera, Biglan, Ary, & Li, 2001). For instance, poor family management and parental prodrug norms have been shown to lead to drug-using peers, with peer effects on soft drug initiation either exerted directly or indirectly through adolescents' drug norms and intentions (Dishion et al., 1991).

In addition to the need for more assessments of distal-proximal mediation, much more research needs to consider the drug- and age-varying effects of these processes. This underdeveloped area has precluded comprehensive reviews of the literature from coming to tentative conclusions about these issues (Donovan, 2004). This line of inquiry may be fruitful, particularly since a limited number of studies have found that intrapersonal factors fully mediate the relationships between alcohol initiation and low family management, parental drug use, and peer factors, while only partial mediation has been observed for marijuana initiation, and no mediation processes have been found for cigarette initiation (Dishion et al., 1999).

Conclusion

An indepth examination of 30 empirical tests of Kandel's (2002) drug sequencing hypothesis brought three major issues to the forefront. First, Kandel's drug sequence has considerable empirical support, with few exceptions. Progression from the initiation of legal drug use to marijuana use has been empirically confirmed across age, race, and geographic lines; at different times in history (1970s-2000); with the use of both prospective and retrospective data; and with different analytic techniques. Soft drug initiation appears to follow a well defined order, although not all adolescents who experience a particular stage go on to initiate a drug in a stage later in the sequence.

Second, empirical ambiguity primarily lies in the sequencing of alcohol and cigarette initiation. While additional confirmation is needed, evidence from four prospective studies suggests that alcohol initiation occurs prior to cigarette initiation for most adolescents who initiate legal drug use, while the initiation of these two soft drugs occurs prior to marijuana initiation among those adolescents who initiate marijuana use.

Third, although a few longitudinal studies ensured the temporal ordering of polydrug initiation, the bulk of the extant research did not. Guttman scalogram analysis, the most common analytic technique used to test Kandel's (2002) sequencing hypothesis, only infers temporal ordering in drug initiation sequences. Since the element of time clearly is evident in Kandel's hypothesis, a proper test of her proposition requires that some type of time data be incorporated into analyses.

Other knowledge generated by theory and empirical investigations examining predictors of adolescent soft drug initiation have led to significant advancements in understanding the etiology of this problem behavior, and how it may be prevented or delayed. Virtually all aspects of social life can have an impact on the soft drug initiation behavior of youth; however, influence is disproportionately located in the characteristics of peers, parents, and adolescents themselves. Five basic issues were brought the forefront concerning predictors of soft drug initiation among youth of varying ages.

First, the bulk of longitudinal research (supported by cross-sectional studies) has distinguished soft drug abstainers and initiates on the following grounds. Prior to initiation, soft drug users typically: 1) are exposed to drug behavioral models and individuals who hold tolerant views about drug use (particularly peers); 2) are not committed to or involved in school to any large degree; 3) live in areas where soft drugs are widely available, or they perceive that soft drugs are easy to obtain; 4) hold positive views about soft drug use; and 5) have initiated at least one legal drug. Hence, the most salient individual-level risk factors for soft drug initiation include prior initiation of one or more other drugs and the perception that drug use is an acceptable behavior and drugs are easily available. At the interpersonal level, peer and parental drug use, prodrug norms, and low school commitment and involvement are the strongest risk factors.

Second, also in support of SDM is the consistent finding that agents of social control and learning primarily impact soft drug initiation through three socialization processes: 1) bonding, in the form of attachment and commitment; 2) behavioral modeling, whereby adolescents emulate the drug-using behaviors they observe in their environment; and 3) definitions or beliefs, whereby adolescents internalize prodrug norms, beliefs, expectations, and attitudes that others exhibit. Of these three processes of socialization, however, behavioral modeling and normative standards are the most important.

Third, of all agents of socialization, parents and peers exert the strongest influence on soft drug initiation, although peers have consistently been shown to exert the strongest effects of the two. Parents and peers tend to impact adolescent soft drug initiation in different ways, however. Whereas parental influence primarily is attributed to the normative standards they establish (Ennett et al., 2001; Kosterman, Hawkins, Guo, Catalano, & Abbott, 2000), peer influence primarily stems from behavioral modeling. With the strong and consistent effects attributed to peer drug use, soft drug initiation among youth (particularly youth during mid-adolescence) largely reflects peer example.

Fourth, as an exogenous variable, the initiation of one or more soft drugs exerts strong risk effects on the likelihood of initiating another soft drug. Research examining progression in adolescent involvement in soft drug use typically finds that cigarette initiation (as opposed to alcohol initiation) generally is found to be a stronger predictor of marijuana initiation. Although this finding mirrors and reinforces the empirical support for Kandel's (2002) drug sequencing hypothesis, it does not explain why the temporal ordering in soft drug initiation (i.e., alcohol>cigarettes>marijuana initiation) exists. Nonetheless, it is reasonable to expect that future research will uncover similar findings.

Fifth, ecological domains of influence do not exert the same level of risk and protective effects on soft drug initiation. In research controlling for age effects, community and school-level risk factors typically exert the weakest effects, while the peer and individual domains exert the strongest. Family domain predictors tend to fall in the middle.

Gaps in the Literature

Although advancements have been made, there are several glaring gaps in the literature that are worthy of redress. Major research calls center on the following issues: 1) determinants of soft drug initiation among rural youth; 2) multivariate research that incorporates constructs from all ecological domains; 3) predictors of age of initiation, particularly early age of initiation; 4) drug-specific risk factors; and 5) community-level influences. In addition, other researchers have stressed the need for theoretically-derived hypotheses and more research on the direct and mediating effects of risk and protective factors.

The most glaring and easily addressable gaps in the literature involve age-graded and drug-specific risk factors for soft drug initiation, determinants of time to initiation, and mediating relationships. Future research designed to address these areas can be based on the following hypotheses. First, since predictors of initiation have been found to change in saliency as youth mature, and they tend to universally predict the initiation of all three soft drugs, future research can seek to verify these findings.

Second, there is some evidence that community-level predictors may be more important for older versus younger youth; higher levels of individual domain risk factors appear more predictive of early versus later ages of initiation; and peer domain risk factors are stronger predictors of initiation during mid-adolescence than early or late adolescence. Moreover, future research can confirm whether parents play a larger role in the soft drug behavior of early adolescents, as compared to older youth. As well, research examining soft drug initiation while holding age effects constant can further assess whether individual domain predictors explain a larger proportion of variance in marijuana initiation than legal drug initiation.

Third, research also can consider if the same predictors that determine later ages of initiation also predict earlier ages. Some research suggests the risk effects for early age of initiation will be stronger in magnitude. Fourth, and finally, future research on mediating processes can establish better evidence for Petraitis et al.'s (1995) distal-proximal mediation hypothesis. Available evidence suggests that peer and individual domain predictors constitute full or partial mediators that condition the relationships between community, school, and family risk factors on soft drug initiation.

With the theoretical and empirical framework for the current study in place, the following two chapters turn to the methodologies that found the research. The next chapter deals with the systematic review of the extant literature, while Chapter 6 focuses on the quantitative component of the current study.

CHAPTER 5

SYSTEMATIC REVIEW METHODS

The current study constituted a comprehensive examination of adolescent soft drug initiation. The overarching goal of the research was to reaffirm and build upon extant research findings and address some empirical ambiguities found in the literature, thereby providing several contributions to the prevention science knowledge base. A four-fold purpose founded this endeavor: (1) provide a systematic assessment of the soft drug initiation literature; (2) test Kandel's drug sequencing hypothesis; (3) determine if predictors of soft drug initiation differ in kind or saliency by stage of adolescent development and drug type; and (4) examine the timing of soft drug initiation in terms of predictors that distinguish early versus later ages of initiation. In an effort to assess the validity of the quantitative findings, a dual cross-validation scheme was employed.

The current chapter explicates the methodology that founded the first major component of the current research. In short, a systematic, comprehensive review of the extant literature on adolescent soft drug initiation was conducted in an effort to address several gaps in this type of literature. Findings served to: 1) generally organize and update this large body of literature, and 2) provide a means by which select findings from the quantitative component of the current research could be cross-validated.

To address each of these areas, this chapter first discusses the nature of comprehensive literature reviews, including the types of techniques employed in reviewing a body of literature and a comparison of the strengths and weaknesses of various strategies. Gaps in past reviews of the adolescent soft drug initiation literature then are discussed in an effort to provide some context and purpose for the current comprehensive review. Next, the methodology underlying the assessment is explained, along with the analysis plan. In the end, major threats to four types of validity are described, along with the systematic review cross-validation.

Nature of Comprehensive Literature Reviews

A comprehensive review of a body of literature serves as a mechanism by which knowledge in a particular field of study may be advanced. In general, reviewing a body of literature necessitates synthesizing existing knowledge about a particular topic, identifying gaps in the literature, and suggesting lines of future empirical inquiry. The following discussion turns attention to two interrelated issues. First, readers are oriented to the major types of comprehensive reviews typically conducted within the social sciences; the associated methodology typically employed by each; and respective advantages and limitations. Second, this foundational information provides some context for the results of an assessment conducted on those extant literature reviews utilized in Chapter 4 (when predictors of adolescent soft drug initiation were identified and discussed). The purpose of this evaluation was to identify weaknesses in methodology, content, and focus, and to reveal gaps in this body of literature worthy of resolve.

Types of Literature Reviews

Two major types of literature reviews are found in virtually all disciplines of study: a qualitative (narrative) literature review and a quantitative meta-analysis.

The discursive, qualitative literature review constitutes the traditional method of synthesizing a body of literature (Cooper & Rosenthal, 1980). In short, this type of review involves identifying patterns of relationships across studies in a narrative format.

In contrast to a qualitative literature review, a statistical meta-analysis is essentially a statistical summary of a body of research (Welsh & Farrington, 2006a). Specifically, when a statistical meta-analysis is conducted on a body of risk and protective factor research, estimates of the predictive power of risk and protective factors for a criterion (typically in the form of Pearson *r* correlations) are culled from individual studies and translated into effect sizes so that findings may be compared across studies. These effect sizes then are used in calculating an average effect size that takes into account the sample size of individual studies. Studies with larger samples sizes are given greater weights in calculating the average effect size than studies using smaller sample sizes (Preiss & Allen, 1995).

Strengths and Limitations

A qualitative review generally is less rigorous and methodologically objective than the quantitative meta-analysis. Although a large number of studies may be included in a qualitative review, and details concerning the attributes of primary studies are able to be discussed, researcher bias is a plausible threat to the validity of findings. Researcher bias typically begins during the literature collection process, where most searches are limited to published studies. Excluding relevant studies lends to an increased likelihood of making inaccurate conclusions concerning the predictive utility of risk and protective factors for adolescent drug use.

Conducting a statistical meta-analysis can be advantageous in terms of the ability to summarize results from a large number of studies. Moreover, this type of review, if conducted and presented correctly, also can be quite transparent, which aids in the degree to which it is replicable (Cook & Leviton, 1980; Light & Pillemer, 1982).There are a three notable drawbacks, though, associated with conducting a meta-analytic review.

First, a meta-analysis is unable to summarize complex relationships, particularly those in which mediating or moderating processes are found to operate (Wilson, 2001). Second, conducting a statistical meta-analysis is very time-consuming and can be costly. Finally, calculating an average effect size when outcomes in individual studies are measured differently can produce invalid results. For example, drug research within the field of prevention science tends to be plagued by multiple and divergent measures of the criterion (Kandel, 1975a).

A qualitative review typically is larger in scope than a statistical meta-analysis, in terms of the number of predictors that can be examined. As opposed to a qualitative review, a meta-analysis is less well-suited for summarizing relationships between criterion measures and many predictors. In fact, those examining more than 15 predictors have been noted as relatively rare in the social sciences (Preiss & Allen, 1995). Hence, an advantage of conducting a qualitative assessment of a relatively large body of literature, relative to employing statistical meta-analytic techniques, is the large number of predictors (and studies) that may be assessed. Qualitative reviews and quantitative meta-analyses are similar in their respective trade-offs, however. Both are able to generate aggregate-level information, although individual differences between studies may be masked when a large volume of research is synthesized.

Reinventing the Traditional Narrative

Over the past 20 years, researchers have begun to employ a systematic methodology when conducting comprehensive reviews, a method that typically was reserved for meta-analytic reviews. In contrast to the traditional, qualitative literature review, the systematic review (when conducted properly) provides for a more comprehensive and objective review of a body of literature. Researcher bias may be tempered when this systematic methodology is employed, thereby enhancing the validity of findings. Moreover, the opportunity for replicability increases because the researcher strives to employ a transparent study design.

As with the meta-analytic review, the systematic review method uses a rigorous and standardized method of identifying, appraising, and synthesizing a body of literature. When done correctly, systematic reviews "provide the most reliable and comprehensive statement about what works" (Petrosino, Boruch, Soyden, Duggan, & Sanchez-Meca, 2001, p. 20). While both meta-analytic and systematic reviews focus on the results of individual studies, the latter method pays particular attention to the methodology underlying these studies.

According to Welsh and Farrington (2006a), five major characteristics of the systematic review distinguish it from the basic narrative method. First, systematic reviews have explicit objectives that are made clear to the reader.

The underlying rationale for the review is discussed, as are the research questions used in framing the review and the methodology that is employed.

Second, all relevant studies, regardless of publication status, are obtained. Locating all studies on a particular topic aids in reducing researcher bias. Third, eligibility criteria for including or excluding studies are made explicit. The final report typically includes a listing of all studies excluded from the review, along with justifications for exclusion.

Fourth, the methods employed in identifying and assessing the body of studies included in the review typically are made known to readers. Including an indepth discussion of the methods employed aids the reader in determining the extent to which researcher bias exists, and how this bias may have compromised results. A listing of computer search engines and bibliographic databases are discussed so that gaps in literature searches may be identified (Welsh & Farrington, 2006a).

Fifth, standardized techniques typically are utilized in the organizational and analytical aspects of the project. Two major techniques include matrix-building and vote-counting. Matrix-building typically is developed and used as an organizational framework, a means by which findings from studies may be organized during the analytic and presentation phases of the research. Developing and using matrices enables findings from a large number of studies to be visually displayed, and common or inconsistent findings to be more easily identified (Petraitis et al., 1998).

The second technique, vote-counting, is utilized in the analytical phase of the research, and involves evaluating the degree to which a given research hypothesis is supported. This typically is done by summing the number of individual studies with statistically significant findings that favor the particular research hypothesis under consideration, and comparing this number to the total number of studies that support the null hypothesis (Wilson, 2001, p. 73).

There are two notable drawbacks associated with the vote-count method. First, equal consideration is given to studies of varying degrees of methodological quality. With few exceptions (e.g., see scientific methods scale, Sherman, Gottfredson, MacKenzie, Eck, Reuter, & Bushway, 1997), most systematic reviews using the vote-count method do not assess primary studies in terms of the degree to which various threats to internal, external, statistical conclusion, and construct validity have been ruled out.

The second drawback of the vote-count method is that equal consideration is given to studies with different sample and effect sizes. Since effect sizes are partly contingent on the size of the sample, a relationship in one study may meet the significance threshold simply because the sample utilized was relatively large. In this instance, the effect size may actually be small. The reverse also may be true. In essence, when the vote-count method is employed, often it is not known whether those relationships identified as meeting a significance threshold are significant due to the size of the sample employed or the actual size of the effect. It is important to note, however, that vote-counting is a commonly used method of summarizing results, particularly by reviewers who place more of an emphasis on describing or updating a body of research than drawing inferences (Light & Smith, 1971).

A keystone characteristic of the systematic review is the structure and detail of the final report. These types of reviews provide the reader with a clear understanding of each stage of the research process, how and why decisions were made, and how and why particular conclusions were drawn (Farrington & Welsh, 2001; Welsh & Farrington, 2006b).

In sum, the qualitative narrative and quantitative meta-analysis constitute the two major types of literature reviews conducted within the social sciences. Qualitative reviews have been recast to be more reputable in recent years, with the emergence and use of more objective and systematic methodologies. In terms of standardization and objectivity, systematic reviews are more comparable to quantitative meta-analyses than the traditional, qualitative narrative. Systematic, comprehensive reviews are relatively new to the social science field and researcher bias still is an issue, but they are undoubtedly an improvement over the traditional narrative, which often is difficult to replicate and is often wrought with biases and questions concerning comprehensiveness. Hence, the preferred method of synthesizing a body of research is to employ a systematic methodology.

Quality and Focus of Extant Reviews

Keeping in mind the foundational information just presented, the following discussion outlines the major results stemming from an assessment conducted on those literature reviews that were utilized in Chapter 4 when predictors of adolescent soft drug initiation were identified and discussed. The purpose of conducting this assessment was to examine the content, focus, methodological rigor, and general quality of these reviews in an effort to identify gaps in this type of literature that are worthy of further investigation.

A total of 29 literature reviews were assessed. Table 11 lists each of these reviews, with relevant methodological and major study characteristics noted (e.g., systematic, focus on soft drug initiation, drug-specific predictors examined). These literature reviews were obtained through electronic searches of major social science databases, with delimits set for 1970-2006. Reviews subject for inclusion in this evaluation included those that synthesized research on adolescent soft drug use. Reviews that explicitly focused on predictors of adolescent drug dependence, hard drug use, or drug abuse were excluded. An effort was made to try and locate as many relevant comprehensive reviews as possible.

Taking into consideration as much of the methodological issues that should be attended to when conducting a systematic, comprehensive review of a body of literature, and the fact that the current research centers on examining predictors of adolescent alcohol, cigarette, and marijuana initiation, these 29 literature reviews were assessed on the following grounds: (1) degree of methodological rigor; (2) relevance of focus and content for adolescent soft drug initiation; and (3) degree of comprehensiveness in terms of examining predictors of initiation from the five ecological domains of influence.

Table 11.

Drawbacks of Extant Comprehensive Literature Reviews

Author/year	Systematic	Focus on soft drug initiation [a]	Ecological domains assessed [b]	Predictors as drug-specific	Predictors as age-graded	Drug initiation/use as predictor
Allen et al. (2003)	X		P, F	X		
Bauman & Ennett (1996)		Alc, Cig, Mar	P			
Charlesworth & Glantz (2005)	X	Cig	C			
Chassin (1984)			I, P, F	X	X	
Clayton et al. (1995)			I, P, F			
Conrad et al. (1992)	X	Cig	I, P, F, S, C		X	X
Derzon & Lipsey (1999a)	X	Cig	I, P, F, S			X
Derzon & Lipsey (1999c)	X	Mar	I, P, F, S			
Dewey (1999)	X		S			
Dielman et al. (1991)			P, F, S			
DiFranza et al. (2006)	X	Cig	C			

(Table 11 continues)

(Table 11 continued)

Author/year	Systematic	Focus on soft drug initiation [a]	Ecological domains assessed [b]	Predictors as drug-specific	Predictors as age-graded	Drug initiation/use as predictor
Donnermeyer (1992)	X		I, P, F, S, C			
Donovan (2004)	X	Alc	I, P, F		X	
Glynn (1981)		Alc, Mar	P, F	X		
Gorsuch & Butler (1976)			I, P, F			
Halebsky (1987)			P, F	X		
Halfors et al. (2002)	X		S		X	
Hastings et al. (2005)			C			
Hawkins et al. (1992a)			I, P, F, S, C		X	X
Hawkins et al. (1990)			I, P, F, S			
Kandel (1980b)			I, P, F	X		X
Leukefeld et al. (1998)			I, P, F, S, C			
Meschke & Peterson (2003)			I, P, F, C			
Penning & Barnes (1982)			I, P, F, S			X

(Table 11 continues)

(Table 11 continued)

Author/year	Systematic	Focus on soft drug initiation [a]	Ecological domains assessed [b]	Predictors as drug-specific	Predictors as age-graded	Drug initiation/use as predictor
Petraitis et al. (1998)	X		I, P, F, S, C			
Randolph (2004)					X	X
Swadi (1999)			I, P, F	X		
Vakalahi (2001)			F			
Wakefield et al. (2003)		Cig	C			

[a] Alc = Alcohol Initiation; Cig = Cigarette Initiation; Mar = Marijuana Initiation.
[b] I = Individual Domain; P = Peer Domain; F = Family Domain; S = School Domain; C = Community Domain.

Methodological Rigor

Of the 29 reviews assessed, less than half (n = 11) employed methodologies that may be characterized as systematic in nature (Allen et al., 2003; Charlesworth & Glantz, 2005; Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Dewey, 1999; DiFranza et al., 2006; Donnermeyer, 1992; Donovan, 2004; Halfors et al., 2002; Petraitis et al., 1998). Specifically, these 11 reviews took an objective and replicable approach to the collection of primary empirical studies, discussing the underlying methodology employed, sources referred to in collecting studies, and inclusion (or exclusion) criteria utilized in producing a candidate pool of studies subject to assessment. These reviews also were founded on a set of research questions and analytic strategies were discussed. Three of these reviews (Allen et al., 2003; Derzon & Lipsey, 1999a, 1999c) used meta-analytic methods to arrive at the results. Only one review employed vote-counting and matrix-building methods in analyzing and presenting the findings (Petraitis et al., 1998).

Aside from these 11 reviews, the remainder were traditional, narrative reviews, and they provided no indication as to how primary studies were obtained. Moreover, no specific research questions were posed and answered. Aside from simply discussing the literature, these reviews do not appear to have served any expressed purpose in advancing knowledge concerning adolescent drug use. It also is not known how the conclusions were generated. Two of these reviews (Chassin, 1984; Dielman et al., 1991), in particular, came to conclusions concerning the most salient predictors of adolescent drug initiation. Since it is not clear whether a thorough review of the literature was conducted, all applicable studies were included, or how the results were generated, the findings should not be given as much weight as those derived from a systematic methodology.

Relevance of Focus and Content

The majority of the literature reviews did not assess drug initiation in isolation of other variants of adolescent drug use (i.e., frequency of use, drug abuse, drug dependence). In fact, only nine reviews focused specifically on predictors of adolescent soft drug initiation (Bauman & Ennett, 1996; Charlesworth & Glantz, 2005; Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; DiFranza et al., 2006; Donovan, 2004; Glynn, 1981; Wakefield et al., 2003). Three reviews published in the past twenty years focused on predictors of cigarette initiation (Conrad et al., 1992; Derzon & Lipsey, 1999a; DiFranza et al., 2006), while a recent review examined longitudinal predictors of adolescent alcohol initiation (Donovan, 2004). Derzon and Lipsey's (1999a) statistical meta-analysis of 67 longitudinal studies of tobacco use included both tobacco initiation and frequency of use studies and excluded studies using cross-sectional designs. DiFranza et al.'s (2006) assessment focused solely on the predictive utility of tobacco advertising and promotion for adolescent initiation of cigarette use. Moreover, Donovan's (2004) review of risk factors for alcohol initiation only focused on longitudinal studies and did not examine school- and community-level risk factors.

Of the nine literature reviews that did focus on predictors of adolescent soft drug initiation, only one (Bauman & Ennett, 1996) examined predictors for all three soft drugs. The remaining reviews examined predictors of cigarette initiation (Charlesworth & Glantz, 2005; Conrad et al., 1992; Derzon & Lipsey, 1999a; DiFranza et al., 2006; Wakefield et al., 2003), or alcohol (Donovan, 2004; Glynn, 1981) or marijuana initiation (Derzon & Lipsey, 1999c; Glynn, 1981).

Comprehensive reviews that synthesize and critically assess research on risk factors for specific types of drugs have tended to examine predictors of marijuana use (e.g., Derzon & Lipsey, 1999c; Penning & Barnes, 1982; Petraitis et al., 1998) and alcohol use (see, e.g., Dielman et al., 1991; Donovan, 2004; Halebsky, 1987; Hastings et al., 2005). Petraitis et al.'s (1998) review of 58 longitudinal studies of adolescent marijuana use, conducted almost ten years ago, was the first systematic, comprehensive assessment of the adolescent marijuana use literature to assess a wide variety of predictors. This assessment, however, included studies that measured marijuana use in varying ways (e.g., lifetime; six-month; monthly; daily; problematic; heavy; experimental; regular; moderate; continued; and age at initiation). Literature reviews examining adolescent cigarette use in isolation from other types of drugs are relatively sparse. In fact, only four such comprehensive reviews were identified (Charlesworth & Glantz, 2005; Conrad et al., 1992; Derzon & Lipsey, 1999a; DiFranza et al., 2006).

Very few reviews examined whether predictors of adolescent soft drug initiation vary as a function of biological age. Of the four reviews that did, only two (Conrad et al., 1992; Donovan, 2004) were drug initiation reviews. A third

(Halfors et al., 2002) only assessed age-graded differences in the predictive utility of school truancy and low grade point average.

Although numerous empirical studies have assessed the sequence and temporal ordering of alcohol, cigarette, and marijuana initiation, it is surprising that only four reviews (Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Penning & Barnes, 1982) examined the predictive utility that prior “other drug initiation” (or use) has for predicting alcohol, cigarette, or marijuana initiation. Given the prevention implications stemming from research that finds past drug use to be a strong correlate and predictor of the initiation of other drugs, there clearly is a need for more systematic reviews assessing the utility of this type of risk factor.

Comprehensiveness

The majority of reviews did not assess predictors originating from all five ecological domains of influence (i.e., individual, peer, family, school, and community). For instance, school-based risk factors for adolescent drug use have been examined in isolation (e.g., Dewey, 1999; Halfors et al., 2002), as have various types of community-level risk factors, such as tobacco promotion and forms of media influence (see, e.g., Charlesworth & Glantz, 2005; DiFranza et al., 2006; Wakefield et al., 2003). Bauman and Ennett (1996) focused specifically on peer influence factors, with minor attention to parental influence factors, while others have centered exclusively on family-based risk factors (Vakalahi, 2001). Still other research has considered intrapersonal risk factors in isolation (e.g., Swadi, 1999). Only five reviews assessed predictors from all domains (Conrad et al., 1992; Donnermeyer, 1992; Hawkins et al., 1992a; Leukefeld et al., 1998; Petraitis et al., 1998). Of these five reviews, only one (Conrad et al., 1992) focused specifically on predictors of adolescent soft drug initiation.

Summary

The majority of the literature reviews assessed were not systematic or comprehensive in nature, and most did not examine predictors of adolescent soft drug initiation in isolation of other variants of use. Results from this assessment also indicate that of those systematic reviews that did assess predictors of adolescent soft drug initiation, none discussed or examined initiation in terms of: (1) predictors common or unique to cigarette, alcohol, and marijuana use; and (2) predictors common or unique to different developmental stages of adolescence.

It appears necessary that an updated systematic, comprehensive review of the adolescent soft drug initiation literature be conducted. Such an assessment can advance knowledge within the field of prevention science by calling attention to salient correlates and predictors that have received less attention in previous reviews (e.g., prior drug use). Moreover, since primary drug prevention programs attempt to take into account the developmental stage of their audiences and tailor prevention initiatives according to age-specific risk factors, assessing the degree to which predictors of soft drug initiation are age-graded may be a fruitful and informative line of inquiry.

Systematic Review Methodology

The literature on predicting adolescent initiation of soft drug use is relatively large and rather cumbersome. Within this body of research, the methods and procedures employed, sample characteristics, and timing of measurement between risk factors and outcome measures (in longitudinal studies) are quite diverse. To sort out and update this complex body of literature, and to provide a means by which findings from the quantitative component of the present study may be cross-validated, an initial investigation was conducted through a systematic, comprehensive review of the literature on adolescent initiation of soft drug use (i.e., alcohol, cigarettes, marijuana).

A dual purpose founded this undertaking. First, as discussed earlier, an examination of previous reviews failed to identify a comprehensive review that summarizes and critically assesses empirical studies examining predictors of the adolescent initiation of all three soft drugs. Cigarettes, alcohol, and marijuana are the most commonly used substances among adolescents, so a comprehensive assessment of studies examining predictors of adolescent soft drug initiation is warranted. The proposed comprehensive assessment filled this gap in the literature, providing a much needed systematic review that identified, critically assessed, and synthesized the empirical evidence in this area. Research Questions #2 and #3 of the proposed study constituted the key questions that guided this effort[4].

[4] (RQ#2) What factors predict soft drug initiation and age of initiation among adolescents? (RQ#3) Since the initiation of soft drug use appears to vary, in part, as a function of biological age, what factors predict soft drug initiation and age of initiation for youth at different stages of adolescence?

Second, conducting a systematic, comprehensive review provided a means by which findings from the quantitative component of the present study could be cross-validated. For instance, this comparison provided a way for plausible threats to the internal and external validity of the quantitative findings to be ruled out.

The following sections detail the standardized procedures that were employed in identifying and retrieving studies relevant for inclusion in the systematic review. The analysis plan also is discussed, with attention paid to how this body of research was critically examined and synthesized, and how conclusions were drawn. Finally, the first component of the dual cross-validation scheme is introduced, with consideration to how results from the systematic review were compared to the quantitative findings.

Inclusion Criteria

Table 12 outlines the inclusion criteria that were employed in identifying study candidates for the assessment. Inclusion criteria were employed for two primary reasons. First, establishing a priori inclusion criteria structures the review in a more systematic and objective fashion, which, in turn, provides for a more objective assessment of the extant literature (Cooper, 1982; Hauser-Cram, 1983; Jackson, 1980). It also provides a means by which others may attempt to replicate the findings, an important principle in social and behavioral science research (Lieberson, 1985).

Second, in order to the enhance the cross-validation of quantitative findings with those of the systematic review, it is important to decrease the degree to which differences in sample characteristics, research design and methodology, and outcome measures exist. These inclusion criteria constituted a mechanism by which differences between studies were controlled. Granted, while it was not possible to completely ameliorate all differences without drastically decreasing the number of studies included in the comprehensive assessment, constructing a priori inclusion criteria based upon characteristics of the quantitative study provided a greater degree of semblance in study purpose, design, methodology, population characteristics, outcome measures, and cross-validation. The inclusion criterion are detailed below.

Table 12.

Inclusion Criteria for Systematic Review

Study Component	Inclusion Criteria
Time period:	▪ 1970 - 2007
Publication and study type:	▪ U.S. studies published in refereed sources
Language:	▪ English
Study design:	▪ Longitudinal; cross-sectional
Population characteristics:	▪ 12-17 years of age (6^{th}-12^{th} grade) at time of outcome measurement who are eligible for primary prevention programming, if targeted
Type of Predictors:	▪ Risk and protective factors; correlates
Type of Outcome Measures:	▪ Initiation of alcohol, cigarettes, marijuana; Time to alcohol, cigarette, marijuana initiation
Timing of Measurement:	▪ Criterion variables measured when study participants were 12-17 years of age
Analytic techniques:	▪ Multivariate statistical techniques

Time Period, Publication and Study Type, Language, and Study Design

Data for the systematic review were drawn from longitudinal and cross-sectional studies conducted in the U.S. and published in refereed journals (in English) from the 1970s until present. Studies conducted outside of the U.S. were excluded based on the generally accepted assumption that social structure, economic forces, and social tolerance of adolescent and adult drug use differ as a function of the culture endemic to particular countries (Brook, Brook, Arencibia-Mireles, Richter, & Whiteman, 2001; Patrick, 1970).

Population Characteristics

The quantitative component of the present research involves determining what psychosocial factors predict soft drug initiation (and age of initiation) among youths from the general population who were in 6th, 9th, and 12th grade at the time criterion variables were measured. As such, studies that were eligible for inclusion in the systematic review were those that utilized adolescent samples drawn from the general adolescent population, consisting of youth who were between the ages of 12 and 17 years when the criterion variables were measured, and were not participating in drug intervention initiatives or programs. Studies of college students were excluded due to the vastly different social contexts in which these late adolescents and early adults interact.

Youth from the general adolescent population are those who, if targeted for drug prevention programming, would be provided primary (universal) prevention designed to reduce the risk of initiating drug use. Since the magnitude of risk effects are generally larger in studies utilizing selective and indicated samples, compared to studies utilizing universal samples (Gorman, 1996; Norman & Turner, 1993), and the focus of the present research was on elucidating predictors of soft drug initiation among adolescents not at a high-risk for initiation, only studies utilizing samples from the general population were included. Empirical studies utilizing adolescent samples deemed to be at high-risk for substance use, abuse, and/or dependence, including children of alcoholics, juvenile delinquents, institutionalized youth, and runaway and homeless youth, were excluded. Also excluded were studies utilizing adolescent samples clinically diagnosed with (or suspected of coping with) one or more substance abuse or dependence problems or disorders.

Studies must also have measured criterion variables when study participants were between 12 and 17 years old. In cases where grade-level was employed as a proxy measure of biological age, and the age of study participants was not reported, studies were included if criterion variables were measured when youths were in 6^{th}-12^{th} grade.

Type of Predictors

To be clear, in order for a variable to be considered a risk factor for initiation of a given soft drug, two criteria must be met. First, evidence of an association must exist. Specifically, there needs to be evidence of a statistically significant relationship between the candidate predictor and initiation of the drug under consideration (Kraemer et al., 2001). Second, there must be evidence that proper temporal ordering has been established. Specifically, there needs to be evidence that the candidate predictor occurred or was present prior to the adolescent’s involvement with the soft drug in question. Without meeting the second criteria, the candidate predictor should be viewed as a correlate of initiation, not a risk factor for initiation.

Cross-sectional data, which have constituted much of the focus of previous comprehensive reviews, can establish that a predictor correlates with the initiation of a drug, but can not determine the direction of influence between the two variables (Kraemer et al., 2001). Hence, the second criteria can not be met when cross-sectional data are utilized. To establish whether a predictor functions as a risk factor for initiation requires the collection of longitudinal data. However, to have excluded studies from the systematic review based on the inability to meet the second criteria constitutes ignoring a rich source of knowledge that, many times, has identified associations between predictors and initiation outcomes that have been replicated and consequently confirmed in longitudinal research. Since the goal of the systematic review was to synthesize knowledge and update this body of literature, cross-sectional studies were included. The discussion of the literature is explicit where common findings are found across study designs, or where conflict exists.

Type and Timing of Outcome Measures

For consistency, all studies that were included in the systematic review were those that either focused upon the adolescent transition from lifetime abstention (never used) into lifetime initiation (ever used) of alcohol, cigarettes, and marijuana use, or time to alcohol, cigarette, or marijuana initiation. With respect to soft drug initiation, investigations that were subject to inclusion were cross-sectional studies that compared abstainers and initiates on a variety of psychosocial variables, or longitudinal studies that followed youth over time and compared psychosocial variables for adolescents who transitioned to initiation from abstention (at baseline) to youths who remained abstainers throughout the follow-up periods. Studies defining initiation on the basis of questions about alcohol, cigarette, or marijuana use in the past year (e.g., Miller & Miller, 1997), past month (see, e.g., Cohen, Richardson, & LaBree, 1994; Killen et al., 1997; Yu & Williford, 1992), or past week or few days (e.g., Ennett, Bauman, Hussong, Faris, Foshee, & Cai, 2006; Weber, Graham, Hansen, Flay, & Johnson, 1989) were excluded, due to the likelihood that some of those youth considered abstainers actually were initiates who simply had not had the opportunity to use the given soft drug within the narrower timeframes that were specified.

Other excluded studies were those that examined (1) predictors of smokeless tobacco and hard drug use (e.g., Colder & Stice, 1998; Krohn & Lizotte, 1996); (2) the temporal sequencing of soft drug initiation (e.g., Kandel, 1975b); (3) abuse and dependence, or other consequences of use (e.g., Ellickson et al., 2001; White, 1987); (4) soft drug use as a component of a general problem behavior construct (see, e.g., Agnew, 1991a; Williams, Ayers, Abbott, Hawkins, & Catalano, 1996); and (5) the frequency, quantity, and extent of soft drug use (see, e.g., Dishion, Capaldi, & Yoerger, 1999; Hill, Shen, Lowers, & Locke, 2000b). In addition, research that examined gender-specific (see, e.g., Andrews, Hops, Ary, Lichenstein, & Tildesley, 1991; Hops, Davis, & Lewin, 1999; Killen et al., 1997; van den Bree, Whitmer, & Pickworth, 2004) and race-specific

(Griesler & Kandel, 1998; Wallace, Brown, Bachman, & LaVeist, 2003) determinants of soft drug initiation, or time to initiation (Fleming, Kellam, & Brown, 1982), also were excluded.

Due to the nature of the unique characteristics of prospective data, two inclusion standards were applied to longitudinal studies. First, longitudinal studies subject for inclusion were those in which two or more waves of data collection were employed, predictors were measured at baseline, and lifetime initiation was measured at each wave of data collection.

Second, eligible longitudinal studies were those in which analyses were restricted to adolescents who reported baseline abstention of the soft drug in question. Studies that merely predicted Time 2 drug use status from Time 1 predictors without excluding Time 1 initiates were not examined because these studies included both Time 1 abstainers and initiates (see Peterson, Hawkins, Abbott, & Catalano, 1994). Third, analyses had to involve predicting Time 2 (or later wave) drug use status (abstainer versus initiate).

Analytic Techniques

The quantitative component of the present research employed multivariate statistical techniques (i.e., binary logistic regression and Cox regression) that permitted the variance in multiple predictors to be controlled or accounted for while the utility of individual factors was simultaneously assessed. As such, only studies that employed multivariate statistical techniques (e.g., regression, structural equation modeling, discriminant function analysis, and survival analysis) were eligible for inclusion in the pool of studies that were assessed.

Excluded were studies employing univariate and bivariate (correlational) techniques only, as well as those that only assessed group membership (such as through Analysis of Variance).

Study Retrieval

Standardized procedures commonly recommended for identifying potential study candidates were followed (see, e.g., Cook & Levitan, 1980; Cooper, 1982; Cooper & Arkin, 1981; Cooper & Rosenthal, 1980; Farrington & Weisburd, 2007; Glass, 1977; Graham, 1995; Hauser-Cram, 1983; Jackson, 1980). Specifically, multiple comprehensive searches were performed for the dual purpose of locating and including as many relevant published studies on adolescent soft drug initiation as possible, and reducing the chance that data supporting a particular conclusion would be preferentially identified while other published findings would be missed. Keyword searches of studies published in books, multiple electronic databases, and published studies indexed in various organization websites (see Table 13) all were examined to identify potential candidates for assessment.

Each study abstract was assessed to determine whether the full study was suitable for retrieval. Studies were considered appropriate when their abstracts: 1) explicitly stated that risk and protective factors were examined in relation to adolescent substance use; or 2) did not mention that risk and protective factors were examined, but alluded to employing predictors characterized in the literature as risk or protective factors. After full copies of the studies were retrieved, a more detailed assessment of each was conducted in order to determine whether the study fully met the eligibility criteria.

Table 13.

Electronic Databases and Organization/Agency Internet Websites Searched

Type/Source	
Electronic Databases	
Academic Search Premier	MEDLINE
CINAHL Select	Primary Development Collection
CINAHL with Full Text	PsycARTICLES
ERIC	PsycINFO
Health Source- Consumer Edition	Psychology/Behavioral Sciences Collection
Health Source- Nursing/Academic Edition	PubMed
InfoTrac	SocINDEX with Full Text
JSTOR	Sociological Collection
Organization/Agency Websites	
Alcohol and Alcohol Problems Science Database (ETOH)	
Alcohol Studies Database	
Drug Policy Alliance	
JTO Direct Archive	
National Clearinghouse for Alcohol/Drug Information	
National Institute on Alcohol Abuse and Alcoholism	
Society for Prevention Research	

Articles meeting the eligibility criteria were sorted and categorized according to the type of drug used as an outcome measure. After this was completed, the literature review portion of each study was read, and bibliographic scans were conducted in an attempt to locate additional studies. These methods identified 71 statistical models (see Table 14) found in 36 primary studies (see Table 15) that met the inclusion criteria. The majority (n = 23) employed a longitudinal research design.

Table 14.

Prediction Models Eligible for Systematic Review, by Drug Type

Type of Drug Initiation Predicted	N [a]
Alcohol	20
Cigarettes	27
Marijuana	24

[a] Summing the number of prediction models does not equate to the total number of empirical studies eligible for assessment. Fifteen studies examined the initiation of more than one type of soft drug, while one study examined predictors of alcohol, cigarette, and marijuana abstention.

Table 15.

Primary Studies for Systematic Review

Time Period, Author(s), and Publication Year

1970s:

- Kandel, Treiman, Faust, & Single (1976)
- Smith & Fogg (1978)

1980s:

- Chassin, Presson, Sherman, Corty, & Olshavsky (1984)
- Brook, Whiteman, Gordon, Nomura, & Brook (1986)
- Chassin, Presson, Sherman, Montello, & McGrew (1986)
- Kandel & Andrews (1987)
- Marcos & Bahr (1988)
- Skinner, Massey, Krohn, & Lauer (1985)

1990s:

- Bailey & Hubbard (1990)
- Flewelling & Bauman (1990)
- Walter, Vaughan, & Cohall (1991)
- Webb et al. (1991)
- Foshee & Bauman (1992)
- Flay et al. (1994)
- Epstein, Botvin, Diaz, & Schinke (1995)
- Robinson, Klesges, Zbikowski, & Glaser (1997)
- Urberg, Degirmencioglu, & Pilgrim (1997)
- Werch, Carlson, Pappas, Dunn & Williams (1997)
- Amey & Albrecht (1998)
- Duncan, Duncan, & Hops (1998)
- Flay, Hu, & Richardson (1998)
- Epstein, Botvin, Baker, & Diaz(1999)
- Unger & Chen (1999)

2000s:

- Ennett et al. (2001)
- Hawkins, Hill, Guo, & Battin-Pearson (2002b)
- Gritz et al. (2003)
- Pokorny, Jason, & Schoeny (2003)
- Urberg, Luo, Pilgrim, & Degirmencioglu (2003)
- Ellickson, Tucker, Klein, & Saner (2004)
- Kandel, Kiros, Schaffsan, & Hu (2004)
- Tilson, McBride, Lipkus, & Catalano (2004)
- Ramirez et al. (2004)
- Crum, Storr, & Anthony (2005)
- D'Amico & McCarthy (2006)
- Shears, Edwards, & Stanley (2006)
- Williams et al. (2007)

Systematic Review Analysis Plan

The analysis plan for the systematic review consisted of four major stages, several of which were preparatory in nature. Overall, the stages included documenting general study characteristics; recording significant relationships; organizing individual findings; and synthesizing individual results, identifying similarities and dissimilarities in findings, and drawing conclusions.

First, major study characteristics were systematically documented. As research has found, inconsistent findings across studies can sometimes be explained by differences in subjects, sample size, sampling error, setting, the quality of research methods, the nature of other variables that were controlled, and the nature and type of measurements utilized (Feldman, 1971; Jackson, 1980). The purpose in documenting important features of each primary study was to use this information in trying to explain divergent findings, as well as provide a means by which individual or aggregate findings may be contextualized. Study characteristics (see Table 16) were culled from each study and have been placed in Appendix F for easy referral.

Second, final prediction models (after all independent variables have been entered) in each study were assessed in order to identify and document the direction (+/-) and significance ($p \leq .05$) of each coefficient (for each predictor-outcome relationship). Following the lead of other researchers (Light & Smith, 1971, p. 433), three specific outcomes were defined in relation to $p \leq .05$: 1) positive significant relationships; 2) negative significant relationships; and 3) non-significant relationships (+/-). Given that the primary studies used different statistical analysis techniques, and standardized coefficients were not reported for each predictor examined across studies, the magnitude of relationships was not assessed.

Table 16.

Major Study Characteristics with Examples

Study Characteristics	Examples of Study Characteristics
Design:	▪ Longitudinal
Sample size:	▪ 520
Geographic location of sample:	▪ Urban
Location of data collection:	▪ Classroom
Data source:	▪ Self-report survey
Probability sampling method:	▪ No (school district-wide)
Efforts to reduce under/over-reporting:	▪ No
Cross-validation:	▪ No
Age/grade predictor variable(s) measured:	▪ 10-11 years
Age/grade criterion variable(s) measured:	▪ 15-16 years
Criterion variable(s) and operationalization:	▪ Alcohol initiation = Dichotomous initiation
Domains and Predictors:	▪ Individual = Temperament ▪ Family = Perceived parental drug use
Mediation/moderation:	▪ No
Analytic technique(s):	▪ Binary Logit
Major Limitations:	▪ 40% attrition between T1 and T2 ▪ Limited number of domains and predictors

Third, multiple matrices were used to organize significant and non-significant relationships and provide a systematic means by which similar and dissimilar findings across studies were identified. Three matrices were developed for each drug type (e.g., initiation of alcohol, initiation of cigarettes, and initiation of marijuana) and six periods of adolescent development. In part, to account for the fact that numerous studies obtained data from samples of youth whose biological ages (or grade-levels) spanned more than one conventional period of adolescence (i.e., early adolescence), findings were organized according to six specific age groups: early-late adolescence (12-17 years, or 6th-12th grade), early adolescence (12-14 years, or 6th-8th grade), early-mid adolescence (12-16 years, or 6th-10th grade), mid-adolescence (14-16 years, or 9th-10th grade), mid-late adolescence (14-17 years, or 9th-12th grade), and late adolescence (16-17 years, or 11th-12th grade). Age group classifications for early, mid-, and late adolescence are similar to those used by other researchers (see, e.g., D'Amico et al., 2005; Scheier, Newcomb, & Skager, 1994).

Within each period of adolescent development (for each of the three drug types), findings were organized by the type of study design employed (i.e., longitudinal, cross-sectional), ecological domain from which predictors originate (i.e., individual, peer, family, school, community), and the type of construct (e.g., social bonding, social learning, or SDM) found within each ecological domain. Table 17 illustrates the scheme that was used in organizing theoretical constructs.

To reinforce this organizational scheme and aid in synthesizing findings, EndNote, version 8.0 (Thompson ISI ResearchSoft, 2004) was used. Keywords were used to link primary studies according to the type of drug outcome measure; study design; period of adolescent development; ecological domain; general theoretical construct; and findings.

Table 17.

Ecological Domains and Related Predictor Categories

Domains/Summary Categories	Examples of Predictors
Community Domain	
Social learning constructs:	Non-familial adult drug use; non-familial pro-drug norms
Community characteristics:	Availability of drugs; neighborhood disadvantage
School Domain	
School bonding constructs:	School attachment, involvement, commitment
School behavior constructs:	Poor school performance; truancy; detention/suspension
School characteristics:	Strict smoking policy
Family Domain	
Family bonding constructs:	Attachment to parents; family involvement
Family social learning constructs:	Parental drug use; parental prodrug norms
Family relationship factors:	Low parental monitoring; family conflict
Family characteristics:	Single-parent family; Step-parent family
Peer Domain	
Peer bonding constructs:	Attachment to drug-using peers
Peer social learning constructs:	Peer drug use; peer prodrug norms
Peer relationship factors:	Peer support
Individual Domain	
Traits/states:	Impulsivity; sensation-seeking; depression
Attitudes and intentions:	Alcohol use intention; prodrug norms
Behaviors:	Marijuana initiation; delinquency; work-for-pay

Fourth, vote-counting methods were used as a tool to identify common patterns of relationships and mixed findings, as well as synthesize findings from primary studies into aggregate results. Using the three outcomes that were previously discussed (i.e., using the $p \leq .05$ threshold to identify positive significant, negative significant, and non-significant relationships), the number of studies falling into each outcome category (for each predictor) were summed. The modal category was assumed to provide the best estimate of the direction (and significance status) of the relationship between the given predictor and soft drug initiation outcome measure.

Mentioned earlier, vote-counting has some weaknesses. Differences in sample sizes across studies are not taken into account, as well as the differential saliency or importance of predictors. However, given the fact that this methodology for integrating research findings is common in the social science, education, and medical fields (see, e.g., Cook, Mulrow, & Haynes, 1997; Graham, 1995; Jackson, 1980; McCormick, Rodney, & Varcoe, 2003; Mulrow & Cook, 1997), and meta-analytic techniques were not employed, the drawbacks of this approach are acknowledged and viewed as a limitation of the assessment.

Illustrated in Table 17, this systematic review was guided by the same theoretical framework that underlies the quantitative component of the research. Specifically, explanatory constructs of interest are grounded either in SDM (Chapter 3), or the traditional theories of social control and social learning that SDM subsumes (Chapter 2). Explicit attention was directed toward findings that speak to several issues highlighted in the discussion of the prediction literature

(Chapter 4). The utility of parent and peer behaviors and norms were of interest, as was the value of prior initiation of soft drugs (i.e., cigarettes or marijuana) for predicting other soft drugs (i.e., alcohol). Drug-specific and age-graded risk and protective factors also were of interest.

Finally, the presentation of findings is organized according to the five ecological domains and theoretical constructs that frame the focus of the review (see Table 17). Tables are used to illustrate the key features of the primary studies, present the directional relationships found for each predictor, and facilitate comparisons. Following the lead of other researchers (Petraitis et al., 1998), all primary studies assessed were assigned a reference number. Reference numbers link tabularized results for individual studies to respective study characteristics (Appendix F). Other comprehensive literature reviews have taken a similar approach to organizing, presenting, and discussing predictors of adolescent drug use (see, e.g., Chassin, 1984; Clayton et al., 1995; Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Donovan, 2004; Hawkins et al., 1992a).

Systematic Review Cross-Validation

The methodological quality of an empirical study is gauged, in large part, by the degree to which findings may be characterized as valid. Validity refers to the accuracy of “inferences about cause and effect” (Shadish, Cook, & Campbell, 2002, p. 34). Although validity is conceptualized as a unitary concept, it is assessed in terms of four specific criteria: statistical conclusion validity, internal validity, construct validity, and external validity (Welsh & Farrington, 2006b).

Some threats to validity may be manipulated in an effort to minimize the degree to which they are plausible. Other threats can not be manipulated, however, and may only be assessed in terms of the likelihood they exist and the type of impact they may have had on research findings.

In an effort to assess the validity of the quantitative findings, a dual cross-validation scheme was employed. Characterized as a comparison of findings from the study of interest to the results of another study (Collins et al., 1987), a cross-validation typically is designed to achieve three goals: (1) provide a general indication of the stability of research findings; (2) determine the extent to which various threats to the validity of findings are plausible; and (3) identify the type of impact that plausible threats may have had on results (Mills & Noyes, 1984).

This dual cross-validation scheme involved comparing the quantitative findings derived from an adolescent sample (i.e., the derivation sample) who completed a survey in 2004 to those generated from: 1) the systematic, comprehensive review; and 2) quantitative findings obtained from a similar, but distinct, sample of youth (i.e., the validation sample) who completed a similar survey instrument in 2001[5]. Comparing quantitative findings to those generated from two other sources made it possible to identify how plausible threats to the validity of findings may have operated in impacting the results. When quantitative findings converge with those from both sources, more confidence may be placed in those results (Collins et al., 1987). The quantitative assessment, which will be

[5] In an effort to clearly distinguish between these two cross-validations, the cross-validation involving the systematic, comprehensive review will be referred to as the “systematic review cross-validation” from this point forward, while the second cross-validation will be referred to as the “quantitative cross-validation.”

taken up at the end of Chapter 6 (Quantitative Methods), was primarily designed to assess plausible threats to the internal and external validity of the quantitative findings. The systematic review cross-validation also assessed likely threats to the internal and external stability of findings; however, this cross-validation also took explicit strides in considering the statistical conclusion and construct validity of the results.

Most of the primary studies that were examined through the systematic review used a self-report methodology when data were gathered in classroom settings. The limitations of this methodology, coupled with the sensitive nature of drug use, and the standard design and analytic issues associated with non-experimental research, pose challenges for adolescent drug researchers who strive to make accurate inferences about causal relationships between psychosocial factors and soft drug initiation.

Since a variety of factors can impact the validity of results (and subsequent causal inferences), divergent findings revealed through the systematic review cross-validation may be the result of one or more limitations intrinsic to the research design, methodology, sample, or analytic strategies upon which findings from primary studies (from the comprehensive review) are based. Keeping this in mind, it is important to place the issue of validity (and its threats) within the broader context of adolescent drug research before discussing the logistics that founded the systematic review cross-validation.

Statistical Conclusion Validity

Statistical conclusion validity refers to the accuracy of conclusions about the relationship between hypothesized predictors and the outcome variable under inspection (Shadish et al., 2002; Welsh & Farrington, 2006b). Regardless of the type of research being conducted, two factors serve as major threats to the statistical conclusion validity of findings: inappropriate statistical techniques and low statistical power.

Inappropriate Statistical Techniques

The use of inappropriate statistical techniques increases the likelihood that inaccurate results are obtained concerning the relationship between a predictor and an outcome. For instance, employing ordinary least squares regression when the outcome measure is a dichotomous variable violates an assumption endemic to ordinary least squares regression (the criterion must be operationalized as a continuous measure) and poses a threat to the statistical conclusion validity of the results, since findings may be either un-interpretable or inaccurate.

If a considerable number of the primary studies that were examined in the comprehensive review did not use appropriate statistical techniques, the findings from this review may not be valid. In turn, these compromised results may explain, in part, any disjuncture between findings from the systematic review and those from the quantitative component of the current research.

Low Statistical Power

The second major threat to statistical conclusion validity centers on statistical power and Type II error. Statistical power is essentially the power of the significance test. Expressed as 1- β (where β is the probability of committing a Type II error), statistical power is the probability of not committing a Type II (beta) error when testing the null hypothesis, given a certain effect size (ES), significance criterion (α), and sample size (Cohen, 1988; Green, 1991; Hansen & Collins, 1994). As a false negative, a Type II error is committed when the null hypothesis is accepted when a relationship does exist between a predictor and the outcome in the population. In contrast to a Type II error, a Type I (alpha) error is a false positive and occurs when the null hypothesis is rejected when no relationship exists between the predictor and outcome (Hansen & Collins, 1994; Pedhazur, 1982).

Low statistical power jeopardizes the ability to detect a relationship between a predictor and outcome variable and increases the risk of committing a Type II error. When a significance criterion is selected, what essentially is being established is the probability (or risk) of committing a Type I versus a Type II error. For example, given several statistical considerations, with α = .05, the probability of committing a Type II error could be calculated as .20. Power then could be computed as 1-.20, which equates to .80. With α = .05, and power = .80, the α:β ratio is 4:1 (Cohen, 1988). Hence, there is a higher probability of erring on the side of caution. Producing a false negative result

(Type II error) generally is afforded a higher probability of occurring than producing a false positive result (Type I error).

Given certain statistical conditions for the significance criterion (α), level of power, and effect size (ES) desired, the size of a sample can impact the ability to detect significant relationships. A small sample poses a plausible threat to statistical conclusion validity, since a statistical model developed with a small sample may fail to detect significant relationships between predictors and an outcome (Welsh & Farrington, 2006b). Within the context of the current research, one possible explanation for disconcordant findings between those of the quantitative research and those stemming from the systematic review is small samples upon which findings from some of the primary studies were based.

Construct Validity

Although the main purpose of the systematic review cross-validation is to assess the internal and external validity of the quantitative findings, it is possible that any disjuncture between the findings from the systematic review and those from the quantitative research may be a partial consequence of construct validity threats endemic to the primary studies that were examined. Construct validity is characterized as the degree to which operational definitions and measures accurately represent theoretical constructs at the broader, conceptual level (Shadish et al., 2002; Welsh & Farrington, 2006b).

Given that the primary studies that were examined in the systematic review already have been conducted, much of the front-end research design elements that could have been manipulated to enhance construct validity already either have or have not been employed. Overall, four factors pose major threats to the construct validity of findings generated from cross-sectional, adolescent drug research employing a self-report survey methodology: (1) invalid or unreliable measures; (2) reporting bias; (3) single source of data collection; and (4) lengthy periods of recall (Shadish et al., 2002).

Invalid or Unreliable Measures

Invalid or unreliable outcome measures do not properly capture the constructs of interest. When these types of measures are used in a study, not only is the construct validity of measures threatened, but the internal validity of findings also becomes questionable (Shadish et al., 2002). As a whole, adolescent drug research is plagued with inconsistent measurement of psychosocial factors and drug initiation outcomes (see, e.g., Allen et al., 2003; Bauman & Ennett, 1996; Kandel, 1975a, 1996; Schaps, DiBartolo, Moskowitz, Palley, & Churgin, 1981). Any contradiction in findings from the quantitative research and the systematic review may partly reflect differences in the operationalization of psychosocial factors.

Reporting Bias

As with all other validity threats discussed thus far, divergent findings between the systematic review and the quantitative research may be partially due to reporting biases that have impacted findings from the primary studies. Reporting (or response) bias can take several forms, including forward and backward telescoping, overreporting, underreporting, and recall decay, which can result in heaping (Bachman & O'Malley, 1981; Golub et al., 2000a, 2000b).

Backward telescoping is not a common occurrence in adolescent drug research, although forward telescoping does constitute a plausible threat to the construct validity of drug measures and the internal validity of the findings (Golub et al., 2000b). Compared to younger students, older students are more apt to bring forward in time the periods at which they report initiating drugs (Johnston, Bachman, & O'Malley, 1992). Further research suggests that forward telescoping serves to attenuate estimates for early ages of drug initiation (referred to as downward bias), while inflating estimates for older ages of drug initiation, resulting in upward bias (Johnson & Schultz, 2005; Johnston et al., 2007).

All adolescent drug research utilizing a self-report methodology must contend with the likelihood that underreporting has occurred. This poses a plausible threat to both the construct validity of drug initiation outcome measures and the internal validity of the findings (Golub et al., 2000a; Harrison & Hughes, 1997). Drug use is a sensitive topic for many youth, with some adolescents fearful of disclosing involvement for fear of chastisement (Junger-Tas & Marshall, 1999). If a considerable proportion of a sample underreports soft drug initiation or the occurrence of behavioral risk factors (e.g., frequency of truancy or cheating on schoolwork), relationships between predictors and soft drug initiation measures will be attenuated (Johnston et al., 1992).

Heaping, or inaccurately reporting one time period for the occurrence of multiple events (Pickles et al., 2001), constitutes another plausible threat to the construct validity of findings for any cross-sectional study examining self-report data for age of soft drug initiation. Research suggests that heaping (for age of drug initiation) is a common occurrence in cross-sectional drug research. Compared to younger adolescents, this phenomenon appears to occur more often among older adolescents who, many times, must recall drug initiation that occurred several years prior to survey administration (Golub et al., 2000a).

Self-Report Data

In general, almost all adolescent drug research relies exclusively on self-report measures. Corroborating this information with other sources (e.g., parents or other adults) is a rarity in adolescent drug research; however, the use of multiple sources of data has increased in recent years (Harrison, 1997; Kandel, 1996). Although sole reliance on self-report data calls into question the accuracy of measures and responses, there is a considerable line of research indicating that self-report data is both a reliable and valid approach to gathering information (see Harrison, 1995, 1997; Hser, 1997; Magura & Kang, 1997). Since the majority of the primary studies that were examined in the systematic review used self-report measures, conflicting findings that were uncovered through the systematic review cross-validation possibly could be linked to those primary studies that used multiple sources to gather data.

Lengthy Recall Periods

Due to poor memory recall, longer recall periods tend to produce less valid responses than those generated with shorter referent periods (Junger-Tas & Marshall, 1999). Some of the systematic review studies are cross-sectional in nature, with biological age of initiation data derived from youths themselves who were required to recollect the ages at which they first initiated soft drugs. Other studies used prospective research designs, however, with drug initiation data collected within several short intervals over time. As with the other plausible threats to construct validity, this particular threat was partially addressed through the systematic review cross-validation, with the consideration of inconsistent findings and the type of research designs utilized by the primary studies.

Internal Validity

Internal validity concerns the degree to which a correlation between a predictor and an outcome actually represents a causal relationship, whereby changes in the predictor consequently cause changes in the outcome (Shadish et al., 2002). With regard to adolescent drug research, three factors constitute plausible threats to the internal validity of findings: selection bias, history, and causal ordering. The systematic review cross-validation attempted to assess the likelihood that these threats impacted the quantitative findings. In instances where results from this comparison of findings revealed inconsistencies, those primary studies (e.g., conducted through longitudinal research) that reached conclusions counter to the quantitative research were examined in a more indepth manner to assess whether these threats partly explained differential results.

Selection Bias

There are two major issues related to sampling in adolescent drug research that can lead to selection bias, thereby posing a likely threat to the internal validity of findings. First, in contrast to experimental and quasi-experimental research, which involves random assignment or matching to treatment and control groups (Shadish et al., 2002), the current adolescent drug research was non-experimental in nature. Since youth present pre-existing differences from each other on varying levels of explanatory factors (e.g., high versus low prosocial bonding), researchers must measure and take into account all factors that may constitute third variable explanations. Regardless of research design, it is unlikely that any study can account for all possible explanations. One major goal of the systematic review cross-validation was to rule out any alternative explanations for relationships identified in the quantitative research.

Second, due to the ethical implications associated with coerced research participation, all adolescent drug researchers must gain the voluntary cooperation of youth when drug data are collected directly. While voluntary cooperation is a standard that all researchers should uphold, a drawback of this requirement centers on the implications that stem from youth self-selecting themselves into research. Most adolescent drug research is school-based, with surveys completed in standard classroom settings in public school systems. The voluntary nature of survey participation, coupled with the requirement that youth be in school on the day of survey administration, lends to the exclusion of certain segments of the adolescent population (e.g., school drop-outs, truants, institutionalized youth, and youth attending alternative schools). Research finds that not only are school drop-out and truancy established as risk factors for soft drug initiation, but these segments of the adolescent population also have relatively higher rates of drug use than youth who attend school regularly (Johnston et al., 2007).

History

History pertains to the confounding of results stemming from one or more events that occur at the same time that outcome variables (or predictors) are measured. In the context of adolescent drug use, events that can impact rates of soft drug initiation (or levels of risk and protection) include school-wide or audience-specific drug prevention programming and anti-drug media campaigns (Rouse, Kozel, & Richards, 1985). These influences can lead to Type II errors, since actual rates of soft drug initiation (or levels of risk and protection) may be attenuated, thereby potentially leading to an artificial suppression of the true relationship between predictors and soft drug initiation.

Causal Ordering

In order to make valid inferences about causal relationships, the temporal ordering of variables must be accurate (predictors measured prior to outcome) and alternative explanations for observed relationships should be ruled out (Shadish et al., 2002). Given the complexity of behavior and the underlying interaction between an individual and the environment, rarely is the latter criteria met in adolescent drug research (Schulenberg, Maggs, & O'Malley, 2003). In an effort to account for alternative explanations of observed relationships, characteristics of respondents known to impact soft drug initiation (e.g., gender, race, and age) need to be taken into account in adolescent drug research. Given that the systematic review examined findings from both cross-sectional and longitudinal research, this source of findings were useful in validating the temporal ordering of variables and related findings of the current quantitative research.

External Validity

External validity refers to the degree to which research findings from one particular sample generalize to different conditions, including different time periods, settings, and people (Shadish et al., 2002). Regardless of research design, these conditional factors pose plausible threats to the external validity of findings from all studies examining predictors of adolescent soft drug initiation.

Investigating the degree to which research findings are externally valid is difficult, particularly given the constraints posed by research replication. If feasible, primary researchers can utilize some type of probability sampling frame (e.g., random, cluster, or stratified sampling) prior to data collection to ensure that adolescents who vary on important attributes are included in the sample. In contrast, secondary researchers typically have a limited number of options available for reducing plausible threats to the external validity of findings. One feasible option, however, is to cross-validate findings with those derived from other samples at different points in time (Welsh & Farrington, 2006b).

Cross-Validation Logistics

The stability and generality of the quantitative findings were examined in terms of plausible threats to all four types of validity. More of an emphasis was placed, however, on plausible threats to internal and external validity. The plausible threats to internal validity that were of interest included selection bias, history, and causal ordering. With respect to external validity, the major threat of interest centered on the characteristics of the sample used in the quantitative research.

The systematic review cross-validation constituted a broader and less exhaustive verification of the quantitative findings than the quantitative cross-validation. Due to the fact that the magnitude of predictor effects were not assessed in the systematic review, the systematic review cross-validation primarily will dealt with confirming the findings from two directional (+/-) hypotheses that were tested. These directional hypotheses (H2 and H3) are introduced and discussed in the following chapter.

The characteristics of each primary study (culled during the preparatory stages of the systematic review) were helpful in attempting to explain divergent findings in the directionality and significance of relationships. Since vote-counting was used to organize the systematic review findings according to three different outcomes (i.e., positive significant, negative significant, and non-significant results), referring to these findings (and related studies) constituted a starting point in identifying relevant primary studies whose results conflicted with the quantitative findings from the current research.

Once these primary studies were isolated, background information concerning the sample (e.g., sample size and demographic characteristics), sampling procedures, operationalization of constructs, and many other study features outlined in Table 12 were considered. An attempt was made to identify patterns in the methodology, sampling, and study characteristics of those primary studies whose results countered those from the quantitative research.

Specifically, by comparing differences between relevant primary studies and the quantitative research on several fronts (e.g., sample size and characteristics, research designs, and operationalization of constructs), patterns identified through the course of these comparisons were used in an attempt to explain differences in findings between the quantitative research and the systematic review. Explanations of differences were approached and will be discussed within the context of validity, with links made between differential findings and relevant plausible threats to statistical conclusion, construct, internal, and external validity.

Conclusion

This chapter introduced and described the systematic, comprehensive review and systematic review cross-validation, one of two cross-validations that were employed in the current research. The systematic review used various inclusion criteria in seeking to address numerous gaps in the body of literature on predictors of adolescent soft drug initiation.

In order to assess the validity of the quantitative findings, a dual cross-validation scheme was employed. The systematic review cross-validation constituted a broader and less exhaustive confirmation of the quantitative findings, although major threats to all four types of validity were assessed. This particular cross-validation centered on verifying the quantitative results stemming from tests of two directional hypotheses. The use of three outcomes (positive significant, negative significant, and non-significant) in the systematic review, coupled with the vote-count method, enabled the identification of divergent findings. Once conflicting findings were linked to primary studies, further examination of study characteristics (e.g., sampling, measurement, and time period of data collection) revealed and spoke to alternative explanations for the findings in the quantitative research.

The next chapter turns to the quantitative component of the current research. The research hypotheses and methodology and analytic techniques are detailed. In the end, attention is directed at the analysis plan underlying the quantitative cross-validation.

CHAPTER 6

QUANTITATIVE METHODS

The quantitative component of the current research used secondary data in conducting a comprehensive examination of soft drug initiation among youth. This research was grounded in three major areas of inquiry. The first area constituted testing a modified version of Kandel's (2002) stage theory, while the second line examined the utility of psychosocial factors for predicting soft drug initiation. The final area of the study used these same predictors in assessing determinants of time to soft drug initiation.

Several major aspects of the study will be discussed in this chapter. Attention first is directed toward the survey instrument from which secondary data were derived. The survey administration procedure, data set development, and human subject issues are discussed. Next, research questions are formally presented, along with the related hypotheses and outcome variables of interest. The independent and control variables then are described, with coding schemes and major recoding issues highlighted. The chapter concludes with the major statistical techniques that were used in testing the hypotheses, the logistics that founded model development and analyses, and a discussion of the quantitative cross-validation.

Secondary Data Source and Sample Attributes

Survey Instrument and Data Set Development

In the spring of 2004, 6th, 9th, and 12th grade students from a rural school district in western Pennsylvania completed the 2004 Primary Prevention

Awareness, Attitude, and Use Survey (PPAAUS; see Appendix A), a 98-item, machine-readable questionnaire. The survey asked a variety of questions related to individual problem behaviors, including soft and hard drug use, fighting, bullying, cheating, and skipping school. Other questions centered on issues related to academic performance, involvement in extracurricular activities, school safety, delinquency victimization, degree of involvement with drug-using peers, and perceived peer problem behaviors. Data from the 2004 PPAAUS constituted the secondary data that were used in the quantitative component of the research.

Funded by the Safe and Drug-Free Schools and Communities Act, the PPAAUS is a triennial survey that has been administered to 6th, 9th, and 12th grade students in the school district since 1995 (J.S. White Surveys, 2004). Developed with input from the school district, and informed by the Communities that Care ® Youth Survey (Arthur et al., 2002), the purpose of the PPAAUS is to track trends in behaviors and attitudes. An independent research company located in State College, Pennsylvania, J. S. White Surveys, was responsible for scanning, maintaining, and initially analyzing 2004 PPAAUS data for the school district (J. S. White Surveys, 2004).

Administration Procedures

Surveys were administered in standard classroom settings, with students marking their responses directly on the questionnaires. Teachers were provided written instructions and a script (see Appendix B), as well as envelopes for survey collection. Instructed to remain at their desks during completion of the survey, teachers delegated a student to collect the completed questionnaires, place them in an envelope, and take them to a designated collection area within the school (J. S. White Surveys, 2004).

Student participation was gained through a passive consent procedure. Both students and their parents were notified by letter prior to survey administration that the school district would be asking students for voluntary participation in the survey. Parents were asked to notify the school district in advance if they did not want their child to participate in the survey. Through both written and verbal instructions, all 6th, 9th, and 12th grade students present on the day of survey administration were again informed that survey participation was voluntary (J. S. White Surveys, 2004). Specifically, students were informed that they could choose not to participate in the survey, and they could leave blank any item or section without any adverse consequences. To guarantee confidentiality and response anonymity, students were instructed not to include any personal information or identifying marks on their questionnaires.

Questionable Response Filtering

The school district mailed all completed surveys to J. S. White Surveys, who scanned them, created the 2004 PPAAUS codebook, developed the SPSS data set, ran descriptive statistics for the school district, and placed the surveys in onsite storage. Before the SPSS data set was created, however, J. S. White Surveys employed a questionable response (QR) filtering technique to identify inconsistent and questionable survey responses, and generally ensure the quality of students' responses. QR filtering is designed to identify respondents who may have been exaggerating or careless in their responses (J. S. White

Surveys, 2004). Surveys that contained questionable or inconsistent responses were excluded from the data set.

As a result of QR filtering, survey data from 13 respondents were excluded from the data set. Subject to automatic exclusion were respondents who reported using “amactin” (a fictitious drug), or those who reported they were in a school grade that was not surveyed (e.g., 8th grade). Surveys not excluded according to these criterion were subject to additional QR filtering. Specifically, five response inconsistencies were assessed, with one QR point assigned for each inconsistency (J. S. White Surveys, 2004). Survey data from respondents who were assigned two or more QR points were eliminated from the data set. The five inconsistencies assessed included: (1) reporting a higher frequency of being drunk than claiming to drink alcohol; (2) reporting cigarette abstention and claiming to have smoked several cigarettes per day; (3) reporting marijuana abstention and claiming to have smoked marijuana several times within the past 30 days; (4) reporting abstention from fighting and claiming to have been in more than one school fight within the past year; and (5) reporting a higher frequency of driving after drinking alcohol (or smoking marijuana) than claiming to drink (or smoke marijuana).

Sample and Setting

Mentioned earlier, 2004 PPAAUS data for this study were derived from a district-wide sample of 6th, 9th, and 12th grade students in the spring of 2004. The school system serves rural youth from two boroughs and two surrounding rural townships. Within this school system, 6th grade students attend one of four elementary schools, 9th grade students attend one junior high school, and 12th grade students attend one senior high school. A total of 766 students completed the 2004 PPAAUS. Of these respondents, responses from 13 surveys were excluded according to QR filtering. A total of 753 students provided usable data and constituted the sample for the current research.

The majority of these students were white (86%) and they were fairly evenly distributed across gender (52% male), grade-level, and adolescent stage of development. Specifically, 6th, 9th, and 12th grade students each comprised roughly one-third of the sample, with 6th grade students (n = 281) constituting 37% of the sample, 9th grade students (n = 238) comprising 32% of the sample, and 12th grade students (n = 234) constituting 31% of the sample. Students ranged in age from 11-19 years, with 11-13 year olds, 14-16 year olds, and 17-19 year olds each comprising about one-third of the sample (37%, 32%, 31%, respectively).

In terms of 6th, 9th, and 12th grade response rates, school district enrollment records indicate that 291 students were enrolled in 6th grade, 287 students were registered for 9th grade, and 280 students were enrolled in 12th grade during the 2003-2004 school year (Hruska, 2004a, 2004b). Considering these enrollment numbers in conjunction with the number of 6th, 9th, and 12th grade students who provided useable data, the 2004 PPAAUS response rate for 6th, 9th, and 12th grade students (after QR filtering) was 97%, 83%, and 84%, respectively, while the total response rate (after QR filtering) for the 2004 PPAAUS was 88%.

The school district is located in a medium-size, northeastern U.S. college town and serves a population of approximately 32,000 persons (The United Way of Pennsylvania [UWP], 2006). As the seat of county government, the community's economic base is primarily industrial, manufacturing, and service in nature, with wholesale and retail trade sectors constituting 22% of employment. A medium-size university serves as the community's largest employer. The town also serves as a "bedroom community" for a major city (Pittsburgh) within relatively short commuting distance (UWP, 2006).

The county in which the sample was drawn may be characterized as follows. The majority of county residents are white (97%), between 18 and 64 years of age (66%), employed (5.5% unemployment rate), and conservative (City-Data.com, 2007). A sizable number of county residents attend church (177 congregations). Less than 25% of households with children are single-parent families; the majority (78%) of these single-parent families are female-headed. In 2003, the median household income was estimated at $32,443; the estimated county poverty rate was 14.2% (with a 7% food stamp program participation rate); and the juvenile poverty rate was estimated at 19.8%.

Although there are two private schools located within the school district providing kindergarten-6th grade education, relatively few children attend these schools (230 total youth in 2006), as the majority of youth are enrolled in the public school system (City-Data.com, 2007). Compared to 33% nationally, roughly 25% of youth who attend schools within the district are eligible for free or reduced lunches (Pennsylvania Department of Education, 2007). The county school drop-out rate has been low in recent years (1.2%), with the majority of 2006 high school graduates (80%) self-reporting plans to continue some form of post-secondary education. Among adult county residents, roughly 45% report not ever having attended college, and 20% report not graduating from high-school (Indiana Area School District, 2007; UWP, 2006).

Human Subject Protections

There were no serious human subject issues related to any component of the current research. The systematic, comprehensive review involved examining empirical studies that are available to the public through electronic databases and hard-bound journals. The quantitative component of the research constituted examining secondary data, with survey administration and data collection having been previously completed. No component of the current study required that the researcher interact with any of the survey respondents, any students who declined participation, or any individual associated with primary data collection.

The quantitative component of the study required the examination of data contained in two existing data sets. One data set included responses to the 2004 PPAAUS, while the other data set included responses to the 2001 PPAAUS. These data sets were stored on the researcher's personal computer, which was located in a secure home environment. These data sets contain no identifying information about survey respondents. In fact, during primary data collection, students complied with the request for anonymity and did not provide any identifying information on the survey instruments (J. S. White Surveys, 2004).

Since the focus of research is on aggregate results, not individual results or intraindividual changes in predictors over time, data were analyzed and reported in the aggregate.

Research Questions and Hypotheses

The quantitative component of the current research was founded upon three basic research questions. Discussed and referred to throughout the previous chapters, these areas of inquiry are formally introduced in Table 18. Also provided are the nine alternative hypotheses that were tested and the related analytic techniques that were utilized. What follows is a discussion of the theoretical and empirical rationale underlying these hypotheses and the operationalization of dependent variables.

Research Question #1

The first area of the quantitative research involved testing a modified version of Kandel's (2002) stage sequencing hypothesis. Expressed as H1, alcohol, cigarette, and marijuana initiation were examined in terms of the degree to which the initiation of these drugs (together) represent a cumulative, hierarchical, and latent continuum of drug involvement that begins with alcohol initiation, proceeds to cigarette initiation, and ends with marijuana initiation.

Discussed in Chapter 3, Kandel (2002) posits that drug use is a developmental phenomenon comprised of three discrete stages. Involvement begins with the most socially acceptable drugs, alcohol or cigarettes (legal drug use), proceeds to marijuana use (Stage 2) and finally to the least socially acceptable drugs, other illegal, hard drugs (Stage 3). This three-stage drug

Table 18.

Overview of Research Questions, Attendant Hypotheses, and Analytic Techniques

Research Questions		Alternative Hypotheses	Analytic Technique
RQ#1: *Is involvement in soft drug use a sequential and hierarchical phenomena? If it is, what is the typical sequence?*	H_a1:	Among youth who initiate soft drug use, the most common hierarchical and cumulative pattern of initiation is one in which alcohol initiation occurs prior to cigarette initiation, and cigarette initiation occurs prior to marijuana initiation.	Guttman Scalogram Analysis
RQ#2: *What factors predict soft drug initiation and age of initiation among adolescents?*	H_a2:	Predictors conceptualized as risk factors increase risk for alcohol, cigarette, and marijuana initiation and lower age of initiation.	Binary Logistic Regression Survival Analysis (Total Sample Models)
	H_a3:	Predictors conceptualized as protective factors decrease risk for alcohol, cigarette, and marijuana initiation and delay age of initiation.	Binary Logistic Regression Survival Analysis (Total Sample Models)
	H_a4:	Relative to other ecological domains, peer domain predictors explain the largest proportion of variance in alcohol and cigarette initiation and age of initiation.	Binary Logistic Regression Survival Analysis (Total Sample Models)
	H_a5:	Relative to other ecological domains, individual domain predictors explain the largest proportion of variance in marijuana initiation and age of initiation.	Binary Logistic Regression Survival Analysis (Total Sample Models)

(Table 18 continues)

(Table 18 continued)

Research Questions		Alternative Hypotheses	Analytic Technique
	H_a6:	Relative to alcohol initiation, cigarette initiation is a stronger predictor of marijuana initiation and age of initiation.	Binary Logistic Regression Survival Analysis (Total Sample Models)
RQ#3: *Since the initiation of soft drug use appears to vary, in part, as a function of biological age, what factors predict soft drug initiation and age of initiation among youth at different stages of adolescence?*	H_a7:	Community domain predictors explain a larger proportion of variance in alcohol, cigarette, and marijuana initiation and age of initiation among 12th grade students as compared to alcohol and cigarette initiation and age of initiation among 6th and 9th grade students, and marijuana initiation and age of initiation among 9th grade students.	Binary Logistic Regression Survival Analysis (Age-Graded Models)
	H_a8:	Parental pro-drug norms is a stronger predictor of alcohol and cigarette initiation and age of initiation among 6th grade students as compared to 9th or 12th grade students.	Binary Logistic Regression Survival Analysis (Age-Graded Models)
	H_a9:	Peer drug use and pro-drug norms are stronger predictors of alcohol, cigarette, and marijuana initiation and age of initiation among 9th grade students as compared to alcohol and cigarette initiation and age of initiation among 6th and 12th grade students, and marijuana initiation and age of initiation among 12th grade students.	Binary Logistic Regression Survival Analysis (Age-Graded Models)

sequence is hypothesized to be hierarchical in nature, with progression experienced by successively smaller numbers of people. Higher-order stages (e.g., Stages 2 and 3) are not attained unless individuals have used drugs (e.g., legal drugs) at lower stages in the drug sequence. There are two major aspects of Kandel's (2002) stage theory that require investigation: sequencing in drug use and association between drugs. The focus of the initial aspect of the proposed research centers on testing Kandel's (2002) sequencing proposition.

As revealed in Chapter 4, there is considerable empirical evidence to suggest that involvement in drug use is a time-ordered, cumulative, and hierarchical phenomenon. Longitudinal, prospective research (see, e.g. Andrews et al., 1991; Ellickson et al., 1992; Hawkins et al., 2002b), as well as cross-sectional, retrospective research (see, e.g. Costello et al., 1999; Federman et al., 1997) supports the features and ordering of drug initiation that Kandel (2002) proposes.

The review of the extant literature revealed two major issues in need of further attention, however. First, there is some empirical ambiguity concerning the role of cigarette initiation in Kandel's drug sequence. Of the 30 studies assessed, 10 did not examine cigarette initiation separately from alcohol initiation (see, e.g. Brook, 1993; Brook et al., 1983; Fleming et al., 1989); three studies did not examine the role of cigarette initiation at all (Donovan & Jessor, 1983; Martin et al., 1996; White et al., 1986); and findings from two cross-sectional studies suggest that cigarette initiation does not constitute a stage (whole or in part) in Kandel's drug sequence (Huba et al., 1981; Gould et al., 1977).

In contrast, four longitudinal, prospective studies (Andrews et al., 1991; Ellickson et al., 1992; Hawkins et al., 2002b; Kandel, 1975b) suggest that alcohol initiation does occur prior to cigarette initiation for most adolescents who initiate legal drug use, and the initiation of these two soft drugs occurs prior to marijuana initiation among those adolescents who initiate marijuana use. Three cross-sectional studies also found this sequence in soft drug initiation (Costello et al., 1999; Federman et al., 1997; Yu & Williford, 1992).

These seven studies suggest that cigarette initiation may not only play an important role in drug involvement, but the initiation of cigarettes also may follow alcohol initiation and occur prior to marijuana use (among those who progress to this stage). Since there is some empirical ambiguity in this area, however, and none of the studies examined utilized data collected in the 21st century, examining the role of cigarette initiation in Kandel's drug sequence appears to be an area ripe for further investigation.

A second issue in need of attention centers on the temporal ordering of drug initiation. The majority of the empirical tests did not incorporate a measure of time (e.g., age of initiation) into the analyses. The element of time clearly is evident in Kandel's (2002) drug sequence hypothesis, with legal drugs hypothesized as being initiated prior to marijuana. Only five of the longitudinal studies examined utilized prospective drug data, with the remaining longitudinal studies collecting drug data at only one point in time without making any effort to establish temporal ordering in initiation. Most of the cross-sectional studies, which used Guttman scalogram analysis, also did not incorporate an element of time into analyses of drug sequencing. Incorporating age of drug initiation data into a Guttman scalogram analysis, the most commonly used method of testing Kandel's (2002) hypothesis, is important for verifying the temporal ordering of soft drug initiation. Briefly discussed in Chapter 4, the temporal ordering inferred by a Guttman scale only implies, but does not necessarily prove, that temporal ordering in the initiation of multiple drugs exists (Kandel, 1980a).

Research Question #2

The second area of the quantitative research examined data from the total sample in determining the independent and combined utility of various psychosocial factors for predicting alcohol, cigarette, and marijuana initiation, as well as time to initiation. Aside from determining the efficacy of all of the psychosocial factors, explicit effort was directed toward understanding whether predictors tend to differ in kind or marked salience by drug type. Discussed in Chapter 4, evidence of the former can be interpreted as providing some support for Kandel's (2002) notion of drug-specific risk factors. In contrast, evidence that most predictors are universal, whereby they significantly predict the initiation of all three soft drugs (although differing in salience), constitutes partial support for the common factor hypothesis (Jessor, 1992).

The broad review of the extant literature, along with insight from stage theory and SDM (as well as the traditional theories that SDM subsumes), guided the development of H2-H6. The overarching purpose in testing H2 and H3 was to empirically verify the risk and protective capacities (i.e., direction of influence) and theoretical conceptualizations that found all of the independent variables.

Research suggests that a plethora of psychosocial factors, at all ecological levels of social life, increase risk for soft drug initiation and lower age of initiation. In support of SDM and social learning theory, exposure to parents and peers who model drug use or espouse prodrug norms; adolescents' own prodrug norms and intentions to use soft drugs; antisocial behavior; and prior (and early) initiation of other soft drugs constitute some of the most salient risk factors (Derzon & Lipsey, 1999a, 1999c; Donovan, 2004).

Although less researched, other factors have been found to serve protective functions in decreasing risk for soft drug initiation and delaying age of initiation. Major protective factors, all of which are associated with SDM (and traditional theories), include prosocial parents and peers who abstain from drug use and espouse antidrug norms; adolescents' antidrug norms and intentions to abstain from drug use; high school performance and attachment to school; and abstention from other soft drugs (Meschke & Patterson, 2003; Swadi, 1999).

Concerning H4, meta-analytic findings from Derzon and Lipsey's (1999a) comprehensive assessment of predictors of cigarette initiation indicate that peer domain predictors explain more variation in cigarette initiation than a variety of predictors emanating from the individual, family, school, and community domains of influence. Although their study did not assess determinants of alcohol initiation or age of cigarette and alcohol initiation, a review of the extant literature indicates that peer-related predictors of legal drug initiation and age of initiation are more similar in kind (and saliency) than those for marijuana initiation (Allen et al., 2003; Glynn, 1981; Swadi, 1999). As well, research consistently finds that peer drug use and prodrug norms exert stronger effects on alcohol initiation and age of initiation than parental drug use, prodrug norms, and adolescent prodrug norms and intentions, although parental influence remains important (Allen et al., 2003; Glynn, 1981). Hence, in proposing H4, it was expected that the peer domain predictors, relative to other ecological domains, would explain a larger proportion of variance in alcohol and cigarette initiation and age of initiation.

In contrast to their findings concerning predictors of cigarette initiation, results from Derzon and Lipsey's (1999c) meta-analysis on predictors of marijuana initiation indicate that individual domain predictors explain a larger proportion of variance in marijuana initiation than peer, family, school, and community domain predictors. Although their research did not assess determinants of age of marijuana initiation, findings from other studies converge in underscoring the utility that prior (and early) initiation of alcohol use, prior (and early) initiation of cigarette use, and prodrug marijuana use norms have for predicting both marijuana initiation and early age of initiation (see, e.g., Andrews et al., 1991; D'Amico & McCarthy, 2006; Duncan et al., 1998; Ellickson et al., 1992; Yamaguchi & Kandel, 1984b). Taking these findings together, it was expected that individual domain predictors, relative to other ecological domains, would explain the largest proportion of variance in both marijuana initiation and age of initiation (H5).

In an attempt to explain progression in Kandel's (2002) well-substantiated drug sequencing hypothesis (legal>marijuana>hard drugs), numerous researchers have examined the association between the initiation of one soft drug and that of another, while controlling for age and a variety of empirically-verified psychosocial predictors. Results from this line of extant research founded the basis of H6. The bulk of the research (see, e.g., Conrad et al., 1992; D'Amico & McCarthy, 2006; Duncan & Duncan, 1994; Duncan et al., 1998; Ellickson et al., 1992; Flay et al., 1998; Kandel et al., 1992; Yamaguchi & Kandel, 1984b) indicates that cigarette initiation, relative to alcohol initiation, is a stronger predictor of marijuana initiation. For example, one five-wave longitudinal study (Duncan et al., 1998) found that of the two legal drugs, only prior initiation of cigarette use was significantly associated with an increased risk for marijuana initiation.

Research Question #3

In addition to examining the utility of predictors for the total sample (holding current biological age constant), further analyses sought to determine whether the effects of factors on soft drug initiation and age of initiation are age-graded. Similar to addressing Research Question #2, a broad assessment of findings was made in determining whether psychosocial predictors tend to differ in kind or marked saliency by stage of adolescent development, both within and across drug type.

Sample Decomposition

In order to determine whether predictors of soft drug initiation and time to initiation differ in kind (or saliency) by stage of adolescent development, the total sample was stratified by school grade, and prediction models were developed for each soft drug among subsamples of 6th, 9th, and 12th grade students. School grade is a commonly used proxy measure of biological age, and more specifically, stage of adolescent development (Dielman, Butchart, & Shope, 1993; Margulies et al, 1977). Sixth grade was conceptualized as early adolescence, 9th grade as middle adolescence, and 12th grade served as late adolescence.

Age decomposition was conducted using students' responses to a PPAAUS item that asked students to notate their current grade-level in school (see Appendix A). Possible response options ranged from 5th-12th grade. Discussed earlier, respondents who notated being in a school grade not actually surveyed (e.g., 5th grade) were excluded from the data set.

Hypotheses #2-#6

H2-H6 not only were tested with soft drug initiation and time to initiation data from the total sample (under Research Question #2), but these hypotheses also were tested with age-graded soft drug initiation and time to initiation data from three subsamples of youth (i.e., 6th, 9th, and 12th grade students). The overarching purpose in testing these hypotheses with age-graded data was to determine whether research expectations hold for each of the three major stages of adolescent development. Discussed in Chapter 4, far less research has examined the age-graded nature of relationships between risk and protective factors and soft drug initiation than has assessed determinants of initiation with data from samples of youth who vary on age. In fact, other than expressing the need for further research, few of the literature reviews examined were able to offer substantive direction, insight, or expectations that could be used to found the basis of age-graded research hypotheses. Hence, testing these hypotheses with age-graded data constituted an attempt to fill this glaring gap in the literature.

Discussed in Chapter 4, past research is clear in establishing that risk factors increase the likelihood of subsequent soft drug initiation and lower age of initiation (H2), while protective factors decrease the likelihood of subsequent soft drug initiation and delay age of initiation (H3). Although the magnitude of effects posed by major predictors tend to vary by age (Conrad et al., 1992; Donovan, 2004 ; Petraitis et al., 1998), the direction of influence does not. Hence, in testing H2 and H3 with age-graded data, it was expected that no age-factor interaction effects would exist with respect to the direction of predictors in influencing soft drug initiation or age of initiation.

Discussed previously, research conducted with total sample data (controlling for age effects) indicates that peer domain predictors explain a larger proportion of variance in alcohol and cigarette initiation (and age of initiation) than variables from other ecological domains of influence (H4). As well, there is evidence to suggest that relative to other ecological domains, individual domain predictors explain the largest proportion of variation in marijuana initiation and age of initiation (H5). Aside from testing these hypotheses with data from the total sample, H4 and H5 also were tested with age-graded data. These tests should be viewed as more exploratory in nature than grounded in past empirical findings. Due to the lack of age-graded studies that speak to these hypotheses, the overarching goal was to determine the degree to which H4 and H5 hold for the three stages of adolescent development.

Discussed above, H6 deals with the stronger risk effects on marijuana initiation and age of initiation posed by cigarette initiation (versus alcohol initiation). A broad review of the research was not able to identify any studies that examined the degree to which this expectation varies by biological age, grade-level, or stage of adolescent development. In fact, all of the studies that support H6 are based upon prediction models in which the effects of biological age were held constant (see, e.g., D'Amico & McCarthy, 2006; Duncan & Duncan, 1994; Duncan et al., 1998; Ellickson et al., 1992; Flay et al., 1998; Kandel et al., 1992; Yamaguchi & Kandel, 1984b). Hence, without empirical research to inform otherwise, there is no basis for anticipating that H6 will vary by biological age.

Hypotheses #7-#9

Little research has examined the age-graded nature of community domain risk factors for increasing the likelihood of soft drug initiation among youth. Hawkins (Catalano & Hawkins, 1996) hypothesized that community-level risk factors are more influential in shaping the behavior of older youth than younger adolescents. Providing indirect support for this hypothesis, Werner (1994) found that sources of social support found in the community (e.g., neighbors and clergy) decreased the risk for soft and hard drug initiation among older adolescents, while this type of protection had relatively little impact on the drug abstaining behavior of younger adolescents. H7 is based upon Werner's results concerning community-level protective factors and the fact that little research has tested Hawkins' community influence hypothesis with soft drug initiation data.

Discussed in Chapter 4, the most sweeping age-graded changes in the saliency of risk factors center on the effects of parent and peer modeling and normative standards. In contrast to peer influence, which generally increases from early adolescence to just prior to late adolescence (Allen et al., 2003; Glynn, 1981), the influence of parents on soft drug initiation takes on a curvilinear pattern when plotted across adolescent age. Research finds that the impact of parental influence on soft drug initiation is strongest during early adolescence (Biddle et al., 1980), weakest during mid-adolescence, and possibly as strong (or stronger) during late as opposed to early adolescence (Huba & Bentler, 1980; Kandel & Wu, 1995). Based on these findings, H8 was proposed with the expectation that parental prodrug norms would exert stronger risk effects on alcohol and cigarette initiation and age of initiation among 6th grade students than among 9th or 12th grade students.

Finally, there is a general consensus in the literature concerning the age-graded saliency of peer influence on the soft drug initiation behavior of youth. Peer influence increases in magnitude as youth navigate through adolescence (Allen et al., 2003; Glynn, 1981). By the time adolescents reach mid-adolescence, peers play a more important role than parents in shaping drug use attitudes, beliefs, and soft drug initiation behavior (Beal et al., 2001; Donovan, 2004; Li et al., 2002b). The literature clearly identifies mid-adolescence as a time period in which the impact of peers on soft drug initiation is strongest, particularly the risk effects posed by peer drug use (see, e.g., Donovan, 2004; Urberg et al, 1991). This line of research founds the basis of H9. Specifically, it was anticipated that both peer drug use and prodrug norms would have stronger impacts on alcohol, cigarette, and marijuana initiation and age of initiation among 9th grade students (middle adolescents) than 6th grade students (early adolescents) or 12th grade students (late adolescents).

Dependent Variables

Three types of dependent variables were utilized in addressing Research Questions #1-#3. Whereas *age of initiation* was used in attending to Research Question #1 (H1), the dependent variables *initiation* and *time to initiation*, which were used to address Research Question #2 (H2-H7), also were employed in addressing Research Question #3 (H2-H9). These measures are discussed below.

Age of Initiation

In testing H1, *age of initiation* for alcohol, cigarette, and marijuana use was utilized. Specifically, continuous data for *age of initiation* were derived from an item posed in the 2004 PPAAUS, which stated: "If you have ever used alcohol, cigarettes, or marijuana, mark the age at which you first used it" (see Appendix A). Aside from "never used," students were able to notate a biological age ranging from 8-18 years for each of the three soft drugs.

Initiation

In examining initiation in terms of its' occurrence (H2-H9), a dichotomous dependent variable, *initiation*, was developed for each drug (alcohol, cigarettes, and marijuana). In order to obtain these dichotomous initiation variables, continuous age of initiation data for alcohol, cigarettes, and marijuana were recoded into three distinct dependent variables. The same survey items used to operationalize age of initiation (discussed above) were used in developing these dichotomous variables. Respondents who notated an age of initiation for a given soft drug were ascribed a “1” (initiation) for that particular drug, while those who notated that they “never used” the given soft drug were coded a “0” (abstention).

Time to Initiation

In order to predict *time to initiation* for alcohol, cigarette, and marijuana use (H2-H9), a two-part dependent variable was developed for each soft drug. *Time to initiation* takes into account *initiation status*, which refers to whether initiation for the given soft drug did occur, and *age of initiation*, which constitutes the biological age at which initiation of the given soft drug occurred. The first part of each dependent variable, *initiation status*, constitutes a dichotomy. Respondents who notated an age of initiation for a given soft drug were coded as “1” (initiation) for that respective drug, while those who “never used” the given soft drug were ascribed a “0” (abstention).

The second component of each dependent variable, *age of initiation*, constitutes the same continuous variable that was used in testing H1 (the modified version of Kandel’s drug sequencing hypothesis). Aside from “never used,” students were able to notate a biological age ranging from 8-18 years for each soft drug. For this part of each dependent variable, students who provided an age of initiation were coded accordingly. For example, a student who reported initiating marijuana use at age 14 was coded as “14” for this age of marijuana initiation. For analytical purposes, students who reported never using the soft drug under consideration were coded according to their biological age at the time of survey administration. For example, a 13 year-old student who reported never using marijuana was coded as “13,” but received a “0” for the initiation status variable to indicate abstention from that drug.

Discussed later when the analytic plan for this set of analyses is presented, *initiation status* and *age of initiation* were considered together in calculating the aggregate risk that predictors posed for initiating a given drug at an earlier age. Hence, although students who reported abstaining from a particular drug were coded according to their respective biological age for the *age of initiation* component of the dependent variable, their *initiation status* (i.e., 0 = abstention) also was considered simultaneously.

Independent and Control Variables

There are a host of psychosocial factors included in the 2004 PPAAUS that were available for study. The broad review of risk and protective factors for soft drug initiation (provided in Chapter 4) informed the 38 predictors chosen for examination. Since H1 did not require the use of any extraneous variables (only initiation outcome data), predictors only were used in estimating the likelihood that alcohol, cigarette, and marijuana initiation occurs and the relative risk of initiating at early versus later biological ages (H2-H9).

Appendix C contains the coding schemes for all of the extraneous variables that were examined. Explained in detail when the analytic plan is introduced, predictors were entered into regression equations in clusters (i.e., blocks), according to the ecological domain from which they emanate. The following discussion examines each block of predictors in turn.

Block #1: Community Domain

The predictive value of six community-level psychosocial factors was assessed (see Table 19). Given past research, which suggests that these influences increase risk for soft drug initiation, all were conceptualized as risk factors.

Table 19.

Prediction Analyses: Community Domain Predictors, Theoretical/Empirical Grounding

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
MessMedAlc-- Media alcohol tolerance	SDM SLT	Prodrug reinforcement (RF)	Hastings et al. (2005)
MessMedCig-- Media cigarette tolerance	SDM SLT	Prodrug reinforcement (RF)	Wakefield et al. (2003)
MessMedMar-- Media marijuana tolerance	SDM SLT	Prodrug reinforcement (RF)	SDM
EasyAlc-- Community alcohol availability	SDT	Drug availability (RF)	Donovan (2004)
EasyCig-- Community cigarette availability	SDT	Drug availability (RF)	Swadi (1999)
EasyMar-- Community marijuana availability	SDT	Drug availability (RF)	Swadi (1999)

[a] SDM = Social Development Model; SLT = Social Learning Theory; SDT = Social Disorganization Theory.
[b] RF = Risk Factor.

Perceived media tolerance of alcohol use (*MessMedAlc*), cigarette use (*MessMedCig*), and marijuana use (*MessMedMar*), all dichotomous measures, represent the social learning and SDM constructs of prodrug reinforcement. Three proxy indicators of soft drug availability also were investigated: the perceived ease in obtaining alcohol (*EasyAlc*), cigarettes (*EasyCig*), and marijuana (*EasyMar*). Although these three predictors were not direct measures of the degree to which given soft drugs are available in the community, it can be argued that the ease with which drugs are secured is dependent (in part) on the degree to which they are available (Jessor, 1981). Hence, operating according to the assumption that ease in obtainment and availability are highly correlated, these variables were designed to capture (in part) the degree to which these soft drugs are available.

Block #2: School Domain

Drawing upon SDM, traditional theory, as well as the extant research, the utility of nine school-related predictors was assessed (see Table 20). Five predictors (*Truancy*, *Cheat*, *MessTchrAlc*, *MessTchrCig*, and *MessTchrMar*) were viewed as risk factors that increase risk for soft drug initiation and decrease age of initiation. Both *Truancy* and *Cheat* constituted SDM measures (involvement in problem behaviors). Conceptualized as measures of prodrug reinforcement, teachers' tolerance of soft drug use (i.e., *MessTchrAlc*, *MessTchrCig*, and *MessTchrMar*) coalesces with both SDM and social learning theory.

Table 20.

Prediction Analyses: School Domain Predictors, Theoretical/Empirical Grounding

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
Truancy-- Frequency of skipping school	SDM	Involvement problem behavior (RF)	Derzon/Lipsey (1999c) Dewey (1999)
Cheat-- Frequency of cheating on schoolwork	SDM	Involvement problem behavior (RF)	Donovan (2004)
MessTchrAlc-- Teacher alcohol tolerance	SDM SLT	Prodrug reinforcement (RF)	Petraitis et al. (1998)
MessTchrCig-- Teacher cigarette tolerance	SDM SLT	Prodrug reinforcement (RF)	Petraitis et al. (1998)
MessTchrMar-- Teacher marijuana tolerance	SDM SLT	Prodrug reinforcement (RF)	Petraitis et al. (1998)
CllgeAsp-- Educational aspirations	SDM SCT	School commitment (PF)	Derzon/Lipsey (1999c)
AttachSchool-- School attachment (Index)	SDM SCT	School attachment (PF)	Conrad et al. (1992) Derzon/Lipsey (1999c)
Academic-- Extracurricular academic involvement	SDM SCT	SDM prerequisite for school bonding (PF) SCT bonding element(PF)	Conrad et al. (1992) Derzon & Lipsey (1999c)
HiAcadPerf-- High academic performance	SDM SCT	School commitment (PF)	Conrad et al. (1992) Derzon/Lipsey (1999c)

[a] SDM = Social Development Model; SLT = Social Learning Theory; SCT = Social Control Theory.
[b] RF = Risk Factor; PF = Protective Factor.

Four predictors (*CllgeAsp*, *HiAcadPerf*, *Academic*, and *AttachSchool*) were conceptualized as protective factors. All represented theoretical constructs from social control theory and SDM. Specifically, college aspirations (*CllgeAsp*) and high academic performance (*HiAcadPerf*) constituted measures of school commitment, while extracurricular academic involvement (*Academic*) was representative of prosocial involvement (viewed as a prerequisite for bonding in

SDM and an element of social bonding in social control theory). *AttachSchool*, a three item index (range = 3-21, Cronbach's alpha = 0.82) designed to capture attachment to school, consisted of three items: degree to which school is enjoyable, teachers are helpful, and subjects are interesting.

Block #3: Family Domain

Within the family domain of influence, the efficacy of three predictors was evaluated (see Table 21). Based upon SDM and social learning theory's construct of prodrug reinforcement, parental tolerance of alcohol use (*MessParAlc*), cigarette use (*MessParCig*), and marijuana use (*MessParMar*) were conceptualized as risk factors.

Table 21.

Prediction Analyses: Family Domain Predictors, Theoretical/Empirical Grounding

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
MessParAlc-- Parental tolerance of alcohol use	SDM SLT	Prodrug reinforcement (RF)	Donovan (2004)
MessParCig-- Parental tolerance of cigarette use	SDM SLT	Prodrug reinforcement (RF)	Derzon/Lipsey (1999a)
MessParMar-- Parental tolerance of marijuana use	SDM SLT	Prodrug reinforcement (RF)	Derzon/Lipsey (1999c)

[a] SDM = Social Development Model; SLT = Social Learning Theory.
[b] RF = Risk Factor.

Block #4: Peer Domain

Six peer influence factors also were investigated (see Table 22). Taking SDM, social learning theory, and findings from past research into account, all were viewed as risk factors. In short, these variables represented two theoretical constructs: behavioral modeling (*PeerAlc*, *PeerCig*, and *PeerMar*) and prodrug reinforcement (*MessPeerAlc*, *MessPeerCig*, and *MessPeerMar*).

Table 22.

Prediction Analyses: Peer Domain Predictors, Theoretical/Empirical Grounding

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
PeerAlc-- Peer alcohol use	SDM SLT	Behavior models/imitation (RF)	Donovan (2004)
PeerCig-- Peer cigarette use	SDM SLT	Behavior models/imitation (RF)	Conrad et al. (1992)
PeerMar-- Peer marijuana use	SDM SLT	Behavior models/imitation (RF)	Derzon/Lipsey (1999c)
MessPeerAlc-- Peer alcohol tolerance	SDM SLT	Prodrug reinforcement (RF)	Donovan (2004)
MessPeerCig-- Peer cigarette tolerance	SDM SLT	Prodrug reinforcement (RF)	Conrad et al. (1992)
MessPeerMar-- Peer marijuana tolerance	SDM SLT	Prodrug reinforcement (RF)	Derzon/Lipsey (1999c)

[a] SDM = Social Development Model; SLT = Social Learning Theory.
[b] RF = Risk Factor.

Block #5: Individual Domain

The current research also examined the predictive value of 14 variables from the individual domain (Table 23). Touched upon later in this chapter (and detailed in Chapter 9, Multivariate Results), some of these extraneous variables only were investigated for particular age-graded subsamples. Ten predictors were conceptualized as risk factors (i.e., *AlcInit*, *AlcEarly*, *AlcLate*, *CigEarly*, *CigLate*, *AlcIntent*, *CigIntent*, *MarIntent*, *Steal*, and *ViolBeh*), while four were viewed as protective factors (i.e., *Sports*, *Work*, *Social*, and *Religious*). Early initiates (i.e., *AlcEarly* and *CigEarly*) were operationalized as students who self-reported initiating alcohol or cigarettes between eight and fifteen years of age, while respondents who self-reported initiating alcohol (*AlcLate*) or cigarettes (*CigLate*) between 16 and 18 years of age were considered late initiates. All individual domain predictors represented theoretical constructs outlined in one or more of the major traditional and developmental theories that were discussed in Chapters 2 and 3 (i.e., SDM, stage theory, social learning theory, and social control theory).

Table 23.

Prediction Analyses: Individual Domain Predictors, Theoretical/Empirical Grounding

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
AlcInit-- Alcohol initiation	SDM ST	RF for initiation of cigarettes and marijuana	Derzon/Lipsey (1999a)
AlcEarly-- Early age of alcohol initiation	SDM ST	RF for initiation of cigarettes and marijuana	Derzon/Lipsey (1999a)
AlcLate-- Late age of alcohol initiation	SDM	RF for initiation of cigarettes and marijuana	Derzon/Lipsey (1999a)

(Table 23 continues)

(Table 23 continued)

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
CigEarly-- Early age of cigarette initiation	SDM ST	RF for initiation of alcohol and marijuana	Donovan (2004)
CigLate-- Late age of cigarette initiation	SDM	RF for initiation of alcohol and marijuana	Donovan (2004)
AlcIntent-- Alcohol use intention	SDM SLT	Perceived rewards from antisocial behavior (RF)	Donovan (2004)
CigIntent-- Cigarette use intention	SDM SLT	Perceived rewards from antisocial behavior (RF)	Conrad et al. (1992)
MarIntent-- Marijuana use intention	SDM SLT	Perceived rewards from antisocial behavior (RF)	Derzon/Lipsey (1999a)
Steal-- Frequency of stealing	SDM	Involvement problem behaviors (RF)	Donovan (2004)
ViolBeh-- Frequency of actual/attempted violent behavior	SDM	Involvement problem behaviors (RF)	Donovan (2004)
Sports-- Frequency of sports activities	SDM SCT	SDM prereq. for prosocial bonding, SCT bonding element (PF)	Donovan (2004)
Work-- Frequency of work-for-pay	SDM SCT	SDM prereq. for prosocial bonding, SCT bonding element (PF)	Donovan (2004)
Social-- Frequency of social activities	SDM SCT	SDM prereq. for prosocial bonding, SCT bonding element (PF)	Donovan (2004)
Religious-- Frequency of religious activities	SDM SCT SLT	Prosocial beliefs (PF)	Donovan (2004)

[a] SDM = Social Development Model; ST = Stage Theory; SLT = Social Learning Theory; SCT = Social Control Theory.
[b] RF = Risk Factor; PF = Protective Factor.

Block #6: Control Variables

Depicted in Table 24, five variables constituted covariates in various prediction models: *Gender*, *Race*, *Age*, and lifetime frequency of alcohol (*AlcFreq*) and cigarette (*CigFreq*) use. Discussed earlier (see Chapter 1 or Chapter 4), controlling for variation in gender, race, and age was warranted, given the considerable amount of research that suggests rates of soft drug initiation vary along these demographic lines. While the impact of gender and race on outcome measures was held constant in all prediction models, the nature of the analyses determined when biological age effects were taken into account. Age constituted a control variable in total sample models, but was not be entered as a covariate in age-specific models. Explained earlier, the total sample was partitioned by school grade-level for these models.

Table 24.

Prediction Analyses: Control Variables and Theoretical Constructs

Predictor	Relevant Theory [a]	Theoretical Construct and Predictor Type [b]	Empirical Support
Gender	---	RF/PF	Johnston et al. (2007)
Race	---	RF/PF	Johnston et al. (2007)
Age [c]	---	RF	Johnston et al. (2007)
AlcFreq [c] -- Frequency of alcohol use (Index)	SDM ST	RF for initiation of other drugs	Donovan (2004)
CigFreq [c] -- Frequency of cigarette use	SDM ST	RF for initiation of other drugs	Conrad et al. (1992)

[a] SDM = Social Development Model; ST = Stage Theory.
[b] RF = Risk Factor; PF = Protective Factor.
[c] Control variable in select models.
--- not applicable.

Both Kandel's (2002) stage theory and extant research (see Table 24) suggest that the extent to which youth are involved in drug use has an impact on the risk for initiating another soft drug. In an effort to diminish differences between alcohol initiates and abstainers, variation in the frequency of alcohol use (*AlcFreq*) was taken into account. As a four-item index that measured lifetime frequency of beer, wine, coolers, and hard liquor consumption, *AlcFreq* ranged from 0-13 (Cronbach's alpha = 0.88). To reduce differences between cigarette initiates and abstainers, the frequency of cigarette use (*CigFreq*) also was taken into account.

Analytic Techniques

In totality, 22 prediction models were developed to test hypotheses and address the three research questions posed. H1 only concerned the status and temporal ordering of alcohol, cigarette, and marijuana initiation among adolescents (not the effects of independent variables), so the prediction model (i.e., scale) that was developed to test this hypothesis did not require the use or examination of extraneous variables. With an upwards of 40 predictors eligible for examination, multivariate analyses was used in addressing Research Questions #2 and #3 (H2-H9). Through the use of multivariate analytic techniques, the effects of one or more predictors on outcome measures can be evaluated while controlling for the impact of other predictors (Weisburd & Britt, 2003). The following discussion describes each analytical line of inquiry: sequencing, initiation, and time to soft drug initiation.

Sequencing in Initiation

Guttman scalogram analysis was used to test H1 (the modified version of Kandel's drug sequencing hypothesis). Briefly discussed in Chapter 4, Guttman scaling is a suitable technique for examining the cumulation and hierarchical properties of drug involvement, since both constitute two assumptions that underlie this scaling technique. In addition to testing this hypothesis, a supplemental Guttman scale also was developed and analyzed.

In contrast to accounting for temporal ordering in soft drug initiation (as was done in testing the hypothesis), the second scale was developed with dichotomous drug initiation data. This coding procedure merely accounted for the initiation of each soft drug, and did not further distinguish initiation patterns in terms of temporal ordering. In the end, the two scales were compared on a set of indices (discussed below) to determine which strategy (i.e., not taking or taking age of initiation into account) provided the best fitting Guttman scale. Since none of the 30 studies reviewed developed these two types of scales and compared them in terms of their relative fit, it was expected that this comparison would be informative, regardless of the findings.

Given the fact that Guttman scaling is used less frequently than other types of analytic techniques (e.g., regression or survival analysis), the following discussion goes into some detail in explicating the assumptions underlying this scale and the coding and error-counting procedures that were employed. Briefly described are the major steps involved in developing and analyzing the scale that was used to test H1. Appendix D presents for a fuller explanation of these steps.

Guttman Scaling

The purpose of Guttman scaling (a.k.a. cumulative scaling or scalogram analysis) is to determine whether scale items (e.g., individual types of drugs) capture progressively higher levels of a unidimensional, latent construct (McIver & Carmines, 1982). Since a latent variable (or construct) cannot be observed or measured in itself, scale items constitute both manifest indicators and varying levels of its existence (McIver & Carmines, 1982). Hence, the latent construct is conceptualized as constituting a continuum.

A Guttman scale may be defined as follows: “For a given population of objects, the multivariate frequency distribution of a universe of attributes will be called a “scale” if it is possible to derive from the distribution a quantitative variable with which to characterize the objects such that each attribute is a simple function of that quantitative variable. Such a quantitative variable is called a scale variable” (Guttman, 1950, p. 64). As this description underscores, a Guttman scale is deterministic in the sense that each “attribute” or scale item constitutes one required component of the latent continuum.

Assumptions. Several assumptions (unidimensionality, hierarchy, and cumulation) underlie Guttman scaling. First, in contrast to Likert or other types of scaling models, a Guttman scale assumes that scale items (together) represent a single dimension of a latent construct (McIver & Carmines, 1982). The degree to which a latent variable exists is evidenced by the scale’s ability to accurately predict responses to all of its component items. In the context of Kandel’s (2002) drug sequencing hypothesis, for example, each type of drug (e.g., alcohol, cigarettes, and marijuana) is assumed to constitute a scale item. Each scale item (or stage) constitutes a required component of the larger, underlying construct of drug involvement.

Second, not only are scale items conceptualized as representing a unidimensional construct, but these items are assumed to differ from each other in terms of magnitude or extremeness. This difference in extremeness, referred to as the assumption of hierarchy, is based on the notion that individuals (and their responses) are not evenly distributed across the latent continuum. With respect to drug involvement, for example, progression along the drug involvement continuum, which only occurs one way, constitutes movement from one stage of less serious drug involvement (e.g., alcohol initiation) to the next higher-stage of drug involvement that is more serious (e.g., marijuana initiation) in nature.

Given the increase in extremeness, progression from one stage to another is experienced by successively smaller numbers of individuals. Applying this notion of hierarchy to Kandel's (2002) drug sequencing hypothesis, more individuals should receive a scale score of one (i.e., indicative of reaching and stopping at the first stage in the drug involvement sequence) than a scale score of two or three. Hence, more individuals should reach (and stop at) the first stage (i.e., first scale item) of drug involvement than reach the last stage in the sequence.

Third, Guttman scaling also assumes cumulation. Respondents who provide an affirmative response to more extreme scale items also should provide affirmative responses to less extreme items. Hence, by summing the number of affirmative responses an individual provides to drug scale items, the resultant scale score signifies the extent to which the individual is involved in drug use.

Typical coding scheme. Traditional Guttman scale tests use a dichotomous coding scheme (0, 1) to signify affirmative (1) and negative (0) responses to scale items. In Guttman tests of Kandel's (2002) drug sequencing hypothesis, a respondent's scale score should indicate two things. First, the score should indicate position in the Guttman scale, which is indicative of the last stage of drug involvement that was reached.

Second, a scale score in a Guttman scale should indicate how many and what types of drugs have been initiated. For example, if a student scores a three on a 3-item drug initiation scale, this should mean that the student responded affirmatively to initiating all three drugs on the scale. In contrast, if a student scores a two on the same scale, this scale score should mean that the only items the student responded to affirmatively are the first and second drugs items on the scale.

Discussed earlier, most Guttman scalogram tests of Kandel's (2002) drug sequencing hypothesis did not incorporate temporal ordering into scaling features. Instead, the typical dichotomous (0,1) coding scheme was employed. Unfortunately, this coding scheme does not provide a way to identify and organize affirmative responses according to the temporal order in which drugs were initiated. For example, in a typical Guttman scalogram analysis using dichotomous response data, a scale score of 2 should mean that the respondent initiated alcohol and cigarette use, but not marijuana use (i.e., 1-1-0). This scale score does not reveal, however, which soft drug was initiated first, alcohol or cigarettes. In order to test H1 and determine the temporal ordering in the initiation of two or more drugs, a modified coding scheme had to be employed.

Scale Development and Analysis

Soft drug measures. Discussed earlier, data on soft drug initiation were derived from a set of questions included in the 2004 PPAAUS that asked respondents to indicate the biological age at which they initiated alcohol, cigarettes, and marijuana. Following common convention (see Clogg & Sawyer, 1981; Ellickson et al., 1992), scalogram analysis was restricted to students who provided useable biological age of initiation data for all three soft drugs. In other words, analysis was restricted to students who provided an age of initiation for each soft drug or who notated that they had not yet initiated a given drug. Soft drug initiation data for students who have missing data on one or more items was excluded. It was expected that almost all students provided usable data, although the limited generality of results stemming from this restricted analysis is acknowledged. Since analyses was restricted to students with complete data, inferences drawn about the unidimensionality and cumulation of the drug sequence pertain only to the scalability of these drug items in student populations comprised of respondents who provided usable data for all three soft drug items (Ellickson et al. 1992).

Coding for the hypothesis test. Respondents' drug initiation data was subjected to two types of independent coding procedures. The first procedure pertained to testing H1, while the second procedure was used in the supplemental analysis. Concerning the former, support for H1 equates to a 1-2-3 response pattern, with alcohol initiation occurring prior (i.e., at a younger age) to cigarette initiation, and cigarette initiation occurring prior in time to marijuana initiation. Three drug initiation sequences (1-2-3, 1-2-0, 1-0-0) actually support the hypothesized pattern, since hierarchy in the data is assumed. The response patterns for cases #32, #87, and #532 (in Table 25) illustrate these three drug initiation sequences.

In order to identify the temporal ordering in soft drug initiation, responses to the three PPAAUS questions (discussed above) were coded such that the number of soft drugs and order of initiation were both taken into account. Any student who reported abstention from a given drug was coded as “0” for that given drug. Hence, all soft drug abstainers had the same response pattern (0-0-0). Students who reported initiating one soft drug only were coded as “1” for that particular drug. This code signifies that the particular drug was the first to be initiated. For example, case #7 (Table 25) presents initiation for marijuana use only, which results in a response pattern of 0-0-1.

Among respondents who notated an age of initiation for two soft drugs, the ages at which these drugs were initiated were taken into account in coding their response patterns. A “1” was used to signify the drug that was initiated first and a “2” to signify the drug that was initiated second. For example, case #41 (Table 25) provided an age for alcohol (15 years) and marijuana (16 years) initiation only (not shown in table), resulting in a response pattern of 1-0-2. In contrast, case #24 provided an age of cigarette (13 years) and marijuana (15 years) initiation, thereby resulting in a response pattern of 0-1-2.

Table 25.

Example of Guttman Response Matrix that Incorporates Age of Drug Initiation

Case No.	(Scale Item #1) Alcohol Initiation	(Scale Item #2) Cigarette Initiation	(Scale Item #3) Marijuana Initiation	Scale Score	Error
32	1	2	3	3	0
120	1	**3**	**2**	3	2
87	1	2	0	2	0
41	1	**0**	**2**	2	2
3	**0**	**1**	**2**	2	3
24	**0**	**1**	**2**	2	3
532	1	0	0	1	0
7	**0**	0	**1**	1	2
610	0	0	0	0	0

Note. As a modified approach to Guttman scaling, this strategy uses age of initiation to identify temporal ordering in the initiation of 2+ soft drugs; errors notated in bold; error beneath lines = 4; error above lines = 8; total error = 12.

Finally, response patterns for students who self-reported biological ages of initiation for all three soft drugs also reflected the temporal order in which these drugs were initiated. For example, case #120 provided an age for alcohol (9 years), cigarette (15 years), and marijuana (11 years) initiation, resulting in a response pattern of 1-3-2. In contrast, case #32 provided an age for alcohol (15 years), cigarette (16 years), and marijuana (18 years) initiation, thereby resulting in a response pattern of 1-2-3.

When biological ages of drug initiation are reported, ties in these respective ages can occur, whereby two or more drugs are reported as having been initiated at the same biological age. Following other researchers (Yamaguchi & Kandel, 1984a), respondents with three-way ties (i.e., biological age ties for all three soft drugs), were excluded from analysis, since it is not possible to identify any sequencing pattern in soft drug initiation. These respondents also were excluded from the supplemental analysis, since comparing the fit of these scales requires that drug data from the same respondents be used.

For respondents who report a two-way tie (e.g., alcohol and cigarette initiation at 17 years of age), proportional probabilities (discussed below) were used to break these ties and code temporal ordering accordingly (Golub & Johnson, 2001, 2002; Yamaguchi & Kandel, 1984a). In short, the proportion of respondents initiating each soft drug were calculated first without using data from cases with ties for those given drugs (e.g., 70% of untied respondents initiated alcohol use first versus 40% of untied respondents who initiated cigarette use first). These resultant proportions were then used in imputing the order of initiation for those respondents with tied ages of initiation.

Coding for the supplemental analysis. In contrast to the coding scheme employed to test the hypothesis, the supplemental analysis did not take temporal ordering of initiation into account (see Table 26). Hence, the traditional dichotomous (0, 1) coding scheme was employed. For each soft drug scale item, biological age of initiation data was recoded into dichotomous variables, with "1" signifying that the given soft drug was responded to affirmatively (i.e., initiated), and "0" indicative of a negative response (i.e., abstention) to a given scale item. For example, case # 32 (Table 26), who self-reported initiating all three soft drugs, would receive a response pattern of 1-1-1, while case #24, who reported initiating cigarettes and marijuana, would receive a response pattern of 0-1-1.

Table 26.

Example of Error-Counting for Supplemental Analysis

Case No.	(Scale Item #1) Alcohol Initiation	(Scale Item #2) Cigarette Initiation	(Scale Item #3) Marijuana Initiation	Scale Score	Error
32	1	1	1	3	0
120	1	1	1	3	0
87	1	1	0	2	0
41	1	**0**	**1**	2	2
3	**0**	1	**1**	2	2
24	**0**	1	**1**	2	2
532	1	0	0	1	0
7	**0**	0	**1**	1	2
610	0	0	0	0	0

Note. As the traditional approach to Guttman scaling, this strategy uses a dichotomous coding scheme to notate affirmative and negative responses; errors notated in bold; error beneath lines = 4; error above lines = 4; total error = 8.

Scale development. Older versions of SPSS included a subprogram, GUTTMAN SCALE, used to develop and evaluate Guttman scales (Mueller, 1986). Since versions published since the 1990s do not include this scaling function, Guttman scaling development and analysis was done manually.

Discussed earlier, two separate Guttman scales were developed and analyzed. The first tested H1, while the second scale examined the scalability of responses in terms of whether respondents did or did not initiate the three soft drugs.

Three major steps were involved in developing the Guttman scale that was used in testing H1. First, a Guttman scalogram response matrix was developed in Microsoft Excel to display response patterns for the three soft drug initiation scale items (see Table 25). Scale scores for each case were calculated and placed in the last column of the matrix. Regardless of temporal ordering in soft drug initiation, a scale score is equal to the sum of all affirmative responses.

Second, the proportion of cases who responded affirmatively to initiating each drug was calculated (McIver & Carmines, 1982). These scale item proportions were used to break any two-way ties in the temporal ordering of soft drug initiation. After all two-way ties are broken, and all cases had a response pattern that reflects temporal ordering of initiation, these proportions were recalculated with all cases and listed as marginal statistics at the end of each drug item column (Golub & Johnson, 2001).

Third, after this calculation was performed, both scale items and cases were arranged in order of magnitude, resulting in a hierarchical pattern of responses that resemble a triangle (McIver & Carmines, 1982). Rearranging both drug items and cases in this manner aided in identifying and counting errors.

Error identification and counting. After drug scale items and cases were rearranged, attention was directed toward identifying errors in response patterns. Discussed in detail in Appendix D, the method by which errors are counted can have a considerable impact on the scalability of drug items. As a general rule of thumb, the less error there is in soft drug initiation response patterns, the more scalable the items (Babbie, 2004; McIver & Carmines, 1982). To identify errors, scale scores were referred to in drawing a horizontal line within each scale item column to constitute the cut-point between one-point differences in scale scores (see Table 25). Once these lines were drawn, response errors were identified and counted.

Illustrated in Table 25, an error constituted a response that lies above or below a given horizontal line and counters the hypothesized response (Champion, 2000). For the marijuana initiation scale item, all responses above and below the horizontal line should be “3” and “0,” respectively. Hence, errors constituted any number other than “3” found above the line and any number other than “0” found below the horizontal line. For the cigarette initiation scale item, all responses above and below the horizontal line should be “2” and “0,” respectively, while all responses above and below the horizontal line for the alcohol initiation scale item should be “1” and “0,” respectively.

Evaluating scale fit. Although errors violate the assumption of cumulation, violating this assumption is expected; rarely is a perfect Guttman scale obtained (DeVellis, 2003; McIver & Carmines, 1982). More often than not, some form of deviation from the perfect scale exists (e.g., cigarette only initiates or marijuana only initiates). Since it is acknowledged that all Guttman scales contain some error, two indices were used in evaluating how much deviation from a perfect scale is tolerable: the index of reproducibility and the index of scalability.

Guttman (1950) developed the index of scale reproducibility to estimate the goodness of fit between observed responses patterns and the hypothesized response pattern. This index, which results in a *coefficient of reproducibility* (CR), indicates how well one can reproduce (or predict) a student's scale item responses given only knowledge of the student's scale score (McIver & Carmines, 1982). The formula for the CR is expressed as:

$$CR = 1.0 - (E)/TR \tag{1}$$

where, E = total response errors
TR = total responses or [(# items) X (# responses)]

With CR ranging from 0-1, a CR ≥ .90 constitutes the minimum standard of acceptability (Guttman, 1950). A CR of .90 means that not only can one predict (with 90% accuracy) the scale item responses of a given student simply by knowing that student's scale score, but the hypothesized sequence of scale items also can be predicted (with 90% accuracy) given knowledge of students' scale scores.

According to Guttman (1950), the CR is a necessary, but insufficient, benchmark for determining scalability, because scale reproducibility can be impacted by the marginal distributions of scale items. In an effort to provide a "check" against an inflated CR, Menzel (1953) developed the index of scalability, which produces a *coefficient of scalability* (CS). In protecting against attributing excessively high scalability (i.e., high CR) to response frequencies of extreme scale items, the CS reflects the degree to which responses to scale items can be predicted given only knowledge of the marginal frequencies (Smith, 1968).

Hence, as a second measure of scalability, the CS does not take into account scale scores, but takes into account the marginal frequencies of scale item responses. Ranging from 0 to 1, an indicator of scalability is CS ≥ .60. According to Brown and Hudson (2002), the CS is expressed as:

$$CS = PI/1\text{-}MMR \qquad (2)$$

where, PI = percentage improvement, or CR-MMR; and
MMR = minimal marginal reproducibility, or
$\sum$ p (or q, whichever is larger)/k,
with p = % initiates for each scale item
q = % abstainers for each scale item
k = # scale items

A CS of .60 means that 60% of total possible errors actually are not errors, but are responses that are consistent with those that are hypothesized (Menzel, 1953). Hence, in testing H1, obtaining a CS ≥ .60 means that at least 60% of all drug scale items that might have been errors are, in fact, not errors, thereby providing evidence that the soft drug scale items are scalable.

H1 is supported in the event that the CR ≥ .90 and CS ≥ .60, since involvement in soft drug use by students in the sample can be deemed (with confidence) as constituting a unidimensional, time-ordered phenomena that is characterized by cumulation and hierarchy. This involvement typically begins with alcohol. Among alcohol initiates, cigarettes constitute the second soft drug that is most commonly initiated. Compared to the number of alcohol only initiates, however, the number of alcohol and cigarette initiates is smaller. Finally, a smaller proportion of alcohol and cigarette initiates proceed to initiate marijuana use. This drug involvement sequence is time-ordered, with alcohol, cigarettes, and marijuana initiated at discrete biological ages.

Predicting Initiation

In addition to testing a modified version of Kandel's (2002) drug sequencing hypothesis, the current research also focused on examining predictors of soft drug initiation. The following discussion describes the analytic techniques that were used in testing the first component of H2-H9.

Analytic Technique

In testing the first aspect of H2-H9, outcome measures for alcohol initiation, cigarette initiation, and marijuana initiation initially were measured as dichotomies (0 = abstention, 1 = initiation). Due to the dichotomous nature of these dependent variables, binary logistic regression (i.e., binary logit) was utilized. In contrast to ordinary least squares regression (OLS), which requires dependent variables to be coded as continuous measures, binary logit requires that dependent variables have two categories (George & Mallery, 2006). In addition to this requirement, predictors (and control variables) can be continuous or dichotomous in nature. Predictors measured with three categories (e.g., age of alcohol initiation) were recoded into three separate dummy variables, with each dummy variable constituting one category of the original three-category variable (e.g., alcohol abstention, early alcohol initiation, and late alcohol initiation). For this type of predictor, one category served as the reference variable (e.g., alcohol abstention) and was excluded from respective prediction models.

Binary logit produces two types of coefficients that can be used to assess the independent impact of a predictor on the likelihood of initiating a given soft drug, while controlling for other predictors. Initially, a binary logit equation is expressed in terms of log odds (i.e., natural logarithm of the odds, or logit of Y). Specifically, the binary logit equation is expressed as:

$$\ln\left\{\frac{P\,(Y=1)}{1-P(Y=1)}\right\} = a+b_1x_1+b_2x_2+b_3x_3\ldots b_jx_j \tag{3}$$

where, $\ln\left\{\frac{P\,(Y=1)}{1-P(Y=1)}\right\}$ = log odds of soft drug initiation occurring;
a = estimated log odds of soft drug initiation occurring when all predictors equal 0;
x = predictor; and
b = slope for the corresponding predictor (Menard, 2002)

For a continuous predictor, a positive slope indicates that a one-unit increase in the given predictor is associated with an increased log odds (i.e., greater likelihood) that soft drug initiation will occur. Conversely, a negative slope indicates that when all other predictors are controlled, a one-unit increase in the given predictor is associated with a decreased log odds (i.e., decreased likelihood) that soft drug initiation will occur (Meyers, Gamst, & Guarino, 2006). In the case of a dichotomous predictor, the log odds coefficient constitutes the impact that the group classified as “1” has on the log odds of soft drug initiation, compared to the impact exerted by the reference category (0).

Due to interpretation challenges, many researchers bypass evaluation of this coefficient in favor of the exponentiated coefficient, or Exp(B). Referred to as an odds ratio, an exponentiated coefficient is obtained by removing the natural log function from the respective coefficient (Menard, 2002). Removing the natural log transforms the log odds into simple odds, which provides a somewhat easier interpretation. In the context of soft drug initiation, the simple odds essentially constitutes the probability that soft drug initiation will occur versus the probability that it will not occur (Meyers et al., 2006). For continuous independent variables, Exp(B) refers to the change in the simple odds that initiation will occur given a one-unit increase in the predictor, with the impact of other predictors controlled. Similar to interpreting the log odds for dichotomous measures, an odds ratio for a dichotomous variable shows the impact that membership in the "1" category of the variable has on the simple odds of soft drug initiation, compared to the impact exerted by membership in the reference category (Mertler & Vannatta, 2005).

In testing H2-H9, odds ratios for each respective predictor were assessed. Since an odds ratio of 1.0 means that the given predictor has no impact on the simple odds that soft drug initiation occurs (Menard, 2002), an odds ratio > 1.0 (for a positive slope) indicates that a one-unit increase in the given predictor is associated with an increase in the simple odds that soft drug initiation occurs. In the current research, this type of finding suggests that the given predictor (e.g., peer drug use) possesses risk qualities. In contrast, an odds ratio < 1.0 (for a negative slope) suggests an inverse relationship, whereby a one-unit increase in the given predictor is associated with a decreased likelihood that soft drug initiation occurs (Menard, 2002). This type of finding suggests that the psychosocial factor under consideration operates in a protective fashion.

Predicting Time to Initiation

In addition to examining soft drug initiation in terms of psychosocial factors that predict its occurrence, the current research also examined soft drug initiation in terms of how quickly it occurs. Since time is conceptualized in terms of biological age, this set of analyses involved estimating the relative risk that psychosocial factors pose for initiating alcohol, cigarette, and marijuana use at earlier versus later biological ages. Both total sample and age-specific models were developed in testing H2-H9.

Analytic Technique

For this area of the research, the dependent variable, time to initiation (for alcohol, cigarettes, and marijuana use), constituted a continuous measure. Cox regression is a statistical technique well-suited for testing attendant hypotheses, since this type of regression analysis takes into account both initiation status (abstention versus initiation) and age of initiation among those who reported initiating, two components of the two-part dependent variable.

Other types of regression are not appropriate for examining this type of dependent measure, with their use resulting in biased estimates (Allison, 1984). For example, although ordinary least squares regression requires that the dependent variable be continuous in nature, all respondents who reported abstention from the given drug would be excluded from analysis (Lawless, 2003). This would produce biased prediction estimates, particularly if a considerable proportion of the sample reported abstention from the drug (e.g., marijuana) in question. Logistic regression, which requires that the dependent variable be measured categorically, also is not suitable for predicting time to initiation because age of initiation, one component of the dependent variable to be examined, constitutes a dependent measure that binary and multinomial logistic regression is not designed to consider when producing regression estimates (Lawless, 2003).

Of the two major Cox regression models (i.e., proportional and nonproportional hazards) that can be developed to examine survival data, the proportional hazards model was used. This type of Cox regression enables multiple predictors to be examined. It also takes into account right-censored cases, or respondents who reported soft drug abstention (i.e., survival) up to the end of the observation period (i.e., time of survey administration). Cox's proportional hazards model was employed using data for both the total sample and age-graded subsamples.

In Cox regression, predictors must either constitute continuous or dichotomous variables (Allison, 1984). Similar to the logistic regression analyses, predictors measured with three categories were recoded into three separate dummy variables, with each dummy variable constituting one category of the original three-category variable. In order to make comparisons between categories, one of the variables (the reference category) was excluded from the model.

Soft drug initiation is conceptualized as an event that occurs only one time. The occurrence of initiation is regarded as failure, while non-occurrence (i.e., abstention) until the end of the observation period is characterized as survival (Lawless, 2003). The dependent variable, time to initiation, is a two-part criterion. The first component, the time indicator, must be continuous in nature, while the second component, the status (or conditional) indicator, must be measured dichotomously (Allison, 1984). Mentioned earlier, these measurement requirements were met, since age of initiation, the time indicator, constituted a continuous variable, and initiation status, the status indicator, served as a dichotomous variable (0 = abstention, 1 = initiation).

The dependent variable, time to initiation, is conceptualized as the hazard of failure, or the risk of initiating the given soft drug within the observation period (Allison, 1984). More specifically, the hazard of failure (or hazard rate) indicates the probability that soft drug initiation will occur at any time in the observation period among those students who are at risk (i.e., abstainers). The hazard rate takes into account both the timing of soft drug initiation (in terms of biological years) among those who initiated and its occurrence (initiation status) for those who initiated (Lawless, 2003). Instead of actually predicting risk, the hazard rate is an "unobserved" dependent variable that represents the risk associated with initiating soft drug use at any point in the observation period. In statistical terms, time to initiation (i.e., risk of failure) is expressed as the natural log of the hazard rate (Allison, 1984; Lawless, 2003):

$$\ln [h(t)] = a(t)+b_1x_1+b_2x_2+b_3x_3+\ldots b_jx_j \tag{4}$$

where, $\ln [h(t)]$ = log hazard of failure (i.e., soft drug initiation) within the observation period;
$a(t)$ = the baseline hazard function, or the estimated log hazard of failure within the observation period when all predictors = 0;
x = predictor; and
b = slope for the corresponding predictor

Interpreting coefficient estimates for each predictor is conducted in a similar manner as interpreting unstandardized regression coefficients in other types of regression equations. A negative slope indicates that an increase in the predictor is associated with a decrease in the log hazard of soft drug initiation (while controlling for other predictors) during the observation period. This decrease equates to a decreased risk for failure (i.e., initiation), or increased survival time (Allison, 1984). In other words, an increased survival time equates to an older or later age of soft drug initiation. From a risk-protection perspective, a negative coefficient is indicative of a protective factor that serves to delay time to initiation. Conversely, a positive slope indicates that an increase in the predictor is associated with an increase in the log hazard of soft drug initiation (while controlling for other factors) during the observation period (Allison, 1984). This increase equates to an increased risk for initiation, or decreased survival time. Further, a decreased survival time equates to an earlier or younger age of soft drug initiation. Hence, a positive slope is indicative of a risk factor that contributes to an earlier age of soft drug initiation.

Another way to assess the utility of a predictor is to take the antilog of the respective coefficient. This equates to exponentiating the value of the coefficient (*b*) by roughly 2.718 (Allison, 1984). Expressed as Exp(B), an exponentiated coefficient for a continuous predictor that is greater than 1.00 indicates that a one-unit increase in the predictor results in an increased hazard, or increased risk of earlier versus later soft drug initiation. In contrast, an Exp(B) less than 1.00 indicates that a one-unit increase in the independent variable results in a decreased risk of failure, or a later versus earlier age of initiation. For continuous predictors, the Exp(B) also indicates the percentage change in relative risk for soft drug initiation associated with a one-unit increase in the predictor (Allison, 1984). For example, if Exp(B) = .75 for the relationship between school attachment and time to alcohol initiation, this indicates that for every one-unit increase in school attachment, the relative risk of failure decreases by 25%. Conversely, if Exp(B) = 4.10 for the relationship between peer drug use and marijuana initiation, this means that for every one-unit increase in peer drug use, the relative risk of initiation increases by 310%.

For a dichotomous (or dummy) variable, the Exp(B) refers to the relative difference in the hazard rates between the two groups to which the value of the exponentiated coefficient refers (when controlling for other predictors). For example, if Exp(B) = 1.75 for the relationship between gender and time to marijuana initiation (with female constituting the reference category), males have a 75% higher risk of initiating marijuana than females at any point during the observation period (controlling for other factors). In contrast, if Exp(B) = .60 for the same gender variable, the hazard of marijuana initiation among males is roughly 60% of the hazard for marijuana initiation among females. In other words, when controlling for other factors, the hazard for marijuana initiation among females is roughly 40% greater than that for marijuana initiation among males (during the observation period).

Model Development and Logistics

To properly address Research Questions #2 and #3, several important aspects of model development and analyses were considered. These features centered on how and what predictors were entered into models, how the regression equations were produced, and various other issues related to the interpretation of specific regression results. A supplemental mediation analysis also was conducted.

Blocking Procedure

In contrast to entering all independent and control variables into one regression equation, predictors were grouped into clusters based upon the ecological domain from which they emanate. In turn, these clusters of predictors, which constitute distinctive blocks, were entered in regression models in a successive fashion. As in the case of the current research, pre-established blocking procedures typically are used for theoretical or empirical reasons, such as testing for mediating effects or examining R^2 differences in the predictive utility of distinct groups of predictors (Weisburd & Britt, 2003).

A total of six blocks of predictors were developed for each regression model, five of which constitute the ecological domains of influence. Community domain predictors constituted Block #1, school influences Block #2, family predictors Block #3, peer factors Block #4, and individual domain predictors Block #5. Control variables were entered last (Block #6). The rationale underlying the ordering of these blocks is reserved for discussion of the supplemental mediation analysis.

Worth noting is that as blocks were developed, predictors in previous blocks served as controls. In this manner, sets of predictors were examined to determine their respective independent (and combined) effects while controlling for the effect of variables that were entered previously (Weisburd & Britt, 2003).

Although all coefficients during each stage of the model-building process were examined, the coefficients in the final models ultimately were the most informative, since all predictors were taken into account at this stage (George & Mallery, 2006).

Backward Stepwise Procedure

Discussed earlier, many of the regression models developed included more than 20 predictors. In an effort to obtain adequate-fitting models while reducing the number of predictors (and generating reliable and unbiased estimates), the backward stepwise regression technique was used in developing both binary logit and Cox regression equations.

The backward stepwise function was employed in conjunction with the blocking procedure discussed above, so all variables in each block were entered into the model at the outset, one block at a time. Following the lead of several statisticians (Cohen, 1991; Dorsett & Webster, 1983; Menard, 2002), irrelevant predictors were removed from the model, with p <.10 specified for model significance. This significance threshold corresponded to the significance of the regression model (e.g., -2 log likelihood model statistic in binary logit), not the significance of each individual parameter estimate. Since this is a relaxed threshold (compared to the p < .05 default), those variables that were marginally significant remained in the model. This proved beneficial for identifying mediation processes, a topic reserved for later discussion.

Beginning with the variable that has the least predictive utility, predictors within each block that did not contribute significantly to R^2 (using the p <.10

criteria for model significance) were removed from the equation (George & Mallery, 2006; Meyers et al., 2006). As blocks of predictors were entered successively, and individual variables within respective blocks were assessed for removal, the *p*-values for variables retained in the model changed (Meyers et al., 2006). Since backward stepwise regression is an iterative process, *p*-value changes were monitored continually. Those predictors whose significance levels changed the significance level of the overall model to $p > .10$ were dropped from the equation.

Forward stepwise regression also may be used to arrive at similar results; however, there are several weaknesses associated with this technique. Unlike backward elimination, the forward insertion procedure tends to overfit the data, which can lead to an inflated R^2 (Flack & Chang, 1987). Also, forward stepwise regression increases the chance of committing a Type II error, since suppressor effects[6] are less apt to be identified (Menard, 2002). Finally, simulation studies indicate that multicollinearity tends to pose more of a problem when forward insertion versus backward elimination is used (Cohen, 1991; Dorsett & Webster, 1983; Flack & Chang, 1987).

There is one major drawback to using the backward elimination technique, but this limitation was not a serious concern in the present study. Research indicates that in instances where backward stepwise regression is employed with a large number of predictors and a relatively small sample size, low statistical power can produce unstable estimates at the beginning stages of the model-

[6] A suppressor effect occurs when a predictor is significant only when a second independent variable is controlled (Cohen, 1991; Menard, 2002).

building process (Cohen, 1991). Power analysis results (to be discussed shortly) indicated that low statistical power was not an issue in the current research. In further support of backward stepwise regression, blocks of variables were entered into models on an incremental basis. Since it was expected that not all predictors across blocks would be significantly related to soft drug initiation outcomes, it was further expected (and substantiated) that the final stages of the model-building process would not include close to all of the predictors subject to model retainment.

Total Sample Models

In addressing Research Question #2, H2-H6 were tested for the entire sample of 6^{th}, 9^{th}, and 12^{th} grade students. A total of 6 regression models were developed: three binary logit models and three Cox regression models. Aside from testing H2-H6, the two-fold purpose in developing these six models was to conduct a preliminary investigation of the strength and direction of predictors for the total sample and determine whether independent variables differ in kind or marked saliency by drug type. Three independent binary logit models (Models #1-3) were developed to determine the impact of relevant predictors on the simple odds of initiating alcohol, cigarette, and marijuana use, while three independent Cox regression models (Models #4-6) constituted a preliminary investigation of the hazard effects of relevant psychosocial factors on time to alcohol, cigarette, and marijuana initiation. To account for biological age effects, the covariate, *Age*, was entered into the last block of each prediction model with the other relevant control variables.

Although a detailed discussion is reserved for Chapter 9 (Multivariate Results), it is important to mention here that the results of H1 (using Guttman scalogram analysis) informed the type of extraneous variables that were entered into each respective prediction model. Specifically, the most common soft drug sequence that was identified with data from the total sample had a direct bearing on the type of drug-related predictors (i.e., *EasyAlc*, *EasyCig*, *EasyMar*, *AlcEarly*, *CigEarly*, *PeerAlc*, *PeerCig*, *PeerMar*, *AlcIntent*, *CigIntent, MarIntent*, and the soft drug tolerance measures) that were eligible for assessment in predicting alcohol, cigarette, and marijuana initiation (and time to initiation). For instance, if H1 findings indicate that most marijuana initiates began marijuana use after initiating alcohol and cigarettes, the marijuana-related predictors (e.g., *EasyMar*, *PeerMar*, *MarIntent*, and the marijuana tolerance variables) would not be eligible for examination in the alcohol and cigarette initiation prediction models. These marijuana-related measures would only be entered into the regression equations predicting marijuana initiation and time to initiation. Developing these six prediction models according to the most common soft drug sequence found in the total sample data provided a way to identify predictors that may explain progression in the most common soft drug sequence.

Age-Graded Models

Aside from examining predictor effects on soft drug initiation and time to initiation for the total sample (holding current biological age constant), further analyses sought to determine whether the simple odds for soft drug initiation and the hazard effects for age of soft drug initiation vary according to stage of adolescent development (i.e., biological age, or grade-level). Specifically, to address Research Question #3, 16 age-specific regression models were developed. Eight binary logit models were used to test the *initiation* component of H2-H9, while eight Cox regression models were developed in testing the *time to initiation* component of H2- H9.

In testing H2-H9, as it relates to predicting the occurrence of soft drug initiation, eight age-graded binary logit models were employed. One model each was developed to predict alcohol initiation among 6th (Model #7), 9th (Model #8), and 12th (Model #9) grade students, as well as predictors of cigarette initiation (Models #10-12) among these students, respectively. One model each also was developed to predict marijuana initiation among 9th (Model #13) and 12th (Model #14) students. Predictors of marijuana initiation among 6th grade students could not be investigated, since only 2.1% (n = 6) of 6th grade students self-reported initiating this soft drug.

In order to test H2- H9 in terms of predicting time to soft drug initiation, eight age-graded Cox regression models were developed and analyzed. One model each was developed to predict time to alcohol initiation among 6th (Model #15), 9th (Model #16), and 12th (Model #17) grade students, as well as predictors of time to cigarette initiation (Models #18-20) among these students, respectively. One model each also was developed to predict time to marijuana initiation among 9th (Model #21) and 12th (Model #22) students. The lack of variation in marijuana initiation among 6th grade students precluded the examination of predictors of time to marijuana initiation among this subsample.

The goals underlying the investigation of predictors of soft drug initiation and age of initiation among the total sample (Models #1-6) similarly applied to the age-graded models just described (Models #7-22). Specifically, the strength and direction of predictors were examined for each age-specific model. An assessment also was made concerning the degree to which extraneous variables differ in kind or saliency by drug type (within and across grade-level).

The type of extraneous variables that were entered into each respective age-graded model were informed by two factors: the results of H1 (similar to the total sample models) and the biological age of the subsample under inspection. With respect to the latter issue, the biological age of the subsample under inspection dictated what extraneous age of initiation variables (early or late age of initiation, or both) were relevant for assessment. In predicting initiation and time to initiation among 6th and 9th grade students, for example, the effects of late age of alcohol (*AlcLate*) and cigarette (*CigLate*) initiation were not assessed, since late initiation was operationalized as that occurring between 16 and 18 years, ages that 6th and 9th grade students had not yet reached. For these subsamples, the predictive utility of early age of initiation (i.e., initiation between 8 and 15 years of age) was examined. Among 12th grade students, however, the extraneous measures of early and late age of initiation were entered into models and assessed.

Supplemental Mediation Analysis

Discussed in Chapters 2 and 4, Petraitis et al. (1995) contend that in main effects models, risk factors conceptualized as distal in nature (e.g., school and community-level factors) may be less salient predictors of adolescent drug use than more proximal risk factors (e.g., family, peer, and individual-level factors). This contention is rooted in their argument that the effects of distal predictors likely are mediated by more proximal influences. This notion of distal-proximal mediation also parallels the propositions put forth by Hawkins (Catalano & Hawkins, 1996) and Akers (1977) concerning distal constructs being mediated by more proximal influences. Although the extant research (see Chapter 4) provides support for Petraitis et al.'s (1995) argument, much more research is needed in this area. Hence, the current research sought to answer this research call in providing a broad-based assessment of the mediating capacity of proximal factors in reducing relationships between distal influences and soft drug initiation. This assessment was conducted within the same binary logit and Cox regression models that were discussed above.

Assessing Mediation

A variable may be referred to as a mediator "to the extent that it accounts for the relation between the predictor and the criterion" (Baron & Kenny, 1986, p. 1176). There are several ways to assess mediation; however, the typical and more broad-based way of identifying mediating relationships in a multivariate regression model is to examine the difference in two key relationships: 1) the size and significance of the total effect of the primary independent variable on the outcome (b_1) before the effects of one or more predictors (b_2, b_3…) are controlled, and 2) the size and significance of the direct effect of the primary independent variable on the outcome (b_1') after the effect of one or more variables has been controlled (Baron & Kenny, 1986).

Evidence of mediation is found in the value of the indirect effect, which equates to the difference between the two slopes, or d = b_1 - b_1' (Clogg, Petkova, & Haritou, 1995; Schwalbe, Fraser, Day, & Cooley, 2006). The value of the indirect effect can provide evidence of full mediation (b_1' = 0, so d = b_1), partial mediation (d = non-0, but < b_1), or no mediation (b_1 = b_1', so d = 0). Evidence of full mediation also can be observed when the change in the significance of the slope exceeds the significance threshold set for the model (Baron & Kenny, 1986; McGloin & Pratt, 2003).

Analysis Plan

The order in which the ecological domains were entered into binary logit and Cox regression models was particularly important for assessing the empirical validity of Petraitis et al.'s (1995) contention concerning distal-proximal mediation, since the successive entry of domains into the models constitutes movement from more distal to more proximal forms of influence. Discussed earlier, the entry of predictors was ecologically-graded, with the most distal factors (community-level) entered first, followed by the school predictors, family, peer, and individual domain predictors.

The size and significance of coefficients were examined both before and after subsequent blocks of predictors were entered into the model. To illustrate, the slopes and significance levels of the community-level predictors (Block #1) were examined before and after school domain predictors (Block #2) were entered. After the family domain predictors (Block #3) were entered, the size and significance of the slopes for the community-level and school domain predictors each were examined to assess reductions. This continued throughout the model-building process until all domain-specific predictors were entered. Thus, beginning with Block #2 (school domain), each subsequent domain of predictors constitute more proximal influences, whose effects may explain all of the effects (in the case of full mediation) of the more distal predictors that were entered previously (Schwalbe et al., 2006).

Tracking the significance levels and magnitude of individual predictors throughout the model-building process, and notating which domains are responsible for the exclusion of particular predictors, provided a way to assess whether full mediation tends to occur in the distal-proximal manner purported by Petraitis et al. (1995). The liberal significance threshold ($p < .10$) for the models served to reinforce evidence of full mediation.

Quantitative Cross-Validation

Discussed at the end of Chapter 5, a dual cross-validation scheme, consisting of a systematic review cross-validation and a quantitative cross-validation, was employed in an effort to assess the validity of the quantitative findings. In verifying findings based on the directional hypotheses that were tested, the systematic review cross-validation provided a broad assessment of the plausible threats to the statistical conclusion, construct, internal, and external validity of the quantitative findings, with an emphasis on internal and external validity. In contrast, the quantitative cross-validation involved comparing the statistical results for H2-H9 with those from the validation sample.

Validation Sample

The validation sample is a distinct, but similar, sample of 6th, 9th, and 12th grade students who completed the PPAAUS in 2001 (see Appendix E). The survey administration procedures used to collect 2004 PPAAUS data also were employed in the collection of 2001 PPAAUS data. PPAAUS instruments are alike in terms of readability and format; question wording, ordering, and font; and the number and nature of response categories. A few items were added to the 2004 PPAAUS that did not appear in the 2001 PPAAUS, including three measures (i.e., perceived ease in obtaining alcohol [*EasyAlc*], cigarettes [*EasyCig*], and marijuana [*EasyMar*]) that were examined in the quantitative component of the current research. With the exception of these three measures, all questionnaire items of interest for the current research were included in both surveys.

Not only were both samples obtained using the same district-wide survey methodology, but they both represent 6th, 9th, and 12th grade students from the same rural school district. Comparatively, the demographic profiles of each sample also are similar. The validation sample constitutes 723 6th, 9th, and 12th grade students who provided usable data that withstood QR filtering conducted by J. S. White Surveys. Similar to the derivation sample, the majority of students in the validation sample were white (90%) and were fairly evenly distributed across gender (52% male) and grade-level. Sixth grade students (n = 236) constituted 33% of the validation sample, 9th grade students (n = 244) comprised 34%, and 33% of the validation sample were 12th grade students (n = 243).

In terms of 6th, 9th, and 12th grade response rates, school district enrollment records indicate that 258 students were enrolled in 6th grade, 277 students were registered for 9th grade, and 287 students were enrolled in 12th grade during the 2000-2001 school year (Hruska, 2001). Considering these enrollment numbers in conjunction with the number of 6th, 9th, and 12th grade students who provided useable data, the 2001 PPAAUS response rate for 6th, 9th, and 12th grade students (after QR filtering) was 91%, 88%, and 85%, respectively (for a 88% total response rate).

Plausible Threats to Validity

Findings from both the derivation and validation sample suffer from similar plausible threats to validity, since the same survey and sampling methodology were used to generate data, and the same type of analytic techniques and strategies will be used to arrive at results. The following discussion primarily frames these plausible threats within the context of the quantitative component of the research (2004 PPAAUS); however, these threats apply equally to the validation results.

Statistical Conclusion Validity

Inappropriate statistical techniques. The statistical techniques (i.e., binary logistic regression and survival analysis) that were used in the current research are warranted given the level at which the outcome variables are measured. Hence, plausible threats to statistical conclusion validity that are attributable to inappropriate statistical techniques should not be an issue with either the quantitative component of the current research or with findings generated from the validation sample.

Low statistical power. Discussed in the previous chapter, given certain statistical conditions for α, level of power, and ES desired, the size of a sample can impact the ability to detect significant relationships. A small sample poses a plausible threat to statistical conclusion validity, since a statistical model developed with a low sample may fail to detect significant relationships between predictors and an outcome (Welsh & Farrington, 2006b). Although the derivation and validation samples differ slightly in terms of size (N = 753 versus N = 723, respectively), data from over 700 students will be examined. In an effort to determine whether this sample (and age-graded subsamples) was large enough to detect significant relationships, Cohen's (1992) statistical power analysis was employed.

Following common convention in social science research, the traditional level of significance, .05, was chosen for α, while level of power was set at .80 (Cohen, 1992; Green, 1991). Statistical power is related to ES in that the greater the ES, the greater the statistical power (Cohen, 1992). In regression analyses, a population ES is the degree of discrepancy between the null and research hypotheses. The null hypothesis is formulated on the assumption that ES = 0 in the population, meaning there is no relationship between one or more predictors and the criterion. The research hypothesis assumes there is a relationship in the population, and thus ES > 0. Aside from viewing regression coefficients for individual predictors in terms of ES, R^2 also may be viewed as a form of an ES. From this perspective, the larger the ES, the more variation in the criterion variable that is being explained by the predictors in the model.

ES is operationalized as a scale, ranging from 0-1. Within this scale, the magnitude of an ES typically is classified as small, medium, or large. How ES values are classified varies according to the type of statistical test employed and the field in which research studies are conducted (Cohen, 1988). According to Cohen's (1992) population ES categorization scheme for regression coefficients and R^2, a small ES is .02, a medium ES is .13, and a large ES is considered to be .26 or larger. Standard practice in the social sciences is to use a medium ES when conducting a power analysis, since social science research typically produces medium effect sizes (Cohen, 1992).

Power analysis results revealed that in order to detect a medium population ES of .13 (or R^2 = .13) when examining 20 predictors in a multivariate regression model, with β = .20 (or .80 power) at the .05 α, the minimum sample size required is 156 cases (Cohen, 1988). Not only does this minimal number of students fall well below the 753 students who completed the 2004 PPAAUS (and 723 students who completed the 2001 PPAAUS), but this minimal sample size also falls short of the roughly 250 students who comprised each of the three age-graded subsamples in both sets of data. Moreover, since the backwards stepwise function was used in conjunction with the blocking procedure in developing models, it was not anticipated that any one regression equation would include more than 30 independent variables, including the final models. Hence, the size of both the total samples and the age-graded subsamples were large enough to avoid a plausible threat to statistical conclusion validity.

In contrast to power analysis, several statisticians have offered sample size “rules-of-thumb” in determining appropriate sample sizes for conducting multivariate regression analyses (see, e.g., Green, 1991; Kraemer & Thiemann, 1987; Nunnally & Bernstein, 1994; VanVoorhis & Morgan, 2007). When statistical power is set at .80, α at .05, and a medium ES of .07 is desired, Green (1991) proposed the following formula: $N \geq 104+m$ (where m is the number of predictors). To establish a worst case scenario, this formula was used to determine the minimal sample size needed for assessing the predictive utility of 60 predictors in one regression equation. If 60 predictors are entered simultaneously, at minimum, the sample size should consist of 164 students. Again, not only does this minimal number of students fall well short of the sizes of the 2004 and 2001 total samples and age-graded subsamples, but the strategies that were employed in building regression models (i.e., predictor blocking coupled with backward stepwise regression) limit the number of predictors that were entered and assessed at any one time.

Green (1991) further proposed a second formula ($N \geq 50+8m$, where m is the number of predictors) for determining the minimal sample size needed for obtaining a valid R^2, given the same statistical conditions outlined above (α = .05, .80 power, medium ES). Although 60 predictors were not available for entry into any one statistical model, doing so would require a minimum sample size of 530 students, which again falls well short of the approximate 750 students who completed the 2004 PPAAUS (and 720 who completed the 2001 PPAAUS).

As the results from the power analysis and “rules-of-thumb” computations indicate, the size of the derivation and validation samples (and age-graded subsamples) were sufficiently large enough to conduct a variety of multivariate analyses. Consequently, it does not appear that low statistical power constitutes a plausible threat to the statistical conclusion validity of findings.

Construct Validity

Since the quantitative data used in the current research is secondary in nature, the front-end research design elements that could be manipulated to enhance construct validity cannot be adjusted. Discussed in the previous chapter, four major factors pose a threat to the construct validity of quantitative findings: (1) invalid or unreliable measures; (2) reporting bias; (3) single source of data collection; and (4) lengthy periods of recall (Shadish et al., 2002).

The majority of these plausible threats could not be assessed fully within the quantitative component of the research itself, or through the quantitative cross-validation, since data from the derivation and validation samples were gathered using equivalent survey methodologies, and constructs were conceptualized and operationalized in the same manner. Due to these similarities, it is difficult to locate any divergent findings identified through the quantitative cross-validation in differences in the construct validity of respective findings. Discussed in the previous chapter, the construct validity of the quantitative findings will be assessed through the systematic review cross-validation.

Invalid or unreliable measures. Invalid or unreliable outcome measures do not properly capture the constructs of interest. When these types of measures are used in a study, not only is the construct validity of measures threatened, but the internal validity of findings also becomes questionable (Shadish et al., 2002). As with all secondary data, variables that were available for study in the current research already had been measured, and data pertaining to the constructs already were collected.

A “bird’s eye” assessment (Shadish et al., 2002) of the operationalization of the drug initiation measures suggests that these outcome measures are logically sound and appear to properly capture the soft drug initiation constructs of interest. Consequently, it does not appear that these measures posed a plausible threat to the construct validity of soft drug initiation estimates. Moreover, the selection of suspected predictors of adolescent soft drug initiation was guided by both theory and findings from extant research.

Reporting bias. The self-report survey methodology used to collect secondary data brings to the forefront several factors that constitute plausible threats. Discussed in the previous chapter, these factors include underreporting, forward telescoping, overreporting, and recall decay. Similar to all adolescent drug research based upon a self-report methodology, underreporting poses a plausible threat to both the construct validity of drug initiation outcome measures and the internal validity of the quantitative findings.

Since forward telescoping attenuates estimates for early ages of drug initiation, while inflating estimates for older ages of drug initiation, it is possible that the ages of initiation reported by 12th grade students in the derivation and validation sample may partly be an artifact of forward telescoping (resulting in an upward bias). Although 12th grade students comprise only 30% of the derivation sample, the cross-sectional nature of the data precludes a determination of the degree to which this threat to construct and internal validity is plausible. The systematic review cross-validation provided some insight into the extent to which this threat was operating.

Concerning overreporting, J. S. White Surveys employed a questionable response (QR) filtering strategy in identifying and excluding from data sets those students who were identified as overreporting drug use, or whose responses to survey items were questionable. The fact that this technique excluded 13 respondents from the derivation sample speaks to the likelihood that overreporting may have been reduced by some degree and was not a plausible threat to the construct validity of measures or the internal validity of quantitative findings.

Both the 2001 and 2004 PPAAUS data were obtained in classroom settings under conditions of confidentiality and anonymity. Not only have these parameters been found to reduce underreporting and generally enhance the validity of survey responses (Harrison & Hughes, 1997; Junger-Tas & Marshall, 1999), but compared to household data collection, data collected in classroom settings also have been found to be more accurate (Harrison, 2001). Although there is no practical way to determine whether the survey administration conditions successfully reduced underreporting (thereby enhancing the construct validity of measures), the last item on both survey instruments asked students to indicate the degree to which they felt their responses were anonymous. Specifically, students were asked, “Are you reasonably sure that no one at your school can identify anyone’s questionnaire?” Possible responses included “yes,” “no,” and “not sure.” In order to assess the plausibility that underreporting occurred, responses to this survey item were examined.

Single data source. Data derived from the derivation and validation samples constitute self-report data collected from students themselves. Since the research relies exclusively on self-report measures, and multiple sources of data were not obtained, the validity of student responses to questions about soft drug initiation and suspected predictors of such could not be verified through the quantitative cross-validation.

Three major research findings support the use of self-report data in the current research: 1) only a small proportion of adolescents report the use of fictitious drugs; 2) adolescent accounts of soft drug use have been found to remain relatively consistent over time; and 3) as a rule, information provided by adolescents is generally in agreement with corroborating sources, including archival records, urinalysis, and reports from parents and peers (see, e.g., Dittus & Jaccard, 2000; Johnston & O’Malley, 1997; Litrownik, Elder, & Campbell, 2000; Maisto, Connors, & Allen, 1995; Sieving, McNeely, & Blum, 2000; Sieving, Perry,

& Williams, 2000; Tobler, Roona, Ochshorn, Marshall, Streke, & Stackpole, 2000; Winters, Anderson, Bengston, Stinchfield, & Latimer, 2000; Winters et al., 1990). Research also suggests that the validity of self-report data is enhanced with the assurance of confidentiality (Aquilino, 1997; Harrell, 1997). Although self-report data has been found to be both a valid and reliable source of information, this plausible threat to the construct validity of the quantitative findings was assessed through the systematic review cross-validation.

Lengthy recall periods. Some aspects of the quantitative research required an examination of age of soft drug initiation data. Discussed earlier, these data were derived from responses to survey items that asked students to recall the biological ages at which they initiated alcohol, cigarettes, and marijuana. It is plausible that poor memory recall, particularly among older adolescents, may have impacted the validity of these responses. There was no practical way to assess this plausible threat within the quantitative aspect of the current research, or the quantitative cross-validation. Again, this issue was assessed through the systematic review cross-validation.

Internal Validity

Selection bias. Similar to all adolescent drug research, two issues related to selection bias bring this factor to the forefront as a plausible threat to the internal validity of derivation and validation findings. First, the research was non-experimental in nature (i.e., there is a lack of random assignment or matching to treatment and control groups), and students varied on levels of numerous explanatory factors.

Second, the nature and logistics underlying both the derivation and validation sampling frame enabled students to self-select themselves into the research. Not only was survey participation voluntary, but students also had to have been in school on the day of survey administration in order to participate. Consequently, particular segments of the adolescent population (e.g., school drop-outs, institutionalized youth, and chronic truants) were not represented in either sample.

Since the relationship between predictors and soft drug initiation in both samples may be an artifact of these two sampling biases, it is plausible that the effects of these biases took the form of attenuated or inflated regression coefficients (Shadish et al., 2002). To address this issue, an effort was put forth in accounting for as many factors as possible that distinguish between soft drug initiates and abstainers. This aided in reducing the impact of selection bias on the findings, while accounting for third variable explanations, reducing the regression error term, and increasing R^2 (Aiken & West, 1991).

Although sampling bias was a plausible threat, it is important to note that actual impact on the findings may have been limited. Although no data are available on rates of truancy, the school district drop-out rate was low (1.3% in 2001 and 0.8% in 2004) during both years of the survey (Hruska, 2003, 2005). Furthermore, the number of out-of-home delinquency placements also appears to have been small during the years of survey administration. County-level data indicate that only 13 youth in 2001 and 6 juveniles in 2004 were placed in juvenile institutions (Pennsylvania Juvenile Court Judges' Commission, 2005).

Finally, the number of 12-18 year olds who were home-schooled within the school district also was low during the survey years, with 26 and 15 students home-schooled in 2001 and 2004, respectively (Pennsylvania Department of Education, 2005a, 2005b).

History. It is likely that results from both the derivation and validation samples were confounded by the effects of various drug prevention initiatives carried out either within the school district or the local community since 1999. In the late 1990s, the community adopted the Communities that Care ® (CTC) prevention strategy in an effort to reduce five major risk factors and enhance numerous protective factors for youth problem behaviors, including soft drug initiation (Myers & Arter, 2005a). Theoretically grounded in SDM, the five risk factors included availability of alcohol and drugs; early initiation of problem behaviors (including soft drug use); extreme economic deprivation; parental tolerance of and involvement in problem behaviors; and youth alienation and rebellion. Protective factors targeted for enhancement primarily centered on parent-child relationships, including consistent discipline, prosocial familial bonding, familial reinforcement of youth prosocial behavior, and prosocial familial interaction.

One prevention initiative in particular, a parental networking project, may have impacted rates of soft drug initiation and levels of individual, family, and peer-related factors. This initiative, which consisted of several smaller-scale projects (e.g., the Caring Homes Campaign, the Parents Who Care ® program, and a social marketing strategy), targeted both youth and their parents in reducing adolescent alienation and rebelliousness, the availability of alcohol and drugs, and parental prodrug attitudes and involvement in drug use (Myers & Arter, 2005a). An explicit effort also was directed at reducing rates of soft drug initiation among 6th and 9th grade students (i.e., early initiation), along with strengthening familial bonding and increasing parental monitoring and consistent child discipline.

Although changes in rates of soft drug use were not assessed longitudinally within cohorts of 6th, 9th, and 12th grade students (nor were cohort and secular effects controlled), outcome results based upon cohort-sequential data indicate that the CTC prevention strategy may have been helpful in reducing rates of adolescent soft drug use (Myers & Arter, 2005a). For example, between 1998 and 2001, monthly rates of alcohol, cigarette, and marijuana use declined among 6th, 9th, and 12th grade students, with the largest downturns observed among 6th and 9th graders. Among 12th graders, annual rates of soft drug use also declined.

Declines in soft drug use between 1998 and 2001 speak to the likelihood that the 2001 PPAAUS data on soft drug initiation and psychosocial factors may have been confounded by targeted prevention efforts. Since these prevention efforts still continue today, data from the 2004 PPAAUS also may have been influenced by these initiatives. However, since both sets of data probably are confounded in similar ways (i.e., in the same direction), it unlikely that conflicting findings for directional effects identified through the quantitative cross-validation were the result of differential “treatment.” Rates of soft drug initiation and levels of risk factors likely are conservative, however, compared to rates for similar youth not exposed to these prevention initiatives.

Due to likelihood that rates are conservative, it also is probable that the magnitude of relationships between predictors and outcome measures are artificially attenuated and may not be generalizable to similar-age students who have not been exposed to community and school-level drug prevention efforts. Because both 2001 and 2004 PPAAUS data are confounded by history, the quantitative cross-validation was not able to properly assess the impact of this threat on quantitative findings. Discussed in the previous chapter, though, the verification of directional hypotheses through the systematic review cross-validation was helpful in determining the impact of history on the quantitative results.

Causal ordering. Valid inferences about causal relationships require that the temporal ordering of variables be accurate and alternative explanations for observed relationships be ruled out (Shadish et al., 2002). Due to the complexity of behavior and the underlying interaction between an individual and the environment (Schulenberg et al., 2003), the current research (like all adolescent drug research) was not able to account for all alternative explanations for observed relationships. In an effort to rule out as many third variables as possible, however, characteristics of respondents known to impact soft drug initiation (e.g., gender, race, and age) were taken into account in prediction models.

With respect to temporal ordering, the quantitative aspect of the research utilized cross-sectional data on predictors and soft drug initiation outcomes. Cross-sectional research on adolescent drug use is both common and helpful when testing hypotheses and producing models that may be replicated with longitudinal data (Clogg & Arminger, 1993; Harrison, 1997; Hawkins et al., 1992a; Reidel, 2000). Unfortunately, this research design cannot fully rule out the possibility that causes and consequences have been confounded (Shadish et al., 2002). Since data generated from the validation sample also were collected using a cross-sectional methodology, the temporal ordering of variables could not be properly verified through the quantitative cross-validation. This salient threat was assessed through the systematic review cross-validation.

External Validity

External validity refers to the extent to which research findings from one particular sample generalize to different conditions, including different time periods, settings, and people (Shadish et al., 2002). Due to the type of samples from which derivation and validation data were derived, all of these conditional factors posed plausible threats to the external validity of derivation and validation findings.

Not only is each respective sample homogeneous in terms of geographical residence, but various segments of the adolescent population (e.g., school drop-outs, truants, and institutionalized youth) were not included in either sampling frame. Although this suggests that estimates of soft drug initiation among both samples should be considered conservative, it is worth mentioning again that county and school district data indicate that these threats pose a limited concern for the external validity of findings. Discussed earlier, low drop-out rates were observed in the school district; county-wide delinquency placements were quite low; and fewer than 30 students were home-schooled during both years of survey administration (Hruska, 2003, 2005; PAJCJC, 2005; PDE, 2005a, 2005b). Moreover, almost all youth residing within the jurisdictional boundaries of the school district were enrolled in the public school system from which survey data were derived.

The characteristics of the derivation and validation samples were similar with regard to gender, race, and age. Moreover, youth in both samples were in the same school grades and attended the same schools in one school district. They also were reared in essentially the same rural environment. Since these samples are similar along these lines, it is not likely that any differences in findings are the result of geographical, cultural, or demographic differences.

In contrast, however, it is possible that conflicting findings identified through the quantitative cross-validation may be located in factors endemic to the different time periods in which each sample completed the PPAAUS. For example, since there was a three-year interval between survey administrations, it is possible that incongruent findings are partly a function of differential secular or cohort effects. If these effects are large enough, they can impact rates of soft drug initiation and estimates of the predictive utility of various explanatory factors (Brunswick & Boyle, 1979; O’Malley et al., 1988), thereby resulting in an incongruence in findings.

Not only is the operation and impact of secular and cohort effects difficult to assess, but isolating these effects as responsible for differential findings also is a challenging endeavor. The quantitative cross-validation was not able to directly determine whether differential findings are rooted in these effects; however, consideration was given to the possibility that divergent findings may be partly the result of these various factors.

Cross-Validation Logistics

In an effort to assess the internal and external stability of quantitative results, the quantitative cross-validation involved comparing binary logit and Cox regression findings from final models on two specific fronts: R^2 (or pseudo-R^2) and regression coefficients. The R^2 test statistic for each final model was compared across samples in an effort to identify shrinkage. Shrinkage refers to any decrease in the predictive utility of a model that is applied to another sample (Collins et al., 1987; Mills & Noyes, 1984). For each model, shrinkage was assessed using a basic computation offered by Collins and colleagues (1987): $R^2_{\text{derivation}} - R^2_{\text{validation}}$. The smaller the shrinkage between corresponding models, the more apt that 2004 PPAAUS findings are internally valid, and the more likely that findings may be generalized to similar youth from different time periods.

Although R^2 for a given outcome is contingent upon the direction and strength of the predictors in the model, two distinct samples producing identical R^2 statistics should not be interpreted as meaning that the direction and saliency of individual predictors is the same in both models (Collins et al., 1987; Pedhazur, 1982). Following the lead of other researchers (Mills & Noyes, 1984), a further comparison of findings was conducted. Specifically, derivation and validation findings for directional hypotheses (H2 and H3) and magnitude hypotheses (H3, H6, H8, and H9) were compared in terms of the direction and size of regression coefficients. In addition, results for H4, H5, and H7 also were compared to ascertain whether the predictive utility of various ecological domains of predictors holds across samples. In instances where hypothesis test results from the derivation sample are congruent with those from the validation sample, threats to the internal validity of 2004 PPAAUS findings, such as third variable explanations, may be deemed less plausible (Collins et al., 1987). As well, some of the threats to external validity (e.g., different time period) also may be ruled out.

The following four chapters present the findings of the current research. Chapter 7 introduces the systematic review results, while Chapters 8 and 9 present the findings from the quantitative component of the research. Finally, the results from the dual cross-validation are put forth in Chapter 10.

CHAPTER 7

SYSTEMATIC REVIEW RESULTS

The current chapter presents the results from the systematic review. Discussed in Chapter 5, this review focused on the predictive utility of, and the directional effects exerted by, predictors of adolescent soft drug initiation and time to initiation. A total of 506 relationships were culled from 36 primary studies, all of which fully met the inclusion criteria set forth in Chapter 5.

A two-fold purpose founded this review. First, in addressing several drawbacks endemic to a number of previous literature reviews (see Chapter 5), this assessment served to update the knowledge base concerning predictors of soft drug initiation and time to initiation among youth. Research Questions #2 and #3 of the current research constituted the key parameters that guided this effort. Unlike many previous reviews of the literature, this assessment applied a systematic methodology to the collection of primary studies and the organization and presentation of results. Predictors of soft drug initiation and time to initiation were investigated for six periods of adolescence: early-late adolescence (12-17 years, or 6^{th}-12^{th} grade), early adolescence (12-13 years, or 6^{th}-8^{th} grade), early-mid adolescence (12-15 years, or 6^{th}-10^{th} grade), mid-adolescence (14-15 years, or 9^{th}-10^{th} grade), mid-late adolescence (14-17 years, or 9^{th}-12^{th} grade), and late adolescence (16-17 years, or 11^{th}-12^{th} grade).

Second, conducting this review provided a means by which the internal and external validity of findings for H2 and H3 (tested in the quantitative aspect of the current research) could be cross-validated. Findings from the systematic review cross-validation are presented in Chapter 10.

This chapter is comprised of three major sections. Before specific findings are presented, the 36 primary studies first are described in terms of research design, sample characteristics, major methodological and analytic features, and study limitations. The chapter concludes by directing attention toward the major findings that emerged, as well as the gaps in the extant literature that the quantitative component of the current research addressed.

Summary Description of the Primary Studies

To provide some context for the review, many of the basic study characteristics that were culled from each of the 36 primary studies (see Appendix F) were placed in tabularized format. Table 27 highlights the common features attendant to the 36 primary studies included in the review.

Table 27.

Primary Studies, Basic Descriptives

Study Component and Corresponding Average Characteristic		Percentage or Number of Studies [a]
Design:	▪ Longitudinal	61%
Geographic Location of Sample:	▪ Urban	44%
Location of Data Collection:	▪ Classrooms	80%
Data Source:	▪ Self-report survey	92%
Probability Sampling Method:	▪ No (school district-wide)	58%

(Table 27 continues)

(Table 27 continued)

Study Component and Corresponding Average Characteristic		Percentage or Number of Studies [a]
Efforts to Reduce Under/Over-Reporting:	▪ No	46%
Cross-Validation:	▪ No	94%
Analytic Technique:	▪ Binary logit	47%
Initiation Models by Age Group and Outcome:	▪ Early-Late Adolescence = 14 models	A = 3, C = 6, M = 5
	▪ Early Adolescence = 19 models	A = 6, C = 7, M = 6
	▪ Early-Mid Adolescence = 4 models	A = 2, C = 2
	▪ Mid-Adolescence = 10 models	A = 2, C = 3, M = 5
	▪ Mid-Late Adolescence = 9 models	A = 3, C = 3, M = 3
	▪ Late Adolescence = 4 models	A = 1, C = 2, M = 1
Major Limitations:	▪ Domains/predictors- limited number	69%
	▪ Major known predictors- not assessed	50%
	▪ Limited generalizability	42%
	▪ Attrition/low participation rate/exclusion	39%
	▪ Direction of NS relationships- no report	28%
	▪ Small T1-T2 interval	23% (of long. studies)

[a] A = Alcohol initiation, C = Cigarette initiation, M = Marijuana initiation.

Research Design, Sample Characteristics, and Methodology

The majority (61%) of the primary studies collected longitudinal data from school district-wide samples (58%) of students in classroom settings (80%). On average, students from which data were derived resided in urban (44%) geographic areas, although 11 studies (41%) collected data from mixed geographic areas (e.g., urban, rural, and suburban; urban and rural; or urban and suburban). Only one study (Skinner et al., 1985) collected data from rural adolescent youth.

Some of the more common study design elements included the following. The bulk (92%) of the data collected by the primary researchers were obtained through the self-report survey method. On average, no overt strides were taken to reduce under- or over-reporting, although some researchers did make use of code names (e.g., Bailey & Hubbard, 1990; D'Amico & McCarthy, 2006; Ellickson et al., 2004), instead of personal identifying information. As well, eight studies (e.g., Chassin et al., 1984; Chassin et al., 1986; Ellickson et al., 2004; Epstein et al., 1999; Foshee & Bauman, 1990; Gritz et al., 2003; Skinner et al., 1985; Werch et al., 1997) took advantage of a CO or saliva pipeline. Five projects (e.g., Brook et al., 1986; Crum et al., 2005; D'Amico & McCarthy, 2006; Duncan et al., 1998; Epstein et al., 1995) issued respondents' a Certificate of Confidentiality. Concerning the cross-validation of findings, Chassin and colleagues (Chassin et al., 1984; Chassin et al. 1986) were the only researchers to compare their results to those derived from similar samples.

By far, binary logistic regression constituted the most common analytic technique used to arrive at findings. The second most common statistical technique included discriminant function analysis, which was employed in four studies (Brook et al., 1986; Chassin et al., 1984; Skinner et al., 1985; Smith & Fogg, 1978). Two studies took advantage of structural equation modeling (Crum et al. 2005; Marcos & Bahr, 1988) and another pair employed survival analysis (Hawkins et al., 2002b; Unger & Chen, 1999). Finally, individual studies employed multinomial logistic regression (Flay et al., 1998), path analysis (Kandel & Andrews, 1987), latent growth modeling (Duncan et al., 1998), multiple classification analysis (Kandel et al., 1976), hierarchical linear modeling (Pokorny et al., 2003), and contemporary log-log modeling (Williams et al., 2007).

Soft Drug Initiation by Period of Development

Systematic review findings were organized by the type of outcome investigated and the biological ages of respective samples. Discussed in Chapter 5, as well as earlier in this chapter (and notated in Table 27), the primary studies that investigated predictors of soft drug initiation obtained data from samples of youth whose biological ages constituted one of six periods of adolescent development. In particular, a total of 14 prediction models centered on alcohol, cigarette, and marijuana initiation among early to late adolescents. Since these youth were between 12-17 years (or 6th-12th grade) at the time that respective measures of alcohol initiation were measured, this sample population coincides with the total sample used in the quantitative component of the current research.

Among early-adolescent youth (12-13 years, or 6th-8th grade), six models were used to predict alcohol and marijuana initiation, while seven models were developed to predict cigarette initiation. Findings from these studies have implicational value for the quantitative component of the current research, particularly as it concerns 6th grade data derived from the 2004 PPAAUS.

A few studies obtained data from youth whose biological ages ranged from early-mid adolescence (12-15 years, or 6th-10th grade) at the time the outcome constructs were measured. Two models each predicted alcohol and cigarette initiation.

A total of 10 soft drug initiation models were developed with initiation outcome data from youth who ranged in age from 14-15 years (or 9^{th}-10^{th} grade). Findings from these samples of mid-adolescent youth coincide with the soft drug initiation results derived from data obtained from the 9^{th} grade students who completed the 2004 PPAAUS.

Some primary researchers also investigated soft drug initiation among samples of youth who ranged in age from 14-17 years (i.e., mid-late adolescence). In particular, the systematic review reports on findings from nine prediction models that were developed with outcome data from this age group. Three models each were used to predict alcohol, cigarette, and marijuana initiation.

Finally, several researchers obtained initiation outcome data from late adolescent youth, all of whom ranged in age from 16-17 years (or 11^{th}-12^{th} grade). One model each was developed to predict alcohol and marijuana initiation, while two prediction models centered on cigarette initiation.

Major Limitations

Six major limitations were identified across the primary studies that were reviewed (Table 27). First, nearly 70% of the 36 primary studies confined analyses to a limited number of ecological domains and predictors. To illustrate, Flewelling and Bauman (1990) assessed the impact of family structure variables (e.g., single- and step-parent family structure) on alcohol, cigarette, and marijuana initiation among youth during early and mid-adolescence. Although both cross-sectional and longitudinal data were utilized, and several controls were employed, these researchers did not examine predictors from other ecological domains (e.g., community, school, peer, or individual).

The second major limitation coincided with the most glaring drawback that was identified. Just as the bulk of the studies confined analyses to limited types of ecological influence, 50% of the 36 studies did not direct attention toward many of the salient predictors that have been identified through extant comprehensive reviews of the literature (see Chapter 4). A second case-in-point may be drawn from Flewelling and Bauman's (1990) research. By limiting their examination to two family structural variables (i.e., single-parent and step-parent family structure), the utility of several major family predictors of soft drug initiation (i.e., parental drug use and attendant norms), as well as other ecological influences, were not evaluated.

Third, judging from the information that was included in published reports, the findings from 42% of the primary studies examined appears to be limited to the specific sample populations from which data were derived. For instance, several researchers (see, e.g., Flewelling & Bauman, 1990; Foshee & Bauman, 1990; Kandel et al., 2004; Pokorny et al., 2003; Shears et al., 2006; Skinner et al., 1985) did not report the demographic make-up of the samples upon which their findings were based. Moreover, of the researchers who did report on the racial distribution of respective samples, several studies obtained data from predominantly white (see, e.g., Duncan et al., 1998; Webb et al., 1991) or black (see, e.g., Crum et al., 2005; Epstein et al., 1999, 1995; Tilson et al., 2004; Werch et al., 1997) samples.

Fourth, although not a significant problem in the aggregate, several studies suffered from attrition, low participation rates, missing data, and the inability to match respondents across longitudinal waves, all of which led to the exclusion of cases (D'Amico & McCarthy, 2006; Flay et al., 1998; Flewelling & Bauman, 1990; Foshee & Bauman, 1990; Tilson et al., 2004). For example, in Flay et al.'s (1998) longitudinal examination of school, peer, family, and individual-related differences between adolescent cigarette abstainers and initiates, experimenters, and regular users, 50% of the sample initially recruited at T1 dropped out of the study by T2.

Fifth, although the social sciences typically regard significant associations as more informative and of higher practical value than insignificant relationships (Kachigan, 1986), publication standards established by the American Psychological Association (APA) suggest that researchers report the directional effects exerted by variables not meeting typical significance thresholds (i.e., p <.05, p <.01; see Morgan, Reichert, & Harrison, 2002). Aside from this technical reporting requirement, making known the direction of insignificant relationships has the potential to inform and advance knowledge on both etiology and prevention fronts, such as furthering an understanding of how constructs operate (i.e., increase or decrease risk) and what theoretical explanations yield the most empirical support.

Tempering the strides that were taken to summarize the nature of all of the non-significant findings, 10 of the 36 primary studies did not report the direction of insignificant effects (e.g., Chassin et al., 1984; Epstein et al., 1999, 1995; Gritz et al., 2003).[7] In instances where vote-counting was used to summarize modal findings, only those relationships for which the direction of effects were reported were eligible for inclusion.

Finally, although not a major problem in the aggregate, it important to mention that 23% of the longitudinal studies included in this systematic review measured extraneous and outcome variables within a relatively short span of time (i.e., one-year or less, see, e.g., Flay et al., 1994; Gritz et al., 2003; Kandel & Andrews, 1987; Urberg et al., 1997). To illustrate, Kandel and Andrews (1987) investigated the relationship between family, peer, and individual influences and alcohol and marijuana initiation among a sample of 1,110 adolescent-best friend-parent triads, by gathering extraneous data 5-6 months prior to measuring alcohol and marijuana initiation.

The following section presents the findings from the systematic review. The direction and significance of each relationship are organized by ecological domain and presented in tabular format. As outlined in Chapter 5, vote-counting was used as a discussionary tool in summarizing relationships and drawing conclusions concerning the degree of importance and the direction of effects most frequently exerted by predictors. Since vote-counting requires that multiple findings be summed, and quite a few of the predictors were evaluated in only one study, this method of reporting was not able to be employed for each of the predictors reported below.

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[7] Since several of these studies investigated the initiation of multiple soft drugs among several distinct periods of adolescent development, this report failure resulted in a relatively large number of findings for which the direction of the effects was unknown. These tabularized findings are notated with a question mark (?). Each study that failed to report the direction of insignificant relationships are identified in Appendix F.

Soft Drug Initiation

Community Domain Predictors

A total of 12 primary studies examined the efficacy of one or more community domain predictors of soft drug initiation (see Table 28). The majority of the findings stemmed from data derived from early adolescent youth. In fact, only four findings have relevance for soft drug initiation among mid-adolescents, and none of the primary studies examined the utility of community influences and characteristics for predicting soft drug initiation from distinct samples of late adolescent youth.

Of the community domain influences and characteristics that were investigated, social learning constructs, particularly non-familial adult drug use and norms, were the most frequently assessed. Surprisingly, none of the primary studies evaluated the importance of community bonding constructs on soft drug initiation. Another notable pattern across findings centered on the types of substances that alcohol, cigarette, and marijuana use by non-familial adults were used to predict. Along with drug-related norms, the efficacy of these measures tended to be evaluated in models predicting the initiation of the soft drugs to which these measures referred. For example, non-familial alcohol use only was examined within the context of alcohol initiation, while non-familial cigarette and marijuana use were examined solely in the context of cigarette and marijuana initiation, respectively.

Many community domain variables constitute salient risk factors for soft drug initiation. Of the 18 social learning constructs and community characteristics

Table 28.

Community Domain Predictors of Soft Drug Initiation: Directional Relationships by Period of Adolescent Development [a b]

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Social Learning:						
Non-familial adult alcohol use	11(+, E) 35(+, E)					
Non-familial adult cigarette use			04b(+, M-L) 04c(+, E-L)	04(?, E)		
Non-familial adult marijuana use					09d(+, E)	09a(+, E) 09c(+, M)
Non-familial adult pro-drug norms						02a,b(-, E) 02c(-, M)
Pro-tobacco messages				17(+, E-L)		
Exposure to smoking in media			04a(+, E)			
Tolerant cigarette media messages/depictions				04a(?, E-L)		
Cues to use alcohol		35(?, E)				
Marijuana offers					09a,d(+, E) 09b,c(+, M)	

(Table 28 continues)

(Table 28 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Community Characteristics:						
Alcohol availability		11(?, E)				
Cigarette availability			25(+, E)			
Rate of tobacco retailers				23(?, E)		
Prohibition of cigarette vending machines			20(-, E-L)			
Prohibition of tobacco marketing			20(+, E-L)			
Low cigarette sales tax			20(+, E-L)			
Low community SES					26(+, E-L)	
Neighborhood disadvantage	06(-, E)					

Note. ? = direction of relationship unknown; not reported in publication.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.
[b] E-L = Early-Late Adolescence; E = Early Adolescence; M = Mid-Adolescence; M-L = Mid-Late Adolescence.

examined in previous research, vote-counting revealed full convergence for the directional findings attendant to four predictors (i.e., non-familial adult alcohol use, non-familial adult cigarette use, non-familial adult marijuana use, and marijuana offers).

Investigated by both Epstein et al. (1999) and Werch et al. (1997), non-familial adult alcohol use serves as a salient risk factor for alcohol initiation among early adolescents. Cigarette use by non-familial adults increases risk for cigarette initiation among early-late and mid-late adolescent youth (Chassin et al., 1984). Marijuana use among non-familial adults, evaluated by Ellickson et al. (2004) in predicting marijuana initiation among early and mid-adolescent youth, was found to increase risk for initiation, although only one association was significant at the .05 level. Finally, Ellickson et al. (2004) also assessed the utility of marijuana offers in predicting marijuana initiation among two samples of early and two samples of mid-adolescent youth. All four findings indicate that early and mid-adolescent youth who are offered marijuana by individuals in the community are at an increased risk for marijuana initiation.

Of the 18 community domain predictors that were investigated, the directional effects exerted by three variables (i.e., non-familial adult pro-drug norms, prohibition of tobacco marketing, and neighborhood disadvantage) countered theoretical and intuitive expectations. First, using self-report longitudinal survey data from a sample of adolescents during early adolescence (7th and 8th grade students at Time 2) and mid-adolescence (9th graders at

Time 2), Bailey and Hubbard (1990) found an inverse (but insignificant) association between the pro-drug norms of non-familial adults and adolescent marijuana initiation. This inverse relationship held across both periods of adolescence.

Second, in their longitudinal examination of predictors of cigarette initiation, Kandel et al. (2004) found that among 5,347 early-late adolescent youth from urban, suburban, and rural communities across the U.S., youth who lived in communities in which tobacco marketing was prohibited were at a greater risk for cigarette initiation than adolescents who resided in communities that did not enact such a ban. This is an interesting finding, particularly given the fact that the official data used in developing this extraneous variable was collected before the self-report cigarette initiation data were measured. In line with MacCoun and Reuter's (2001) notion of the "forbidden fruit" effect that drug law mechanisms constitute, it is possible that adolescents' knowledge of the marketing ban, coupled with their knowledge of cigarette use as a status offense, may have inadvertently elicited such an effect, thereby inadvertently promoting the initiation and use of cigarettes among these adolescents. Kandel and associates did not assess nor report on the sample's knowledge of a ban on tobacco marketing, so it is unclear how likely this "forbidden fruit" effect may have been operating.

Finally, the sole finding concerning the direction of effects exerted by neighborhood disadvantage on risk for alcohol initiation counters one of the major propositions put forth by Shaw and McKay's (1969) social disorganization theory. Specifically, Crum et al.'s (2005) longitudinal analysis found that this measure of social disorganization served as a salient protective factor in decreasing risk for alcohol initiation among predominantly inner-city, black 8th grade students.

School Domain Predictors

The directional relationships for the association between school-related factors and soft drug initiation are presented in Table 29. Of the school domain factors investigated, school bonding constructs have received the most attention, while school-related behavior and school characteristics have been studied the least.

Some general findings centered on the selective attention paid to the predictive utility of school factors for explaining soft drug initiation within various periods of adolescence. None of the primary studies included in this review evaluated school predictors of soft drug initiation among early-mid adolescent youth or alcohol initiation during late adolescence. Further, limited research investigated school predictors of alcohol initiation during early-late (Urberg et al., 1997), mid- (Walter et al., 1991), and mid-late (Kandel et al., 1976) adolescence; cigarette initiation during late adolescence (Flay et al., 1998); and marijuana initiation among early-late (Shears et al., 2006) and late (Smith & Fogg, 1978) adolescent youth.

Full convergence. Evidence of full convergence was observed for seven of the eight school factors for which multiple investigations were conducted. Of the four school factors evaluated in models predicting alcohol initiation, consistent directional findings were found for the risk effects exerted by three variables.

Table 29.

School Domain Predictors of Soft Drug Initiation: Directional Relationships by Period of Adolescent Development [a b]

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
<u>Bonding</u>:						
Poor school performance	21(+, M-L)	06(+, E) 33(?, M) 34(?, E)	25(+, E)	13(+, L) 17(?, E-L)	02b(+, E) 02c(+, M) 09a,d(+, E) 09b,c(+, M) 12(+, E) 21(+, M-L)	02a(+, E) 28(?, L) 33(?, M)
Low school commitment	21(+, M-L)	06(+, E) 32(?, E-L)	04(+, E, E-L)	32(?, E-L)	21(+, M-L)	
High school commitment			04b(-, M-L) 16(-, M-L) 27a,c(-, M-L)	27b (-, M)		28(?, L)
Low school attachment						02c(-, M)
High school attachment			20(-, E-L)		22(-, M-L) 26(-, E-L)	02a(-, E) 02b(+, E)

(Table 29 continues)

(Table 29 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Comfort among classmates						02a(+, E) 02b(+, E) 02c(+, M)
Behavior:						
Absenteeism/truancy	11(+, E) 21(+, M-L)	33(?, M)		33(?, M)		33(?, M)
Detention/suspension		33(?, M)	17(+, E-L)	33(?, M)		33(?, M)
% students smoke			20(+, E-L)			
School Characteristics:						
% minority				20(?, E-L)		
Strict smoking policy				20(+, E-L)		

Note. ? = direction of relationship unknown; not reported in publication.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.
[b] E-L = Early-Late Adolescence; E = Early Adolescence; M = Mid-Adolescence; M-L = Mid-Late Adolescence; L = Late Adolescence.

In particular, evidence from both longitudinal and cross-sectional work indicates that poor school performance (Crum et al., 2005; Kandel et al., 1976), low school commitment (Crum et al., 2005; Kandel et al., 1976), and absenteeism and truancy (Epstein et al., 1999; Kandel et al., 1976) constitute salient risk factors for alcohol initiation. The sole study (Walter et al., 1999) that investigated the predictive utility of detention/suspension did not report the direction of insignificant effects.

With respect to cigarette initiation, the directional findings for one risk factor and one protective factor were consistent. Poor school performance serves as a risk factor for cigarette initiation during both early (Robinson et al., 1997) and late (Flay et al., 1998) adolescence, while the five findings from three longitudinal studies (Chassin et al., 1984; Foshee & Bauman, 1990; Skinner et al., 1985) indict low school commitment as a salient risk factor for cigarette initiation among mid-late adolescent youth. No studies evaluated the utility of this construct for predicting cigarette initiation during early adolescence.

Finally, multiple findings were consistent in underscoring the risk capacity of poor school performance for predicting marijuana initiation among youth of all ages (Bailey & Hubbard, 1990; Ellickson et al., 2004; Epstein et al., 1995; Kandel et al., 1976). As the findings for the initiation of all three soft drugs indicate, poor school performance constitutes a generic risk factor that is associated with an increased risk for initiating all three soft drugs.

Consistent support also was found for the risk effects exerted by a second type of bonding construct, comfort among classmates. In Bailey and Hubbard's

(1990) longitudinal study, 7th, 8th, and 9th grade students who perceived high levels of comfort among classmates were found to be at an increased (but insignificant) risk for marijuana initiation.

Mixed findings. Although 75% of the findings are consistent, more research is needed to clarify the age-graded impact of high levels of school attachment on marijuana initiation. Two cross-sectional studies found that this bonding construct was important in decreasing risk for marijuana initiation during early-late (Shears et al., 2006) and mid-late (Marcos & Bahr, 1988) adolescence. In Bailey and Hubbard's (1990) longitudinal study of marijuana initiation among 7th-9th grade students, high school attachment also was associated with a decrease (although insignificant) in the likelihood of marijuana initiation among 7th and 9th grade students. Among 8th graders, however, these researchers found an inverse relationship.

Family Domain Predictors

Family influences play an important role in explaining soft drug initiation among youth. Of the 34 primary studies that examined predictors of soft drug initiation, almost 50% (n = 15) investigated one or more familial determinants (see Table 30). Before an overview of family risk and protective factors is presented, it is important to direct attention toward five thematic findings that emerged.

First, with the exception of marijuana initiation among mid-late and late adolescent youth, the importance of family factors has been studied with samples of youth from all major periods of adolescence. The evidence is scant, however,

Table 30.

Family Domain Predictors of Soft Drug Initiation: Directional Relationships by Period of Adolescent Development[a b]

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
<u>*Bonding*</u>:						
Strong familial bond	21(-, M-L)	19(?, M-L)			21(-, M-L) 24(-, E-L)	19(?, M-L)
Maternal attachment			16(-, M-L) 27a,c(-, M-L)	27b(-, M)		
Paternal attachment			16(-, M-L) 27a,c(+, M-L)	27b(+, M)		
Value spending time with parents		32(?, E-L)		32(?, E-L)		
Importance of parents versus peers						02a(-, E) 02b(+, E) 02c(+, M)
<u>*Social Learning*</u>:						
Parental drug use	21(+, M-L)				21(+, M-L)	
Parental alcohol use	19(+, M-L) 21(+, M-L)	10(+, E-M)		10(-, E-M)	21(+, M-L)	19(?, M-L)

(Table 30 continues)

(Table 30 continued)

Predictors	Alcohol Initiation *p* <.05 (+/-)	Alcohol Initiation *p* >.05 (+/-)	Cigarette Initiation *p* <.05 (+/-)	Cigarette Initiation *p* >.05 (+/-)	Marijuana Initiation *p* <.05 (+/-)	Marijuana Initiation *p* >.05 (+/-)
Maternal alcohol use	11(+, E)					
Paternal alcohol use	11(+, E)	34(?, E)				
Older sibling alcohol use	11(+, E)					
Parental cigarette use	10(+, E-M)		04a,c(+, E, E-L) 05(+, E-L) 10(+, E-M) 13(+, L)	04b(?, M-L) 20(+, E-L) 23(+, E) 25(+, E)		
Maternal cigarette use				16(-, M-L) 27a,c(-, M-L) 27b (-, M)		
Paternal cigarette use				16(+, M-L) 27a,c(+, M-L) 27b(+, M)		
Older sibling cigarette use			04a,c(+, E, E-L)	04b(?, M-L)		
Household smoker			17(+, E-L)			
Older sibling marijuana use					09d(+, E) 09b(+, M)	09a(+, E) 09c(-, M)

(Table 30 continues)

(Table 30 continued)

Predictors	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
	p <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Parental pro-alcohol norms		19(?, M-L)				
Parental pro-cigarette norms				05(+, E-L) 13(+, L) 17(?, E-L)		
Parental pro-marijuana norms						09a,d(+, E) 09b(+, M) 09c(-, M) 19(?, M-L)
Parental pro-drug norms		10(+, E-M)		10(-, E-M)		
Relationship Factors:						
Little clarity of family rules	36(+, E-L)		04c(+, E-L)		36(+, E-L)	
Clear rules about legal drug use		10(+, E-M)		10(+, E-M)		
Parental consistency in expectations				04b(?, M-L) 34(?, E)		
Parent-child conflict	34(+, E)					
Low family conflict				13(-, L)		

(Table 30 continues)

(Table 30 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Low parental support	33(+, M)		33(+, M)			33(?, M)
Parental prosocial support		10(+, E-M)		04a,c(?, E, E-L) 04b(?, M-L) 05(-, E-L) 10(-, E-M)		12(?, E)
Good communication with parents					02a(+, E) 02b(-, E)	
Good communication—media portrayals & consequences of legal drug use		10(+, E-M)		10(-, E-M)		
Importance of comm. & harmony w/ parents					02a,b(-, E)	02c(-, M)
Parental strictness				04b(?, E-L) 04c(?, M-L) 05(+, E-L) 34(?, E)		
Parental harmony & warmth	03(-, L)					
Good parent-child relationship	01(-, E-L)	34(?, E)	01(-, E-L)	20(-, E-L)	09a(-, E)	01(-, E-L) 02a,c(-, E, M) 02b(+, E) 09b,c,d(-, M, E)

(Table 30 continues)

(Table 30 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Good relationship with mother		32(?, E-L)		32(?, E-L)		
High parental monitoring	36(-, E-L)	01(-, E-L) 10(-, E-M) 32(?, E-L)		01(+, E-L) 10(+, E-M) 27a,b(-, M, M-L) 27c(+, M-L) 32(?, E-L)	24(-, E-L) 36(-, E-L)	01(-, E-L)
<u>*Family Characteristics*</u>:						
Divorce		01(+, E-L)	01(+, E-L)		01(+, E-L)	
Step-parent family	15(+, E, M-L)	01(+, E-L)	15(+, E)	01(+, E-L) 15(+, M-L)	01(+, E-L) 15(+, M-L)	
Single-parent family	15(+, E)	01(+, E-L) 15(+, M-L)	01(+, E-L) 15(+, E, M-L) 20(+, E-L) 29(+, E)		15(+, M-L) 26(+, E-L)	01(+, E-L) 12(?, E)
Two-parent family		10(+, E-M)		10(-, E-M)		09a,d(-, E) 09b,c(-, M)

Note. ? = direction of relationship unknown; not reported in publication.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.
[b] E-L = Early-Late Adolescence; E = Early Adolescence; E-M = Early-Mid Adolescence; M = Mid-Adolescence; M-L = Mid-Late Adolescence; L = Late Adolescence.

with respect to certain developmental periods. For example, one study investigated the utility of family constructs for predicting alcohol and cigarette initiation among early-mid adolescents (Ennett et al., 2001), while two others evaluated family predictors of alcohol initiation during mid- (Walter et al., 1991) and late (Brook et al., 1986) adolescence. Finally, one longitudinal study (Flay et al., 1998) used extraneous data from 7th grade students in predicting cigarette initiation by 12th grade.

Second, parents predominantly influence the soft drug behavior of youth by modeling the use of these drugs and providing environmental contexts for transmitting norms concerning the acceptability of use (Donovan, 2004). As this review revealed, the social learning constructs of parental soft drug use and attendant norms constitute some of the more widely investigated familial influences, particularly parental soft drug use.

Third, in addition to parents, older siblings constitute role models and influential agents who also can increase risk for soft drug initiation. The mechanisms through which siblings influence adolescent drug behavior include modeling, access to these substances, and direct social influence, such as overt acts of pressure to initiate (Donovan, 2004). Of the three types of influence, sibling drug use is the only mechanism to which primary studies directed attention, and this social learning construct was investigated far less frequently than parental drug use.

Fourth, of the four elements of Hirschi’s (1969) social bond, parental attachment was the most widely investigated. Unfortunately, though, assessments of the utility of this construct were evaluated with respect to cigarette initiation only.

Finally, of the diverse number of family relationship factors that have been studied, parental monitoring has been the most frequently investigated. Several familial characteristics also have been evaluated, particularly single-and step-parent family structure. By far, the differential impact of single-parent family structure versus two-parent structure has received the most attention.

Full convergence. Of those predictors for which multiple investigations were conducted, 18 sets of directional findings for 13 factors fully converged. Concerning alcohol initiation, vote-counting only was possible for the multiple findings directed at four variables, three of which constitute salient risk factors. Parental alcohol use constitutes a risk factor for alcohol initiation among early-late adolescent youth (Ennett et al., 2001; Kandel & Andrews, 1987; Kandel et al., 1976), while early-late adolescent youth from both step-parent and single-parent families are at a higher risk for alcohol initiation, as compared to youth from two-parent families (Amey & Albrecht, 1998; Flewelling & Bauman, 1990). Finally, the directional results from three studies (Amey & Albrecht, 1998; Ennett et al., 2001; Williams et al., 2007) suggest that high parental monitoring decreases risk for alcohol initiation among youth of all ages.

Consistent findings were yielded for six family risk factors that increase risk for cigarette initiation: parental and older sibling cigarette use (for all periods of adolescence); paternal cigarette use (among mid-late adolescents); parental pro-cigarette norms (for youth of all ages); and step-parent and single-parent family structure (for all periods of adolescence). Full convergence in multiple findings identified four variables as protective factors for cigarette initiation. These constructs include maternal attachment (Foshee & Bauman, 1990; Skinner et al., 1985), maternal cigarette use (Foshee & Bauman, 1990; Skinner et al., 1985), parental prosocial support (Chassin et al., 1984; Chassin et al., 1986; Ennett et al., 2001), and good parent-child relationship (Amey & Albrecht, 1998; Kandel et al., 2004).

Finally, with respect to predictors of marijuana initiation that have received consistent support, findings indicate that complete convergence in directional effects is far more common for protective factors than risk factors. In particular, among youth of all ages, a strong familial bond decreases the likelihood of marijuana initiation (Kandel et al., 1976; Ramirez et al., 2004). The likelihood of marijuana initiation also is lower among early and mid-adolescent youth who believe that communication and harmony with parents is important (Bailey & Hubbard, 1990). Similar to the findings for the protective effects observed for alcohol and cigarette initiation, adolescents of all ages who self-report high levels of parental monitoring are less likely to initiate marijuana use (Amey & Albrecht, 1998; Ramirez et al., 2004; Williams et al., 2007). Although the four findings from Ellickson et al.'s (2004) work did not meet the conventional level of significance (i.e., $p < .05$), the direction of effects observed was consistent in identifying the two-parent family structure as a protective factor for marijuana initiation during early and mid-adolescence.

Only two risk factors for marijuana initiation were consistently identified through past research. The findings from Amey and Albrecht (1998), Flewelling and Bauman (1990), and Shears et al. (2006) indicate that among youth of all ages, those who live in step-parent and single-parent families are at a higher risk for marijuana initiation, as compared to their counterparts living in two-parent families.

Mixed findings. Some of the respective findings for the directional effects exerted by seven family predictors conflicted, but vote-counting results indicate that the majority of these respective findings did largely coalesce. Perhaps due to the fact that the bulk of the variables assessed with respect to alcohol initiation were evaluated in one study only, no mixed findings were identified for this outcome measure.

Mixed results were yielded for the directional effects exerted by two factors (paternal attachment and high parental monitoring) in predicting cigarette initiation. Although 75% of the findings for paternal attachment suggest that this factor increases risk for cigarette initiation, Foshee and Bauman's (1990) longitudinal research indicates that a high level of parental attachment constitutes a salient protective factor for cigarette initiation. Since the point of conflict centers on Foshee and Bauman's (1990) and Skinner et al.'s (1985) longitudinal findings for mid-late adolescent youth, more research is needed for this period of adolescence.

The findings for five predictors (i.e., importance of parents versus peers, older sibling marijuana use, parental pro-marijuana norms, good communication with parents, and good parent-child relationship) of marijuana initiation did not fully converge, suggesting that more research should be conducted on these fronts. First, the three findings from Bailey and Hubbard's (1990) longitudinal study of 3,454 adolescents suggest that the degree to which early and mid-adolescent youth prioritize parents over peers does not have a significant impact on their risk for marijuana initiation. The direction of effects exerted by this construct conflicted, however. During 7^{th} grade, the prioritization of parents over peers resulted in a decreased risk for marijuana initiation, while this construct increased risk for marijuana initiation during both 8^{th} and 9^{th} grade. The differential effects of this predictor may partly be a result of age effects.

Second, of the four longitudinal and cross-sectional findings reported by Ellickson et al. (2004) for the directional effects exerted by older sibling marijuana use, three converged in underscoring the importance of this predictor as a risk factor for marijuana initiation during 7^{th}, 8^{th}, and 9^{th} grade. During 7^{th} and 8^{th} grade, marijuana use by older siblings served as a salient risk factor. By 10^{th} grade, however, these researchers found that youth who self-reported having older siblings who used marijuana were less likely to initiate marijuana use. This inverse relationship was insignificant at the .05 level, however.

Third, the direction of effects exerted by parental pro-marijuana norms among Ellickson et al.'s (2004) sample of 10^{th} grade students countered the direction of effects observed during 7^{th}, 8^{th}, and 9^{th} grade. All four findings were non-significant, however, suggesting that parents' permissiveness concerning marijuana use is not an important factor in predicting marijuana initiation among early and mid-adolescent youth. Nonetheless, three of Ellickson et al.'s (2004) findings indicate that this factor increases risk for marijuana initiation among 7th, 8th, and 9th grade students, while decreasing risk for initiation among 10th grade students.

Fourth, not only is more research needed to clarify the relationship between marijuana initiation and good parent-child communication, but more insight is required in determining whether the direction of effects differ by biological age. In their longitudinal research examining predictors of marijuana initiation by 7th, 8th, and 9th grade, Bailey and Hubbard (1990) found that regardless of the biological age at which marijuana initiation was measured, good communication with parents served as a salient predictor. During 8th grade, students who self-reported having strong communication with their parents were at a significantly lower risk for marijuana initiation. During 7th grade, however, adolescents who self-reported strong parent-adolescent communication were at a significantly higher risk for marijuana initiation.

Finally, almost 90% of the directional findings for the relationship between good parent-child relationship and marijuana initiation converged. Seven findings across three studies (Amey & Albrecht, 1998; Bailey & Hubbard, 1990; Ellickson et al., 2004) speak to the protective capacity of this factor for youth of all ages. Among Bailey and Hubbard's (1990) sample of 8th grade students, however, youth who reported having a strong relationship with their parents were at an increased (but insignificant) risk for marijuana initiation.

Peer Domain Predictors

Table 31 presents the directional relationships between soft drug initiation and a variety of peer domain variables. In general, peer constructs consistent with social learning theory and the SDM were investigated more frequently than social bonding variables. In fact, only four primary studies examined measures of peer bonding. Three major thematic findings are worth discussing.

First, no primary studies evaluated peer domain predictors of marijuana initiation among early-mid and late adolescent youth. Only two studies investigated peer factors related to alcohol initiation during mid-adolescence (Walter et al., 1991) and late adolescence (Brook et al., 1986), while two others assessed peer constructs in prediction models explaining cigarette initiation during early-mid adolescence (Urberg et al., 1997) and late adolescence (Flay et al., 1998).

Second, of the peer domain factors assessed by the primary studies, the social learning construct of peer soft drug use (e.g., alcohol, cigarette, and marijuana), as well as peer drug norms (to a lesser extent), were the most frequently investigated and empirically supported predictors identified in this review. These general findings are consistent with those revealed through other literature reviews (see, e.g., Conrad et al., 1992; Halebsky, 1987; Penning & Barnes, 1982; Petraitis et al., 1998; Windle et al., 2008).

Finally, as was found for non-familial adult soft drug use (and parental and sibling soft drug use and norms), the bulk of the research on peer alcohol, cigarette, and marijuana use (and norms) tended to evaluate the efficacy of

Table 31.

Peer Domain Predictors of Soft Drug Initiation: Directional Relationships by Period of Adolescent Development [a b]

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Bonding:						
Attachment to peers			27a(+, M-L)	27b(+, M) 27c(+, M-L)		
Involvement with peers	21(+, M-L)				21(+, M-L)	
Importance of peers versus family						02a,b(+, E) 02c(+, M)
Dependence on peers						02a,b(+, E) 02c(+, M)
Strong friendship	32(+, E-L)		32(+, E-L)			
Social Learning:						
Peer alcohol use	06(+, E) 07(+, E) 11(+, E) 32(+, E-L) 33(+, M) 35(+, E)	34(?, E) 19(?, M-L)			07(+, E)	

(Table 31 continues)

(Table 31 continued)

Predictors	Alcohol Initiation *p* <.05 (+/-)	Alcohol Initiation *p* >.05 (+/-)	Cigarette Initiation *p* <.05 (+/-)	Cigarette Initiation *p* >.05 (+/-)	Marijuana Initiation *p* <.05 (+/-)	Marijuana Initiation *p* >.05 (+/-)
Same-grade alcohol-using peers		33(?, M)				
Best friend alcohol use	11(+, E)	19(?, M-L)				
Peer cigarette use		07(-, E)	05(+, E-L) 14(+, E) 16(+, M-L) 17(+, E-L) 20(+, E-L) 23(+, E) 25(+, E) 32(+, E-L) 33(+, M)	07(+, E) 13(+, L)		07(-, E)
Same-grade cigarette-using peers				33(?, M)		
Best friend cigarette use			04b(+, E-L) 04c(+, M-L)	04a(?, E)		
Number of smoking friends			13(+, L)			
Male peer cigarette use			04a,c(+, E, E-L)	04b(?, M-L)		
Female peer cigarette use			04a,c(+, E, E-L)	04b(?, M-L)		
Peer alcohol and cigarette use	03(+, L)					

(Table 31 continues)

(Table 31 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Peer marijuana use		07(+, E)		07(-, E)	07(+, E) 09d(+, E) 12(+, E) 19(+, M-L) 26(+, E-L) 33(+, M)	09a(+, E) 09b(+, M) 09c(-, M)
Same-grade marijuana-using peers						33(?, M)
Best friend marijuana use						19(?, M-L)
Peer alcohol and marijuana use					02b(-, E) 02c(+, M)	02a(+, E)
Peer drug use	21(+, M-L)					
Close friend drug use	31(+, E-M)		31 (+, E-M)			
Friendship group drug use		31(+, E-M)		31(+, E-M)		
Peer deviance/delinquency	03(+, L) 36(+, E-L)				36(+, E-L)	
Peer pro-alcohol norms	33(+, M)	34(?, E)				
Peer anti-alcohol norms		19(?, M-L)				

(Table 31 continues)

(Table 31 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Peer pro-cigarette norms			33(+, M)	05(+, E-L) 13(+, L) 17(?, E-L)		
Peer pro-marijuana norms					02c(+, M) 09d(+, E) 09c(+, M) 33(+, M)	09a(+, E) 09b(+, M) 12(?, E)
Peer anti-marijuana norms					19(-, M-L)	
Peer pro-alcohol/marijuana norms					02b(+, E)	02a(-, E)
Peer tolerance of youth's drug use					12(+, E)	
Association with male smoking peers				27a,c(+, M-L) 27b(+, M)		
Association with female smoking peers				27a,c(+, M-L) 27b(-, M)		
<u>*Relationship Factors*</u>:						
Peer conflict		32(?, E-L)		32(?, E-L)		
Peer strictness				05(?, E-L)		

(Table 31 continues)

(Table 31 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Peer support		32(?, E-L)		04a,c(?, E, E-L) 04b(?, M-L) 05(?, E-L) 32(?, E-L)		12(?, E)
Consistency in peer-youth prosocial values			04b(-, E-L) 04c(-, M-L)	04a(?, E)		
Strong communication with peers					02b(+, E)	02a(+, E) 02c(+, M)
Peer academic expectations for youth			05(-, E-L)			

Note. ? = direction of relationship unknown; not reported in publication.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.
[b] E-L = Early-Late Adolescence; E = Early Adolescence; E-M = Early-Mid Adolescence; M = Mid-Adolescence; M-L = Mid-Late Adolescence; L = Late Adolescence.

these constructs for predicting the initiation of those specific drugs to which they correspond. For example, D'Amico & McCarthy (2006) were the only researchers who investigated the predictive utility of peer alcohol use for explaining marijuana initiation, as well as peer cigarette use for explaining alcohol and marijuana initiation. Since their data were derived from early adolescent youth, it is unknown whether their findings hold for older adolescents. Results suggest, however, that peer alcohol use serves as a significant risk factor for marijuana initiation among early adolescents. Interestingly, peer cigarette use was associated with a decreased (but insignificant) risk for both alcohol and marijuana initiation.

Full convergence. In general, of those predictors for which multiple investigations were conducted, the direction of effects for 12 factors not only fully converged, but they met the expectations of the theories (e.g., social bond theory, social learning theory, and the SDM) from which they were derived. With respect to alcohol initiation, vote-counting only was possible for two peer domain predictors. Multiple findings fully converged in indicting peer alcohol use and delinquency as salient risk factors for alcohol initiation. Further, with increases in risk for alcohol initiation observed among all periods of adolescence, the findings suggest that the risk effects of peer alcohol use and delinquency are not restricted to a particular period of adolescence.

Consistent findings were yielded for the following peer factors, all of which were found to increase risk for cigarette initiation: attachment to peers (among mid- and mid-late adolescent youth); peer cigarette use, including best friends' use and male and female peer use (among all periods of adolescence); peer pro-cigarette norms (among all stages of adolescence); and association with male smoking peers (among mid- and mid-late adolescence).

Worth noting is that all three findings for peer attachment counter Hirschi's (1969) proposition concerning the relationship between measures of attachment and delinquency abstention. Instead of reducing the likelihood of cigarette initiation, attachment to peers serves as a risk factor, increasing the likelihood of initiation (Skinner et al., 1985). Notably, although cause may have been confounded by consequence, Kandel et al.'s (1976) cross-sectional research found that involvement with peers served as a salient risk factor for alcohol initiation among mid-late adolescent youth.

The three findings from Chassin et al.'s (1984) longitudinal study provide some solid evidence for the protective capacity demonstrated by a convergence in adolescent-peer prosocial values. Among 7th-8th grade students, 9th-12th graders, and 7th-12th grade students, youth whose prosocial values are consistent with those of their friends are less likely to initiate cigarette use.

Finally, multiple findings were consistent in underscoring the risk capacity of the following five predictors for predicting marijuana initiation: importance of peers versus family (among early and late adolescent youth); dependence on peers (among early and late adolescents); peer pro-marijuana norms (among early and mid-adolescent youth); and strong communication with peers (among early and mid-adolescent youth). With the exception of Ellickson et al.'s (2004) finding of an insignificant negative relationship, peer marijuana use has consistently been shown to increase risk for marijuana initiation among youth of all ages.

Mixed results. Although insignificant at the .05 level, worth mentioning are the two inconsistent findings for the impact that association with female smoking peers exerted in predicting cigarette initiation. Among their sample of junior-high students, Skinner et al. (1985) found an inverse association, while a positive relationship was observed for both their total sample and subsample of high-school students. No other primary studies investigated this differential association construct.

Individual Domain Predictors

Table 32 presents the individual domain predictors of soft drug initiation that were examined in relevant primary studies. In general, adolescents' personal attitudes and intentions were the most widely researched predictors. Three major thematic findings emerged.

First, no primary studies evaluated individual domain predictors of soft drug initiation among early-mid adolescent youth, while only two studies examined individual-related predictors of alcohol initiation among early-late (Urberg et al., 2003) and late (Brook et al., 1986) adolescent youth. Flay et al.'s (1998) longitudinal study constituted the only investigation of individual predictors of cigarette initiation among late adolescent youth. Finally, two other studies investigated individual domain variables associated with marijuana initiation during early-late (Ramirez et al., 2004) and late (Kandel et al., 1976) adolescence.

Table 32.

Individual Domain Predictors of Soft Drug Initiation: Directional Relationships by Period of Adolescent Development [a b]

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Traits/States:						
Sensation-seeking	32(+, E-L)	34(?, E)	25(+, E) 32(+, E-L)			12(?, E)
Risk-taking				13(-, L)		
Impulsivity						28(?, L)
Rebellious					09a(+, E)	09d(-, E) 09b(+, M) 09c(-, M)
Emotional maladjustment		34(?, E)				
Depression	21(+, M-L)	33(?, M)	33(+, M)	17(?, E-L) 20(-, E-L)	21(+, M-L)	33(?, M)
Anxiety	33(+, M)			33(?, M)		33(?, M)
Low self-esteem				32(?, E-L)		

(Table 32 continues)

(Table 32 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Low locus of control		35(+, E)	04a,c(+, E, E-L) 04b(+, M-L)			
Low alcohol refusal self-efficacy		33(?, M) 35(+, E)				
Low cigarette refusal self-efficacy			33(+, M)	13(+, L)		
Low marijuana refusal self-efficacy					09d(+, E) 09b(+, M)	09a(+, E) 09c(+, M) 22(+, M-L) 33(?, M)
Poor life skills					12(+, E)	
Poor decision-making skills					12(+, E)	
Low orientation to work	03(+, L)					
Sociable						28(?, L)

(Table 32 continues)

(Table 32 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Attitudes/Intentions:						
Alcohol use intention	35(+, E)					
Cigarette use intention			04a,c(+, E, E-L) 04b(+, M-L) 14(+, E) 17(+, E-L)		22(-, M-L)	
Marijuana use intention					09a,d(+, E)	09b,c(+, M)
Pro-alcohol norms		33(?, M)				
Pro-cigarette norms				33(?, M)		
Pro-marijuana norms					12(+, E)	33(?, M)
Tolerance of deviance	34(+, E)		04a,c(+, E, E-L) 04b(+, M-L)			
Alcohol use as harmful		19(?, M-L) 32(?, E-L)		32(?, E-L)		
Cigarette use as harmful		32(?, E-L)		32(?, E-L)		

(Table 32 continues)

(Table 32 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Marijuana as harmful					09b,d(-, M, E) 19(-, M-L)	09a,c(-, E, M)
Alcohol use benefits		35(+, E)				
Positive alcohol expectancies	35(+, E)					
Marijuana use benefits						02c(-, M) 09a,d,b(-, E, M) 09c(+, M)
Marijuana use costs						02a(-, E) 02b(+, E)
Occasional alcohol use- not risky		33(?, M)				
Occasional cigarette use- not risky			33(+, M)			
Occasional marijuana use- not risky					33(+, M)	
Marijuana as not addictive					09a(+, E)	09b,c(+, M) 09d(+, E)
Marijuana knowledge						24(-, E-L)

(Table 32 continues)

(Table 32 continued)

Predictors	Alcohol Initiation *p* <.05 (+/-)	Alcohol Initiation *p* >.05 (+/-)	Cigarette Initiation *p* <.05 (+/-)	Cigarette Initiation *p* >.05 (+/-)	Marijuana Initiation *p* <.05 (+/-)	Marijuana Initiation *p* >.05 (+/-)
Views smokers negatively					28(-, L)	
Belief in appropriate use of alcohol and cigarettes			27a,b(-, M-L, M)	27c(-, M-L)		
Belief in appropriate use of drugs				27a,c(+, M-L) 27b(+, M)		
Low religiousness	21(+, M-L)				21(+, M-L)	
High religiousness	34(-, E)			27c(+, M-L) 27a,b(-,M-L, M)		
Prosocial beliefs				04a,c(?, E, E-L) 04b(?, M-L) 27a,c(-, M-L) 27b(-, M)		
Future plans/goals						02a(+, E) 02b,c(-, E, M)
Importance of respect						02a,c(-, E, M) 02b(+, E)

(Table 32 continues)

(Table 32 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Behavior:						
Frequent alcohol use			07(+, E) 13(+, L)	08(+, M)	09a,d(+, E) 21(+, M-L)	07(+, E) 08(+, M) 09b,c(+, M)
Increase in extent of alcohol use				08(+, M)		08(+, M)
Cigarette initiation	11(+, E)					
Frequent cigarette use		07(+, E) 08(?, M)			07(+, E) 08(+, M) 09a,d(+, E) 09b,c(+, M) 21(+, M-L)	28(?, L)
Increase in extent of cigarette use	08(+, M)				08(+, M)	
Marijuana initiation	11(+, E)					
Frequent marijuana use		07(-, E)	07(+, E) 13(+, L)			

(Table 32 continues)

(Table 32 continued)

	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Deviance/delinquency	03(+, L) 11(+, E)		20(+, E-L)			09a(+, E) 09d(-, E) 09b,c(+, M)
Work-for-pay				20(+, E-L) 27b(-, M) 27a,c(+, M-L)		
Obedient					28(-, L)	
Law-abiding					28(-, L)	
Events:						
Stressful life events		33(?, M)		33(?, M)	33(+, M)	

Note. ? = direction of relationship unknown; not reported in publication.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.
[b] E-L = Early-Late Adolescence; E = Early Adolescence; M = Mid-Adolescence; M-L = Mid-Late Adolescence; L = Late Adolescence.

Second, the bulk of the primary studies assessing the efficacy of traits and states tended to evaluate these predictors within the context of one soft drug. For example, risk-taking, low self-esteem, and low cigarette refusal self-efficacy all were used to predict cigarette initiation only. Further, the utility of impulsivity, rebelliousness, low marijuana refusal self-efficacy, poor life and decision-making skills, and sociability all were assessed within the context of marijuana initiation only.

Finally, important for the current research are the findings for the predictive utility of soft drug intentions. Of the six studies to include these constructs in prediction models, the bulk evaluated the importance of alcohol, cigarette, and marijuana use intentions in predicting the initiation of the corresponding substance. Of this research, the majority examined the impact that cigarette use intentions have for predicting cigarette initiation (Chassin et al., 1984; Flay et al., 1994; Gritz et al., 2003). Hence, little of the extant research has investigated these intention variables for predicting the initiation of other soft drugs.

The evidence concerning use intentions deserves comment. In general, the extant research indicates that youth of varying ages who intend (or are willing) to use a given soft drug are at a considerably higher risk for initiating that drug, in comparison to youth who are not so inclined. Interestingly, the sole study (Marcos & Bahr, 1988) that evaluated cigarette use intentions for predicting the initiation of another soft drug (in this case, marijuana) found that mid-late adolescent youth were at a decreased risk for marijuana initiation if they were inclined to smoke cigarettes. The validity of this finding is difficult to gauge within the context of this review, since Marcos and Bahr's research constituted the sole study that examined the nature of this construct in this manner.

Full convergence. Vote-counting only was able to be conducted for one predictor of alcohol initiation, since either the direction of effects for other variables were not reported in corresponding primary studies, or only one directional finding was identified through the review. Findings from Brook et al. (1986) and Epstein et al. (1999) indicate that among early and late adolescent youth, involvement in delinquency (or deviance) constitutes a salient risk factor for alcohol initiation.

Multiple findings fully converged in underscoring the risk capacity that the following eight predictors exhibit in increasing the likelihood of cigarette initiation: sensation-seeking, low locus of control, low cigarette refusal self-efficacy, cigarette use intention, tolerance of deviance, belief in the appropriate use of drugs, frequent alcohol use, and frequent marijuana use. In contrast, multiple findings for two protective factors (i.e., prosocial beliefs and a belief in the appropriate use of alcohol and cigarettes) converged in underscoring the types of beliefs that are associated with a reduced likelihood of cigarette initiation.

With respect to predicting marijuana initiation, multiple findings were consistent in underscoring the risk capacity of the following five predictors: low marijuana refusal self-efficacy, marijuana use intention, the belief that marijuana is not addictive, frequent alcohol use, and frequent cigarette use. Finally, all five findings from two published reports (Ellickson et al., 2004; Kandel & Andrews,

1987) provide evidence that youth who believe marijuana use is harmful are at a decreased risk for initiating this substance, compared to adolescents who view marijuana use as not harmful.

Mixed findings. The directional effects exerted by the following nine individual domain variables yielded mixed findings: rebelliousness, depression, costs and benefits of marijuana use, high levels of religiousness, future plans/goals, importance of respect, delinquency/deviance, and involvement in work-for-pay activities. These predictors should be the focus of future research, particularly since the inconsistent findings for the directional effects of these variables may simply be the product, in part, of a number of study differences (e.g., sample size, measurement, power, or analytic technique). As well, until more research is conducted, it will remain unclear whether some of these inconsistent findings are due to genuine age effects.

One study (Ellickson et al., 2004) examined the utility of rebelliousness in predicting marijuana initiation among two samples of early and mid-adolescent youth. This personality trait was found to increase risk for marijuana initiation among one sample of early adolescent youth and one sample of mid-adolescent youth. Among the other two samples, however, rebelliousness served as an insignificant protective factor for marijuana initiation.

Walter et al.'s (1991) cross-sectional study found that depression serves as a salient risk factor for cigarette initiation during mid-adolescence. According to Kandel et al.'s (2004) longitudinal research, however, youth of all ages who self-reported high levels of depression were less likely to initiate cigarette use than their counterparts. Although Kandel and colleagues' finding was insignificant, the fact that their measure of depression was obtained prior to self-reported cigarette initiation suggests that their finding may have more credence. More research is needed on this front, particularly since age effects may partly explain these differential findings.

Results concerning the benefits and costs ascribed marijuana use are mixed with respect to marijuana initiation, which points to the need for more research in this area. The bulk of the findings suggest that 7th and 8th grade students who believe benefits stem from marijuana use are less likely to initiate marijuana use (Ellickson et al., 2004). This same finding was yielded for 9th grade students (Bailey & Hubbard, 1990; Ellickson et al., 1990). In contrast, Ellickson et al. (2004) found a positive (but insignificant) association between marijuana use benefits and marijuana initiation among a sample of 10th grade students.

It also is unclear how perceived costs concerning marijuana use operate in impacting the likelihood of marijuana initiation. In their longitudinal research, Bailey and Hubbard (1990) found that during 7th grade, youth who believed that many costs stem from marijuana use were less likely to initiate the use of this drug. By 8th grade, however, the direction of effects exerted by this variable changed, such that increases in perceived costs were associated with an increased (but insignificant) likelihood of marijuana initiation. This change in effects could be the result of an increase in associating with individuals who use marijuana.

Using longitudinal data, Skinner et al. (1985) developed three prediction models in assessing the utility that high levels of religiousness has for predicting cigarette initiation among junior and senior high students, junior-high students alone, and high-school students alone. Although insignificant, high levels of religiousness served as a protective factor for cigarette initiation among junior-high students and the combined sample, but increased risk for cigarette initiation among high-school students.

The insignificant findings for the association between marijuana initiation among early adolescents and their future plans and views on respect were inconsistent. Since all of these results stem from one study (Bailey & Hubbard, 1990), additional research should be conducted in this area.

Another set of inconsistent findings centered on the directional impact of delinquency on marijuana initiation. Ellickson et al. (2004) constituted the only group of researchers who examined the impact of delinquency on marijuana initiation. Among 8th, 9th, and 10th grade students, frequent involvement in delinquency placed youth at an increased (but insignificant) risk for marijuana initiation. In contrast, 7th grade students who self-reported frequent delinquency were less likely to initiate marijuana use, as compared to 7th grade students who reported less frequent involvement in delinquent behavior.

Finally, although not an important determinant of cigarette initiation, the directional effects demonstrated by involvement in work-for-pay activities are inconsistent with regard to this outcome, suggesting this construct deserves more attention. In Kandel et al.'s (2004) longitudinal study of 5,347 youth (whose ages spanned early-late adolescence), frequent involvement in work-for-pay activities increased risk for cigarette initiation. Skinner et al.'s (1985) longitudinal research found the same directional relationship with a combined sample of junior and high-school students, as well as a decomposed sample consisting of only high-school students. In contrast, another finding from Skinner et al.'s (1985) study indicated that involvement in work-for-pay activities decreased risk for cigarette initiation among junior-high students.

Time to Soft Drug Initiation

The current review was only able to identify two studies (Hawkins et al., 2002b; Unger & Chen, 1999) that examined family, peer, and individual predictors of time to soft drug initiation and met the inclusion criteria set forth in Chapter 5. Hence, although these studies did yield some important findings, much more research is needed in drawing solid conclusions about the importance and direction of effects exerted by these constructs. Investigations of community and school-related determinants of time to soft drug initiation also are needed.

It is important to mention that the findings from both studies stem from data derived from samples of early-late adolescent youth (12-17 years, or 6^{th}-12^{th} grade). The direction and significance of relationships discussed below may differ for youth from divergent periods of adolescence (e.g., early or late adolescence).

Family, Peer, and Individual Domain Predictors

Tables 33-35 present the direction of effects for various family, peer, and individual domain constructs. Within the family domain (Table 33), social learning constructs were investigated more frequently than familial bonding or relationship factors, largely due to the longitudinal work of Hawkins et al. (2002b). The direction of most of the relationships coincides with the propositions put forth by social learning theory, social bond theory, and the SDM.

Among youth of all ages, strong maternal bonding delays age of alcohol, cigarette, and marijuana initiation, while proactive family management serves as a salient protective factor in delaying age of marijuana initiation. Although not an important predictor of time to alcohol and cigarette initiation, Hawkins and colleagues did find that proactive family management serves to lower age of initiation for these substances.

Parental permissiveness of alcohol use constitutes a salient risk factor for time to alcohol initiation, while parental and sibling cigarette use both are important in lowering age of cigarette initiation. Although not an overly important determinant, parental pro-marijuana norms serve to lower age of marijuana initiation.

Listed in Table 34, only four peer-related predictors were examined by Hawkins et al. (2002b) and Unger and Chen (1999). All constitute social learning variables that are designed to capture drug use modeling. As was observed with the initiation research, these drug-related variables were only examined within the context of the soft drug to which they refer, although all of the findings parallel the directional effects postulated in social learning theory and the SDM. Peer alcohol use constitutes a salient risk factor for time to alcohol initiation, while male and female peer cigarette use lowers age of cigarette initiation. Finally,

Table 33.

Family Domain Predictors of Time to Soft Drug Initiation: Directional Relationships, Early-Late Adolescence [a]

	Time to Alcohol Initiation		Time to Cigarette Initiation		Time to Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Bonding:						
Maternal bonding		18(-)	18(-)			18(-)
Social Learning:						
Parental cigarette use			30(+)			
Sibling cigarette use			30(+)			
Parental pro-alcohol norms	18(+)					
Parental pro-marijuana norms						18(+)
Relationship Factors:						
Proactive family management		18(+)		18(+)	18(-)	

Note. + = less survival time, hence the predictor is associated with an earlier age of initiation.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.

Table 34.

Peer Domain Predictors of Time to Soft Drug Initiation: Directional Relationships, Early-Late Adolescence [a]

	Time to Alcohol Initiation		Time to Cigarette Initiation		Time to Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Social Learning:						
Peer alcohol use	18(+)					
Male peer cigarette use			30(+)			
Female peer cigarette use			30(+)			
Peer/sibling marijuana use					18(+)	

Note. + = less survival time, hence the predictor is associated with an earlier age of initiation.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.

Table 35.

Individual Domain Predictors of Time to Soft Drug Initiation: Directional Relationships, Early-Late Adolescence [a]

	Time to Alcohol Initiation		Time to Cigarette Initiation		Time to Marijuana Initiation	
Predictors	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)	*p* <.05 (+/-)	*p* >.05 (+/-)
Attitudes/Intentions:						
Tobacco use intention			30(+)			
Pro-alcohol norms		18(+)				
Pro-cigarette norms			18(+)			
Pro-marijuana norms					18(+)	
Favorite cigarette advertisement			30(+)			
Behavior:						
Alcohol initiation			18(+)		18(+)	
Cigarette initiation	18(+)				18(+)	
Accepted tobacco promo item			30(+)			

Note. + = less survival time, hence the predictor is associated with an earlier age of initiation.
[a] The reference numbers link tabularized results for respective primary studies to corresponding research parameters, found in Appendix F.

peer/sibling marijuana use serves as a salient risk factor for time to marijuana initiation.

Within the individual domain, both studies focused solely on adolescents' attitudes, intentions, and behavior (see Table 35). Similar to investigations of the utility of cigarette use intentions for explaining cigarette initiation, Unger and Chen (1999) evaluated the efficacy of tobacco use intentions for predicting time to cigarette initiation, but not time to alcohol or marijuana initiation.

Both pro-alcohol norms and cigarette initiation constitute risk factors for time to alcohol initiation, while five variables (tobacco use intention, pro-cigarette norms, favorite cigarette ad, alcohol initiation, and acceptance of tobacco promotion items) are important determinants in lowering age of cigarette initiation. Finally, among youth of all ages, adolescents who hold permissive views about marijuana use, or who have initiated alcohol or cigarette use, are significantly more likely to initiate marijuana use at younger ages, as compared to adolescents who do not tolerate marijuana use or who have not initiated alcohol or cigarette use.

Overview of Key Findings and Attendant Gaps

With few exceptions, the direction of relationships identified in this systematic review met theoretical expectations. The most frequently investigated constructs were those put forth by social learning theory (Akers, 1977), the social development model (Catalano & Hawkins, 1996), and social control theory (Hirschi, 1969). Discussed below are some of the overarching themes that characterized the research, as well as four major types of predictors that were revealed.

Ecological and Predictor Foci

Taking each of the three soft drug initiation and time to initiation outcomes in turn, Table 36 provides a break-down of the ecological domains and predictors that primary studies most frequently examined for each of the six periods of adolescent development. Not only does this synopsis place some of the major design characteristics and foci of the primary studies into context, but this summary enables numerous comparisons across drug types to be made, while identifying gaps in this body of literature that future research can resolve.[8]

In short, across outcomes and periods of adolescent development, the family domain constituted the most widely assessed ecological domain. In fact, of the 506 relationships presented in this review, 30% (n = 154) involved family-related variables. The utility of family constructs were most frequently evaluated in models predicting alcohol, cigarette, and time to alcohol initiation, as well as marijuana initiation and time to initiation (along with individual domain constructs).

The last column of Table 36 notates the predictors that were most frequently investigated. Across outcomes, ecological domains, and stages of adolescent development, constructs rooted in social learning theory and the SDM (e.g., parental and peer soft drug use and drug-related norms) tended to be examined most often.

[8] Recommendations for bridging several of these gaps are offered in the final chapter (Chapter 11) of this study.

Table 36.

Primary Studies: Select Design, Domain, and Predictor Descriptives by Outcome and Sample Population

Outcome by Sample Population [a]	Design Type [b]	Ecological Domains, Number of Models Appraising [c]	Ecological Domain Most Commonly Examined	Predictor Most Commonly Assessed
Alcohol Initiation (n = 17 models)				
Early-Late Adolescence	L = 2, C-S = 1	C = 0, S = 1, F = 3, P = 2, I = 1	Family	High Parental Monitoring
Early Adolescence	L = 3, C-S = 3	C = 3, S = 3, F = 4, P = 5, I = 4	Individual	Peer Alcohol Use
Early-Mid Adolescence	L = 2	C = 0, S = 0, F = 1, P = 1, I = 0	Family	N/A (No One More Common)
Mid-Adolescence	L = 1, C-S = 1	C = 0, S = 1, F = 1, P = 1, I = 2	Individual	Peer Alcohol Use
Mid-Late Adolescence	L = 3	C = 0, S = 1, F = 3, P = 2, I = 2	Family	Strong Familial Bond, Par. Alc. Use
Late Adolescence	L = 1	C = 0, S = 0, F = 1, P = 1, I = 1	Family, Peer, Individual	N/A (No One More Common)
Cigarette Initiation (n = 23 models)				
Early-Late Adolescence	L = 5, C-S = 1	C = 3, S = 4, F = 6, P = 5, I = 4	Family	Parental Cigarette Use
Early Adolescence	L = 3, C-S = 4	C = 3, S = 2, F = 5, P = 5, I = 4	Peer	Peer Cigarette Use
Early-Mid Adolescence	L = 2	C = 0, S = 0, F = 1, P = 1, I = 0	Family	N/A (No One More Common)

(Table 36 continues)

(Table 36 continued)

Outcome by Sample Population [a]	Design Type [b]	Ecological Domains, Number of Models Appraising [c]	Ecological Domain Most Commonly Examined	Predictor Most Commonly Assessed
Mid-Adolescence	L = 2, C-S = 1	C = 0, S = 2, F = 2, P = 2, I = 3	Individual	Low School Commitment, Parental Attachment, Parental Cigarette Use, Association with Smoking Peers
Mid-Late Adolescence	L = 3	C = 1, S = 3, F = 3, P = 3, I = 3	Family	Peer Cigarette Use
Late Adolescence	L = 2	C = 0, S = 2, F = 2, P = 2, I = 2	Family	Parental Cigarette Use
<u>Marijuana Initiation</u> (n = 20 models)				
Early-Late Adolescence	L = 1, C-S = 4	C = 1, S = 1, F = 4, P = 2, I = 1	Family	High Parental Monitoring
Early Adolescence	L =3, C-S = 3	C = 5, S = 5, F = 5, P = 6, I = 6	Individual	Frequent Alcohol Use
Early-Mid Adolescence	---	---	---	---
Mid-Adolescence	L = 4, C-S = 1	C = 3, S = 4, F = 4, P = 4, I = 5	Individual	Poor School Performance, Peer Pro-Marijuana Norms
Mid-Late Adolescence	L = 3	C = 0, S = 2, F = 3, P = 2, I = 3	Family	Strong Familial Bond, Parental Alcohol Use, Peer Marijuana Use
Late Adolescence	L = 1	C = 0, S = 1, F = 0, P = 0, I = 1	Individual	N/A (No One More Common)

(Table 36 continues)

(Table 36 continued)

Outcome by Sample Population [a]	Design Type [b]	Ecological Domains, Number of Models Appraising [c]	Ecological Domain Most Commonly Examined	Predictor Most Commonly Assessed
Time to Alcohol Initiation (n = 1 model)				
Early-Late Adolescence	L = 1	C = 0, S = 0, F = 1, P = 1, I = 1	Family	N/A (No One More Common)
Time to Cigarette Initiation (n = 2 models)				
Early-Late Adolescence	L = 1, C-S = 1	C = 0, S = 0 , F = 2, P = 2, I = 2	Individual	Peer Cigarette Use
Time to Marijuana Initiation (n = 1 model)				
Early-Late Adolescence	L = 1	C = 0, S = 0 , F = 1, P = 1, I = 1	Family, Individual	N/A (No One More Common)

[a] Early-Late Adolescence = 12-17 years, 6th-12th grade; Early Adolescence = 12-14 years, 6th-8th grade; Early-Mid Adolescence = 12-16 years, 6th-10th grade; Mid-Adolescence = 14-16 years, 9th-10th grade; Mid-Late Adolescence = 14-18 years, 9th-12th grade; Late Adolescence = 16-18 years, 11th-12th grade
[b] L = Longitudinal; C-S = Cross-Sectional
[c] C = Community Domain; S = School Domain; F = Family Domain; P = Peer Domain; I = Individual Domain

The final major theme that emerged centered on the periods of adolescent development that received the most and least attention with respect to ecological-specific determinants of soft drug initiation. Illustrated in Table 37, among the primary studies that assessed predictors of soft drug initiation, early adolescence constituted the most commonly researched stage of adolescent development. In turn, soft drug initiation during early-mid and late adolescence was investigated the least frequently.

Table 37.

Soft Drug Initiation: Most and Least Researched Stages of Adolescent Development by Ecological Domain

Domain	Stage of Adolescence Most Widely Researched	# Findings	Stage of Adolescence Least Frequently Researched	# Findings
Community:	▪ Early Adolescence	13	▪ Early-Mid Adolescence ▪ Late Adolescence	0 0
School:	▪ Early Adolescence	14	▪ Early-Mid Adolescence ▪ Late Adolescence	0 3
Family:	▪ Early-Late Adolescence	42	▪ Late Adolescence	4
Peer:	▪ Early Adolescence	39	▪ Early-Mid Adolescence ▪ Late Adolescence	4 5
Individual:	▪ Mid-Adolescence	49	▪ Early-Mid Adolescence ▪ Late Adolescence	0 11

Major Types of Predictors

In synthesizing the directional findings for variables that were investigated in more than one primary study, evidence emerged to support the existence of four types of variables: universal, drug-specific, age-specific, and age- and drug-specific predictors.

Universal predictors. As non-specific or generic factors, universal predictors are variables that are significantly associated with the initiation of all three soft drugs, and across all periods of adolescent development (Windle et al., 2008). A total of six factors were found to exert salient universal effects in increasing risk for alcohol, cigarette, and marijuana initiation among youth of all ages. Within the school domain, poor school performance and low school commitment served as salient risk factors for soft drug initiation. Family-related constructs included unclear family rules concerning behavior and a poor parent-child relationship. Two familial structure variables (i.e., stepparent and single-parent family structure) also exerted universal risk effects.

Drug-specific predictors. With respect to soft drug initiation, 12 family, peer, and individual domain variables exerted drug-specific effects at the .05 level of significance. Possibly due to the fact that minimal attention has been paid to time to soft drug initiation, only two drug-specific predictors were identified for this type of outcome.

Six family factors demonstrated drug-specific effects in predicting soft drug initiation. First, parental alcohol use serves as a salient risk factor for alcohol (Kandel & Andrews, 1987; Kandel et al., 1976) and marijuana (Kandel et al., 1975) initiation among mid-late adolescent youth, but constitutes an unimportant protective factor for cigarette initiation among early-mid adolescent youth (Ennett et al., 2001). Second, low parental support serves as a salient risk factor for alcohol and cigarette initiation, but an insignificant risk factor for marijuana initiation during mid-adolescence (Walter et al., 1991).

Third and fourth, although all findings for parental prodrug norms and "good communication with parents about media portrayals and consequences of legal drug use" were insignificant, extant research suggests that these variables may serve as risk factors for alcohol initiation among mid-late and early-mid adolescents, respectively, while decreasing risk for cigarette initiation among these same subpopulations (Ennett et al., 2001).

Fifth, with respect to high parental monitoring, this construct operates as an important protective factor in decreasing initiation risk for alcohol (Williams et al., 2007) and marijuana (Amey & Albrecht, 1998). In contrast, high parental monitoring does not make a significant contribution to explaining cigarette initiation (Amey & Albrecht, 1998; Ennett et al., 2001; Skinner et al., 1985; Urberg et al., 2003).

Parental divorce constitutes the final family domain variable found to exert drug-specific effects. Although examined in only one study (Amey & Albrecht, 1998), results suggest that adolescents of all ages whose parents are divorced are not at a significantly higher risk for alcohol initiation. In contrast, divorce constitutes a salient risk factor for cigarette and marijuana initiation.

Several other drug-specific findings for soft drug initiation emerged from this review. Although more research may be needed on the impact of peer marijuana use on alcohol and cigarette initiation, this social learning construct appears to exert drug-specific effects. High levels of peer marijuana use have a significant, positive impact on the likelihood of adolescent marijuana initiation (D’Amico & McCarthy, 2006; Ellickson et al., 2004; Epstein et al., 1995; Kandel & Andrews, 1987; Shears et al., 2006; Walter et al., 1991), while the insignificant results from one study (D’Amico & McCarthy, 2006) suggest that peer marijuana use constitutes a risk factor for alcohol initiation, but a protective factor for cigarette initiation.

Five factors from the individual domain pose drug-specific effects on soft drug initiation: sensation-seeking, anxiety, religiousness, cigarette use intentions, and stressful life events. Sensation-seeking is a salient risk factor for alcohol (Urberg et al., 2003) and cigarette (Robinson et al, 1997; Urberg et al., 2003) initiation among early-late adolescent youth, but is not an important factor in predicting marijuana initiation among early adolescents (Epstein et al., 1995). With respect to one personality state, anxiety, Walter et al. (1991) found that mid-adolescent youth who self-reported high levels of anxiety were at a significantly higher risk for alcohol initiation, but not cigarette or marijuana initiation.

Findings from Kandel et al. (1976) and Webb et al. (1991) converged in underscoring the importance that adolescents’ level of religiousness can have in predicting alcohol initiation. Low levels of religiousness are associated with an increased risk for alcohol initiation (Kandel et al., 1976), while high levels decrease risk (Webb et al., 1991). In contrast, the degree to which youth are religious appears to have an unimportant impact on the likelihood of cigarette initiation (Skinner et al., 1985).

The results from three studies converged to show that, compared to early-late adolescent youth who do not intend to smoke cigarettes, youth who intend to do so are at a significantly higher risk for cigarette initiation (Chassin et al., 1984; Flay et al., 1994; Gritz et al., 2003). In contrast, Marcos and Bahr (1988) found that this factor significantly decreased the likelihood of marijuana initiation among mid-late adolescent youth. More research is needed to substantiate this finding for marijuana initiation.

Finally, although only investigated in one study (Walter et al., 1991), stressful life events were found to exert a significant, positive impact on marijuana initiation. This construct did not make an important contribution to explaining alcohol and cigarette initiation, although it served to increase risk.

Although Hawkins et al. (2002b) and Unger and Chen (1999) were the only two groups of researchers who investigated predictors of time to soft drug initiation, two drug-specific factors were identified. Both findings are relevant for youth of all ages. First, maternal bonding makes a significant contribution to explaining age of cigarette initiation, but not alcohol or marijuana initiation.

The second major drug-specific finding for time to soft drug initiation centers on the effects exerted by proactive family management. Discussed earlier, this construct is a salient protective factor in delaying age of marijuana initiation, but operates as a possible risk factor for time to alcohol and cigarette initiation. Although the risk effects demonstrated by this construct were not significant at the .05 level, the findings speak to the possibility that other family-related factors tied to the home environment also may be operating, such as parents' permissive norms concerning adolescent alcohol or cigarette use, or parental use of these drugs. More research on this issue is suggested.

Age-specific predictors. Drawing upon the findings for predictors that were investigated with two or more samples of youth from different stages of adolescence, results from this review indicate that eight peer and individual domain factors exert age-specific effects on cigarette and marijuana initiation. No age-specific factors were identified in relation to alcohol initiation.

Three constructs (peer pro-cigarette norms, low cigarette refusal self-efficacy, and frequent alcohol use) demonstrate age-specific effects in impacting the likelihood of cigarette initiation. Peers who condone cigarette use place mid-adolescent youth at significant risk for cigarette initiation (Chassin et al., 1986; Flay et al., 1998). In comparison, this construct does not make a significant contribution to explaining cigarette initiation among early-late (Chassin et al., 1986) and late (Flay et al., 1998) adolescents. Low cigarette refusal self-efficacy serves as a salient risk factor for cigarette initiation among mid-adolescent youth (Walter et al., 1991), but not among late adolescents (Flay et al., 1998). Finally, taking together the findings from three studies, there is some evidence to suggest that frequent alcohol use is not an important factor for cigarette initiation among mid-adolescent youth (Duncan et al., 1998), but is a salient risk factor for cigarette initiation among youth during early and late adolescence (D'Amico & McCarthy, 2006; Flay et al., 1998). Additional research again is needed to confirm this set of findings.

With respect to marijuana initiation, several findings suggest that five constructs (peer alcohol use, peer marijuana use, rebelliousness, low marijuana refusal self-efficacy, and marijuana use intention) demonstrate age-specific effects. In particular, peer alcohol and marijuana use constitute salient risk factors for marijuana initiation among mid-adolescent youth, while serving as significant protective factors for marijuana initiation among early adolescents (Bailey & Hubbard, 1990).

There is both longitudinal and cross-sectional evidence (Ellickson et al., 2004) to suggest that early adolescent youth who initiate marijuana use tend to be disproportionately more rebellious than their early adolescent counterparts. Among mid-adolescent youth, however, Ellickson et al. (2004) found that level of rebelliousness was not an important factor in explaining the initiation of this soft drug.

During mid-late adolescence, teenagers' ability to refuse marijuana use when offered does not appear to constitute an important risk factor for marijuana initiation (Marco & Bahr, 1988). In contrast, low marijuana refusal self-efficacy has been found to increase significantly the risk for marijuana initiation among early and mid-adolescent youth (Ellickson et al., 2004).

The final age-specific predictor identified for marijuana initiation centers on adolescents' intentions to use this substance. Ellickson et al.'s (2004) findings for two early adolescent and two mid-adolescent prediction models indicate that although this predictor increases risk for marijuana initiation during both periods of adolescence, this construct makes a more important contribution to explaining marijuana initiation among early adolescents than among mid-adolescent youth.

Age- and drug-specific predictors. Only one construct (delinquency or deviance) was found to demonstrate both age and drug-specific effects. Among Epstein et al.'s (1999) sample of early adolescent youth, delinquency served as a salient risk factor for alcohol initiation, but was not an important determinant of marijuana initiation. Further, seven findings from four additional studies (Brook et al., 1986; Ellickson et al., 2004; Epstein et al., 1999; Kandel et al., 2004) suggest that this factor is a salient risk factor for alcohol and cigarette initiation, but not marijuana initiation.

Gaps Addressed by the Current Research

The systematic review identified several gaps in the extant literature. A full discussion of many of these voids is reserved for the conclusion chapter of this study (Chapter 11); however, the remainder of this chapter turns attention to those gaps that specifically were addressed in the quantitative aspect of the current research.

Community domain. Three major gaps concerning community domain predictors of soft drug initiation and time to initiation were identified through the systematic review and subsequently addressed in the quantitative component of the current study. First, the systematic review was unable to identify any published studies that investigated community-related determinants of time to soft drug initiation. Further, compared to research on family, peer, and individual predictors of initiation, relatively little research has evaluated the utility of community domain factors for predicting soft drug initiation among youth. The quantitative component of the current research addressed these respective issues by examining the relative importance of alcohol, cigarette, and marijuana availability, as well as media soft drug norms, for predicting both soft drug initiation and time to initiation. The current research also investigated the age-graded nature of these influences.

Second, none of the primary studies derived outcome data from distinct samples of late adolescent youth. This is unfortunate, particularly given the fact that a proper test of Hawkins' (Catalano & Hawkins, 1996) proposition concerning late adolescent behavior (i.e., community influences have a stronger and disproportionate impact on late adolescent behavior versus the behavior of younger adolescents) requires that researchers consider and compare the impact of community influences on the soft drug initiation behavior of older versus younger adolescents. The current research addressed this gap, through a comprehensive test of H7.

Finally, the generic nature of alcohol, cigarette, and marijuana availability for predicting the initiation and time to initiation of other soft drugs was not investigated in any of the 36 primary studies included in the systematic review. The current study took up this line of inquiry, thereby expanding the research base in this area.

School domain. With respect to school domain influences, three major research gaps were identified through the systematic review and subsequently addressed in the current research. First, neither of the two primary studies that investigated time to soft drug initiation examined the utility of school domain factors. This line of inquiry is investigated in the prediction component of the current research, by evaluating the total and age-graded impact of numerous school-related constructs (e.g., college aspirations, involvement in academic activities, strong school performance, and cheating).

Second, the bulk of the existing research assessed school attachment within the context of marijuana initiation. Not only was the current research able to confirm past findings for this relationship, but the current study also expanded this line of research by assessing the utility of school attachment for predicting alcohol and cigarette initiation, as well as time to soft drug initiation.

Third, additional research should investigate the predictive utility of truancy, particularly for cigarette and marijuana initiation among youth of all ages. Although Walter et al.'s (1991) cross-sectional study did assess the impact of truancy on cigarette and marijuana initiation among 10th grade students, the direction of insignificant effects were not included in the published report. In addressing this void, the current research evaluated the impact of truancy on soft drug initiation and reported the direction of effects. Further, the current study extended this line of research by investigating the predictive utility of school truancy for explaining time to soft drug initiation among a combined sample and three age-graded samples of youth.

Family domain. One major gap in the literature, which was addressed through analyses of the 2004 PPAAUS data, centers on the generic nature of parental soft drug norms. In particular, the current research filled a void with respect to the universal effects that parental alcohol, cigarette, and marijuana norms exert in predicting the initiation and time to initiation of other soft drugs. The current research also extended the extant work in this area by assessing the age-graded nature of these social learning constructs.

Peer domain. Three gaps in the literature concerning peer influence factors were subsequently addressed by the current research. First, the direction of the insignificant findings for most peer constructs investigated in the primary studies were not reported in published materials. This issue was partly resolved by reporting the direction of effects exerted by those peer factors retained in regression models at some point during the model-building process. In presenting the findings for the peer variables (Chapter 9), attention is directed at directional relationships that countered theoretical or hypothesized expectations.

Second, the need for more research on the nature of peer alcohol, cigarette, and marijuana use and attendant norms also was addressed through the current research. Peer alcohol use and norms were assessed within the context of alcohol, cigarette, and marijuana initiation (as well as time to initiation), while peer cigarette use and pro-cigarette norms were evaluated in models predicting cigarette and marijuana initiation (and time to initiation). In models predicting marijuana initiation and time to initiation, peer alcohol, cigarette, and marijuana initiation, as well as peer norms concerning these substances, were subject to investigation.

Finally, none of the primary studies that investigated soft drug initiation evaluated the predictive utility of peer soft drug use and prodrug norms with data from distinct samples of late adolescent youth. The current research bridged this gap and extended this line of research by investigating the importance of peer soft drug use and attendant norms for predicting time to soft drug initiation among 12th grade students.

Individual domain. Four major gaps concerning individual domain predictors were subsequently addressed through the current research. First, similar to the literature voids concerning the universal impact of drug-related constructs found within the community, family, and peer domains of social life, the current study assessed the capacity that alcohol, cigarette, and marijuana use intentions have for predicting the initiation and time to initiation of other drugs. Not only is this line of inquiry under-developed with respect to soft drug initiation, but the two studies (Hawkins et al., 2002b; Unger & Chen, 1999) that examined predictors of time to soft drug initiation did not evaluate the cross-cutting nature of these intention variables.

Second, the current research expanded the knowledge base concerning the efficacy of alcohol and cigarette initiation for predicting alcohol, cigarette, and marijuana initiation. None of the primary studies included in the systematic review directed attention toward the efficacy of alcohol initiation as a determinant of cigarette initiation. Further research in this area is needed, particularly if additional insight is to be yielded with respect to the predictive validity of Kandel's (2002) stage hypothesis.

Third, the current research addresses the efficacy that involvement in work-for-pay activities has for predicting the initiation and time to initiation of all three substances. Discussed earlier, the extant literature indicates that this construct may be an unimportant determinant of cigarette initiation. Due to a paucity of research, it is unclear if and how involvement in work activities impacts the ages at which youth initiate alcohol, cigarettes, and marijuana.

Fourth, the findings from Hawkins et al. (2002b) and Unger and Chen (1999) stemmed from data derived from youth whose ages spanned early-late adolescence. The current research not only examined predictors of time to initiation among this sample population, but further analyses were conducted with subsamples of early, mid-, and late adolescent youth.

As this concluding discussion underscored, numerous gaps in the extant literature were addressed through the quantitative component of the current study. Before these multivariate findings are revealed (Chapter 9), the following chapter presents the results for H1 (modified version of Kandel's sequencing hypothesis) and introduces the univariate and bivariate findings from the 2004 PPAAUS.

CHAPTER 8

UNIVARIATE AND BIVARIATE RESULTS

Three research questions were posed in the current study. To address these questions, nine hypotheses were tested, eight of which were assessed through multivariate prediction models. Before multivariate results are introduced, however, it is important to first present the univariate and bivariate findings, as well as the results for H1, which speak to Research Question #1.

The chapter is organized as follows. Univariate information concerning the outcome and extraneous variables is presented first, along with the bivariate correlations. Findings that speak to Research Question #1 (H1) are presented next. Research Questions #2 and #3 and attendant hypotheses are addressed in the following chapter.

Sample Descriptives

Outcome Measures

Table 38 presents the descriptive information for the three dichotomous initiation and three continuous age of initiation outcome variables.[9] Indirect support for Kandel's (2002) proposition concerning the hierarchical nature of legal and illegal drug prevalence is found in the frequency percentages for alcohol, cigarette, and marijuana initiation. Among the total sample (N = 753), 51% self-reported prior initiation of alcohol use, while 34% and 21% indicated prior initiation of cigarette and marijuana use, respectively. A more complete picture of the soft drug initiation behavior of the total sample emerges when

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[9] Roughly 1% (n = 9), 2% (n = 13), and 2% (n = 12) of cases had missing data for alcohol, cigarette, and marijuana initiation (and age of initiation), respectively. These cases were recoded as respective alcohol, cigarette, and marijuana abstainers.

average ages of initiation are examined. On average, cigarette initiates (n = 256) began cigarette use at a slightly younger age (13.12 years) than alcohol initiates (n = 384) began alcohol use (13.33 years). Among marijuana initiates (n = 159), the average age of initiation was 14 years.

Table 38.

Dependent Measures: Descriptives for Total Sample

Variable	Min	Max	Mean	SD
Alcohol Initiation	0	1	.510	.500
Cigarette Initiation	0	1	.340	.474
Marijuana Initiation	0	1	.211	.408
Age of Alcohol Initiation [a]	8	18	13.335	2.490
Age of Cigarette Initiation [b]	8	18	13.125	2.349
Age of Marijuana Initiation [c]	8	18	14.081	1.778

Note. Dichotomous initiation measures include all respondents, N = 753.
[a] Only includes alcohol initiates, valid n = 384
[b] Only includes cigarette initiates, valid n = 256
[c] Only includes marijuana initiates, valid n = 159

Extraneous Measures

The predictive utility of 38 extraneous measures were examined in the research.[10] Table 39 presents the percentage breakdowns for the 25 dichotomous predictors that were assessed, while Table 40 presents the descriptives for the 13 ordinal/continuous variables that were employed.[11]

[10] Skewness and kurtosis diagnostics were performed for all continuous predictors and controls. These statistics indicated acceptable distributions.
[11] No more than 3% of cases had missing data for any of the extraneous measures. For those few respondents who had missing data, mean replacement was used. This imputation strategy has been deemed an acceptable method of handling missing data in instances where only a small proportion of data are missing (Little, 1992; Sande, 1982).

Table 39.

Dichotomous Predictors/Controls: Percentage Frequencies (N = 753)

Variable	0	1	Variable	0	1
		Community Domain			
MessMedAlc 0 = Not OK to use 1 = OK to use, mixed/no message	.195	.805	*MessMedMar* 0 = Not OK to use 1 = OK to use, mixed/no message	.612	.388
MessMedCig 0 = Not OK to use 1 = OK to use, mixed/no message	.305	.699			
		School Domain			
MessTchrAlc 0 = Not OK to use 1 = OK to use, mixed/no message	.811	.189	*MessTchrMar* 0 = Not OK to use 1 = OK to use, mixed/no message	.896	.104
MessTchrCig 0 = Not OK to use, 1 = OK to use, mixed/no message	.857	.143	*CllgeAsp* 0 = No 1 = Yes	.208	.792
		Family Domain			
MessParAlc 0 = Not OK to use, 1 = OK to use, mixed/no message	.744	.256	*MessParMar* 0 = Not OK to use, 1 = OK to use, mixed/no message	.919	.081
MessParCig 0 = Not OK to use, 1 = OK to use, mixed/no message	.861	.139			
		Peer Domain			
MessPeerAlc 0 = Not OK to use, 1 = OK to use, mixed/no message	.304	.695	*MessPeerMar* 0 = Not OK to use, 1 = OK to use, mixed/no message	.546	.454
MessPeerCig 0 = Not OK to use, 1 = OK to use, mixed/no message	.442	.558			
		Individual Domain			
AlcInit 0 = Alcohol abstention 1 = Alcohol initiation	.490	.510	*AlcIntent* 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.324	.676

(Table 39 continues)

(Table 39 continued)

Variable	0	1	Variable	0	1
Individual Domain (continued)					
AlcEarly 0 = Alcohol abstainers, Late alcohol initiates (16-18 yrs) 1 = Early alcohol initiates (8-15 yrs)	.592	.408	*CigIntent* 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.660	.340
AlcLate 0 = Alcohol abstainers, Early alcohol initiates (8-15 yrs) 1 = Late alcohol initiates (16-18 yrs)	.898	.102	*MarIntent* 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.736	.264
CigEarly 0 = Cigarette abstainers, Late cigarette initiates (16-18 yrs) 1 = Early cigarette initiates (8-15 yrs)	.717	.283	*Steal* 0 = Never stole before 1 = Stole before	.699	.311
CigLate 0 = Cigarette abstainers, Early cigarette initiates (8-15 yrs) 1 = Late cigarette initiates (16-18 yrs)	.943	.057	*ViolBeh* 0 = No violent acts 1 = 1 or more violent acts	.764	.236
Control Variables					
Gender 0 = Female 1 = Male	.490	.510	*Race* 0 = Nonwhite 1 = White	.110	.890

Table 40.

Ordinal/Continuous Predictors and Controls: Descriptives (N = 753)

Variable	Min	Max	Mean	SD
Community Domain				
EasyAlc	0	4	2.544	1.484
EasyCig	0	4	2.610	1.643
EasyMar	0	4	1.686	1.563
School Domain				
Truancy	0	4	.709	1.060

(Table 40 continues)

(Table 40 continued)

Variable	Min	Max	Mean	SD
		School Domain (continued)		
Cheat	0	3	1.443	1.204
AttachSchool	3	21	13.775	3.997
Academic	0	3	2.253	1.045
HiAcadPerf	0	5	3.516	1.121
		Peer Domain		
PeerAlc	0	4	1.622	1.167
PeerCig	0	4	1.402	.992
PeerMar	0	4	.917	.857
		Individual Domain		
Sports	0	3	2.211	1.024
Religious	0	5	2.851	1.473
Work	0	5	3.468	1.397
Social	0	5	3.564	1.041
		Control Variables		
Age	11	19	15.000	2.487
AlcFreq	0	13	3.470	4.124
CigFreq	0	5	.845	1.441

Based on the response distributions for these 38 measures, the total sample can be described as follows. Roughly half (51%) of the students were male, the majority (89%) were white, and on average, students were 15 years of age at the time of data collection. Although they reported somewhat low average levels of alcohol (*AlcFreq*) and cigarette (*CigFreq*) frequency, average responses indicate that 6th, 9th, and 12th grade students perceived alcohol (*EasyAlc*) and cigarettes (*EasyCig*) as “somewhat easy” to access, while students were “not sure” how easy marijuana (*EasyMar*) was to access.

Most students perceived that both the media and their peers espoused tolerant views of alcohol and cigarette use and intolerant views of marijuana use. Comparatively, a larger proportion of students perceived media intolerance than peer intolerance of marijuana use (61.2% versus 54.6%, respectively). Perceptions concerning parents’ and teachers’ tolerance of soft drug use were similar, with the majority of respondents reporting that their parents and teachers were intolerant of alcohol use (74.4% versus 81.1%), cigarette use (86.1% versus 85.7%), and marijuana use (91.9% versus 89.6%), respectively.

Taking into account responses to six school bonding measures, students’ bonds to school can be described as fairly strong, on average. At the univariate-level, roughly 80% of the sample self-reported aspirations of attending college (*CllgeAsp*). Although most students self-reported cheating on schoolwork (*Cheat*) and skipping school *(Truancy*) within their lifetime, the average student had not engaged in these behaviors within the past year. The bulk of respondents also reported fairly positive attitudes about school, school subjects, and teachers (items that comprised *AttachSchool*), with the average student exhibiting a level of school attachment (13.775) that can be characterized as medium in strength (on a scale of 3-21, with 3 representing low attachment). Most students also were involved in extracurricular academic activities (*Academic*) on a fairly frequent basis, with the average student engaging in these activities 1-2 times a week.

Regardless of grade-level, the average student also reported earning “good” grades in school (*HiAcadPerf*).

Aside from the three peer soft drug tolerance measures that were employed, the utility of perceived peer alcohol (*PeerAlc*), cigarette (*PeerCig*), and marijuana (*PeerMar*) use also were assessed in multivariate regression models. On average, 6^{th}, 9^{th}, and 12^{th} grade students perceived that 50% of peers in their respective grades engaged in alcohol use, and 25% engaged in cigarette use and marijuana use, respectively.

Within the individual domain, 10 predictors constituted dichotomous variables, while four variables were measured at the ordinal/continuous level. Among alcohol and cigarette initiates, the majority began use at earlier (8-15 years) versus later (16-18 years) biological ages. In fact, of the 384 alcohol and 256 cigarette initiates, roughly 80% ($n = 307$) and 83% ($n = 213$) self-reported initiating these respective drugs between 8 and 15 years of age.

Concerning respondents’ intentions to use soft drugs, univariate-level percentages indicate that students’ intentions to use alcohol, cigarettes, and marijuana mirror the availability and tolerance of these soft drugs in society (see Chapter 1). In particular, 68% of students indicated some degree of willingness to engage in alcohol use, while 34% and 26% indicated some degree of willingness to use cigarettes and marijuana, respectively.

Two problem behaviors, involvement in stealing (*Steal*) and violent behavior (*ViolBeh*), constituted dichotomous variables, while four involvement measures were measured at the ordinal/continuous level. Although most students indicated that they never stole before or engaged in (or threatened) overt violent acts, students were more apt to engage in the former (31%) than the latter (24%). Students were involved in social or entertainment activities, as well as sports or physical activities, an average of 1-2 times a week, while religious and work-for-pay activities were attended to 1-2 times a month.

Bivariate Correlations

Presented and discussed below, a total of five Pearson *r* correlation matrices were developed and assessed. Four of these matrices dealt with the bivariate associations between relevant extraneous variables and alcohol, cigarette, and marijuana initiation, as well as age of alcohol, cigarette, and marijuana initiation. The purpose underlying the development of the fifth matrix, which contained the bivariate correlations between independent/control variables, was to identify serious collinearity problems. Further collinearity diagnostics were performed, including an assessment of tolerance statistics and variance inflation factors. In short, no major collinearity issues were revealed.[12]

Discussed in detail following the presentation of the Guttman scaling results, H1 findings dictated the type of drug-related variables that were used in developing each respective regression equation. Since not all drug-related measures were examined for each outcome, most of the bivariate correlation tables presented in this section contain some empty cells. These empty cells

[12] All tolerance coefficients were >.20 and all variance inflation factors were < 5.0. Common convention holds that collinearity between extraneous variables poses a major problem when tolerance coefficients are ≤ .20 (Menard, 2002) and variance inflation factors are ≥ 10.0 (Kutner, Nachtsheim, Neter, & Li, 2005).

indicate those extraneous variables that were excluded from respective multivariate models.

It is important to note that at the bivariate level, the impact of other predictors and controls on the soft drug initiation (or age of initiation) outcome under consideration were not taken into account. Hence, although significant bivariate associations were found, it is possible that the significance and strength of these relationships changed once multivariate models accounted for the risk or protective effects of the other extraneous measures. Examining these bivariate correlations does provide some preliminary insight into the direction, strength, and significance of relationships.

Initiation

Alcohol

Table 41 presents the bivariate relationships between all extraneous measures and dichotomous alcohol, cigarette, and marijuana initiation. Alcohol initiation is of initial consideration. At the bivariate level, numerous variables were predictive of alcohol initiation. Within the community domain, both perceived media tolerance of alcohol use and easy access to alcohol (a proxy measure of alcohol availability) exerted significant risk for alcohol initiation. All school domain measures were highly significant ($p < .01$) predictors, with the exception of college aspirations. Teachers' tolerance of alcohol use ($r = .276$), cheating ($r = .482$), and truancy ($r = .440$) all possessed risk qualities, with increases in these measures associated with increased risks for alcohol initiation. Conversely, high levels of school attachment ($r = -.398$), high academic performance ($r = -.117$), and

Table 41.

Dichotomous Initiation: Bivariate Correlations [a]

Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation	Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation
				Community Domain			
MessMedAlc	.234**	.085*	.050	EasyAlc	.480**	.330**	.275**
MessMedCig	---	.074*	-.029	EasyCig	---	.455**	.358**
MessMedMar	---	---	.149**	EasyMar	---	---	.491**
				School Domain			
MessTchrAlc	.276**	.235**	.216**	AttachSchool	-.398**	-.378**	-.346**
MessTchrCig	---	.226**	.187**	Academic	-.187**	-.241**	-.250**
MessTchrMar	---	---	.209**	HiAcadPerf	-.117**	-.211**	-.166**
Cheat	.482**	.376**	.353**	CllgeAsp	-.026	-.087*	-.079*
Truancy	.440**	.374**	.440**				
				Family Domain			
MessParAlc	.271**	.221**	.188**	MessParMar	---	---	.216**

(Table 41 continues)

(Table 41 continued)

Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation	Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation
MessParCig	---	.237**	.214**				
			Peer Domain				
PeerAlc	.578**	.424**	.346**	MessPeerAlc	.449**	.334**	.257**
PeerCig	---	.427**	.354**	MessPeerCig	---	.368**	.284**
PeerMar	---	---	.410**	MessPeerMar	---	---	.417**
			Individual Domain				
AlcInit	---	.614**	---	AlcIntent	.553**	.359**	.275**
AlcEarly	---	.557**	.498**	CigIntent	---	.663**	.446**
AlcLate	---	.109**	-.014	MarIntent	---	---	.664**
CigEarly	---	---	.607**	Work	.097**	.078*	.059
CigLate	---	---	.125**	Religious	-.178**	-.186**	-.239**
ViolBeh	.227**	.214**	.202**	Sports	-.060	-.091*	-.072*
Steal	.337**	.365**	.305**	Social	.307**	.276**	.251**

(Table 41 continues)

(Table 41 continued)

Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation	Variable	Alcohol Initiation	Cigarette Initiation	Marijuana Initiation
			Control Variables				
Gender	.057	.034	.102**	AlcFreq	---	.661**	.631**
Race	.066	.023	.018	CigFreq	---	---	.695**
Age	.532**	.418**	.354**				

[a] N = 753; * *p* <.05; ** *p* <.001; --- refers to those variables that were not examined in respective multivariate analyses.

frequent involvement in extracurricular academic activities (*r* = -.187) exhibited protective qualities in decreasing risk for alcohol initiation. All community and school domain predictors (including *CllgeAsp*) demonstrated risk and protective capacities consistent with the direction of respective relationships that were hypothesized (H2 and H3) and subsequently tested in multivariate models.

Within the family domain, only one bivariate relationship was estimated: the association between parental tolerance of alcohol use *(MessParAlc*) and alcohol initiation. Supportive of H2 (but at the bivariate level), youth who reported that their parents were tolerant of alcohol use were more apt to initiate alcohol use, compared to youth whose parents were intolerant of alcohol use.

Both peer domain predictors (*PeerAlc* and *MessPeerAlc*) were significantly related to alcohol initiation. Compared to their counterparts, students who believed a large proportion of their peers used alcohol (*r* = .578) and were tolerant of alcohol use (*r* = .449) were more apt to have initiated alcohol use.

All individual domain predictors that were eventually examined in the total sample and age-graded binary logistic models were highly significant (*p* <.01) at the bivariate level, with the exception of *Sports*, which was non-significant altogether. Significant bivariate relationships within the individual domain indicate that compared to their counterparts, students who intended to use alcohol, stole before, and had a history of threatening or over violent behavior were at an increased risk for alcohol initiation. In contrast, frequent involvement in religious activities exerted protective effects in decreasing risk for alcohol initiation.

At the bivariate level, H3 was not supported with respect to the direction of relationships between alcohol initiation and involvement in work-for-pay and social activities. These factors exerted risk effects, not protective effects, with frequent involvement in work (r = .097) and social (r = .307) activities each associated with an increased risk for alcohol initiation.

Although being white and male increased risk for alcohol use, the bivariate relationships (r = .057 and .066, respectively) were not significant at the .05 level. The biological age of students was significantly associated with alcohol initiation (r = .532; p <.01), however, with older ages (versus younger ages) associated with an increased risk for initiation.

Cigarettes

With the exception of gender (r = .034) and race (r = .023), all predictors eligible for assessment in the multivariate cigarette initiation models were significantly associated with cigarette initiation at the bivariate level. Although the strength of the relationships between cigarette initiation and college aspirations (r = -.087), involvement in work (r = .078) and sports (r = -.091), and media tolerance of alcohol use (r = .085) and cigarette use (r = .074) were rather weak, these associations were significant at the .01 level. The hypothesized direction of these relationships were supported at the bivariate level, with the exception of work. Parallel to findings for alcohol initiation, increased frequency in work-for-pay activities was associated with an increased risk for cigarette initiation.

The remaining predictors were significant at the .05 level. Compared to their counterparts, youth who self-reported cigarette initiation were more apt to be older, report teacher, parent, and peer tolerance of alcohol and cigarette use, perceive easy access to alcohol and cigarettes, and perceive that high proportions of their peers use alcohol and cigarettes. Cigarette initiates also were more apt to have: cheated frequently on schoolwork, skipped school frequently, initiated alcohol use (with early age of initiation [8-15 years] exerting a stronger risk effect than late age of initiation [16-18 years]), engaged in stealing and overt or threatening acts of violence, been involved in social activities on a frequent basis; and had intentions to use alcohol and cigarettes. Conversely, high levels of school attachment, high academic performance, and frequent involvement in academic and religious activities all were associated with a decreased risk for cigarette initiation at the bivariate level.

Marijuana

Five extraneous measures were not significantly related to marijuana initiation at the bivariate level: media tolerance of alcohol use (r = .050) and cigarette use (r = -.029), late age of alcohol initiation (r = -.014), work (r = .059), and race (r = .018). The direction of these relationships, however, indicated that media tolerance of cigarette use and late age of alcohol initiation may exert protective functions in decreasing risk for marijuana initiation, while frequent involvement in work-for-pay activities may increase risk for initiation.

The remaining predictors were significantly associated with marijuana initiation. With the exception of two variables (*CllgeAsp* and *Sports*), which were significant at the .05 level, all extraneous measures were highly significant

(p <.01). Reflecting on these highly significant bivariate relationships, marijuana initiates can be characterized as follows. Compared to marijuana abstainers, marijuana initiates are more apt to be older males who engaged in frequent alcohol and cigarette use. Moreover, marijuana initiates (versus abstainers) were more apt to: 1) perceive the media as being tolerant of marijuana use; 2) perceive that their parents, teachers, and peers have permissive norms concerning alcohol and cigarette use; 3) perceive that large proportions of their peers engage in alcohol, cigarette, and marijuana use; 4) report easy access to alcohol, cigarettes, and marijuana; and 5) skip school, cheat on schoolwork, and engage in social activities on a frequent basis.

Compared to marijuana abstainers, marijuana initiates also were more likely to have stolen before and threatened (or engaged in) overt violent acts; initiated alcohol use at an early age (8-15 years); initiated cigarette use, regardless of age of initiation; and self-reported intentions to use alcohol, cigarettes, and marijuana. Frequent involvement in religious (r = -.239) and extracurricular academic (r = -.250) activities, high levels of school attachment (r = -.346), and strong academic performance (r = -.166) all exhibited significant protective functions at the bivariate level in reducing the risk for marijuana initiation.

Comparison of Predictors

The bivariate relationships just discussed existed when the effects of the other predictors and control variables were not considered. Hence, some of the significant relationships that were revealed may be nullified or enhanced at the multivariate level. Nonetheless, this preliminary analysis speaks to the possibility that some independent variables may be drug-specific.

Whereas *MessMedAlc* was significantly associated with alcohol initiation (and to a lesser extent, cigarette initiation), media tolerance of alcohol use was not significantly related to marijuana initiation. Media tolerance of cigarette use (*MessMedCig*) demonstrated a weak, but significant positive effect on cigarette initiation, but was not significantly associated with marijuana initiation. As another example, late age of alcohol initiation (that which occurred between 16-18 years) increased risk for cigarette initiation at the .05 level, but this predictor exhibited a non-significant, but negative impact on marijuana initiation.

Further, while *Work* was associated with an increased risk for alcohol initiation ($r = .097$; $p < .01$), this predictor was not significantly related to marijuana initiation ($r = .059$), and it exerted a relatively weak, but significant, risk effect on cigarette initiation ($r = .078$; p <.05). At the bivariate level, the impact of gender on initiation also varied by drug type. While males were significantly more likely to initiate marijuana use than females, gender had no significant impact on risk for alcohol or cigarette initiation.

Age of Initiation

Aside from examining predictors of initiation, the current research also examined predictors of time to alcohol, cigarette, and marijuana initiation. Discussed in Chapter 7, time to initiation takes into account both dichotomous initiation (i.e., whether or not the given soft drug was initiated) and the biological age at which the given soft drug was initiated. Since this outcome measure incorporates age of initiation data, it is important to assess the bivariate relationships between the extraneous variables and age of alcohol, cigarette, and marijuana initiation. Developed with data from initiates only, the estimated bivariate relationships are presented in Table 42. Again, empty table cells indicate that the given predictor was not examined in the respective multivariate model and those bivariate relationships discussed below only indicate the change in one variable that is associated with the change in another measure (without accounting for the effects of other predictors).

Alcohol

Far fewer predictors were significantly related to age of alcohol initiation at the bivariate level, as compared to those that were related to alcohol initiation. Taking into account age of alcohol initiation data from 384 alcohol initiates, variables that significantly predicted age of alcohol initiation included easy access to alcohol; teacher, parent, and peer tolerance of alcohol use; perceived peer alcohol use; intentions to use alcohol; frequent involvement in social and work activities; violent behavior and stealing; and being an older or white student.

The direction of the bivariate associations between many of these predictors and age of alcohol initiation countered the direction of associations hypothesized (H2 and H3), with the majority of predictors having exerted protective effects in increasing (delaying) age of alcohol initiation. In fact, the only significant bivariate relationships that met hypothesized expectations were those involving violent behavior and stealing. Adolescents who engaged in (or threatened) overt violent acts, and those who self-reported stealing, were more

Table 42.

Age of Initiation: Bivariate Correlations [a]

Variable	Age of Alcohol Initiation [b]	Age of Cigarette Initiation [c]	Age of Marijuana Initiation [d]	Variable	Age of Alcohol Initiation	Age of Cigarette Initiation	Age of Marijuana Initiation
				Community Domain			
MessMedAlc	.049	.022	.039	*EasyAlc*	.112*	.085	.067
MessMedCig	---	-.026	-.028	*EasyCig*	---	.047	.206**
MessMedMar	---	---	.008	*EasyMar*	---	---	-.010
				School Domain			
MessTchrAlc	.152**	-.026	.018	*AttachSchool*	-.043	.116	.078
MessTchrCig	---	-.077	-.036	*Academic*	-.010	.143*	.100
MessTchrMar	---	---	-.059	*HiAcadPerf*	.074	.209**	.166*
Cheat	.055	-.023	-.030	*CllgeAsp*	.092	.157*	.220**
Truancy	.089	-.037	-.071				

(Table 42 continues)

(Table 42 continued)

Variable	Age of Alcohol Initiation	Age of Cigarette Initiation	Age of Marijuana Initiation	Variable	Age of Alcohol Initiation	Age of Cigarette Initiation	Age of Marijuana Initiation
			Family Domain				
MessParAlc	.108*	-.101	-.118	*MessParMar*	---	---	-.076
MessParCig	---	-.035	-.029				
			Peer Domain				
PeerAlc	.342**	.141*	.203*	*MessPeerAlc*	.201**	.046	.080
PeerCig	---	.073	.115	*MessPeerCig*	---	-.009	.049
PeerMar	---	---	-.003	*MessPeerMar*	---	---	.090
			Individual Domain				
AlcInit	---	.124*	---	*AlcIntent*	.160**	.186**	.201*
AlcEarly	---	-.299**	-.366**	*CigIntent*	---	.069	-.047
AlcLate	---	.427**	.466**	*MarIntent*	---	---	-.083
CigEarly	---	---	-.413**	*Sports*	.052	.096	.113
CigLate	---	---	.454**	*Social*	.203**	.057	.118

(Table 42 continues)

(Table 42 continued)

Variable	Age of Alcohol Initiation	Age of Cigarette Initiation	Age of Marijuana Initiation	Variable	Age of Alcohol Initiation	Age of Cigarette Initiation	Age of Marijuana Initiation
			Individual Domain (continued)				
ViolBeh	-.246**	-.245**	-.190*	*Religious*	.061	.159*	.200*
Steal	-.150**	-.151*	-.061	*Work*	.117*	.044	-.040
			Control Variables				
Gender	-.091	-.054	-.159*	*AlcFreq*	---	.078	-.098
Race	.126*	.305**	-.074	*CigFreq*	---	---	-.159*
Age	.602**	.403**	.345**				

[a] * *p* <.05; ** *p* <.001; --- refers to those variables that were not examined in respective multivariate analyses.
[b] Only includes alcohol initiates, valid n = 384
[c] Only includes cigarette initiates, valid n = 256
[d] Only includes marijuana initiates, valid n = 159

apt to initiate alcohol use at younger versus older ages. In addition to the non-significant impact exerted by gender in effecting the age at which alcohol was initiated, it also is notable that none of the school bonding variables exerted significant effects at the bivariate level.

Cigarettes

The bivariate relationships between predictors, controls, and age of cigarette initiation were based upon data from 256 cigarette initiates. Significant predictors of age of cigarette initiation included being an older or white student; frequent involvement in academic and religious activities; high academic performance; college aspirations; perceived peer alcohol use; late age of alcohol initiation; and intentions to use alcohol. All of these predictors served to delay age of cigarette initiation. Significant predictors found to exert risk effects in decreasing age of cigarette initiation included early age of alcohol initiation and involvement in violent behavior and stealing.

Surprisingly, many of the cigarette-related predictors were not significantly related to the ages at which cigarette initiates began cigarette use, including all of the cigarette tolerance measures, easy access to cigarettes, peer cigarette use, and intentions to use cigarettes. Again, these bivariate relationships should be viewed as preliminary insight into the nature of relationships, since the effects of other predictors were not controlled.

Marijuana

The bivariate relationship between predictors and age of marijuana initiation were based upon data from 159 students who self-reported marijuana initiation. In all, 14 predictors and control variables were significant at the .05 level or lower.

Predictors that demonstrated protective functions in delaying age of marijuana initiation included easy access to cigarettes (a proxy measure of cigarette availability), high school performance, college aspirations, peer alcohol use, late age of alcohol and cigarette initiation, intentions to use alcohol, and frequent involvement in religious activities. A significant, positive association also was observed for biological age and age of marijuana initiation. The following factors exerted significant risk effects (thereby lowering age of marijuana initiation) at the bivariate level: early alcohol and cigarette initiation, involvement (or threatened involvement) in violent acts, frequent cigarette use, and being male as opposed to female.

Similar to the bivariate correlations between cigarette-related predictors (e.g., *MessMedCig*, *EasyCig*, and *PeerCig*) and age of cigarette initiation, none of the marijuana-related predictors (e.g., *MessMedMar, EasyMar*, and *PeerMar*) significantly distinguished marijuana initiates in terms of the ages at which they initiated marijuana use. In addition, with the exception of a few predictors (e.g., *EasyCig*, *PeerAlc*, *AlcIntent*, *CigFreq*, and the alcohol and cigarette age of initiation measures), almost all of the alcohol and cigarette-related factors were not significantly associated with age of marijuana initiation at the bivariate level.

Extraneous Variable Correlations

One way to determine whether collinearity between extraneous variables poses a major problem is to examine the bivariate correlations between these variables. Table 43 presents the estimated Pearson *r* correlations for select bivariate relationships between predictors and control variables. All bivariate associations were examined for strength and level of significance; however, only those correlations that were significant at the .05 level or lower, and whose magnitude was ± .400 or greater, are presented. These significant bivariate relationships, coupled with the results of tolerance and variance inflation diagnostics, indicated that multicollinearity did not pose a serious problem.

Table 43.

Predictors and Controls: Bivariate Correlations ± .400 or Greater (N = 753)

Variable		*r*	Variable	*r*
		Community Domain		
MessMedAlc & …	MessMedCig	.567		
EasyAlc & …	Cheat	.434	PeerMar	.415
	EasyCig	.671	MessPeerAlc	.437
	EasyMar	.562	AlcInit	.480
	PeerAlc	.528	Age	.494
	PeerCig	.430	AlcFreq	.474
EasyCig & …	Cheat	.410	MessPeerAlc	.464
	EasyMar	.653	AlcInit	.475
	PeerAlc	.558	Age	.602
	PeerCig	.498	AlcFreq	.477
	PeerMar	.464		
EasyMar & …	Cheat	.448	AlcInit	.453
	PeerAlc	.540	CigEarly	.411

(Table 43 continues)

(Table 43 continued)

Variable		*r*	Variable	*r*
EasyMar & …	PeerCig	.490	MarIntent	.460
	PeerMar	.552	Age	.520
	MessPeerAlc	.426	AlcFreq	.529
	MessPeerMar	.439	CigFreq	.470
		School Domain		
Truancy & …	AttachSchool	-.406	Age	.436
	PeerAlc	.446	AlcFreq	.496
	PeerMar	.415	CigFreq	.420
	AlcInit	.440		
MessTchrAlc & …	MessTchrCig	.607	Age	.443
	MessTchrMar	.549		
MessTchrCig & …	MessTchrMar	.594		
Cheat & …	AlcInit	.482	AlcFreq	.501
	Age	.518		
AttachSchool & …	PeerAlc	-.409		
		Family Domain		
MessParCig & …	MessParMar	.471		
		Peer Domain		
PeerAlc & …	Cheat	.522	AlcEarly	.403
	PeerCig	.739	AlcIntent	.426
	PeerMar	.713	Age	.752
	MessPeerAlc	.550	AlcFreq	.563
	AlcInit	.578		
PeerCig & …	Cheat	.420	Age	.598

(Table 43 continues)

(Table 43 continued)

Variable		*r*	Variable	*r*
PeerCig & …	PeerMar	.725	AlcFreq	.468
	MessPeerAlc	.411	CigFreq	.401
	AlcInit	.478		
PeerMar & …	Cheat	.422	Age	.592
	MessPeerAlc	.401	AlcFreq	.478
	MessPeerMar	.411	CigFreq	.410
	AlcInit	.486		
MessPeerAlc & …	Cheat	.409	AlcInit	.449
	MessPeerCig	.626	Age	.499
	MessPeerMar	.568	AlcFreq	.430
	AlcIntent	.455		
MessPeerCig & …	MessPeerMar	.673		
MessPeerMar & …	MarIntent	.439	AlcFreq	.425
		Individual Domain		
AlcEarly & …	Cheat	.435	MarIntent	.532
	CigEarly	.589	AlcFreq	.635
	AlcIntent	.442	CigFreq	.496
	CigIntent	.460		
AlcLate & …	Age	.430	MarIntent	.526
CigEarly & …	AlcInit	.533	AlcFreq	.564
	CigIntent	.589	CigFreq	.718
AlcIntent & …	AlcInit	.453	AlcFreq	.491
	CigIntent	.425		

(Table 43 continues)

(Table 43 continued)

Variable		*r*	Variable	*r*
CigIntent & …	AlcInit	.468	AlcFreq	.537
	MarIntent	.568	CigFreq	.663
MarIntent & …	AlcInit	.497	CigFreq	.583
	AlcFreq	.592		
		Control Variables		
Age & …	AlcInit	.532	AlcFreq	.518
AlcFreq & …	AlcInit	.763	CigFreq	.672
CigFreq & …	MessPeerMar	.405	AlcInit	.531

In general, some of the stronger bivariate relationships (e.g., $r > \pm .600$) involved the associations between similar, but discrete, predictors. For instance, as would be expected, perceived easy access to alcohol *(EasyAlc*), cigarettes (*EasyCig*), and marijuana (*EasyMar*) all were moderately correlated with each other at the bivariate level. This suggests that youth who perceived that alcohol was easy to access also tended to believe that cigarettes and marijuana were easy to access. In contrast, youth who felt access to alcohol was difficult also tended to perceive cigarettes and marijuana as being difficult to access.

Other notable correlations included the positive relationships between biological age and *EasyAlc* (r = .602) and *PeerAlc* (r = .752). Consistent with developmental theory, these relationships suggest that as youth grow older, they are increasingly more apt to perceive that cigarettes are available (i.e., easily accessible) and large proportions of their peers engage in alcohol use.

Variants of peer soft drug use also exhibited fairly strong relationships with each other. Students who thought large proportions of their peers use alcohol also tended to perceive that large proportions of their peers engage in cigarette use (r = .739) and marijuana use (r = .713). These fairly strong relationships prompted the development of models in which findings for the individual peer drug use measures were compared to those using a composite measure of peer drug use. With no substantive difference in findings, these variables were kept as individual measures to enable a more fine-grained analysis.

Finally, frequent cigarette and alcohol use were strongly associated with several cigarette and alcohol-related variables. Students who engaged in frequent cigarette use tended to engage in frequent alcohol use (r = .672), initiate cigarette use at early ages (r = .718), and self-report intentions to use cigarettes (r = .663), compared to students who did not engage in cigarette use on a frequent basis. As expected, a fairly strong relationship also existed between alcohol initiation and frequent alcohol use (r = .763) at the bivariate level.

Research Question #1

One goal of the research was to test a modified version of Kandel's (2002) drug sequencing hypothesis with age of initiation data. In addressing Research Question #1, H1 centered on the temporal ordering and cumulative and hierarchical properties of alcohol, cigarette, and marijuana initiation. Specifically, H1 posited that the most common drug initiation sequence is one in which alcohol initiation occurs prior to cigarette initiation, and cigarette initiation occurs prior to marijuana initiation. It also was argued that progression in the soft drug sequence is experienced by successively smaller numbers of students, with far more adolescents initiating alcohol use only than adolescents initiating alcohol and cigarette use, or alcohol, cigarette and marijuana use. The following sections provide the sample descriptives, along with the results for H1 and a supplemental analysis.

Sample Description

Roughly 95% (n = 713) of the total sample (N = 753) provided useable data for the Guttman analysis. Hence, the sample size used in testing H1 consisted of 713 6th (n = 273), 9th (n = 226), and 12th (n = 214) grade students. Of the 5% of cases excluded from the analysis, 21 students self-reported three-way drug ties, while 19 respondents had missing data for one or more of the survey items used in generating age of initiation data.

Of the 713 youth who provided useable data, about 16% (n = 115) reported two-way drug initiation ties. Specifically, 8% (n = 57), 4% (n = 28), and 4% (n = 30) reported the same biological age for alcohol and cigarettes, cigarettes and marijuana, and alcohol and marijuana initiation, respectively. To break these two-way ties, the proportion of cases initiating each soft drug was calculated using the temporal initiation data from the untied cases. Among untied cases (n = 598), 30% (n = 180), 12% (n = 71), and 0.16% (n = 1) indicated first initiating alcohol, cigarettes, and marijuana, respectively. Taking these initiation proportions into account, students who reported alcohol-cigarette ties were deemed as having had initiated alcohol first. Youth who had cigarette-marijuana ties were noted as having had initiated cigarettes first, and students who reported alcohol-marijuana ties were deemed as having initiated alcohol first.

With these two-ties broken, the useable sample can be described as follows. Roughly 48% (n = 344) were complete abstainers, having indicated abstention from all three soft drugs, while slightly more than half of the sample (n = 369) initiated at least one soft drug. Among the soft drug initiates, 96% (n = 354), 63% (n = 231), and 36% (n = 131) reported alcohol, cigarette, and marijuana initiation, respectively.

Drug Initiation Sequences

Table 44 presents the soft drug sequencing behavior among the 369 students who reporting initiating one or more soft drugs. These sequences take into account the temporal ordering of initiation. The majority of the soft drug initiates were polydrug initiates. Specifically, no soft drug initiates reported marijuana initiation only, 3.3% (n= 12) of initiates indicated cigarette initiation only, and 36% (n = 131) of initiates only initiated alcohol use.

Table 44.

Temporal Ordering in Soft Drug Sequences

Observed Sequences	n	% of Soft Drug Initiates (n = 369)	% of Sample (N = 713)
Began with Alcohol Initiation…	272	.737	.381
Alcohol → Cigarettes → Marijuana	62	.168	.087
Alcohol → Cigarettes	62	.168	.087
Alcohol only	131	.355	.184

(Table 44 continues)

(Table 44 continued)

Observed Sequences	n	% of Soft Drug Initiates (n = 369)	% of Sample (N = 713)
Alcohol → Marijuana	7	.018	.010
Began with Cigarette Initiation…	96	.260	.135
Cigarettes → Alcohol → Marijuana	36	.097	.050
Cigarettes → Alcohol	31	.084	.043
Cigarettes only	12	.033	.017
Cigarettes → Marijuana → Alcohol	14	.038	.019
Cigarettes → Marijuana	3	.008	.004
Began with Marijuana Initiation…			
Marijuana → Alcohol → Cigarettes	1	.002	.001

The soft drug initiation sequence outlined in H1 was supported, from a percentage frequency perspective. As illustrated in Figure 2, the hypothesized sequence was found to be the most common, with transitions in the sequence (i.e., cumulation in drug initiation) experienced by successively smaller numbers of students. Taking into account the temporal ordering of initiation, the majority (74%) of soft drug initiates began soft drug use with alcohol. Comparatively, only 26% of initiates began soft drug use with cigarettes, while one student reported initiating marijuana first.

The second drug most commonly initiated was cigarettes. Among those who initiated two or three soft drugs (n = 226), 55% (n = 124) initiated cigarettes second, compared to 30% (n = 67) and 15% (n = 34) who initiated alcohol and marijuana use second, respectively. With respect to the hypothesized soft drug initiation sequence, among the 141 alcohol initiates who initiated a second drug,

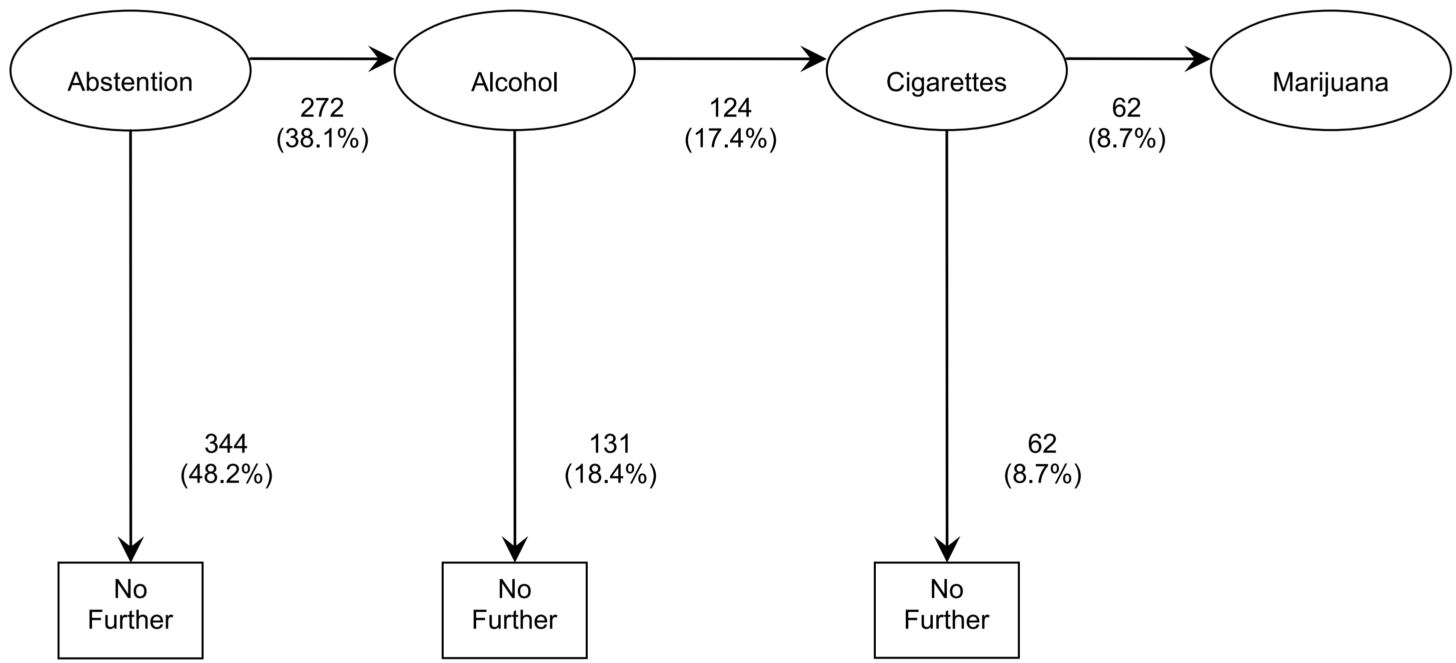

Figure 2. This transition diagram depicts H1, the most common soft drug initiation sequence found in the data (n = 713).

88% (n = 124) initiated cigarette use. Finally, a total of 123 students (17.3% of the sample) initiated all three soft drugs. Of these polydrug initiates, 80% (n = 98) initiated marijuana last, with roughly 63% (n = 62) of these students exhibiting the soft drug initiation sequence outlined in H1.

Worth noting is the second most common soft drug sequence found in the data. Illustrated in Figure 3, this sequence began with cigarette initiation, proceeded to alcohol initiation, and then ended with marijuana initiation (see Figure 4). Although the soft drug initiation sequence outlined in H1 was the most common, a proper test of H1 required that the CR and CS be calculated, as described in Chapter 7.

Scalability of H1 Sequence

In order to determine whether the H1 soft drug initiation sequence fit a valid Guttman scale, Guttman scalogram analysis was used to identify the total number of errors in the sequence and generate various statistics that were used in calculating the coefficients of reliability (CR) and scalability (CS). Taking into account the temporal ordering of soft drug initiation for the 713 students who provided useable data, a total of 246 errors were found in the hypothesized soft drug sequence. Specifically, 97 errors were identified in the alcohol initiation scale item, 144 for the cigarette initiation scale item, and 35 errors were found in the marijuana initiation scale item.

With knowledge of the total number of errors (246), the number of scale items (3), and the number of cases (713), Equation 1 from Chapter 7 was used to

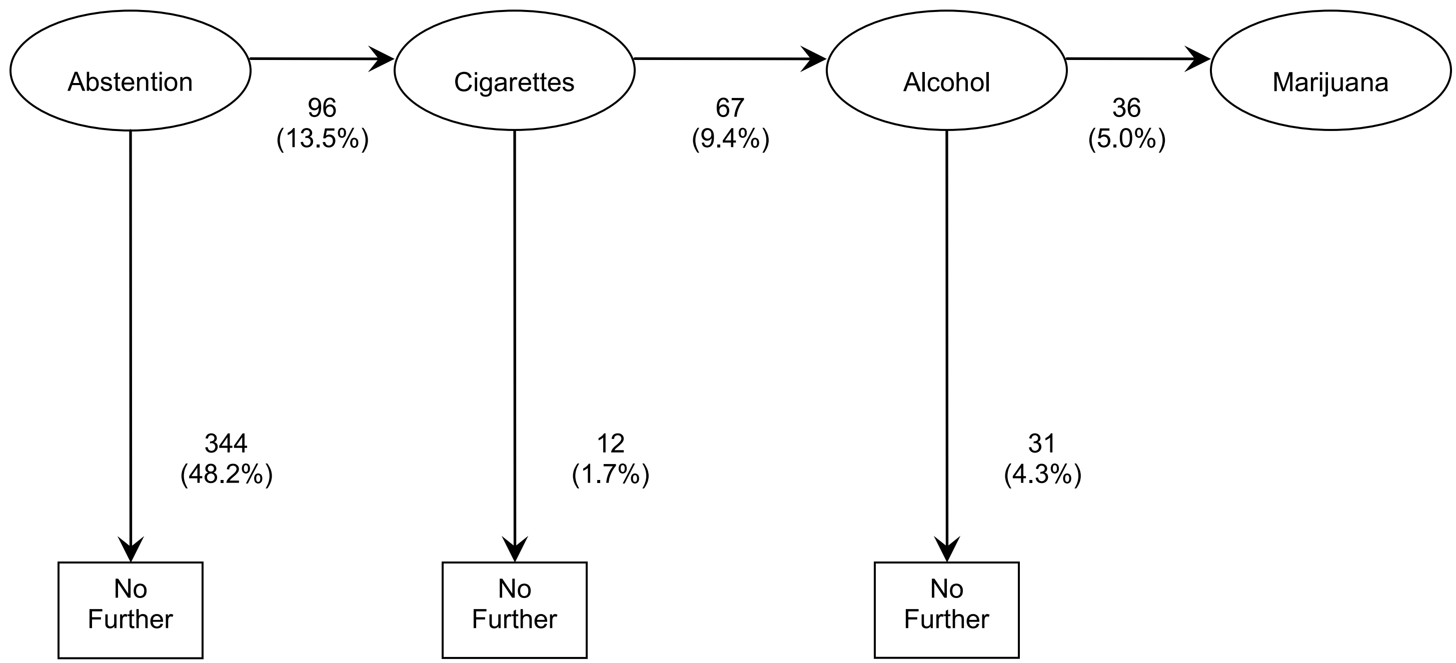

Figure 3. This transition diagram depicts cigarettes>alcohol>marijuana, the second most common soft drug initiation sequence (n = 713).

calculate the coefficient of reproducibility (CR). A CR of .89 was produced, a value that falls just shy of the .90 minimal acceptability benchmark offered by Guttman (1950). A CR of .89 means that not only can one predict with 89% accuracy the scale item responses of a given student simply by knowing that student's scale score, but the initiation sequence outlined in H1 also can be predicted with 89% accuracy given knowledge of students' scale scores.

To determine the scalability of the H1 soft drug sequence, Equation 2 from Chapter 7 was utilized in calculating the coefficient of scalability (CS). With a MMR of .664, and a percentage improvement value of .221, the CS for the hypothesized soft drug initiation sequence was .66, a value that exceeds the minimal scalability benchmark suggested by Menzel (1953) and provides evidence that the soft drug scale items are scalable. This coefficient means that 66% of the total possible errors actually were not errors, but were responses consistent with those hypothesized.

Although the most common soft drug initiation sequence identified in the data was that which was hypothesized, the CR fell slightly short of the threshold for minimal acceptability. Discussed earlier in this chapter, a slightly earlier age of initiation was observed for cigarettes (13.125 years) than alcohol (13.335 years). Given this observation, coupled with the fact that more errors were identified in the cigarette scale item than in the alcohol or marijuana scale items, it is possible that the two-way tie breaks for alcohol-cigarette initiation (i.e., alcohol initiation deemed as having occurred first) may have artificially inflated the number of alcohol initiates, thereby providing inflated support for H1.

Excluding Two-Way Ties

In an effort to determine whether the CR would improve when excluding cases with two-way drug ties, a second Guttman scale was developed and analyzed. For this particular scale, Guttman scalogram analysis was employed using data from 598 of the original 713 students whose drug initiation data were used in testing H1. All soft drug initiates among these 598 students provided discrete biological ages of initiation.

Taking into account the temporal ordering of soft drug initiation for those students who reported initiation, a total of 174 errors were identified: 73 errors in the alcohol scale item, 80 errors in the cigarette scale item, and 21 errors in the marijuana scale item. With knowledge of the total number of errors (174), the number of scale items (3), and the number of cases (598), a CR of .90 was produced. This value equaled the minimal acceptability benchmark for the CR. In turn, with a MMR value of .772, and a percentage improvement value of .131, a CS of .58 was produced, a value that did not quite meet the minimal scalability benchmark.

What this analysis indicated is that a slight, but negligible, positive impact on the CR was observed when two-way drug ties were excluded; however, excluding these two-way ties decreased the CS to a value slightly below the minimal acceptability standard. In essence, whether the two-way drug ties were excluded or included did not have a considerable bearing on the fit of the data.

Supplemental Analysis

Discussed in Chapter 7, an additional Guttman scale was developed with dichotomous initiation data. The purpose of this supplemental analysis was to determine whether the type of data (i.e., dichotomous initiation or age of initiation) utilized had a bearing on the reproducibility and scalability of the soft drug initiation sequence outlined in H1.

The same soft drug initiation data (N = 713) previously used to test H1 was employed in this supplemental analysis, except with this Guttman scale, dichotomous initiation was considered, not the temporal ordering of initiation. Using this method of scale development, 44 errors were identified: 15 errors in the alcohol scale item, 19 errors in the cigarette scale item, and 10 errors in the marijuana scale item.

With knowledge of the total number of errors (44), the number of scale items (3), and the number of cases (713), the CR was calculated. A CR of .98 was produced, a value that far exceeds the .90 minimal acceptability benchmark. With a MMR of .664, and a percentage improvement value of .316, a CS of .94 was obtained, a value that far exceeds the minimal scalability benchmark of .60.

These coefficients indicate that when dichotomous initiation data were used, not only could one predict with 98% accuracy the scale item responses of a given student simply by knowing that student’s scale score, but the initiation sequence outlined in H1 also could be predicted with 98% accuracy given knowledge of students’ scale scores. Moreover, 94% of the total possible errors actually were not errors, but were responses consistent with those outlined in H1.

Had this scale initially been used to test H1, the hypothesis would have been fully supported.

Utility of H1

Guttman scale findings revealed that the dichotomous initiation Guttman scale was less stringent than the temporal ordering scale used to test H1. Since the temporal ordering of initiation was not taken into account in the dichotomous initiation scale, considerably fewer errors were identified. With less errors in the sequence, the dichotomous data provided a better fit, leading to larger reproducibility and scalability coefficients.

Taking into account the CR and CS values for both of these scales, and the fact that the CR value (.89) obtained for the temporal ordering scale fell just shy of the minimum acceptability benchmark (CR = .90) while the CS value (.66) produced exceeded the minimum value of acceptability (CS = .60), considerable evidence was uncovered to support H_a1. Furthermore, among the current sample, the most common soft drug initiation sequence was alcohol>cigarettes> marijuana. In general, these soft drugs tended to be initiated at discrete biological ages, with alcohol use initiated prior to cigarettes, and cigarette use initiated prior to marijuana use. With cumulative and hierarchical properties, each successive transition in this sequence was experienced by fewer youth. As was observed, more students initiated alcohol use only than initiated alcohol and cigarettes, while more students also initiated both alcohol and cigarette use than all three soft drugs.

Conclusion

The current chapter focused on the univariate and bivariate aspects of the quantitative research, as well as sequencing in soft drug initiation. H1 was supported, as among soft drug initiates, alcohol typically was initiated prior to cigarette initiation, while marijuana tended to be initiated following cigarette initiation. As well, progression in the sequence was experienced by successively smaller numbers of students.

A comparison of Guttman scale results for dichotomous initiation versus temporal initiation indicated that the number of errors identified varies according to the type of data utilized. Compared to the Guttman scale that took advantage of temporal initiation data, considerably fewer errors were identified when the dichotomous data were utilized. This difference in errors had a direct impact on the both the reproducibility and scalability of the sequence.

With the presentation of univariate data, bivariate relationships, and H1 findings complete, the following chapter turns to the multivariate results. The findings for H2-H9 are presented, along with insight concerning drug-specific predictors and the efficacy of Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

CHAPTER 9

MULTIVARIATE RESULTS

The current chapter addresses Research Questions #2 and #3, by presenting the multivariate findings for eight hypotheses. H2-H6 were tested with findings from six total sample models (Models #1-6), while the utility of H2-H9 were evaluated with results from 16 age-graded models (Models #7-22). Data were derived from the 2004 PPAAUS.

Discussed in Chapter 7, all regression models were developed through the use of the backward stepwise elimination technique (with a $p > .10$ model exclusion criteria) and a hierarchical blocking procedure. The successive entrance of distal (e.g., family, community, and school) to proximal (e.g., peer and individual) predictors into the models provided a means by which the utility of Petraitis et al.'s (1995) distal-proximal mediation hypothesis could be evaluated.

The chapter is organized into three main sections. An overview of the findings for each total sample model (Research Question #2) is presented first, followed by a synopsis of the age-graded results (Research Question #3). These findings then are taken together in presenting the results for H2-H9, Petraitis et al.'s (1995) distal-proximal mediation hypothesis, and age- and drug-specific predictors.

Research Question #2

One major goal of the research was to identify predictors of alcohol, cigarette, and marijuana initiation and time to initiation among the total sample. Three binary logit models (Models #1-3) dealt with predictors of alcohol, cigarette, and marijuana initiation, while three Cox regression models (Models #4-6) involved predictors of time to initiation. In an effort to model the H1 soft drug sequence from a prediction standpoint, the Guttman scalogram results for H1 informed the types of drug-related variables that were entered into the alcohol, cigarette, and marijuana regression models. Table 45 lists the predictors that were entered into each total sample model.

Table 45.

Dichotomous Initiation and Time to Initiation Models (Total Sample): Predictor Listing

Model Type and Corresponding Predictors					
		Alcohol Initiation and Time to Alcohol Initiation			
Community:	MessMedAlc	EasyAlc			
School:	Truancy	MessTchrAlc	Cheat	CllgeAsp	Academic
	AttachSchool	HiAcadPerf			
Family:	MessParAlc				
Peer:	PeerAlc	MessPeerAlc			
Individual:	AlcIntent	Steal	ViolBeh	Sports	Religious
	Work	Social			
Controls:	Gender	Race	Age		
		Cigarette Initiation and Time Cigarette Initiation			
Community:	MessMedAlc	MessMedCig	EasyAlc	EasyCig	

(Table 45 Continues)

(Table 45 continued)

Model Type and Corresponding Predictors					
	Cigarette Initiation and Time Cigarette Initiation (continued)				
School:	Truancy	MessTchrAlc	MessTchrCig	Cheat	CllgeAsp
	Academic	AttachSchool	HiAcadPerf		
Family:	MessParAlc	MessParCig			
Peer:	PeerAlc	MessPeerAlc	PeerCig	MessPeerCig	
Individual:	AlcIntent	CigIntent	AlcEarly	Steal	ViolBeh
	Sports	Religious	Work	Social	
Controls:	Gender	Race	Age	AlcFreq	
	Marijuana Initiation and Time to Marijuana Initiation				
Community:	MessMedAlc	MessMedCig	MessMedMar	EasyAlc	EasyCig
	EasyMar				
School:	Truancy	MessTchrAlc	MessTchrCig	MessTchrMar	Cheat
	CllgeAsp	Academic	AttachSchool	HiAcadPerf	
Family:	MessParAlc	MessParCig	MessParMar		
Peer:	PeerAlc	MessPeerAlc	PeerCig	MessPeerCig	PeerMar
	MessPeerMar				
Individual:	AlcIntent	CigIntent	MarIntent	AlcEarly	CigEarly
	Steal	ViolBeh	Sports	Religious	Work
	Social				
Controls:	Gender	Race	Age	AlcFreq	CigFreq

In assessing predictors of alcohol initiation and time to initiation (Models #1 and #4), the cigarette and marijuana-related variables (e.g., tolerance, peer use, intentions, and early age of initiation measures) were not examined. Only the alcohol-related predictors (e.g., *MessMedAlc*) were eligible for assessment in these models. In contrast, the alcohol and cigarette-related predictors were entered into the cigarette initiation and time to initiation models (Models #2 and #5). The utility of the marijuana-related measures (e.g., *MessMedMar*) were not examined in these models. Finally, for the marijuana initiation and time to initiation models (Models #3 and #6), the predictive efficacy of all of the alcohol, cigarette, and marijuana-related predictors were subject to assessment.

Alcohol Initiation

Tables 46-48 present the findings for alcohol, cigarette, and marijuana initiation among the total sample. To begin, nine extraneous variables explained roughly 48% of the variance in the log odds of alcohol initiation. With the exception of *Age* (b = .10; p <.10), eight of these nine predictors exerted significant effects at the .05 level (or lower): *EasyAlc*, *Truancy*, *Cheat*, *PeerAlc*, *AlcIntent*, *Steal*, *Religious*, and *Social*.

All of these factors, with the exception of *Religious* (b = -.276; p <.001), exerted risk effects in increasing the log odds of alcohol initiation. In particular, the simple odds of alcohol initiation increased by 32%, 44%, 28%, and 72%, respectively, for every one-unit increase in students' perceptions of access to alcohol (from difficult to easy access), the frequency with which students' skipped school and cheated on schoolwork, and the proportion of peers that students believed used alcohol.

Table 46.

Model #1: Predictors of Alcohol Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.785*** (.228)	2.192	.600** (.266)	1.822	.560** (.268)	1.751	---	---	---	---	---	---
EasyAlc	.730*** (.065)	2.075	.487*** (.074)	1.627	.468*** (.075)	1.596	.311*** (.081)	1.365	.284*** (.087)	1.328	.274*** (.087)	1.316
Truancy			.612*** (.112)	1.844	.592*** (.112)	1.807	.441*** (.116)	1.554	.359*** (.125)	1.432	.361*** (.125)	1.435
MessTchrAlc			.671*** (.267)	1.956	.601*** (.268)	1.825	---	---	---	---	---	---
AttachSchool			-.122*** (.028)	.885	-.117*** (.028)	.890	-.092*** (.029)	.913	---	---	---	---
Cheat			.462*** (.089)	1.588	.469*** (.090)	1.598	.321*** (.095)	1.379	.251** (.108)	1.285	.248** (.107)	1.281
MessParAlc					.442* (.232)	1.556	.470* (.241)	1.600	---	---	---	---
PeerAlc							.644*** (.108)	1.905	.641*** (.124)	1.899	.545*** (.148)	1.725
MessPeerAlc							.776*** (.252)	2.173	.402 (.281)	1.495	---	---

(Table 46 Continues)

(Table 46 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									2.057*** (.277)	7.820	2.092*** (.270)	8.105
Steal									.826*** (.254)	2.284	.866*** (.256)	2.378
Sports									-.199* (.117)	.820	---	---
Religious									-.248*** (.082)	.780	-.276*** (.080)	.759
Social									.310** (.120)	1.363	.228** (.115)	1.256
Age											.103* (.061)	1.109
-2LL	843.695***		679.671***		676.006***		629.898***		544.232***		546.196***	
Cox/Snell R^2	.233		.383		.386		.423		.485		.483	

[a] N = 753; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 47.

Model #2: Predictors of Cigarette Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyCig	.863*** (.083)	2.371	.693*** (.088)	2.000	.669*** (.088)	1.953	.583*** (.090)	1.791	.740*** (.117)	2.095	.562*** (.127)	1.754
Truancy			.192** (.093)	1.212	.172* (.094)	1.187	---	---	---	---	---	---
Cheat			.339*** (.088)	1.403	.352*** (.089)	1.422	.326*** (.091)	1.385	---	---	---	---
AttachSchool			-.115*** (.028)	.891	-.111*** (.028)	.895	-.103*** (.029)	.902	---	---	---	---
HiAcadPerf			-.318*** (.090)	.727	-.308*** (.091)	.735	-.298*** (.094)	.742	-.200* (.120)	.819	-.375*** (.127)	.687
MessParCig					.585*** (.255)	1.795	---	---	---	---	---	---
PeerCig							.402*** (.118)	1.495	.502*** (.149)	1.652	---	---
MessPeerCig							1.002*** (.217)	2.723	---	---	---	---
AlcEarly									1.982*** (.276)	7.254	1.661*** (.309)	5.264

(Table 47 Continues)

(Table 47 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigIntent									2.918*** (.282)	18.497	3.119*** (.316)	22.632
AlcIntent									-.712* (.371)	.490	-1.360*** (.395)	.257
Steal									.462* (.271)	1.587	---	---
Religious									-.184** (.087)	.832	---	---
Work									.159* (.095)	1.172	---	---
Age											.288*** (.069)	1.333
AlcFreq											.207*** (.043)	1.230
-2LL	776.844***		681.382***		676.070***		645.850***		418.466***		378.607***	
Cox/Snell R^2	.221		.314		.319		.346		.516		.541	

[a] N = 753; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 48.

Model #3: Predictors of Marijuana Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.640*** (.230)	1.896	.629** (.250)	1.875	.568** (.253)	1.765	---	---	---	---	---	---
MessMedCig	-.603** (.253)	.547	-.807*** (.275)	.446	-.837*** (.277)	.433	-.871*** (.270)	.419	-1.155*** (.391)	.315	-1.349*** (.426)	.260
EasyMar	.744*** (.099)	2.104	.616*** (.104)	1.851	.600*** (.105)	1.822	.513*** (.107)	1.671	.591*** (.122)	1.805	.425*** (.138)	1.530
EasyCig	.485*** (.135)	1.624	.324** (.139)	1.383	.310** (.140)	1.364	.346** (.147)	1.413	---	---	---	---
Truancy			.339*** (.105)	1.403	.336*** (.105)	1.399	.357*** (.108)	1.430	.395*** (.131)	1.484	---	---
AttachSchool			-.096*** (.034)	.908	-.095*** (.034)	.909	-.111*** (.034)	.895	---	---	---	---
Academic			-.229** (.111)	.796	-.214* (.112)	.807	---	---	---	---	---	---
Cheat			.313*** (.111)	1.368	.325*** (.112)	1.384	.308*** (.115)	1.360	---	---	---	---

(Table 48 continues)

(Table 48 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessParMar					.873** (.346)	2.393	.616* (.354)	1.851	---	---	---	---
MessPeerMar							1.829*** (.330)	6.226	1.371*** (.436)	3.940	1.301*** (.488)	3.674
MessPeerAlc							-.877* (.458)	.416	-1.109** (.548)	.330	-1.852*** (.653)	.157
CigEarly									2.139*** (.351)	8.493	1.530*** (.425)	4.618
AlcEarly									.766** (.390)	2.150	.808* (.450)	2.243
MarIntent									2.369*** (.336)	10.690	2.018*** (.369)	7.525
Religious									-.302*** (.104)	.739	-.283** (.113)	.753
Work									.299** (.117)	1.349	---	---

(Table 48 continues)

(Table 48 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Age											.387*** (.102)	1.472
AlcFreq											.120** (.053)	1.128
CigFreq											.499*** (.140)	1.647
-2LL	554.102***		492.884***		486.426***		457.440		290.097***		244.739***	
Cox/Snell R^2	.256		.314		.320		.345		.476		.506	

[a] N = 753; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Within the individual domain, findings indicated that students who reported some degree of willingness (*AlcIntent*) were over 8 times more likely than their counterparts to have initiated alcohol use. The simple odds of initiation exerted by *Steal* also were quite high. Adolescents who reported stealing from others were over 2 times more likely to have initiated alcohol use, compared to youth who never stole before. Finally, every one-unit increase in the frequency of involvement in social activities (*Social*) was associated with a 26% increase in the simple odds of alcohol initiation.

A comparison of Wald statistics (not tabled) and significance values for the predictors in the final model can be made, in order to determine which factors exerted the most highly significant effects on the log odds of alcohol initiation. Such a comparison indicates that students' alcohol intentions (*AlcIntent*) constituted the most highly significant predictor, followed by involvement in stealing (*Steal*), perceived peer alcohol use (*PeerAlc*), frequent truancy (*Truancy*), frequent involvement in religious activities (*Religious*), and the perception that alcohol is easy to access (*EasyAlc*).

Full mediation was observed for four variables: three alcohol tolerance measures (*MessMedAlc*, *MessTchrAlc*, and *MessParAlc*) and school attachment (*AttachSchool*). While the individual domain predictors mediated the effects of *MessParAlc* and *AttachSchool*, once the impact of *MessParAlc* was taken into account, the risk effects of *MessMedAlc* and *MessTchrAlc* were rendered non-significant.

Cigarette Initiation

The same explanatory factors that were entered into the alcohol initiation model were entered into the cigarette initiation model. In addition, the cigarette-related predictors (i.e., *MessMedCig*, *EasyCig*, *MessTchrCig*, *MessParCig*, *PeerCig*, *MessPeerCig*, and *CigIntent*) were subject to assessment, in an effort to model the first transition of the soft drug initiation sequence that was identified through H1 (i.e., alcohol>cigarette initiation). Also considered was the effect that early age of alcohol initiation (*AlcEarly*) exerted on the likelihood of cigarette initiation. The impact on cigarette initiation exerted by the frequency with which alcohol initiates used alcohol (*AlcFreq*) was controlled.

In the final model, seven variables explained nearly 55% of the variation in the log odds of cigarette initiation (see Table 47). In order of importance, five of these predictors exerted risk effects at the .001 level of significance: *CigIntent* (b = 3.119), *AlcEarly* (b = 1.661), *EasyCig* (b = .562), *Age* (b = .228), and *AlcFreq* (b = .207).

Two variables (*AlcIntent* and *HiAcadPerf*) demonstrated protective capacities in reducing the likelihood of cigarette initiation. Counter to H2, the simple odds of cigarette initiation decreased by 75% for every one-unit increase in *AlcIntent* (b = -1.360; *p* <.001). This counter finding, coupled with the significant risk effects that *AlcIntent* and *CigIntent* posed on the log odds of alcohol and cigarette initiation, respectively, speaks to the possibility that these drug-related predictors may not operate in a generic fashion. In contrast, the operating capacity of these factors may be drug-specific, whereby they serve to increase risk for the initiation of the soft drug to which they correspond, but exert protective effects in reducing the log odds of initiating other soft drugs.

Evidence of full mediation was found for nine variables. The peer domain predictors reduced to non-significance the effects of *Truancy* and *MessParCig*, while the individual domain predictors mediated the effects of *Cheat, AttachSchool*, and *MessPeerCig*. Once the impact of the control variables were taken into account, the risk and protective effects of *PeerCig*, *Steal*, *Religious*, and *Work* were reduced to non-significance.

Marijuana Initiation

In assessing predictors of marijuana initiation among the total sample, the same variables that were entered into the cigarette initiation model were subject to examination. In addition, in an effort to model the full soft drug initiation sequence that was identified through H1 (i.e., alcohol>cigarette>marijuana initiation) from a prediction perspective, the utility of the marijuana-related predictors (i.e., *MessMedMar*, *EasyMar*, *MessTchrMar*, *MessParMar*, *PeerMar*, *MessPeerMar*, and *MarIntent*) also were considered, along with early age of cigarette initiation (*CigEarly*). The frequency with which cigarette initiates smoked cigarettes (*CigFreq*) was controlled.

A total of 10 predictors explained roughly 51% of the variance in marijuana initiation (see Table 48). Although retained in the model, *AlcEarly* ($b = .808$; $p < .10$) did not meet a conventional significance threshold. Three of the constructs (*MessMedCig*, *MessPeerAlc*, and *Religious*) demonstrated protective effects, while the other predictors (*EasyMar*, *MessPeerMar*, *CigEarly*, *MarIntent*, *Age*, *CigFreq*, and *AlcFreq*) increased the log odds of marijuana initiation. Taking Wald statistics (not tabled) and significance levels into account, the most highly significant predictor at the .001 level was *MarIntent* (b = 2.018), followed by *MessPeerAlc* (b = -1.852), *CigEarly* (b = 1.530), *MessMedCig* (b = -1.349), *MessPeerMar* (b = 1.301), *CigFreq* (b = .499), and *EasyMar* (b = .425). Significant age effects also were observed (b = .387; p <.001), with a one-year increase in biological age associated with a 47% increase in the simple odds of marijuana initiation.

Three other findings are worth highlighting. First, not only was frequent cigarette use (b = .499; p <.001) a more highly significant determinant of marijuana initiation than frequent alcohol use (b = .120; p <.05), but early age of cigarette initiation (b = 1.530; p <.001) had a significant bearing on the log odds of marijuana initiation, while early age of alcohol initiation (b = .808; p <.10) had a less significant effect. In culmination, these findings add further support to the H1 soft drug initiation sequence that was observed.

Second, counter to H2, the simple odds of marijuana initiation were 74% lower for youth who felt the media espoused tolerant views about cigarette use, as compared to students who believed the media was intolerant of cigarette use. The direction of the relationship between peer tolerance of alcohol use (*MessPeerAlc*, b = -1.852; p <.001) and marijuana initiation also countered H2. Compared to their counterparts, the simple odds of marijuana initiation were 84% lower for students who felt their peers condoned alcohol use. Although the risk effect exerted by *Work* was reduced to non-significance once the impact of the control variables was accounted, the direction of the relationship indicated that frequent involvement in work-for-pay activities placed students at an increased risk for marijuana initiation, a finding that countered H3.

Third, full mediation was evidenced for six variables. The peer domain predictors mediated the relationship between marijuana initiation and *MessMedMar* and *Academic*. The individual domain served as a salient mediating force in reducing to non-significance the effects of *EasyCig*, *AttachSchool*, *Cheat*, and *MessParMar*.

Time to Alcohol Initiation

Tables 49-51 present the findings for time to alcohol, cigarette, and marijuana initiation among the total sample. First, seven variables (*EasyAlc*, *MessParAlc*, *AlcIntent*, *Steal*, *ViolBeh*, *Religious*, and *Age*) exerted direct, significant effects on the hazard of alcohol initiation (see Table 49). In turn, the individual domain variables fully mediated the impact of three predictors: *Cheat*, *AttachSchool*, and *HiAcadPerf*. An examination of Wald statistics and significance values for those variables exerting direct effects indicated that *AlcIntent* was the most highly significant predictor of time to alcohol initiation among the total sample, followed by *Steal*, *ViolBeh*, *EasyAlc*, *Age*, and *Religious*.

Two predictors, *Religious* (b = -.079; p <.001) and *Age* (b = -.240; p <.001), exerted protective effects in decreasing the hazard of alcohol initiation (i.e., delayed age of alcohol initiation). With respect to *Age*, the negative relationship suggests that while controlling for the other factors, older youth in the sample reported later ages of alcohol initiation. In fact, the hazard of alcohol

Table 49.

Model #4: Predictors of Time to Alcohol Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyAlc	.271*** (.048)	1.311	.198*** (.050)	1.219	.185*** (.051)	1.203	.185*** (.051)	1.203	.103** (.049)	1.109	.207*** (.052)	1.231
Cheat			.179*** (.052)	1.197	.173*** (.052)	1.189	.173*** (.052)	1.189	---	---	---	---
AttachSchool			-.034** (.015)	.967	-.030** (.015)	.971	-.030** (.015)	.971	---	---	---	---
HiAcadPerf			-.135*** (.047)	.874	-.117*** (.048)	.890	-.117*** (.048)	.890	---	---	---	---
MessParAlc					.324*** (.109)	1.383	.324*** (.109)	1.383	.230** (.109)	1.259	.251** (.110)	1.285
AlcIntent									1.273*** (.208)	3.571	1.454*** (.209)	4.282
Steal									.481*** (.108)	1.618	.471*** (.108)	1.601
ViolBeh									.448*** (.115)	1.565	.391*** (.116)	1.479
Religious									-.066* (.034)	.936	-.079** (.035)	.924

(Table 49 continues)

(Table 49 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Age											-.241*** (.032)	.786
-2LL	4392.562		4354.029		4345.418		4345.418		4264.899		4208.995	
Model χ^2	32.748***		72.030***		82.506***		82.506***		153.469***		213.512***	

[a] N = 753; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

Table 50.

Model #5: Predictors of Time to Cigarette Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	-.410** (.178)	.663	-.413** (.182)	.662	-.427** (.182)	.653	-.483*** (.183)	.617	-.343* (.181)	.710	-.308* (.181)	.735
EasyCig	.478*** (.073)	1.613	.398*** (.073)	1.489	.376*** (.074)	1.457	.337*** (.075)	1.400	.293*** (.072)	1.340	.332*** (.079)	1.393
Cheat			.152** (.061)	1.165	.165*** (.061)	1.180	.170*** (.062)	1.185	---	---	---	---

(Table 50 continues)

(Table 50 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AttachSchool			-.065*** (.018)	.937	-.060*** (.019)	.942	-.054*** (.019)	.947	---	---	---	---
HiAcadPerf			-.266*** (.059)	.767	-.251*** (.059)	.778	-.223*** (.060)	.800	-.173*** (.057)	.841	-.168** (.057)	.845
MessParCig					.388*** (.148)	1.475	.281* (.150)	1.324	---	---	---	---
MessPeerCig							.658*** (.167)	1.930	---	---	---	---
AlcEarly									1.350*** (.173)	3.858	1.143*** (.184)	3.135
CigIntent									1.610*** (.171)	5.001	1.491*** (.176)	4.440
AlcIntent									-.651** (.252)	.521	-.659** (.257)	.518
ViolBeh									.263* (.138)	1.301	.262* (.138)	1.300
Religious									-.082* (.041)	.921	-.073* (.041)	.929
Age											-.138*** (.043)	.871

(Table 50 continues)

(Table 50 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcFreq											.056*** (.019)	1.058
-2LL	2981.762		2921.354		2914.900		2897.560		2690.657		2675.325	
Model χ^2	51.064		114.351***		124.307***		137.695***		358.170***		378.219***	

[a] N = 753; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 51.

Model #6: Predictors of Time to Marijuana Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.407** (.169)	1.503	.305* (.169)	1.356	---	---	---	---	---	---	---	---
MessMedAlc	-.675*** (.231)	.509	-.720*** (.230)	.487	-.636*** (.221)	.529	-.567** (.228)	.567	-.431* (.226)	.650	---	---
EasyMar	.628*** (.077)	1.874	.524*** (.078)	1.689	.525*** (.078)	1.691	.467*** (.081)	1.596	.314*** (.075)	1.369	.215*** (.080)	1.240

(Table 51 continues)

(Table 51 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.182** (.073)	1.199	.182** (.074)	1.200	.231*** (.076)	1.260	---	---	---	---
Cheat			.192** (.088)	1.212	.206** (.087)	1.229	.179** (.086)	1.196	---	---	---	---
AttachSchool			-.059** (.023)	.948	-.061** (.024)	.941	-.066*** (.023)	.936	-.043* (.023)	.958	---	---
CllgeAsp			-.408** (.186)	.665	-.397** (.185)	.672	---	---	---	---	---	---
MessParMar					.660*** (.202)	1.935	.413** (.205)	1.511	---	---	---	---
PeerAlc							-.222* (.114)	.801	---	---	---	---
MessPeerMar							1.343*** (.271)	3.832	---	---	---	---
MessPeerAlc							-.815** (.369)	.442	---	---	---	---
CigEarly									1.357*** (.231)	3.884	1.200*** (.249)	3.321
AlcEarly									1.111*** (.290)	3.038	.735** (.307)	2.086

(Table 51 continues)

(Table 51 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MarIntent									1.583*** (.249)	4.869	1.304*** (.261)	3.685
AlcIntent									-1.008*** (.339)	.365	-1.249*** (.364)	.287
Religious									-.197*** (.052)	.821	-.214*** (.053)	.807
Work									.164*** (.063)	1.179	.138** (.063)	1.148
Gender											.473*** (.172)	1.604
Age											-.109* (.061)	.897
AlcFreq											.104*** (.030)	1.110
CigFreq											.139** (.059)	1.150
-2LL	1780.355		1743.374		1737.325		1705.903		1542.081		1522.691	
Model χ^2	95.036***		136.861***		144.815***		169.673***		350.880***		386.045***	

[a] N = 753; * p <.10; ** p <.05; *** p <.001; standard error in parentheses.

initiation declined by 21% for every one-year increase in biological age. At face value, this total sample result indicates that the risk for alcohol initiation declined as age increased, with older adolescents experiencing later ages of alcohol initiation than younger youth. It is worth noting that this finding may have been impacted by forward telescoping on the part of older adolescents, however. If enough older alcohol initiates brought forward in time the biological ages at which they initiated alcohol use, this may have artificially lengthened the time to failure, resulting in an inflated negative coefficient.

The other significant predictors in the final model demonstrated risk capacities in decreasing survival time (i.e., earlier ages of alcohol initiation). A 23% and 29% increase in the hazard of alcohol initiation was observed for every one-unit increase in perceived access to alcohol (*EasyAlc*, b = .207; p <.001) and parental tolerance of alcohol use (*MessParAlc*, b = .251; p <.05), respectively. Compared to their counterparts, the hazard of alcohol initiation was over four times greater for students who had intentions of using alcohol (*AlcIntent*, b = 1.454; p <.001), and 60% and 48% greater among adolescents who stole from others (*Steal*, b = .471; p <.001) or engaged in (or threatened) violent behavior (*ViolBeh*, b = .391; p <.001).

Time to Cigarette Initiation

In order of importance, seven community, school, individual, and control variables exerted direct, significant effects on the hazard of cigarette initiation among the total sample (see Table 50): *CigIntent* (b = 1.491; p <.001), *AlcEarly* (b = 1.143; p <.001), *EasyCig* (b = .332; p <.001), *Age* (b = -.138; p <.001), *AlcFreq* (b = .056; *p* <.001), *AlcIntent* (b = -.659; *p* <.05), and *HiAcadPerf* (b = -.168; *p* <.05). Although retained in the model, *MessMedAlc* (b = -.308; *p* <.10), *ViolBeh* (b = .262; *p* <.10), and *Religious* (b = -.073; *p* <.10) were not significant at a conventional level of significance.

Of the seven significant predictors that impacted the hazard of cigarette initiation, three (*HiAcadPerf*, *AlcIntent*, and *Age*) exerted protective effects in delaying age of cigarette initiation (i.e., decreased hazard). The hazard of cigarette initiation declined by 15% for every one-unit increase in school grades. *AlcIntent* served to decrease the hazard of cigarette initiation, rather than lower the age of cigarette initiation, as expected. In fact, every one-unit increase in willingness to use alcohol was associated with a 48% decrease in the hazard of cigarette initiation. Similar to the time to alcohol initiation model, *Age* demonstrated a protective function in lengthening survival time. The hazard of cigarette initiation declined by 13% for every one-year increase in biological age. As found for time to alcohol initiation, this finding suggests that the risk for cigarette initiation declines as age increases, with older adolescents reporting later ages of cigarette initiation than younger youth. Forward telescoping among the older cigarette initiates may partly explain this inverse relationship, however.

The remaining significant predictors demonstrated risk capacities in contributing to earlier ages of cigarette initiation. After controlling for other factors, a 39% and 6% increase in the hazard of cigarette initiation was observed for every one-unit increase in perceived access to cigarettes (*EasyCig*) and the frequency with which alcohol initiates drank alcohol (*AlcFreq*). Moreover, compared to their counterparts, the hazard of cigarette initiation was over three and four times greater, respectively, for students who initiated alcohol use between 8 and 15 years of age (*AlcEarly*) and who were willing to smoke cigarettes (*CigIntent*).

Two school (*Cheat*, *AttachSchool*), one family (*MessParCig*), and one peer (*MessPeerCig*) variable were fully mediated. Although the initial direction of respective risk and protective effects were consistent with hypothesized expectations, taking the individual domain variables into consideration nullified their respective impacts.

Time to Marijuana Initiation

Outlined in Table 51, a total of 10 predictors exerted significant (p <.05 or lower), direct effects on time to marijuana initiation among the total sample. In order of importance, respective increases in the log hazard of marijuana initiation were observed among students who intended to use marijuana (*MarIntent*); youth who initiated cigarette use between 8 and 15 years of age (*CigEarly*); male adolescents (*Gender*); students who perceived marijuana was easy to access (*EasyMar*); youth who drank alcohol frequently (*AlcFreq*); adolescents who initiated alcohol use between 8 and 15 years of age (*AlcEarly*); students who smoked cigarettes on a frequent basis (*CigFreq*), and frequent involvement in work-for-pay activities (*Work*). *Age* (b = -.109; p <.10) did not meet the conventional level of significance.

Only two predictors (*AlcIntent* and *Religious*) delayed time to marijuana initiation. As the second most highly significant predictor in the model, and counter to H2, the hazard of marijuana initiation was 71% lower for students who intended to use alcohol (*AlcIntent*, b = -1.249; *p* <.001), as compared to students who had no such intentions. The protective capacity of *AlcIntent* also was observed for time to cigarette initiation among the total sample. The direction of the relationship between religious activities (*Religious*, b = -.214; *p* <.001) met hypothesized expectation. Similar to the effects observed for time to alcohol initiation, the hazard of marijuana initiation was 19% lower among adolescents who were frequently involved in religious activities, as compared to students who attended to religious activities on a less frequent basis.

A total of 10 variables were fully mediated by more proximal predictors. The family domain predictors mediated the risk effect of *MessMedMar*, while the peer domain and the control variables reduced to non-significance the protective capacity of college aspirations (*CllgeAsp*) and attachment to school (*AttachSchool*). Finally, the individual domain served as a considerable mediating force in nullifying the significant effects of *Truancy*, *Cheat*, *MessParMar*, *PeerAlc*, *MessPeerMar*, and *MessPeerAlc*.

In sum, similar to marijuana initiation, time to marijuana initiation among the total sample was impacted predominantly by variables found within the individual domain. In fact, aside from *EasyMar*, all of the significant predictors were either individual domain variables or control variables. Tolerance of soft drug use by the media, parents, peers, and teachers had no significant final bearing on the hazard of marijuana initiation. Peer soft drug use, all of the school-related variables, *Race*, and *Age* also were not predictive of time to marijuana initiation at the .05 level (or lower).

Research Question #3

In addition to examining soft drug initiation and time to initiation among the total sample, further analyses sought to determine whether the predictors of soft drug initiation and time to soft drug initiation varied by stage of adolescent development. Discussed in Chapter 7, the total sample was decomposed by school grade-level, resulting in three subsamples (6th, 9th, and 12th grade students). To address Research Question #3, Models #7-9 and Models #10-12 examined predictors of alcohol and cigarette initiation among 6th, 9th, and 12th graders, respectively. Due to a low percentage of marijuana initiation among 6th grade students[13], predictors of marijuana initiation only were assessed for 9th (Model #13) and 12th (Model #14) grade students. Models #15-17 and Models #18-20 included predictors of time to alcohol and cigarette initiation among 6th, 9th, and 12th grade students, while predictors of time to marijuana initiation were assessed for 9th (Model #21) and 12th (Model #22) grade students.

The development of the age-graded models differed from the total sample models on one important front. Although the H1 soft drug sequence informed the types of drug-related predictors that were entered into respective age-graded models (mirroring the strategy employed with the total sample models), the biological ages of each subsample also were considered in assessing the impact

[13] Only 2.1% (n = 6) of 6th grade students self-reported initiating marijuana use.

of the soft drug extraneous measures on cigarette and marijuana initiation and time to initiation. [14]

In particular, the utility of *AlcInit* (whether alcohol initiation occurred) was assessed in the 6th grade cigarette initiation (and time to initiation) model.[15] In examining cigarette initiation (and time to initiation) among 9th grade students, early age of alcohol initiation (*AlcEarly*) was assessed, while the efficacy of both early age of alcohol (*AlcEarly*) and cigarette (*CigEarly*) initiation were considered in the 9th grade marijuana initiation (and time to initiation) model. Finally, both early (*AlcEarly*) and late (*AlcLate*) age of alcohol initiation were entered into the 12th grade cigarette initiation and time to initiation models, while the utility of early (*AlcEarly*) and late (*AlcLate*) age of alcohol initiation and early (*CigEarly*) and late (*CigLate*) age of cigarette initiation were gauged in the 12th grade marijuana initiation (and time to initiation) model.

The presentation of age-graded findings is organized as follows. First, each subsample is described in terms of their respective distributions for each outcome measure. Next, the multivariate results for soft drug initiation are presented, followed by findings for time to initiation. For all regression models, results significant at the .05 level (or lower) are emphasized.

Subsample Descriptives

The descriptive information for alcohol and cigarette initiation and age of initiation among 6th grade students, along with alcohol, cigarette, and marijuana

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[14] Discussed in Chapter 7, early age of initiation was operationalized as that which had occurred between 8 and 15 years, while late age of initiation was viewed as that which had occurred between 16 and 18 years of age.
[15] Infrequent use of alcohol among 6th graders precluded the need to control for *AlcFreq* in the 6th grade cigarette initiation (and time to initiation) model.

initiation and age of initiation among 9th and 12th grade students is presented in Table 52. A total of 281 6th grade students, 238 9th grade students, and 234 12th grade students provided useable data, thereby constituting the sample sizes for these respective subsamples.

Sixth grade students ranged in age from 11 to 15 years, with the majority (65.5%) self-reporting 12 years as their biological age at the time of data collection. Only three students were 15 years of age. Roughly 18% and 8% of 6th grade students self-reported alcohol and cigarette initiation, respectively. On average, 6th grade alcohol initiates (n = 50) and cigarette initiates (n = 23) initiated these drugs at roughly 10 years of age, although alcohol use was initiated at a slightly younger age, on average, than cigarette use.

Ninth grade students ranged in age from 14 to 16 years. The majority (66.4%) of these students were 15 years of age at the time of data collection. Nine students were 16 years of age. Alcohol, cigarette, and marijuana initiation were reported by 60%, 42%, and 28% of 9th graders, respectively. While the average age of alcohol and cigarette initiation was equivalent (12.8 years, respectively), the average age of marijuana initiation was 13.8 years.

The majority (70%) of 12th grade students were 18 years old at the time of data collection (age range = 16-19 years). Two students were 16 years of age, while nine students were 19 years of age. Among 12th graders, 82%, 56%, and 37% self-reported alcohol, cigarette, and marijuana initiation, respectively. The average age of cigarette initiation (13.8 years) was younger than the average

Table 52.

Dependent Measures: Descriptives for Age-Graded Subsamples

Variable	Min	Max	Mean	SD
6th Grade (n = 281)				
Alcohol Initiation	0	1	.177	.383
Cigarette Initiation	0	1	.081	.274
Age of Alcohol Initiation [a]	8	12	9.980	1.449
Age of Cigarette Initiation [b]	8	12	10.260	1.136
9th Grade (n = 238)				
Alcohol Initiation	0	1	.600	.490
Cigarette Initiation	0	1	.424	.495
Marijuana Initiation	0	1	.277	.448
Age of Alcohol Initiation [c]	8	15	12.846	1.602
Age of Cigarette Initiation [d]	8	15	12.861	1.708
Age of Marijuana Initiation [e]	11	15	13.833	.970
12th Grade (n = 234)				
Alcohol Initiation	0	1	.816	.388
Cigarette Initiation	0	1	.564	.496
Marijuana Initiation	0	1	.371	.484
Age of Alcohol Initiation [f]	8	18	14.581	2.310
Age of Cigarette Initiation [g]	8	18	13.825	2.512
Age of Marijuana Initiation [h]	8	18	14.505	1.987

Note. Dichotomous initiation measures derived from initiation data from all respondents in each age-graded subsample.
[a] Only includes alcohol initiates, valid n = 50
[b] Only includes cigarette initiates, valid n = 23
[c] Only includes alcohol initiates, valid n = 143
[d] Only includes cigarette initiates, valid n = 101
[e] Only includes marijuana initiates, valid n = 66
[f] Only includes alcohol initiates, valid n = 191
[g] Only includes cigarette initiates, valid n = 132
[h] Only includes marijuana initiates, valid n = 87

age of alcohol initiation (14.5 years), with alcohol and marijuana initiates beginning to use these respective drugs at roughly the same age.

Alcohol Initiation

6th Grade Students

Tables 53-55 present the multivariate results for alcohol initiation among 6th, 9th, and 12th grade students. Explaining roughly 25% of the variance in alcohol initiation among 6th grade students, six predictors (*EasyAlc*, *Academic*, *PeerAlc*, *AlcIntent*, *Steal*, and *ViolBeh*) increased, and one variable (*Religious*) decreased, the log odds of alcohol initiation (see Table 53). Noted earlier, *EasyAlc*, *PeerAlc*, *AlcIntent*, and *Steal* were predictive of alcohol initiation among the total sample.

As the most highly significant predictor of alcohol initiation, 6th graders who were inclined to use alcohol (*AlcIntent*, b = 1.625; p <.001) were over five times more likely to have initiated alcohol use, as compared to youth who were not so inclined. Compared to their counterparts, youth who were involved in (or threatened) violent behavior (b = 1.126; p <.001), engaged in stealing (b = .954; p <.05), and perceived that many of their peers engaged in alcohol use (b = .816; p <.001) also were at a considerably higher risk for alcohol initiation (over 3.0, 2.5, and 2.0 times, respectively). Counter to H3, 6th grade students who engaged in academic activities on a frequent basis (*Academic*, b = .520; p <.05) were at an increased risk for alcohol initiation.

Table 53.

Model #7: Predictors of Alcohol Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.692* (.408)	1.998	.710* (.434)	2.034	---	---	---	---	---	---	---	---
EasyAlc	.456*** (.116)	1.578	.448*** (.124)	1.565	.391*** (.130)	1.479	.356*** (.129)	1.427	.324** (.134)	1.383	.324** (.134)	1.383
Truancy			.875*** (.245)	2.399	.846*** (.250)	2.331	.698*** (.255)	2.010	---	---	---	---
Academic			.485** (.231)	1.625	.511** (.234)	1.667	.576** (.251)	1.778	.520** (.251)	1.682	.520** (.251)	1.682
AttachSchool			-.158*** (.048)	.854	-.139*** (.048)	.870	-.111** (.050)	.895	---	---	---	---
MessParAlc					.965** (.415)	2.626	---	---	---	---	---	---
PeerAlc							.708*** (.239)	2.030	.816*** (.240)	2.261	.816*** (.240)	2.261
MessPeerAlc							1.088*** (.393)	2.968	---	---	---	---

(Table 53 continues)

(Table 53 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									1.625*** (.429)	5.078	1.625*** (.429)	5.078
Steal									.954** (.447)	2.595	.954** (.447)	2.595
ViolBeh									1.126*** (.429)	3.083	1.126*** (.429)	3.083
Religious									-.328** (.139)	.720	-.328** (.139)	.720
-2LL	238.716***		211.494***		209.086***		197.065***		180.200***		180.200***	
Cox/Snell R^2	.083		.168		.175		.210		.256		.256	

[a] N = 281; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 54.

Model #8: Predictors of Alcohol Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.918** (.405)	2.505	1.131** (.485)	3.098	.959** (.488)	2.610	---	---	---	---	---	---
EasyAlc	.524*** (.111)	1.689	.450*** (.137)	1.569	.407*** (.138)	1.502	.316** (.140)	1.372	---	---	---	---
Truancy			.396** (.195)	1.485	.398** (.192)	1.489	.345* (.199)	1.412	---	---	---	---
Cheat			.795*** (.180)	2.214	.808*** (.180)	2.243	.759*** (.187)	2.136	.821*** (.211)	2.273	.884*** (.217)	2.419
Academic			-.364** (.184)	.695	-.401** (.182)	.670	-.377* (.187)	.686	---	---	---	---
AttachSchool			-.089* (.052)	.915	---	---	---	---	---	---	---	---
HiAcadPerf			-.470*** (.171)	.625	-.520*** (.168)	.594	-.548*** (.173)	.578	-.584*** (.188)	.558	-.520*** (.192)	.594
MessParAlc					.854* (.462)	2.350	1.072** (.466)	2.921	1.094** (.489)	2.986	.983** (.488)	2.673

(Table 54 continues)

(Table 54 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
PeerAlc							.804*** (.230)	2.235	.982*** (.251)	2.669	1.145*** (.264)	3.142
AlcIntent									2.601*** (.506)	13.484	2.720*** (.535)	15.186
Sports									-.438** (.212)	.646	-.539** (.224)	.583
Gender											.958** (.410)	2.606
-2LL	290.035***		219.802***		219.148***		209.575***		185.674***		179.944***	
Cox/Snell R^2	.119		.344		.346		.372		.432		.445	

[a] N = 238; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 55.

Model #9: Predictors of Alcohol Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	-1.775 (1.084)	.170	-1.689 (1.109)	.185	-1.689 (1.109)	.185	-1.689 (1.109)	.185	-2.727* (1.425)	.065	-2.677** (1.355)	.069
EasyAlc	.597*** (.166)	1.817	.499*** (.174)	1.647	.499*** (.174)	1.647	.499*** (.174)	1.647	.364* (.202)	1.439	---	---
Truancy			.669*** (.181)	1.952	.669*** (.181)	1.952	.669*** (.181)	1.952	.720*** (.216)	2.055	.892*** (.230)	2.440
AlcIntent									2.788*** (.531)	16.244	3.238*** (.585)	25.486
Steal									.960* (.531)	2.612	.942* (.531)	2.566
Gender											-.851* (.447)	.427
Race											-1.421* (.782)	.241
-2LL	207.401***		191.607***		191.607***		191.607***		152.442***		147.903***	
Cox/Snell R^2	.066		.127		.127		.127		.261		.275	

[a] N = 234; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Frequent involvement in religious activities (b = -.328; *p* <.05) was the only predictor found to significantly decrease the log odds of alcohol initiation among 6th grade students. For every one-unit increase in frequency of involvement in religious activities, the simple odds of alcohol initiation decreased by 28%.

Evidence of full mediation was found for five variables: *MessMedAlc*, *MessParAlc*, *AttachSchool*, *MessPeerAlc*, and *Truancy*. *MessParAlc* mediated the risk effects of *MessMedAlc*. In turn, the risk effects of *MessParAlc* were reduced to non-significance by the peer domain predictors. The individual domain predictors mediated the protective effect of *AttachSchool*, as well as the risk effects exerted by *Truancy* and *MessPeerAlc.*

9th Grade Students

Seven variables accounted for roughly 45% of the variation in alcohol initiation among 9th grade students (see Table 54). Five of these predictors (*Cheat*, *MessParAlc*, *PeerAlc*, *AlcIntent*, and *Gender*) increased risk for alcohol initiation, while two (*HiAcadPerf* and *Sports*) demonstrated protective capacities. Three of these factors (*Cheat*, *PeerAlc*, and *AlcIntent*) were predictive among the total sample as well.

Similar to findings for alcohol initiation among both the total sample and the 6th grade subsample, *AlcIntent* (b = 2.720; *p* <.001) was the most highly significant predictor of the log odds of alcohol initiation among 9th graders. In addition, compared to their 9th grade counterparts, 9th grade males (*Gender*, b = .958; *p* <.05), students who believed many of their peers engaged in alcohol use (*PeerAlc*, b = 1.145; *p* <.001), youth who cheated frequently on schoolwork

(*Cheat*, b = .884; p <.001), and 9th graders who felt their parents condoned alcohol use (*MessParAlc*, b = .983; p <.05), also were at a considerably higher risk for alcohol initiation (over 2.5, 3.0, 2.0, and 2.5 times, respectively).

Although there were insignificant predictors among 6th grade students, *Sports* and *HiAcadPerf* were important in decreasing the log odds of alcohol initiation among 9th graders. In fact, the simple odds of alcohol initiation were 39% and 42% lower among 9th graders who earned better grades (b = -.520; p <.001) and who were involved in sports on a more frequent basis (b = -.539; p <.05).

Full mediation was found for five variables. The individual domain factors completely mediated the relationship between alcohol initiation and *EasyAlc*, *Truancy*, and *Academic*. The peer domain reduced to non-significance the risk effect of *MessMedAlc*, while parental tolerance of alcohol use mediated the protective impact of *AttachSchool*.

12th Grade Students

Of the 21 variables that were assessed in the 12th grade alcohol initiation model, only three factors (*AlcIntent*, *Truancy*, and *MessMedAlc*) exerted significant, direct effects (see Table 55). These variables were quite important, however, with roughly 28% of the variation in alcohol initiation explained.

As the most highly significant predictor, *AlcIntent* (b = 3.238; p <.001) engendered considerable risk for alcohol initiation. In fact, seniors who were willing to engage in alcohol use were over 25 times more likely to have initiated alcohol use, as compared to 12th grade students who were less willing. This factor also was a salient predictor of alcohol initiation among the total sample and 6th and 9th grade students. Another total sample predictor, *Truancy*, was predictive of alcohol initiation among 12th graders. For every one-unit increase in the frequency of skipping school (b = .892; p <.001), the simple odds of alcohol initiation increased more than two-fold. Contrary to H2, media tolerance of alcohol use (*MessMedAlc*, b = -2.677; p <.05) served to decrease the log odds of alcohol initiation. *Steal*, *Gender*, and *Race* were retained in the final model, but these variables were significant only at the .10 level.

Full mediation was found for one variable. Once gender and race effects were taken into account, impact of *EasyAlc* on alcohol initiation was nullified.

Cigarette Initiation

6th Grade Students

Tables 56-58 present the results for cigarette initiation among 6th, 9th, and 12th grade students. In culmination, eight variables explained roughly 34% of the variance in the log odds of cigarette initiation among 6th grade students (see Table 56). Five of these predictors (*EasyCig*, *MessPeerCig*, *AlcInit*, *CigIntent*, and *Social*) exerted risk effects, while *HiAcadPerf*, *AlcIntent*, and *Religious* served as protective mechanisms. Of these variables, four (*EasyCig*, *CigIntent*, *HiAcadPerf*, and *AlcIntent*) were predictive of cigarette initiation among the total sample. Taking into account Wald and significance values, the most highly significant predictor of cigarette initiation among 6th grade students was *CigIntent*

Table 56.

Model #10: Predictors of Cigarette Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyCig	.697*** (.160)	2.007	.658*** (.191)	1.932	.658*** (.191)	1.932	.403* (.206)	1.496	.687** (.311)	1.987	.687** (.311)	1.987
Truancy			.928*** (.287)	2.529	.928*** (.287)	2.529	.655* (.370)	1.924	---	---	---	---
AttachSchool			-.233*** (.069)	.792	-.233*** (.069)	.792	-.224*** (.081)	.799	---	---	---	---
HiAcadPerf			-1.000*** (.297)	.368	-1.000*** (.297)	.368	-1.150*** (.343)	.317	-1.585*** (.519)	.205	-1.585*** (.519)	.205
PeerCig							.957** (.435)	2.604	---	---	---	---
MessPeerCig							1.724** (.764)	5.607	2.455** (1.027)	11.644	2.455** (1.027)	11.644
AlcInit									3.976*** (1.154)	53.298	3.976*** (1.154)	53.298
CigIntent									4.112*** (1.226)	61.066	4.112*** (1.226)	61.066
AlcIntent									-2.464** (1.173)	.085	-2.464** (1.173)	.085

(Table 56 continues)

(Table 56 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Religious									-.980*** (.350)	.375	-.980*** (.350)	.375
Social									1.563*** (.522)	4.773	1.563*** (.522)	4.773
-2LL	135.115***		90.366***		90.366***		74.021***		43.191***		43.191***	
Cox/Snell R^2	.082		.217		.217		.261		.338		.338	

[a] N = 281; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 57.

Model #11: Predictors of Cigarette Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedCig	.629* (.333)	1.875	.725** (.357)	2.064	.725** (.357)	2.064	---	---	---	---	---	---
EasyCig	.671*** (.125)	1.955	.615*** (.136)	1.850	.615*** (.136)	1.850	.568*** (.138)	1.765	.603*** (.195)	1.827	.603*** (.195)	1.827

(Table 57 continues)

(Table 57 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Cheat			.627*** (.167)	1.872	.627*** (.167)	1.872	.592*** (.166)	1.808	-.469* (.273)	.626	-.469* (.273)	.626
Academic			-.418*** (.149)	.659	-.418*** (.149)	.659	-.415*** (.149)	.661	-.516** (.228)	.597	-.516** (.228)	.597
CllgeAsp			-1.127** (.446)	.325	-1.127** (.446)	.325	-1.053** (.443)	.349	---	---	---	---
MessPeerCig							.748** (.358)	2.113	---	---	---	---
AlcEarly									3.838*** (.817)	46.446	3.838*** (.817)	46.446
CigIntent									3.591*** (.541)	36.266	3.591*** (.541)	36.266
Work									.325* (.199)	1.384	.325* (.199)	1.384
-2LL	280.204***		248.458***		248.458***		248.226***		120.664***		120.664***	
Cox/Snell R^2	.170		.273		.273		.274		.575		.575	

[a] N = 238; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 58.

Model #12: Predictors of Cigarette Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	-1.955** (.751)	.142	-1.955** (.779)	.142	-1.925** (.781)	.146	-1.877** (.780)	.153	-1.961** (.873)	.141	-1.860** (.875)	.156
EasyCig	.532* (.285)	1.702	---	---	---	---	---	---	---	---	---	---
Truancy			.287** (.128)	1.333	.253* (.131)	1.288	.229* (.132)	1.257	---	---	---	---
MessTchrAlc			.574** (.282)	1.776	.485* (.288)	1.624	.603** (.290)	1.827	---	---	---	---
HiAcadPerf			-.301** (.143)	.740	-.279* (.145)	.757	-.249* (.147)	.780	-.433** (.200)	.649	-.416** (.204)	.660
MessParCig					.759** (.388)	2.137	---	---	---	---	---	---
MessPeerCig							1.022*** (.314)	2.779	---	---	---	---

(Table 58 continues)

(Table 58 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcEarly									3.572*** (.754)	35.605	2.921*** (.824)	18.562
AlcLate									2.388*** (.745)	10.894	1.816** (.800)	6.146
CigIntent									3.684*** (.522)	39.802	3.500*** (.536)	33.099
AlcIntent									-1.867** (.738)	.155	-2.010*** (.726)	.134
AlcFreq											.119* (.069)	1.126
-2LL	305.720***		291.307***		287.286***		280.411***		161.304***		158.239***	
Cox/Snell R^2	.061		.117		.132		.158		.494		.500	

[a] N = 234; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

(b = 4.112; *p* <.001), followed by alcohol initiation (b = 3.976; *p* <.001), good school grades (*HiAcadPerf*, b = -1.585; *p* <.001), and involvement in social and entertainment activities (*Social*, b = 1.563; *p* <.001). Contrary to expectations, intentions to use alcohol (*AlcIntent*, b = -2.464; *p* <.05) operated in a protective manner, while involvement in social activities (*Social*) increased risk for cigarette initiation.

The effects of three predictors (*Truancy*, *AttachSchool*, and *PeerCig*) were fully mediated. All were reduced to non-significance, once the individual domain predictors were entered into the model.

9th Grade Students

Among 9th grade students, six variables explained nearly 60% of the variation in the log odds of cigarette initiation (see Table 57). *EasyCig* (b = .603; *p* <.001), *AlcEarly* (b = 3.838; *p* <.001), and *CigIntent* (b = 3.591; *p* <.001) increased risk for cigarette initiation, while involvement in extracurricular academic activities (*Academic*, b = -.516; *p* <.05) reduced the log odds of cigarette initiation. Of these measures, three variables (*EasyCig*, *AlcEarly*, and *CigIntent*) predicted cigarette initiation among the total sample. The most highly significant predictor of cigarette initiation among 9th graders was early age of alcohol initiation, followed by intentions to use cigarettes. Compared to alcohol abstainers and 9th graders who initiated alcohol use after 15 years of age, 9th grade students who initiated alcohol use between 8 and 15 years of age were over 46 times more likely to have initiated cigarette use. Although retained in the final model, *Cheat* (b = -.469; *p* <.10) and *Work* (b = .325; *p* <.10) were not significant factors at the .05 level.

Three predictors were fully mediated by more proximal variables. The peer domain variables mediated the relationship between *MessMedCig* and cigarette initiation, while the individual domain reduced the risk effects of *MessPeerCig* and protective effects of *CllgeAsp* to non-significance.

12th Grade Students

As depicted in Table 58, seven predictors (*MessMedAlc*, *HiAcadPerf*, *AlcEarly*, *AlcLate*, *CigIntent*, *AlcIntent*, and *AlcFreq*) accounted for 50% of the variance in cigarette initiation among 12th grade students. Frequent alcohol use (b = .119; *p* <.10) was not significant at a conventional level, however. Early age of alcohol initiation (b = 2.921; *p* <.001), late age of alcohol initiation (b = 1.816; *p* <.05), and intentions to use cigarettes (b = 3.500; *p* <.001) all increased the log odds of cigarette initiation, while media tolerance of alcohol use (b = -1.860; *p* <.05), good grades in school (b = -.416; *p* <.05), and intentions to use alcohol (b = -2.010; *p* <.001) decreased the log odds of cigarette initiation. *CigIntent* was the most highly significant predictor, followed by early age of alcohol initiation and intentions to use alcohol. Four of these constructs *(HiAcadPerf, AlcEarly, CigIntent*, and *AlcIntent*) were significant predictors of cigarette initiation among the total sample.

Similar to cigarette initiation among the total sample and 6th grade students, *AlcIntent* operated in a protective manner, by reducing the log odds of cigarette initiation. The direction of the relationship between cigarette initiation and media tolerance of alcohol use also countered H2, with an 84% decrease in the simple odds of cigarette initiation associated with a one-unit increase in *MessMedAlc* (from intolerance to tolerance).

Concerning distal-proximal mediation, the risk effects of *EasyCig* were fully mediated by the school domain predictors, while the peer domain variables reduced the risk effects of *MessParCig* to non-significance. Finally, when the individual domain predictors were taken into account, the independent risk effects exerted by *Truancy*, *MessTchrAlc*, and *MessPeerCig* were nullified.

Marijuana Initiation

9^{th} Grade Students

Tables 59-60 present the predictors of marijuana initiation among 9^{th} and 12^{th} grade students. First, five predictors (*MarIntent*, *Religious*, *Social*, *AlcFreq*, and *CigFreq*) explained a considerable amount of variation (R^2 = 55%) in marijuana initiation among 9^{th} grade students (see Table 59). Frequent involvement in religious activities (b = -.586; p <.001) was associated with a 44% decrease in the log odds of marijuana initiation, while the other variables exerted risk effects. *MarIntent* constituted the most highly significant predictor of marijuana initiation among 9^{th} graders. Compared to 9^{th} grade students who were not willing to engage in marijuana use, 9^{th} graders who expressed some degree of willingness to use marijuana (*MarIntent*, b = 2.343; p <.001) were over 10 times more likely to have initiated marijuana use.

Table 59.

Model #13: Predictors of Marijuana Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyAlc	-.321* (.175)	.725	-.490** (.191)	.664	-.409** (.191)	.664	-.384** (.194)	.681	---	---	---	---
EasyMar	.501*** (.153)	1.650	.370** (.165)	1.447	.370** (.165)	1.447	---	---	---	---	---	---
EasyCig	.642*** (.215)	1.901	.709*** (.236)	2.031	.709*** (.236)	2.031	.869*** (.223)	2.385	---	---	---	---
Truancy			.318* (.171)	1.375	.318* (.171)	1.375	---	---	---	---	---	---
MessTchrCig			-1.801*** (.666)	.165	-1.801*** (.666)	.165	-1.603** (.640)	.201	---	---	---	---
AttachSchool			-.187*** (.056)	.829	-.187*** (.056)	.829	-.228*** (.056)	.796	-.141** (.070)	.868	---	---
Cheat			.472** (.202)	1.603	.472** (.202)	1.603	.542*** (.201)	4.756	---	---	---	---
MessPeerMar							1.559*** (.461)	4.756	---	---	---	---

(Table 59 continues)

(Table 59 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigEarly									3.166*** (.670)	23.710	---	---
MarIntent									2.945*** (.622)	19.001	2.343*** (.649)	10.407
Religious									-.389** (.172)	.678	-.586*** (.200)	.557
Social									.743** (.343)	2.101	.891** (.410)	2.437
AlcFreq											.228*** (.074)	1.256
CigFreq											1.064*** (.232)	2.899
-2LL	232.745***		196.996***		196.996***		192.600***		109.088***		92.602***	
Cox/Snell R^2	.184		.297		.297		.310		.514		.547	

[a] N = 238; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 60.

Model #14: Predictors of Marijuana Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.735** (.329)	2.086	.798** (.350)	2.222	.739** (.355)	2.094	---	---	---	---	---	---
MessMedAlc	-2.011*** (.665)	.134	-2.114*** (.718)	.121	-2.161*** (.734)	.115	-1.972*** (.746)	.139	-2.496*** (.743)	.082	-2.597*** (.907)	.075
EasyMar	.887*** (.167)	2.428	.819*** (.171)	2.269	.806*** (.174)	2.240	.763*** (.174)	2.145	.949*** (.246)	2.583	.854*** (.245)	2.350
EasyCig	1.195 (.868)	3.304	1.072 (.828)	2.921	1.269 (.856)	3.557	1.156 (.874)	3.177	---	---	---	---
Truancy			.353** (.149)	1.424	.360** (.150)	1.433	.365** (.161)	1.440	.414* (.219)	1.513	---	---
Academic			-.463*** (.165)	.629	-.458*** (.168)	.632	-.428** (.178)	.652	-.593** (.274)	.553	-.646** (.263)	.524
MessParMar					1.142** (.527)	3.133	---	---	---	---	---	---
MessPeerMar							2.171*** (.452)	8.766	1.621*** (.621)	5.056	1.509** (.644)	4.520
MessPeerAlc							-2.324*** (.879)	.098	-2.652*** (1.139)	.071	-3.104*** (1.069)	.045

(Table 60 continues)

(Table 60 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigEarly									3.089*** (.761)	21.958	1.043* (.567)	2.836
CigLate									2.703*** (.861)	14.931	---	---
AlcEarly									.997* (.556)	2.710	1.372** (.573)	3.944
MarIntent									3.142*** (.609)	23.139	3.469*** (.679)	32.118
CigIntent									-1.958*** (.707)	.141	-3.030*** (.890)	.048
CigFreq											.991*** (.262)	2.695
-2LL	234.080***		216.369***		211.449***		191.318***		110.424***		106.534***	
Cox/Snell R^2	.273		.326		.340		.395		.572		.579	

[a] N = 234; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

With the exception of *Social*, the direction of effects exerted by the significant predictors in the model met hypothesized expectations. Frequent involvement in social activities (b = .891; p <.05) was associated with an increased, not decreased, risk for marijuana initiation.

Some support for the H1 soft drug initiation sequence also was observed. Although both were significant at the .001 level, an examination of the Wald values for *AlcFreq* and *CigFreq* indicated that frequent cigarette use was a more important predictor of marijuana initiation than frequent alcohol use. Moreover, early age of cigarette initiation (*CigEarly*) was retained in the model, before being mediated, while early age of alcohol initiation (*AlcEarly*) never was retained.

The model also yielded substantial support for Petraitis et al.'s (1995) distal-proximal mediation hypothesis. No significant, direct relationships were observed between marijuana initiation and the community, school, family, and peer domain factors that were assessed. The peer domain variables reduced the effects of *EasyMar* and *Truancy* to non-significance, while the individual domain mediated the respective impacts of *EasyCig*, *MessTchrCig*, *Cheat*, and *MessPeerMar*. The effects of *AttachSchool* and *CigEarly* were nullified once the control variables were taken into account.

12th Grade Students

Accounting for almost 60% of the variation in the dependent variable, two community (*MessMedAlc* and *EasyMar*), one school (*Academic*), two peer (*MessPeerMar* and *MessPeerAlc*), and three individual (*AlcEarly*, *MarIntent*, and *CigIntent*) domain variables, along with *CigFreq*, exerted significant direct effects on the log odds of marijuana initiation among 12th grade students (see Table 60). Retained in the final model, *CigEarly* (b = 1.034) was significant only at the .10 level. Of the retained variables, five (*EasyMar*, *MessPeerAlc*, *MessPeerMar*, *MarIntent*, and *CigFreq*) also were significant in the corresponding total sample model.

With the exception of *MessMedAlc* (b = -2.597; *p* <.001), *MessPeerAlc* (b = -3.104; *p* <.001), and *CigIntent* (b = -3.030; *p* <.001), the direction of effects exerted by these variables met hypothesized expectations. Counter to H2, media and peer tolerance of alcohol use, as well as intentions to use cigarettes, operated in a protective fashion, by decreasing the log odds of marijuana initiation.

The most highly significant predictor of marijuana initiation among 12th graders was *MarIntent* (b = 3.469; *p* <.001). After controlling for other factors, 12th grade students who were willing to engage in marijuana use were over 32 times more likely to have initiated marijuana use than 12th graders who were less willing to engage in use. In order of importance, the second most highly significant predictor was *MessPeerAlc*, followed by *CigIntent*, and *MessMedAlc*.

Some support for the H1 soft drug initiation sequence also was observed. Although early age of cigarette initiation (*CigEarly*) did not meet the conventional threshold of significance, and early age of alcohol use (*AlcEarly*, b = 1.372; *p* <.001) was highly significant, frequent cigarette use was much more important in distinguishing marijuana abstainers from initiates than frequent alcohol use. In fact, *AlcFreq* exerted non-significant effects. In contrast, every one-unit increase in the frequency with which 12th graders smoked cigarettes elicited a more than two-fold increase in the simple odds of marijuana initiation.

Five variables were fully mediated by more proximal predictors. The peer domain mediated the risk effects of *MessMedMar* and *MessParMar*, while the individual domain nullified the risk effect elicited by *EasyCig*. Finally, once the control variables were taken into account, the risk capacities of *Truancy* and *CigLate* were reduced to insignificance.

Time to Alcohol Initiation

Using age-graded data, three Cox regression models were developed for time to alcohol (Model #15-17) and cigarette (Model #18-20) initiation among 6th, 9th, and 12th grade students. Two additional models (Models #21-22) were designed to identify predictors of time to marijuana initiation among 9th and 12th grade students. Beginning with time to alcohol initiation (Tables 61-63), a synopsis of findings for each age-graded Cox regression model is provided.

6th Grade Students

Only five predictors wielded significant, direct effects on time to alcohol initiation among 6th grade students (Table 61). In order of importance, respective increases in the log hazard of alcohol initiation were observed among 6th graders who intended to use alcohol (*AlcIntent*), engaged in (or threatened) violent behavior (*ViolBeh*), perceived that large proportions of their peers engaged in alcohol use (*PeerAlc*), and felt that alcohol was easy to access (*EasyAlc*). *Religious*, the least highly significant predictor, was the only direct effect variable found to decrease the log hazard of alcohol initiation among 6th grade students.

Table 61.

Model #15: Predictors of Time to Alcohol Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyAlc	.452***	1.572	.436***	1.547	.371***	1.449	.313***	.240**	.240**	1.272	.240**	1.272
	(.101)		(.103)		(.109)		(.108)	(.103)	(.103)		(.103)	
Truancy			.611***	1.842	.567***	1.763	.439***	1.550	---	---	---	---
			(.146)		(.150)		(.162)					
AttachSchool			-.133***	.875	-.115***	.891	-.087**	.917	---	---	---	---
			(.036)		(.038)		(.039)					
Academic			.402**	1.495	.413**	1.511	.468**	1.597	.296	1.345	.296	1.345
			(.177)		(.176)		(.186)		(.183)		(.183)	
MessParAlc					.599*	1.821	---	---	---	---	---	---
					(.328)							
PeerAlc							.499***	1.646	.611***	1.843	.611***	1.843
							(.169)		(.178)		(.178)	
MessPeerAlc							.916***	2.499	---	---	---	---
							(.339)					

(Table 61 continues)

(Table 61 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									1.285*** (.367)	3.613	1.285*** (.367)	3.613
ViolBeh									.961*** (.299)	2.613	.961*** (.299)	2.613
Religious									-.202** (.101)	.817	-.202** (.101)	.817
Social									.231* (.135)	1.260	.231* (.135)	1.260
-2LL	529.970		504.548		501.283		489.441		476.955		476.955	
Model χ^2	22.111***		53.692***		61.322***		75.522***		85.114***		85.114***	

[a] N = 281; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

Table 62.

Model #16: Predictors of Time to Alcohol Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.535* (.291)	1.707	.471 (.292)	1.602	---	---	---	---	---	---	---	---
EasyAlc	.321*** (.071)	1.379	.199** (.079)	1.220	.198** (.080)	1.219	.153* (.081)	1.165	---	---	---	---
Cheat			.410*** (.104)	1.506	.417*** (.102)	1.517	.355*** (.104)	1.426	.325*** (.111)	1.384	.325*** (.111)	1.384
AttachSchool			-.045* (.025)	.956	---	---	---	---	---	---	---	---
HiAcadPerf			-.157** (.073)	.855	-.177** (.069)	.838	-.176** (.070)	.838	---	---	---	---
MessParAlc					.460** (.184)	1.584	.450** (.184)	1.568	.388** (.181)	1.474	.388** (.181)	1.474
PeerAlc							.340*** (.114)	1.404	.340*** (.114)	1.405	.340*** (.114)	1.405

(Table 62 continues)

(Table 62 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									1.523*** (.375)	4.584	1.523*** (.375)	4.584
ViolBeh									.288* (.173)	1.334	.288* (.173)	1.334
Sports									-.233*** (.082)	.792	-.233*** (.082)	.792
-2LL	1448.438		1411.678		1412.147		1402.954		1377.631		1377.631	
Model χ^2	21.769***		58.537***		59.558***		68.572***		80.799***		80.799***	

[a] N = 238; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

Table 63.

Model #17: Predictors of Time to Alcohol Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	-.522** (.250)	.594	-.579** (.255)	.561	-.579** (.255)	.561	-.579** (.255)	.561	-.498* (.255)	.608	-.549** (.257)	.578
EasyAlc	.358*** (.091)	1.430	.300*** (.092)	1.349	.300*** (.092)	1.349	.300*** (.092)	1.349	.251*** (.095)	1.285	.235** (.096)	1.266
Truancy			.175** (.068)	1.191	.175** (.068)	1.191	.175** (.068)	1.191	.148** (.066)	1.160	.159** (.066)	1.173
Cheat			.128* (.076)	1.316	.128* (.076)	1.136	.128* (.076)	1.136	---	---	---	---
CllgeAsp			-.365* (.189)	.694	-.365* (.189)	.694	-.365* (.189)	.694	-.420** (.185)	.657	-.425** (.185)	.654
AlcIntent									1.307*** (.352)	3.694	1.341*** (.352)	3.822
Steal									.497*** (.156)	1.643	.524*** (.157)	1.688
ViolBeh									.301* (.176)	1.351	.308* (.177)	1.361
Social									.185* (.102)	1.204	.194* (.103)	1.214

(Table 63 continues)

(Table 63 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Race											-.624** (.252)	.536
-2LL	1864.629		1846.687		1846.687		1846.687		1808.770		1803.565	
Model χ^2	19.178***		37.511***		37.511***		37.511***		68.549***		73.920***	

[a] N = 234; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Of these constructs, four (*EasyAlc*, *AlcIntent*, *ViolBeh*, and *Religious*) were significantly associated with time to alcohol initiation among the total sample.

Worth noting is the type of impact exerted by involvement in academic and social activities. Although not statistically significant at the .05 level in the final model, frequent involvement in extra-curricular academic (b = .296; p <.20) and social (b = .231; p <.10) activities were associated with earlier, not later ages of alcohol initiation.

Four factors were fully mediated by more proximal predictors. The peer domain variables nullified the impact of parental tolerance of alcohol use, while the individual domain reduced the risk and protective effects of skipping school, school attachment, and peer tolerance of alcohol use to non-significance.

9th Grade Students

Four risk (*Cheat*, *MessParAlc*, *PeerAlc*, and *AlcIntent*) and one protective (*Sports*) construct significantly predicted time to alcohol initiation among 9th grade students (see Table 62). Parallel to findings from most of the other alcohol regression models, *AlcIntent* (b= 1.523; p <.001) was the most highly significant predictor. Retained in the final model, *ViolBeh* (b = .288) was significant at the .10 level. Unlike that observed among 6th grade students, frequent involvement in sports (*Sports*, b = -.233; p <.001) and parental tolerance of alcohol use (*MessParAlc*, b = .388; p <.05), were salient predictors of time to alcohol initiation among 9th grade students.

Four variables were fully mediated by more proximal predictors. The individual domain mediated the risk effects of *EasyAlc* and the protective capacity of *HiAcadPerf*, while *MessMedAlc* and *AttachSchool* were mediated by *MessParAlc*.

12th Grade Students

Four variables (*EasyAlc*, *Truancy*, *AlcIntent*, and *Steal*) exerted risk effects, and three variables (*MessMedAlc*, *CllgeAsp*, and *Race*) demonstrated protective capacities in predicting time to alcohol initiation among 12th graders (see Table 63). *ViolBeh* and *Social* were significant at the .10 level. Parallel to findings from the 9th grade Cox regression model for time to alcohol initiation, as well as the other total sample and age-graded alcohol regression models, *AlcIntent* (b= 1.341; *p* <.001) was the most highly significant predictor.

Surprisingly, the direction of the impact of media tolerance of alcohol use (*MessMedAlc*, b = -.549; *p* <.05) countered H2 expectations. Compared to 12th grade students who perceived the media as espousing intolerant norms concerning alcohol use, high-school seniors who felt the media condoned alcohol use were 42% less likely to have initiated alcohol use at earlier versus later biological ages. In other words, media tolerance of alcohol use served to delay age of alcohol initiation, not lower it (as expected).

Other notable findings include the following. Among seniors, college aspirations (*CllgeAsp*, b = -.425; *p* <.05) exerted a significant negative impact, delaying the age at which alcohol was initiated. As the second most highly significant factor, past involvement in stealing (*Steal*, b = .524; *p* <.001) also made an important contribution. The log hazard of alcohol initiation was 69%

greater among seniors who stole from others, compared to 12th graders who reported never stealing from others.

Another interesting finding concerns race. With a 46% decrease in the hazard of alcohol initiation among white 12th graders versus minority seniors (*Race*, b = -.624; p <.05), white students tended to initiate marijuana use at later biological ages.

Finally, not only were the family and peer factors unimportant determinants, but only one distal-proximal mediation effect was observed. Once the individual domain predictors were considered, the risk effect of *Cheat* was rendered non-significant.

Time to Cigarette Initiation

6th Grade Students

Predictors of time to cigarette initiation among 6th, 9th, and 12th grade students are presented in Tables 64-66. First, depicted in Block #6 (Table 64), five factors (*PeerCig*, *MessPeerCig*, *AlcInit*, *CigIntent*, and *Social*) lowered and four predictors (*AttachSchool*, *HiAcadPerf*, *MessParCig*, and *Religious*) delayed time to cigarette initiation among 6th grade students. Two of these variables (*HiAcadPerf* and *CigIntent*) also were important determinants in the corresponding total sample model. As the most highly significant predictor, parental tolerance of cigarette use (b = -1.901; p <.001) served to delay, not lower, age of cigarette initiation. The second and third most highly significant predictors were frequency of involvement in social activities (b = .797; p <.001)

Table 64.

Model #18: Predictors of Time to Cigarette Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyCig	.653*** (.152)	1.921	.473*** (.160)	1.605	.476*** (.163)	1.610	.340** (.161)	1.406	---	---	---	---
Truancy			.571** (.242)	1.770	.471* (.254)	1.602	---	---	---	---	---	---
MessTchrCig			1.378* (.763)	3.966	1.865** (.840)	6.455	---	---	---	---	---	---
Cheat			.338* (.191)	1.402	.360* (.196)	1.433	.442** (.191)	1.556	---	---	---	---
AttachSchool			-.204*** (.056)	.816	-.221*** (.057)	.802	-.153*** (.048)	.858	-.124** (.055)	.884	-.124** (.055)	.884
HiAcadPerf			-.668** (.270)	.513	-.693*** (.253)	.500	-.754*** (.254)	.470	-.613** (.303)	.542	-.613** (.303)	.542
MessParCig					-1.260* (.721)	.284	-1.065 (.680)	.345	-1.901*** (.703)	.149	-1.901*** (.703)	.149
MessParAlc					1.000* (.521)	2.718	---	---	---	---	---	---

(Table 64 continues)

(Table 64 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
PeerCig							1.110*** (.346)	3.034	.812** (.373)	2.252	.812** (.373)	2.252
MessPeerCig							1.521** (.663)	4.579	1.479** (.691)	4.388	1.479** (.691)	4.388
AlcInit									1.068** (.469)	2.909	1.068** (.469)	2.909
CigIntent									1.459** (.648)	4.303	1.459** (.648)	4.303
Religious									-.551*** (.160)	.576	-.551*** (.160)	.576
Social									.797*** (.249)	2.218	.797*** (.249)	2.218
-2LL	232.306		186.122		181.820		171.887		151.832		151.832	
Model χ^2	25.113***		87.444***		90.532***		96.943***		132.339***		132.339***	

[a] N = 281; * $p < .10$; ** $p < .05$; *** $p < .001$; standard error in parentheses.

Table 65.

Model #19: Predictors of Time to Cigarette Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedCig	.421* (.250)	1.524	.401 (.250)	1.493	.401 (.250)	1.493	.401 (.250)	1.493	---	---	---	---
EasyCig	.511*** (.102)	1.667	.448*** (.102)	1.566	.448*** (.102)	1.566	.448*** (.102)	1.566	.311*** (.099)	1.365	.311*** (.099)	1.365
Cheat			.359*** (.113)	1.433	.359*** (.113)	1.433	.359*** (.113)	1.433	---	---	---	---
Academic			-.231*** (.087)	.794	-.231*** (.087)	.794	-.231*** (.087)	.794	---	---	---	---
CllgeAsp			-.630*** (.239)	.533	-.630*** (.239)	.533	-.630*** (.239)	.533	---	---	---	---
AlcEarly									2.332*** (.603)	10.298	2.332*** (.603)	10.298
CigIntent									1.718*** (.301)	5.576	1.718*** (.301)	5.576
ViolBeh									.389* (.203)	1.475	.389* (.203)	1.475
-2LL	1020.343		995.848		995.848		995.848		892.630		892.630	
Model χ^2	32.571***		55.809***		55.809***		55.809***		146.793***		146.793***	

[a] N = 238; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table 66.

Model #20: Predictors of Time to Cigarette Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	-.674** (.261)	.510	-.723*** (.271)	.485	-.652** (.268)	.521	-.654** (.267)	.520	---	---	---	---
EasyCig	.436* (.234)	1.547	---	---	---	---	---	---	---	---	---	---
Truancy			.187** (.080)	1.206	.175** (.079)	1.191	.162** (.080)	1.176	---	---	---	---
MessTchrAlc			.472*** (.180)	1.603	.359** (.178)	1.432	.375** (.178)	1.455	---	---	---	---
CllgeAsp			-.440** (.218)	.644	---	---	---	---	---	---	---	---
HiAcadPerf			-.148* (.089)	.862	-.183** (.085)	.833	-.157* (.085)	.854	-.203** (.078)	.816	-.199** (.078)	.819
MessParCig					.510** (.200)	1.665	.401** (.203)	1.494	---	---	---	---
MessPeerCig							.539** (.223)	1.714	---	---	---	---

(Table 66 continues)

(Table 66 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcEarly									2.752*** (.540)	15.666	2.566*** (.563)	13.010
AlcLate									1.791*** (.538)	5.995	1.664*** (.553)	5.279
CigIntent									1.380*** (.212)	3.975	1.271*** (.221)	3.565
AlcIntent									-1.464*** (.394)	.231	-1.658*** (.418)	.191
AlcFreq											.051* (.028)	1.052
-2LL	1344.641		1322.614		1320.412		1314.084		1218.818		1215.529	
Model χ^2	11.200**		36.055***		39.758***		44.020***		130.319***		133.967***	

[a] N = 234; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

and religious (b = -.551; p <.001) activities. As observed in other models, *Social* demonstrated a risk capacity, by lowering age of cigarette initiation.

Among 6th grade students, both peer tolerance (b = 1.479; p <.05) and peer use of cigarettes (b = .812; p <.05) made significant contributions to explaining time to cigarette initiation. Compared to 6th graders who felt their peers were intolerant of cigarette use, 6th grade students who believed their peers condoned cigarette use were over 4 times more likely to have initiated cigarette use at earlier biological ages. As well, a more than two-fold increase in the hazard of cigarette initiation occurred for every one-unit increase in the proportion of peers that 6th graders thought smoked cigarettes.

The peer and individual domain predictors were responsible for nullifying the risk effects of five variables. Once the individual domain constructs were taken into account, the risk effects of *EasyCig*, the sole community domain predictor initially retained in the model, and *Cheat*, were rendered non-significant. *Truancy*, *MessTchrCig*, and *MessParAlc* were fully mediated by peer-related factors.

9th Grade Students

Only three variables wielded significant, direct effects on the hazard of cigarette initiation among 9th grade students (see Table 65). *ViolBeh* (b = .389; p <.10) was retained in the model, but did not meet a conventional level of significance. In order of importance, early age of alcohol initiation (*AlcEarly*, b = 2.332; p <.001), intentions to use cigarettes (*CigIntent*, b = 1.718; p <.001), and easy access to cigarettes (*EasyCig*, b = .311; p <.001) lowered age of cigarette initiation. Two of these variables (*EasyCig* and *CigIntent*) were predictive of time to cigarette initiation among the total sample. Early age of alcohol initiation was a particularly important factor among 9th graders. Compared to 9th grade students who abstained from alcohol (and the few students who initiated alcohol use between 16 and 18 years of age), 9th graders who initiated alcohol use between 8 and 15 years of age were over 10 times as likely to have initiated cigarette use at earlier versus late biological ages.

Evidence of full mediation was observed for four variables. Three school factors (*Cheat*, *Academic*, and *CllgeAsp*), along with *MessMedCig*, were fully mediated by the individual domain variables.

12th Grade Students

Of the 31 total predictors assessed in the 12th grade model, only five variables exerted significant, direct impacts on the hazard of cigarette initiation (see Table 66). In order of importance, early (*AlcEarly*, b = 2.556; p <.001) and late (*AlcLate*, b = 1.664; p <.001) age of alcohol initiation, as well as intentions to use alcohol (*AlcIntent*, b = -1.658; p <.001) and cigarettes (*CigIntent*, b = 1.271; p <.001), were particularly important factors. *HiAcadPerf* (b = -.199; p <.05) was important in delaying age of cigarette initiation. Four of these variables (*HiAcadPerf*, *AlcEarly*, *CigIntent*, and *AlcIntent*) were important for time to cigarette initiation among the total sample. Although retained in the final model, *AlcFreq* (b = .051) was significant only at the .10 level.

Counter to H2, instead of lowering age of cigarette initiation, *AlcIntent* served as a protective mechanism. This finding was observed among the total sample as well. Among seniors, 12th graders who were willing to use alcohol were 81% more likely to have initiated cigarette use at later versus earlier ages, compared to high-school seniors who were less willing to use alcohol.

Alcohol initiation served as the most important determinant of time to cigarette initiation among seniors, particularly alcohol initiation that occurred between childhood and mid-adolescence. Compared to 12th grade alcohol abstainers, high-school seniors who initiated alcohol use between 8 and 15 years of age (*AlcEarly*) were more than 13 times as likely to have initiated cigarette use at earlier versus later biological ages. In contrast, roughly a five-fold difference in the hazard of cigarette initiation was found between alcohol abstainers and late alcohol initiates.

With respect to distal-proximal mediation, all of the community, family, and peer domain predictors that were initially retained at some point in the model, along with the bulk of the school domain variables, were reduced to insignificance by the final model examining the hazard of cigarette initiation among 12th grade students. In particular, the school domain constructs nullified the risk effect of *EasyCig*, while the family domain fully mediated the protective capacity of *CllgeAsp*. Once the effects of the individual domain variables were considered, the effects of one community variable (*MessMedAlc*), two school (*Truancy* and *MessTchrAlc*), and one family (*MessParCig*) and peer (*MessPeerCig*) factor were rendered non-significant.

Time to Marijuana Initiation

9th Grade Students

Tables 67 and 68 outline the predictors of time to marijuana initiation among 9th and 12th grade students. First, similar to time to marijuana initiation among the total sample, time to marijuana initiation among 9th grade students was impacted predominantly by variables found within the individual domain (see Table 67). In fact, no community, school, family, or peer factors exerted significant effects on the hazard of marijuana initiation in the final model.

In order of importance, respective increases in the log hazard of marijuana initiation were observed among students who intended to use marijuana (*MarIntent*); youth who initiated cigarette use between 8 and 15 years of age (*CigEarly*); and adolescents who drank alcohol frequently (*AlcFreq*). The least highly significant predictor, *Religious* (b = -.189; p <.05) operated in a protective fashion in decreasing the log hazard of marijuana initiation. Frequent cigarette use (*CigFreq*, b = .158; p <.10) did not meet a conventional level of significance. All four of the significant predictors in this model were predictive of time to marijuana initiation among the total sample.

Some support for the H1 soft drug sequence was found in this model. Among 9th grade students, early age of alcohol initiation (*AlcEarly*) had no significant bearing on the hazard of marijuana initiation. In contrast, 9th graders who initiated cigarette use between 8 and 15 years of age (*CigEarly*) were over four times more likely to have initiated marijuana use at early ages, as compared

Table 67.

Model #21: Predictors of Time to Marijuana Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
EasyMar	.375***	1.456	.263**	1.301	.263**	1.301	---	---	---	---	---	---
	(.128)		(.123)		(.123)							
EasyCig	.516***	1.675	.563***	1.756	.563***	1.756	.602***	1.825	---	---	---	---
	(.183)		(.181)		(.181)		(.168)					
EasyAlc	-.220*	.803	-.251**	.778	-.251**	.778	-.199*	.820	---	---	---	---
	(.122)		(.127)		(.127)		(.114)					
MessTchrCig			-1.105**	.331	-1.105**	.331	-.955**	.385	---	---	---	---
			(.478)		(.478)		(.475)					
Cheat			.386**	1.471	.386**	1.471	.389**	1.475	---	---	---	---
			(.157)		(.157)		(.155)					
AttachSchool			-.119***	.888	-.119***	.888	-.137***	.872	---	---	---	---
			(.036)		(.036)		(.034)					
HiAcadPerf			-.176*	.839	-.176*	.839	---	---	---	---	---	---
			(.105)		(.105)							
MessPeerMar							.978**	2.660	---	---	---	---
							(.391)					

(Table 67 continues)

(Table 67 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigEarly									2.136*** (.542)	8.463	1.487*** (.569)	4.425
MarIntent									2.316*** (.544)	10.131	1.825*** (.565)	6.203
Religious									-.157* (.081)	.855	-.189** (.087)	.828
CigFreq											.158* (.089)	1.171
AlcFreq											.110*** (.040)	1.116
-2LL	656.883		626.449		626.449		626.011		562.674		549.578	
Model χ^2	34.936***		65.897***		65.897***		63.616***		130.253***		161.256***	

[a] N = 238; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

Table 68.

Model #22: Predictors of Time to Marijuana Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.519** (.229)	1.680	.453** (.228)	1.573	---	---	---	---	---	---	---	---
MessMedAlc	-.956*** (.311)	.384	-.784** (.313)	.457	-.669** (.229)	.512	-.592* (.304)	.553	---	---	---	---
EasyMar	.687*** (.127)	1.988	.607*** (.127)	1.835	.652*** (.125)	1.919	.611*** (.126)	1.843	.506*** (.125)	1.658	.436*** (.128)	1.546
EasyCig	1.080 (.834)	2.943	.983 (.811)	2.674	1.021 (.815)	2.775	1.097 (.833)	2.995	---	---	---	---
Truancy			.237** (.098)	1.268	.289*** (.097)	1.335	.342*** (.103)	1.408	.275** (.104)	1.316	.282** (.112)	1.325
Academic			-.248** (.103)	.780	-.247** (.101)	.781	-.209** (.099)	.812	---	---	---	---
MessParMar					.967*** (.272)	2.630	.671** (.277)	1.956	---	---	---	---
PeerAlc							-.404** (.189)	.668	-.511*** (.188)	.600	-.621*** (.199)	.537
MessPeerMar							1.207*** (.311)	3.342	---	---	---	---

(Table 68 continues)

(Table 68 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigEarly									2.175*** (.436)	8.803	1.796*** (.468)	6.025
CigLate									1.455*** (.479)	4.285	1.070** (.491)	2.915
AlcEarly									1.044*** (.303)	2.839	.987*** (.324)	2.683
MarIntent									1.195*** (.280)	3.304	1.025*** (.290)	2.787
CigIntent									-.645** (.262)	.525	-1.460*** (.378)	.232
Religious									-.136* (.070)	.873	---	---

(Table 68 continues)

(Table 68 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Gender											.437* (.252)	1.547
CigFreq											.340*** (.111)	1.405
AlcFreq											.074* (.042)	1.076
-2LL	843.827		828.047		821.427		798.777		729.312		716.959	
Model χ^2	61.725***		83.445***		87.948***		105.107***		183.402***		197.602***	

[a] N = 234; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

to 9th grade students who abstained from cigarette use or who initiated after 15 years of age.

Distal-proximal mediation was evidenced for eight variables. With the exception of *HiAcadPerf*, which was mediated by the peer domain variables, all other predictors (*EasyMar*, *EasyCig*, *EasyAlc*, *MessTchrCig*, *Cheat*, *AttachSchool*, and *MessPeerMar*) were reduced to non-significance by the individual domain predictors.

12th Grade Students

In contrast to the 9th grade model, one community (*EasyMar*), school (*Truancy*), and peer (*PeerAlc*) factor exerted significant, direct effects on the hazard of marijuana initiation among 12th grade students (see Table 68). The other significant predictors (*CigEarly*, *CigLate*, *AlcEarly*, *MarIntent*, *CigIntent*, and *CigFreq*) constituted the most proximal variables in the model. Of these variables, five (*EasyMar*, *AlcEarly*, *CigEarly*, *MarIntent*, and *CigFreq*) predicted time to marijuana initiation among the total sample. No extraneous measures conceptualized as protective in nature (H3) exerted significant, direct effects on the hazard of marijuana initiation among 12th grade students. *Gender* ($b = .437$; $p < .10$) and *AlcFreq* ($b = .074$; $p < .10$), although retained in the final model, were not significant at the .05 level.

An examination of Wald statistics and significance values indicated that early age of cigarette initiation was more highly significant than early age of alcohol initiation, late age of cigarette initiation, and intentions to use marijuana. In fact, *CigEarly* was the most highly significant predictor in the model. Compared to 12th grade cigarette abstainers, seniors who initiated cigarette use between 8 and 15 years of age were over six times more likely to have initiated marijuana use at early biological ages.

Other support for the H1 soft drug sequence relates to the control variables. Frequent alcohol use was not a strong determinant of the age at which marijuana was initiated. In contrast, for every one-unit increase in frequent cigarette use (*CigEarly*, b = .340; p <.001), the hazard of marijuana initiation increased by 41%.

The direction of the effect exerted by *CigIntent* (b = -1.460; p <.001), the second most significant predictor, as well as perceived peer alcohol use (*PeerAlc*, b = -.621; p <.001), countered H2 expectations. For every one-unit increase in perceived peer alcohol use and willingness to use cigarettes, the hazard of marijuana initiation decreased by 46% and 77%, respectively.

Seven predictors were fully mediated by more proximal variables. The impact of the family domain variables nullified the risk effect of *MessMedMar*, while the protective effect exerted by *Religious* was reduced to non-significance once the control variables were taken into account. The other five variables (*MessMedAlc*, *EasyCig*, *Academic*, *MessParMar*, and *MessPeerMar*) were fully mediated by the individual domain predictors.

Drug- and Age-Specific Predictors

According to Kandel (2002), progression in the H1 soft drug sequence can be explained by drug-specific predictors. In providing empirical evidence on this proposition, one goal of the current research was to determine whether predictors differed in kind by drug type, as well as stage of adolescent development. In conducting this assessment, only main effects were considered. Table 69 notates those extraneous measures whose significant (*p* >.05 or lower) impacts were observed for one or more of the six outcomes among the total sample and the age-graded subsamples. These effects pertain to those that were observed after the impact of all other factors were considered.

Drug-Specific Predictors

A predictor may be characterized as "drug-specific" if one or both of its' properties (i.e., saliency and direction of effect) differ by drug type. In particular, a variable typically is viewed as a drug-specific predictor if it exerts a significant impact on one soft drug outcome (e.g., alcohol initiation), but not another (e.g., cigarette and marijuana initiation), or the direction of the effect (+/-) differs according to the type of drug under inspection (Kandel, 2002).

Discussed in Chapter 7, nine predictors (*MessMedCig*, *EasyCig*, *MessTchrCig*, *MessParCig*, *PeerCig* and *MessPeerCig*, *CigIntent*, *AlcEarly*, and *AlcFreq*) were eligible for assessment in the total sample cigarette and marijuana initiation (and time to initiation) models. The 21 other independent variables and controls that were entered into all of the soft drug initiation and time to initiation models, and were subject to assessment, included one community domain variable (*MessMedAlc*), seven school (i.e., *Truancy*, *MessTchrAlc*, *CllgeAsp*, *AttachSchool*, *Academic*, *HiAcadPerf*, and *Cheat*), one family (*MessParAlc*), two peer (*PeerAlc* and *MessPeerAlc*), most of the individual domain variables (i.e., *AlcIntent*, *Steal*, *ViolBeh*, *Sports*, *Religious*, *Work*, and *Social*), and *Gender*, *Race*, and *Age*.

Four major findings emerged when the direct effects observed among the total sample were compared by drug type (see Table 69). Together, these findings provide some insight into the H1 soft drug sequence that was observed, as well as some support for Kandel's (2002) notion of drug-specific predictors.

First, no one factor universally predicted soft drug initiation and time to initiation among the total sample. *AlcIntent* was a fairly universal determinant, however, as were *Religious* and *Age*. Students' intentions to use alcohol predicted all outcomes among the total sample, with the exception of marijuana initiation. With few exceptions, *AlcIntent* also tended to impact the likelihood and hazard of soft drug initiation among the three subsamples.

Second, although no one factor was a universal predictor, drug-specific effects were observed for a limited number of predictors. For example, three of the 21 predictors that were evaluated for all six outcomes among the total sample exerted drug-specific effects. With the exception of alcohol initiation among 9th grade students, *HiAcadPerf* exerted a salient protective effect on cigarette initiation and time to initiation only. In contrast, *EasyAlc* and *Steal* were predictive of alcohol initiation and time to initiation only.

Further support for the finding of limited drug-specific effects was found in the cigarette and marijuana initiation models. In particular, only two of the nine variables that were assessed in both the cigarette and marijuana models exerted drug-specific effects. *EasyCig* and *CigIntent* were predictive of cigarette initiation

Table 69.

Total Sample and Age-Graded Results: Drug and Age-Specific Predictors (p <.05 or lower)

Domain/ Predictor	Alcohol Initiation				Cigarette Initiation				Marijuana Initiation			Time to Alcohol Initiation				Time to Cigarette Initiation				Time to Marijuana Initiation		
	TS [a]	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th	**TS**	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th
Community Domain																						
MessMedAlc				(-)				(-)			(-)				(-)							
MessMedCig	**---**	---	---	---					**(+)**			**---**	---	---	---							
EasyAlc	**(+)**	(+)										**(+)**	(+)		(+)							
EasyCig	**---**	---	---	---	**(+)**	(+)	(+)					**---**	---	---	---	**(+)**		(+)				
EasyMar	**---**	---	---	---	**---**	---	---	---	**(+)**		(+)	**---**	---	---	---	**---**	---	---	---	**(+)**		(+)
School Domain																						
Truancy	**(+)**			(+)											(+)							(+)
CllgeAsp															(-)							
AttachSchool																	(-)					
Academic		(+)					(-)				(-)											

(Table 69 continues)

(Table 69 continued)

Domain/ Predictor	Alcohol Initiation				Cigarette Initiation				Marijuana Initiation			Time to Alcohol Initiation				Time to Cigarette Initiation				Time to Marijuana Initiation		
	TS	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th	**TS**	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th
HiAcadPerf			(-)		**(-)**	(-)		(-)								**(-)**	(-)		(-)			
Cheat	**(+)**		(+)											(+)								
Family Domain																						
MessParAlc			(+)									**(+)**		(+)								
MessParCig	**---**	---	---	---								---	---	---	---		(-)					
Peer Domain																						
PeerAlc	**(+)**	(+)	(+)										(+)	(+)								(-)
PeerCig	**---**	---	---	---								---	---	---	---		(+)					
MessPeerAlc									**(-)**		(-)											
MessPeerCig	**---**	---	---	---		(+)						---	---	---	---		(+)					
MessPeerMar	**---**	---	---	---	**---**	---	---	---	**(+)**		(+)	---	---	---	---	**---**	---	---	---			

(Table 69 continues)

(Table 69 continued)

Domain/ Predictor	Alcohol Initiation				Cigarette Initiation				Marijuana Initiation			Time to Alcohol Initiation				Time to Cigarette Initiation				Time to Marijuana Initiation		
	TS	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th	**TS**	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th
Individual Domain																						
AlcInit	**---**	---	---	---	**---**	(+)	---	---	**---**	---	---	**---**	---	---	---	**---**	(+)	---	---	**---**	---	---
AlcEarly	**---**	---	---	---	**(+)**	---	(+)	(+)			(+)	**---**	---	---	---	**(+)**	---	(+)	(+)	**(+)**		(+)
AlcLate	**---**	---	---	---	**---**	---	---	(+)	**---**	---		**---**	---	---	---	**---**	---	---	(+)	**---**	---	
CigEarly	**---**	---	---	---	**---**	---	---	---	**(+)**			**---**	---	---	---	**---**	---	---	---	**(+)**	(+)	(+)
CigLate	**---**	---	---	---	**---**	---	---	---	**---**	---		**---**	---	---	---	**---**	---	---	---	**---**	---	(+)
AlcIntent	**(+)**	(+)	(+)	(+)	**(-)**	(-)		(-)				**(+)**	(+)	(+)	(+)	**(-)**			(-)	**(-)**		
CigIntent	**---**	---	---	---	**(+)**	(+)	(+)	(+)			(-)	**---**	---	---	---	**(+)**	(+)	(+)	(+)			(-)
MarIntent	**---**	---	---	---	**---**	---	---	---	**(+)**	(+)	(+)	**---**	---	---	---	**---**	---	---	---	**(+)**	(+)	(+)
Steal	**(+)**	(+)										**(+)**			(+)							
ViolBeh		(+)										**(+)**	(+)									
Sports			(-)											(-)								

(Table 69 continues)

(Table 69 continued)

Domain/ Predictor	Alcohol Initiation				Cigarette Initiation				Marijuana Initiation			Time to Alcohol Initiation				Time to Cigarette Initiation				Time to Marijuana Initiation		
	TS	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th	**TS**	6th	9th	12th	**TS**	6th	9th	12th	**TS**	9th	12th
Religious	**(-)**	(-)				(-)			**(-)**	(-)		**(-)**	(-)				(-)			**(-)**	(-)	
Social	**(+)**					(+)				(+)							(+)					
Work																				**(+)**		
Control Variables																						
Gender			(+)																	**(+)**		
Race															(-)							
Age		---	---	---	**(+)**	---	---	---	**(+)**	---	---	**(-)**	---	---	---	**(-)**	---	---	---		---	---
AlcFreq	**---**	---	---	---	**(+)**	---			**(+)**	(+)		**---**	---	---	---	**(+)**	---			**(+)**	(+)	
CigFreq	**---**	---	---	---	**---**	---	---	---	**(+)**	(+)	(+)	**---**	---	---	---	**---**	---	---	---	**(+)**		(+)

Note. --- refers to those variables that were not examined in respective regression models; +/- refers to the direction of the given relationship, with + indicative of a direct risk effect significant at the .05 level (or lower).
[a] TS = Total Sample.

and time to initiation, but not marijuana initiation or time to initiation. *MessTchrCig* was not associated with any cigarette and marijuana outcomes, while *AlcEarly* exerted important effects on the log odds and hazard of cigarette initiation, as well as the hazard of marijuana initiation. The findings for *AlcFreq* were similar to those for *AlcEarly*.

Third, another major finding that supported Kandel's (2002) notion of drug-specific predictors involved variables that were drug and outcome-specific. *Truancy*, *Cheat, PeerAlc*, and *Social* increased risk for alcohol initiation among the total sample, but had no significant, direct bearing on time to alcohol initiation, or cigarette and marijuana initiation and time to initiation among the total sample. In contrast, *MessMedCig* and *MessPeerAlc* exerted direct effects on marijuana initiation only, while *MessParAlc* and *ViolBeh* wielded direct effects on the hazard of alcohol initiation only. Finally, among the total sample, the impact of *Work* was significant for time to marijuana initiation only.

Finally, the most important evidence for the notion of drug-specific predictors was found in the differential saliency and direction of effects exerted by many of the drug-related variables. Taken together, total sample findings indicated that the bulk of these drug-related variables were drug-specific, not generic predictors of soft drug initiation and time to initiation.

With respect to differential saliency, the utility of *EasyAlc*, which was evaluated for all three soft drugs (and both outcomes), was predictive of alcohol initiation and time to initiation only. In contrast, *EasyCig*, entered into both the cigarette and marijuana outcome models, was predictive of cigarette initiation and time to initiation only. The same pattern of findings was observed for *MessParAlc*, *MessPeerCig*, *CigIntent*, and to a lesser extent, *MessParCig* and *PeerAlc*. Discussed earlier, the only major exception to this thematic finding centered on *AlcIntent*, which was predictive of the initiation and time to initiation of almost all of the three soft drugs.

Aside from differential saliency, the direction of effects for several drug-related variables differed by drug type, although some of these relationships were insignificant. As reported earlier, the direction of effects exerted by *AlcIntent* among the total sample differed according to the soft drug under inspection. Compared to students who were not willing to engage in alcohol use, youth who were willing to engage in alcohol use were more apt to have initiated alcohol use (b = 2.092; p <.001), and at younger biological ages (b = 1.454; p <.001). In contrast, *AlcIntent* served as a protective mechanism in reducing both the log odds and hazard of cigarette initiation (b = -1.360; p <.001 and b = -.659; p <.05), and delaying age of marijuana initiation (b = -1.249; p <.001). Although insignificant, the protective capacity of *AlcIntent* also was observed for marijuana initiation(b = -.372; p <.515).

CigIntent, assessed in the cigarette and marijuana initiation and time to initiation models, exerted a positive effect on cigarette initiation (b = 3.119; p <.001 and time to initiation (b = 1.491; p <.001) and a negative (but insignificant) impact on marijuana initiation (b = -.462; p = .243) and time to initiation (b = -.276; p = .221). The direction of effects exerted by *MessMedAlc* and *MessMedCig* also differed by drug type.

Age-Specific Predictors

Two major themes were revealed when the age-graded results were compared to the corresponding total sample findings. These issues included the masking of age-graded effects amidst the total sample findings and possible interaction effects involving biological age and the predictors.

Masked Effects

A comparison of age-graded and corresponding total sample findings was made, in an effort to determine the extent to which age-graded results were masked (i.e., not observed) in the corresponding total sample findings. Such a comparison revealed that 15 variables, whose effects were non-significant among one or more of the total sample models, exerted significant, direct impacts on the initiation outcomes for one or more of the age-graded subsamples (see Table 69).

The impact of one community (*MessMedAlc*) and six school (*Truancy*, *CllgeAsp*, *HiAcadPerf*, *Cheat*, *AttachSchool*, and *Academic*) factors differed according to biological age, but were masked in the total sample findings. Within the community domain, *MessMedAlc* was not a significant predictor of soft drug initiation among the total sample. Among 12th grade students, however, media tolerance of alcohol use decreased risk for soft drug initiation and delayed age of alcohol initiation.

Concerning the school factors, *Truancy*, *CllgeAsp*, *HiAcadPerf*, and *Cheat* were not important determinants of time to alcohol initiation among the total sample, yet *Truancy* and *CllgeAsp* delayed age of alcohol initiation among 12th

graders, while *CllgeAsp* delayed and *Cheat* lowered age of alcohol initiation among 9th grade students. Unimportant among the corresponding total sample, *Truancy* also delayed time to marijuana initiation among 12th graders. *AttachSchool* was not an important determinant of time to cigarette initiation among the total sample, while this bonding construct delayed age of initiation among 6th graders. Finally, *Academic*, not predictive of soft drug initiation among the total sample, increased risk for alcohol initiation among 6th graders, but decreased risk for cigarette and marijuana initiation among 9th and 12th grade students, respectively.

The effects of two family-related factors (*MessParAlc* and *MessParCig*) were important for students in particular school grades, but were masked in total sample findings. *MessParAlc*, which was not an important factor in predicting alcohol initiation among the total sample, increased risk for alcohol initiation among 9th grade students only. In contrast, *MessParCig*, a non-significant predictor of time to cigarette initiation among the total sample, was important in delaying age of cigarette initiation among 6th grade students only.

Within the peer domain, perceived peer alcohol (*PeerAlc*) did not exert significant, direct effects on time to alcohol or marijuana initiation among the total sample. In contrast to these findings, age-graded results indicated that *PeerAlc* delayed age of alcohol initiation among 6th and 9th graders, while delaying age of marijuana initiation among 12th graders. *PeerCig*, not predictive of time to cigarette initiation among the total sample, worked to lower age of cigarette initiation among 6th graders. Finally, peer tolerance of cigarette use

(*MessPeerCig*) was not an important factor in predicting any of the six initiation outcomes among the total sample. Age-graded results indicated, however, that 6th grade students were differentially impacted by this social learning construct, with peer tolerance of cigarette use increasing risk for cigarette initiation and lowering age of initiation.

The impact of six individual domain variables (*AlcEarly*, *CigIntent*, *ViolBeh*, *Sports*, *Religious*, and *Social*) also differed by biological age. Early age of alcohol initiation increased risk for cigarette initiation among the total sample, while lowering age of cigarette and marijuana initiation. Although not assessed in the 6th grade models, *AlcEarly* increased risk for marijuana initiation among 12th graders, a finding that was masked in the total sample results for this soft drug. *CigIntent* was not significantly predictive of marijuana initiation among the total sample. Among 12th grade students, however, a willingness to use cigarettes was associated with an older age of marijuana initiation, as well as an increased risk for initiating this soft drug.

Concerning *ViolBeh* and *Sports*, past involvement in (or the threatened use of) violent behavior increased risk for alcohol initiation among 6th grade students only. *Sports*, which had not direct bearing on the log odds or hazard of soft drug initiation among the total sample, decreased risk and delayed the age of alcohol initiation among 9th graders only.

The effects of *Religious* and *Social* also were age-graded. Frequent involvement in religious activities, which was not predictive of cigarette initiation among the total sample, both decreased risk and delayed age of cigarette initiation among 6^{th} grade students only. In turn, involvement in social activities, which was not an important determinant of cigarette or marijuana initiation, or time to cigarette initiation among the total sample, increased risk for cigarette initiation and lowered age of initiation among 6^{th} graders, and increased risk for marijuana initiation among 9^{th} grade students only.

Finally, age-graded differences in gender and race effects also were masked in some of the total sample results. Among the total sample, *Gender* had no significant bearing on the likelihood of alcohol initiation. Among 9^{th} graders, however, males were at a higher risk for alcohol initiation than females. The racial composition of the total sample had no significant bearing on time to alcohol initiation, but as the age-graded findings revealed, 12^{th} grade white students were at a higher risk for early age of alcohol initiation than minority students.

Possible Age X Factor Interactions

Taking the findings from the 22 regression models into account, two major issues concerning biological age highlighted the possibility that age X factor interaction effects may have been operating. First, findings for age effects (*Age*) among the total sample indicated that older students were more apt to have initiated cigarettes and marijuana than younger adolescents, while increases in biological age were associated with older ages of alcohol and cigarette initiation. These total sample findings suggest that biological age X predictor interaction effects may exist with respect to cigarette and marijuana initiation and time to alcohol and cigarette initiation, with certain variables predictive of outcomes among particular age-graded subsamples.

Second, the observation that several predictors were important factors in one or more age-graded models, but their effects were masked in the corresponding total sample models, speaks to the importance of decomposing (by stage of adolescent development) samples of youth who vary in age, since biological age may moderate the magnitude or direction of effects. Of the seven constructs (*MessMedAlc*, *MessTchrAlc*, *CllgeAsp*, *AttachSchool*, *Academic*, *Sports*, and *Race*) that were not predictive of soft drug initiation and time to initiation among the total sample, only one (*MessTchrAlc*) was not significant in the age-graded models.

Age-graded versus total sample differences in the nature of predictors underscored the possibility that the direction of effects wielded by these variables also may have differed by biological age. In an effort to identify possible biological age X factor interaction effects, the direction of the relationships between the predictors that were significant among one age-graded subsample for a given outcome, but not another subsample (or the total sample), were examined. Outlined in Table 70, the direction (+/-) of these effects are notated, with significant (p <.05 or lower) relationships bolded.

The results for *Academic* are instructive, in terms of interpreting the other findings outlined in Table 70. Among 6th grade students, frequent involvement in extra-curricular academic activities increased risk for alcohol initiation. Although not significant, *Academic* also increased risk among 12th graders, but decreased

Table 70.

Total Sample and Age-Graded Findings: Possible Age X Factor Interaction Effects

Domain/ Predictor	Alcohol Initiation				Cigarette Initiation				Marijuana Initiation			Time to Alcohol Initiation				Time to Cigarette Initiation				Time to Marijuana Initiation		
	TS [a]	6th	9th	12th	TS	6th	9th	12th	TS	9th	12th	TS	6th	9th	12th	TS	6th	9th	12th	TS	9th	12th
MessMedAlc	(+)	(+)	(+)	**(-)**								(+)	(+)	(+)	**(-)**							
Academic	(-)	**(+)**	(-)	(+)	(-)	(+)	**(-)**	(+)	(-)	(+)	**(-)**											
HiAcadPerf	(-)	(+)	**(-)**	(+)																		
MessParAlc	(+)	(+)	**(+)**	(-)																		
MessParCig																(+)	**(-)**	(-)	(+)			
PeerAlc																				(-)	(+)	**(-)**
ViolBeh	(+)	(+)	(+)	(-)																		
Sports	(-)	(-)	**(-)**	(+)								(-)	(-)	**(-)**	(+)							
Religious					(-)	**(-)**	(+)	(-)								(-)	**(-)**	(+)	(-)			
Gender	(+)	(+)	**(+)**	(-)																		
Race												(-)	(+)	(-)	**(-)**							

Note. Bolded directional effects refer to those relationships that were significant at the .05 level (or lower).
[a] TS = Total Sample.

risk for alcohol initiation among 9th grade students. A significant, negative relationship was found for *Academic* and cigarette initiation among 9th graders. Among 6th grade students, however, *Academic* exerted a non-significant positive impact, by increasing risk for cigarette initiation. Finally, frequent involvement in academic activities exerted a significant, direct impact on marijuana initiation among seniors, by eliciting a decreased likelihood of initiation. Among 9th graders, however, *Academic* a non-significant, but positive, impact on marijuana initiation, by increasing risk for initiation.

Hypotheses Results

To address Research Questions #2 and #3, nine hypotheses were tested. The utility of H2-H7 were evaluated with findings from both the total sample and the age-graded soft drug initiation and time to initiation models. In particular, findings from all 22 regression models were used to test H2 and H3. Results from the total sample and age-graded alcohol and cigarette initiation (and time to initiation) models were used in testing H4, while the utility of H5 and H6 were tested with findings for marijuana initiation (and time to initiation) among the total sample and 6th, 9th, and 12th grade students.

H7-H9 dealt specifically with the age-graded results. The age-graded findings for soft drug initiation and time to initiation were used in testing H7 and H9, while the efficacy of H8 was evaluated with the age-graded results for alcohol and cigarette initiation and time to initiation. Findings from all 22 regression models were used in evaluating the efficacy of Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

Hypothesis 2

H2 posited that predictors conceptualized as risk factors would increase risk for alcohol, cigarette, and marijuana initiation and lower age of initiation. To test H2, the direction of the regression coefficients were examined for those predictors that were retained in the final models and were conceptualized as increasing the likelihood of initiation and lowering age of initiation.

With few exceptions, H2 garnered considerable support. For instance, the direction of effects were supported with respect to alcohol, cigarette, and marijuana initiation, and time to alcohol initiation among the total sample. Concerning the age-graded findings, all of the predictors hypothesized to exert risk effects met expectations for alcohol initiation and time to initiation among 6th graders, alcohol and marijuana initiation and time to initiation among 9th grade students, and cigarette initiation among 9th graders.

Several deviations from H2 were observed, however. The factors that countered hypothesized expectations included *AlcIntent*, *CigIntent*, *PeerAlc*, *MessPeerAlc*, *Cheat*, *MessParCig*, and *MessMedAlc*. Due to these exceptions, partial support was yielded for H2.

Two of the most considerable deviations from H2 concerned intentions to use alcohol and media tolerance of alcohol. With respect to *AlcIntent*, protective functions were observed for time to cigarette (b = -.659; p <.05) and marijuana (b = -1.249; p <.001) initiation among the total sample. These findings were reinforced by those for some of the age-graded models. Among 12th grade students, intentions to use alcohol worked to decrease the likelihood of cigarette initiation (b = -2.010; *p* <.001) and the hazard of cigarette initiation (b = -1.658; *p* <.001). Cigarette initiation also was less likely to have occurred among 6th graders who were willing to use cigarettes (b = -2.464; *p* <.05), compared to 6th grade students who reported being less willing to smoke cigarettes. *CigIntent* also decreased the likelihood of marijuana initiation (b = -3.030; *p* <.001) and hazard of marijuana initiation (b = -1.460; *p* <.001) among 12th grade students.

MessMedAlc exerted significant protective effects. Instead of increasing risk, media tolerance of alcohol use decreased risk for alcohol (b = -2.677; *p* <.001) and marijuana (b = -2.597; *p* <.001) initiation, as well as time to alcohol (b = -.549; *p* <.05) and cigarette (b = -1.860; *p* <.05) initiation among 12th grade students.

A few other deviations from H2 were observed among the age-graded subsamples. Among 6th graders, parental tolerance of cigarette use (*MessParCig*, b = -1.901; *p* <.001) delayed age of cigarette initiation, while peer alcohol use and peer tolerance of alcohol use decreased the log hazard and log odds of marijuana initiation among 12th grade students (b = -.621; *p* <.001; b = -3.104; *p* <.001, respectively). Finally, although not significant at the .05 level, frequent cheating on schoolwork (b = -.469; *p* <.10) decreased the likelihood of cigarette initiation among 9th grade students.

Hypothesis 3

In contrast to H2, H3 argued that those variables viewed as protective factors would decrease risk for alcohol, cigarette, and marijuana initiation, as well as delay age of initiation. To test H3, the direction of the regression coefficients for those predictors conceptualized as reducing the likelihood of initiation and delaying age of initiation were examined in the final models. Similar to H2, H3 garnered partial support.

The direction of effects were supported with respect to cigarette and marijuana initiation and time to alcohol and cigarette initiation among the total sample. Due to the lack of significant protective effects found in the final models for time to alcohol and marijuana initiation among 12th graders, as well as time to cigarette initiation among 9th grade students, H3 could not be fully tested with findings from these models. Full support of H3 was yielded, however, from findings for the following 9th and 12th grade outcomes: alcohol initiation and time to alcohol and marijuana initiation among 9th grade students, and cigarette and marijuana initiation and time to alcohol and cigarette initiation among 12th graders.

Among the total sample, frequent involvement in social activities (b = .220; p <.05) increased risk for alcohol initiation, while frequent involvement in work-for-pay activities (b = .138; p <.05) increased the likelihood of marijuana initiation. The same risk effects were observed for several of the age-graded outcomes. Although not significant at the .05 level, *Work* exerted risk effects for cigarette initiation (b = .325; p <.10) among 9th grade students. The risk effects of *Social* were observed in four age-graded models: cigarette initiation (b = 1.563; p <.10) and time to alcohol (b = .231; p <.10) and cigarette (b = .797; p <.001) initiation among 6^{th} graders, and marijuana initiation (b = .891; p <.05) among 9^{th} grade students.

The direction of the effect exerted by *Academic* also countered H3. Among 6^{th} graders, students who frequently attended to extra-curricular academic activities were more apt to have initiated alcohol use (b = .520; p <.05) and at earlier ages (b = .296; p <.20) than their 6^{th} grade counterparts.

Hypothesis 4

H4 purported that predictors found within the peer domain (relative to predictors in other domains) would explain the largest proportion of variance in alcohol and cigarette initiation and age of initiation. This hypothesis was tested with findings for alcohol and cigarette initiation and time to alcohol and cigarette initiation among the total sample, as well as alcohol and cigarette initiation and time to initiation among 6^{th}, 9^{th}, and 12^{th} grade students.

To test this hypothesis with the initiation data, the percentage decrease in the explained variance in alcohol (R^2 = 48.3%) and cigarette (R^2 = 54.1%) initiation among the total sample, and alcohol and cigarette initiation among 6^{th} (R^2 = 26% and 34%), 9^{th} (R^2 = 45% and 58%), and 12^{th} (R^2 = 28% and 50%) grade students, respectively, was calculated by removing each significant domain from each of the respective final models.

Since Cox regression does not provide coefficient of determination statistics, model chi-square (χ^2) statistics were utilized in testing the time to initiation component of H4 with findings from these models. Specifically, the percentage decrease in the final model χ^2 attributed to removing each significant domain was calculated for time to alcohol (χ^2 = 213.512; p <.001) and cigarette (χ^2 = 378.217; p <.001) initiation among the total sample, and time to alcohol (χ^2 = 85.114, 80.799, and 73.920; p <.001) and cigarette (χ^2 = 132.339, 146.793, and 133.967; p <.001) initiation among 6th, 9th, and 12th grade students, respectively. The ecological domain responsible for the largest percentage reduction in χ^2 for each respective model was deemed as having made the largest contribution to explaining the given outcome.

In short, both total sample and age-graded findings did not support H4. The individual domain made the largest contributions to each total sample and age-graded outcome.

Total Sample Findings

Among the total sample, the largest percentage decrease in explained variation in alcohol and cigarette initiation and time to initiation was attributed to the individual domain, not the peer domain. In particular, the individual domain wielded the largest percentage decrease (16.15% and 20.7%) in explained variance in alcohol and cigarette initiation, respectively. For alcohol initiation, the second largest percentage decrease was attributed to the school domain (2.28%), while the control variables were responsible for the second largest percentage decrease (7.3%) in explained variance in cigarette initiation. The significant peer domain predictors were responsible for a small reduction (1.9%) in the explained variance in alcohol initiation, while no peer domain predictors were retained in the cigarette initiation model.

No peer domain factors were retained in the time to alcohol initiation model. In the time to cigarette initiation model, the sole peer predictor (*MessPeerCig*) was fully mediated by the individual domain. Of the significant domains found in the final time to alcohol and cigarette initiation models, the individual domain variables were responsible for the largest percentage decrease (58% and 37%) in the final model χ^2 values, respectively.

Age-Graded Findings

H4 also was not supported in the age-graded models. Concerning alcohol initiation, the individual domain wielded the largest percentage decrease (48%, 22.3%, and 60%) in explained variance in alcohol initiation among 6th, 9th, and 12th grade students, respectively. Among 6th grade students, the second largest percentage decrease was attributed to the peer domain (12.1%), while the school domain was responsible for the second largest percentage decrease among 9th (14.6%) and 12th (23%) grade students. Since no peer domain predictors were retained in the 12th grade alcohol initiation model, peer factors did not contribute to any of the explained variance in alcohol initiation among this subsample.

The individual domain also was responsible for the largest percentage decrease (41%, 58%, and 42%) in R^2 for cigarette initiation among 6th, 9th, and 12th grade students, respectively. The peer domain only was responsible for a 4.7% reduction among 6th grade students, and no peer factors were significantly related to cigarette initiation among 9th and 12th grade students.

H4 also was not supported with respect to time to alcohol and cigarette initiation among the three subsamples. Concerning time to alcohol initiation, the individual domain was responsible for the largest percentage decrease (46%, 29%, and 49%) in the final model χ^2 values for time to alcohol initiation among 6th, 9th, and 12th graders, respectively. The peer domain predictors were responsible for the second largest reductions (13% and 11%) in χ^2 for time to alcohol initiation among 6th and 9th grade students, respectively, while no peer factors were retained at any point in the 12th grade model.

Finally, the largest percentage decrease (36%, 79%, and 50%) in the final model χ^2 for time to cigarette initiation among 6th, 9th, and 12th graders, respectively, also was attributed to the individual domain variables. The peer domain was responsible for the second largest reduction (14%) in time to cigarette initiation among 6th grade students, while no peer domain variables were retained in the final models for the 9th and 12th grade subsamples.

Hypothesis 5

H5 argued that relative to other ecological domains, individual domain predictors explain the largest proportion of variance in marijuana initiation and age of initiation. To evaluate the utility of H5, results for marijuana initiation and time to marijuana initiation among the total sample were examined, as well as findings for marijuana initiation and time to marijuana initiation among 9th and 12th grade students. The same strategies used to test H4 were used in testing H5. In culmination, findings yielded partial support for H5.

Total Sample Findings

H5 was fully supported with respect to marijuana initiation and time to marijuana initiation among the total sample. The individual domain wielded the largest percentage decrease (10%) in explained variance (R^2 = 51%) in marijuana initiation, followed by the control variables (8.5% decrease) and the community domain predictors (2.8% decrease). The individual domain also was responsible for the largest percentage reduction (19.4%) in χ^2 (386.045) for time to marijuana initiation among the total sample. The second and third largest percentage reductions (11% and 2%) were attributed to the control variables and the community domain predictors, respectively.

Age-Graded Findings

Age-graded findings yielded mixed support for H5. Among 9th grade students, the largest percentage reduction (21.4%) in explained variation in marijuana initiation (R^2 = 55%) was attributed to the control variables (*AlcFreq* and *CigFreq*). The individual domain only was responsible for a 12.3% reduction in the explained variance in this model. In support of H5, however, the individual domain was responsible for the largest percentage decrease (22.8%) in explained variance (R^2 = 58%) in marijuana initiation among 12th grade students, followed by the community domain (7% decrease) and *CigFreq* (6% decrease), the sole significant control variable in the final model.

H5 findings for time to marijuana initiation among 9th and 12th grade students mirrored those for the age-graded marijuana initiation outcomes. Counter to H5, the control variables (*CigFreq* and *AlcFreq*) were responsible for the largest percentage decrease (20%) in χ (161.256) for time to marijuana initiation among 9th grade students. The individual domain accounted for a mere 8% decrease in χ^2. In contrast to time to marijuana initiation among 9th grade students, the individual domain was responsible for the largest percentage reduction (23%) in χ^2 (197.602) for time to marijuana initiation among 12th grade students.

Hypothesis 6

H6 contended that relative to alcohol initiation, cigarette initiation is a stronger predictor of marijuana initiation and age of marijuana initiation. The efficacy of H6 was evaluated with findings for marijuana initiation and time to marijuana initiation among the total sample, as well as results for marijuana initiation and time to marijuana initiation among 9th and 12th grade students, respectively. Due to counter findings for marijuana initiation among 12th grade students, H6 garnered partial support.

The total sample models yielded full support for H6. Early age of cigarette initiation (*CigEarly*, b = 1.530; p <.001) was more highly significant than early age of alcohol initiation (*AlcEarly*, b = .808; p <.10) in predicting marijuana initiation. *CigEarly* (b = 1.200; p <.001) also was more highly significant than *AlcEarly* (b = .735; p <.05) in predicting time to marijuana initiation.

H6 was fully supported with respect to marijuana initiation among 9th grade students. *AlcEarly* never was retained in the model. In contrast, *CigEarly* (b = 3.166; p <.001) was retained, although it was reduced to non-significance once the effects of the control variables were taken into account.

H6 garnered full support with respect to time to marijuana initiation among 9th and 12th grade students. Among 9th grade students, *AlcEarly* never was retained in the model, while youth who initiated cigarettes between 8 and 15 years of age (*CigEarly*) were nearly 4.5 times more likely than cigarette abstainers and late cigarette initiates to have initiated marijuana use at early ages. Both *CigEarly* and *AlcEarly* were significant predictors of time to marijuana initiation among 12th grade students. An examination of respective Wald statistics and *p*-values indicated that the risk effect exerted by *CigEarly* on the hazard of marijuana initiation was more highly significant than that posed by *AlcEarly*.

The sole finding that countered H6 dealt with marijuana initiation among 12th grade students. Early age of cigarette initiation (*CigEarly*, b = 1.043; $p < .10$) was associated with an increased likelihood of marijuana initiation, but early age of alcohol initiation (*AlcEarly*, b = 1.372; $p < .05$) was more highly significant. Compared to 12th grade alcohol abstainers, 12th grade students who initiated alcohol use between 8 and 15 years of age were nearly 4 times more likely to have initiated marijuana use.

Hypothesis 7

Based upon Hawkins' (Catalano & Hawkins, 1996) hypothesis concerning the saliency that community influences have on the behavior of older adolescents, H7 argued that community domain predictors would explain a larger proportion of variance in alcohol, cigarette, and marijuana initiation and age of initiation among 12th grade students as compared to alcohol and cigarette initiation and age of initiation among 6th and 9th grade students, and marijuana initiation and age of initiation among 9th grade students. This hypothesis was tested with findings from the age-graded soft drug initiation and time to initiation regression models for 6th, 9th, and 12th grade students. As Table 71 illustrates, H7 yielded partial support.

Table 71.

Hypothesis 7 Findings, 2004 PPAAUS: Percentage Decrease in Final Model R^2/χ^2 Attributed to the Community Domain

	6th Graders		9th Graders		12th Graders	
Outcome	Proportion Decrease	Final Model R^2/χ^2	Proportion Decrease	Final Model R^2/χ^2	Proportion Decrease	Final Model R^2/χ^2
Alcohol Initiation	.062	25.600	.000	44.500	.062	27.800
Cigarette Initiation	.041	33.800	.033	57.500	.024	50.000
Marijuana Initiation	---	---	.000	54.700	.070	57.900
Time to Alcohol Initiation	.080	85.114	.081	80.799	.130	73.920
Time to Cigarette Initiation	.000	132.339	.032	146.793	.000	133.967
Time to Marijuana Initiation	---	---	.000	161.256	.044	197.602

Initiation

In testing H7 with findings from the soft drug initiation models, the percentage decrease in explained variation attributed to removing the community domain for alcohol (R^2 = 26%, 45%, and 28%) and cigarette (R^2 = 34%, 58%, and 50%) initiation among 6th, 9th, and 12th grade students, respectively, and marijuana (R^2 = 55% and 58%) initiation among 9th and12th grade students, respectively, was calculated and then compared. Mixed support was found for H7 with respect to soft drug initiation.

Removing the community domain was responsible for an approximate 6% reduction in explained variance in alcohol initiation among 6th and 12th grade students, respectively. No community domain predictors were significantly related to alcohol initiation among 9th grade students. Counter to H7, the community domain predictors were responsible for a larger percentage decrease (4% versus 2%) in explained variance for cigarette initiation among 6th grade students than 12th grade students, respectively.

H7 was fully supported with respect to the age-graded findings for marijuana initiation. In particular, the community domain was responsible for a 7% decrease in explained variance in marijuana initiation among 12th grade students. No community domain predictors were significantly related to marijuana initiation among 9th grade students.

Time to Initiation

H7 also garnered full support with respect to time to alcohol and marijuana initiation. Findings for time to cigarette initiation countered H7, however. The community domain was responsible for a 13% decrease in the final model χ^2 for time to alcohol initiation and a 4% reduction in the final model χ^2 for time to marijuana initiation among 12th grade students. In contrast, roughly an 8% decrease in χ^2 was observed for time to alcohol initiation among 6th and 9th graders, while no community domain variables made a significant contribution to explaining time to marijuana initiation among 9th grade students.

Finally, counter to H7, no significant community domain predictors were retained in the final 12th (or 6th) grade time to cigarette initiation model.

Comparatively, the community domain constructs were responsible for a 3% decrease in the final model χ^2 for time to cigarette initiation among 9th graders.

Hypothesis 8

H8 posited that parental prodrug norms is a stronger predictor of alcohol and cigarette initiation and age of initiation among 6th grade students, as compared to 9th or 12th grade students. To test this hypothesis, the magnitude and corresponding *p*-values of the direct impact of parental tolerance of alcohol (*MessParAlc*) and cigarette (*MessParCig*) use on respective outcomes were examined. Outlined in Table 72, only one finding supported H8.

Findings for alcohol and cigarette initiation were not supportive. Parental tolerance of alcohol use (*MessParAlc*) had a significant, direct effect (b = .982; p <.05) on alcohol initiation among 9th grade students only. The direct impact of *MessParAlc* on alcohol initiation among 6th grade students, and parental tolerance of cigarette use (*MessParCig*) on cigarette initiation among 12th grade students, were fully mediated by peer domain predictors. The rest of the findings concerning *MessParAlc* and *MessParCig* were non-significant.

As was observed with alcohol initiation among 9th grade students, *MessParAlc* (b = .388; p <.05) exerted a significant, direct impact on time to alcohol initiation among 9th graders, but was not a significant, direct determinant of time to alcohol initiation among 6th or 12th grade students. Among 6th graders, parental tolerance of alcohol use was fully mediated by the peer domain predictors (*PeerAlc* and *MessPeerAlc*). Parental tolerance of alcohol use never was retained in the 12th grade model.

Table 72.

Hypothesis 8 Findings, 2004 PPAAUS: Direct Effect of Parental Pro-Drug Norms on Respective Outcomes

	6^{th} Graders		9^{th} Graders		12^{th} Graders	
Outcome/Predictor	B	*p*-value (<)	B	*p*-value (<)	B	*p*-value (<)
Alcohol Initiation						
MessParAlc	NS	NS	.982	.05	NS	NS
Cigarette Initiation						
MessParAlc	NS	NS	NS	NS	NS	NS
MessParCig	NS	NS	NS	NS	NS	NS
Time to Alcohol Initiation						
MessParAlc	NS	NS	.388	.05	NS	NS
Time to Cigarette Initiation						
MessParAlc	NS	NS	NS	NS	NS	NS
MessParCig	-1.901	.001	NS	NS	NS	NS

Note. NS = not significant at the .05 level (or lower).

The only finding that somewhat supported H8 centered on time to cigarette initiation. Parental tolerance of cigarette use (b = -1.901; p <.001) was significantly associated with the hazard of cigarette initiation among 6^{th} grade students, but not 9^{th} or 12^{th} graders. However, instead of lowering age of cigarette initiation, *MessParCig* delayed age of cigarette initiation among 6^{th} grade students.

Hypothesis 9

H9 argued that peer drug use and pro-drug norms are stronger predictors of alcohol, cigarette, and marijuana initiation and age of initiation among 9^{th}

grade students, as compared to alcohol and cigarette initiation and age of initiation among 6^{th} and 12^{th} grade students, and marijuana initiation and age of initiation among 12^{th} grade students. In testing H9, the magnitude and corresponding *p* values for relevant peer soft drug use and tolerance measure were assessed.

On balance, not only did peer soft drug use and tolerance of use have no significant, direct bearing on time to marijuana initiation among 9^{th} and 12^{th} graders, but these measures also were not important predictors of soft drug initiation and time to initiation for most of the outcomes assessed. Only one of seven findings supported H9, as revealed in Table 73. *PeerAlc* was a stronger predictor of alcohol initiation among 9^{th} (b = 1.145; p <.001) than 6^{th} (b = .816; p <.001) grade students. This factor was not significant at the .05 level in the 12^{th} grade model.

The remaining findings countered H9. *MessPeerCig* exerted significant, direct effects on cigarette initiation (b = 2.455; p <.05) and time to cigarette initiation (b = 1.479; p <.05) among 6^{th} grade students, but not 9^{th} (or 12^{th}) graders. As well, *PeerCig* had a significant bearing on age of cigarette initiation among 6^{th} graders (b = .812; p <.05), but not among 9^{th} or 12^{th} grade students. Peer alcohol use did exert a significant, direct impact on time to alcohol initiation among 6^{th} (b = .611; p <.001) and 9^{th} (b = .340; p <.001) grade students, but this measure was a more salient predictor among 6^{th} graders.

Finally, the age-graded results for marijuana initiation among 9^{th} and 12^{th} grade students also countered H9. Both peer tolerance of alcohol (b = -3.104;

p <.001) and marijuana (b = 1.509; p <.05) use were important in predicting marijuana initiation among 12th grade students only.

Table 73.

Hypothesis 9 Findings, 2004 PPAAUS: Direct Effects of Peer Soft Drug Use and Pro-Drug Norms on Respective Outcomes

	6th Graders		9th Graders		12th Graders	
Outcome/Predictor	B	*p*-value (<)	B	*p*-value (<)	B	*p*-value (<)
Alcohol Initiation						
PeerAlc	.816	.001	1.145	.001	NS	NS
MessPeerAlc	NS	NS	NS	NS	NS	NS
Cigarette Initiation						
PeerAlc	NS	NS	NS	NS	NS	NS
PeerCig	NS	NS	NS	NS	NS	NS
MessPeerAlc	NS	NS	NS	NS	NS	NS
MessPeerCig	2.455	.05	NS	NS	NS	NS
Marijuana Initiation						
PeerAlc	---	---	NS	NS	NS	NS
PeerCig	---	---	NS	NS	NS	NS
PeerMar	---	---	NS	NS	NS	NS
MessPeerAlc	---	---	NS	NS	-3.104	.001
MessPeerCig	---	---	NS	NS	NS	NS
MessPeerMar	---	---	NS	NS	1.509	.05
Time to Alcohol Initiation						
PeerAlc	.611	.001	.340	.001	NS	NS
MessPeerAlc	NS	NS	NS	NS	NS	NS

(Table 73 continues)

(Table 73 continued)

	6th Graders		9th Graders		12th Graders	
Outcome/Predictor	B	*p*-value (<)	B	*p*-value (<)	B	*p*-value (<)
Time to Cigarette Initiation						
PeerAlc	NS	NS	NS	NS	NS	NS
PeerCig	.812	.05	NS	NS	NS	NS
MessPeerAlc	NS	NS	NS	NS	NS	NS
MessPeerCig	1.479	.05	NS	NS	NS	NS
Time to Marijuana Initiation						
PeerAlc	---	---	NS	NS	NS	NS
PeerCig	---	---	NS	NS	NS	NS
PeerMar	---	---	NS	NS	NS	NS
MessPeerAlc	---	---	NS	NS	NS	NS
MessPeerCig	---	---	NS	NS	NS	NS
MessPeerMar	---	---	NS	NS	NS	NS

Note. --- not applicable; NS = not significant at the .05 level (or lower).

Distal-Proximal Mediation

Petraitis et al. (1995) posited that predictors conceptualized as proximal in nature should mediate the effects of distal variables. The efficacy of this hypothesis was tested in terms of full mediation. Evidence of distal-proximal mediation was highlighted in the previous synopsis of findings for each regression model. All models garnered some degree of support. In order to identify patterns and assess the utility of Petraitis et al.'s (1995) argument in a more holistic fashion, a more formal evaluation was conducted. Specifically, for each of the 22 regression models that were developed, the proportion of the community, school, family, and peer variables that were retained at some point in the model-building process and subsequently were fully mediated by more proximal predictors was calculated.

Table 74 provides the percentages of each ecological domain in each regression model that exerted indirect effects. The average proportion of effects that were indirect (as opposed to direct) are presented in the last column of the table. The last row of the table includes the average proportion of each ecological domain that exerted indirect effects.

Considerable support was yielded for Petraitis et al.'s (1995) distal-proximal mediation hypothesis. On balance, 61% of the community, school, family, and peer variables that initially were retained in the 22 regression models exerted indirect effects. In only six (i.e., time to alcohol initiation among the total sample and 9th and 12th graders; alcohol initiation among 12th graders; cigarette initiation among 6th graders; and time to cigarette initiation among 9th graders) of the 22 models analyzed were the majority of effects direct in nature. For instance, the finding that roughly 66% of the effects observed for time to alcohol initiation among the total sample were direct, was reinforced by the large proportion of direct effects found in the 9th and 12th grade time to alcohol initiation models.

In terms of specific ecological domains, on average, variables found within the family domain were most apt to have been fully mediated by more proximal (peer or individual) predictors. The school domain came in close second, however. Of the effects exerted by the school domain constructs, on average, 70% were indirect, with respective impacts fully mediated by more proximal variables. Surprisingly, the community domain, the most distal domain assessed, exerted more direct effects (50%), on average, than the school, family, and peer domains.

Table 74.

Proportion of Extraneous Effects as Indirect, 2004 PPAAUS: Total Sample and Age-Graded Models

	Proportion of Ecological Domain Effects as Indirect				
Sample/Model Type	Community	School	Family	Peer	Mean Proportion
		Total Sample Models			
Alcohol Initiation	.50	.50	1.00	.50	.63
Cigarette Initiation	0.00	.75	1.00	1.00	.69
Marijuana Initiation	.50	1.00	1.00	0.00	.63
Time to Alcohol Initiation	0.00	1.00	0.00	---	.33
Time to Cigarette Initiation	0.00	.66	1.00	1.00	.67
Time to Marijuana Initiation	.33	1.00	1.00	1.00	.82
		Age-Graded Models			
Alcohol Initiation					
Among 6th Graders	.50	.66	1.00	.50	.67
Among 9th Graders	1.00	.80	0.00	.50	.58
Among 12th Graders	.50	0.00	---	---	.25
Cigarette Initiation					
Among 6th Graders	0.00	.66	---	.50	.39

(Table 74 continues)

(Table 74 continued)

	Proportion of Ecological Domain Effects as Indirect				
Sample/Model Type	Community	School	Family	Peer	Mean Proportion
Among 9th Graders	.50	.66	---	1.00	.72
Among 12th Graders	.50	.66	1.00	1.00	.79
Marijuana Initiation					
Among 9th Graders	1.00	1.00	---	1.00	1.00
Among 12th Graders	.50	.50	1.00	0.00	.50
Time to Alcohol Initiation					
Among 6th Graders	0.00	.66	1.00	.50	.54
Among 9th Graders	1.00	.66	0.00	0.00	.42
Among 12th Graders	0.00	.33	---	---	.17
Time to Cigarette Initiation					
Among 6th Graders	1.00	.60	.50	0.00	.53
Among 9th Graders	.50	1.00	0.00	0.00	.38
Among 12th Graders	1.00	.75	1.00	1.00	.94
Time to Marijuana Initiation					
Among 9th Graders	1.00	1.00	---	1.00	1.00
Among 12th Graders	.75	.50	1.00	.50	.69
Column averages	.50	.70	.72	.58	.61

--- no predictors in the ecological domain made a significant contribution to explaining the given outcome.

Conclusion

Building upon the univariate and bivariate results presented in Chapter 8, the purpose of this chapter was to present the multivariate findings that addressed Research Questions #2 and #3. Predictors of soft drug initiation and time to initiation among the total sample and 6th, 9th, and 12th grade students were discussed. Attention also was directed at drug- and age-specific predictors. A formal presentation of findings for H2-H9 was made, along with a general assessment of the utility of Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

On balance, the multivariate results yielded partial support for H2-H9. The largest deviation from hypothesized expectations centered on H4. Both the total sample and age-graded findings indicated that the individual domain was responsible for the largest contributions in the final model R^2 and χ^2 values for alcohol and cigarette initiation and time to initiation, not the peer domain (as expected).

Both the total sample and age-graded results yielded more support for Kandel's (2002) notion of drug-specific predictors than the common factor hypothesis. The predictive utility of some of the extraneous measures also differed by biological age. This aspect of the research provided some predictive insight into the H1 soft drug sequence that was observed.

Finally, the multivariate findings yielded considerable support for Petraitis et al.'s (1995) distal-proximal mediation hypothesis. The findings for indirect effects, coupled with the size of the individual domain coefficients, indicated that the individual domain predictors constituted a powerful mediating force.

The following chapter presents the results for the dual cross-validation. In particular, the results for H2 and H3 from the 2004 PPAAUS are compared to relevant findings from the systematic review. The results from the quantitative cross-validation also are detailed.

CHAPTER 10

DUAL CROSS-VALIDATION RESULTS

The current chapter presents the findings from the systematic review and quantitative cross-validations. The overarching purpose in conducting this dual cross-validation was to verify the internal and external validity of the 2004 PPAAUS findings for H2-H9 and distal-proximal mediation. The statistical conclusion and construct validity of these results also were relevant.

The chapter is organized as follows. The findings from the systematic review cross-validation are introduced first, followed by a presentation of the results from the quantitative cross-validation. The conflicting findings from these cross-validations then are considered more carefully, in identifying and discussing the major threats to the validity of the 2004 PPAAUS results.

Systematic Review Cross-Validation

As was discussed in Chapter 5, the primary purpose of the systematic review cross-validation was to verify the internal and external validity of the 2004 PPAAUS results for H2[16] and H3.[17] This was accomplished by first identifying those 2004 PPAAUS variables (or similar constructs) that were examined in the systematic review, and then comparing the direction (+/-) of effects across findings.

A total of 35 predictors were examined in both the quantitative aspect of the current research and the systematic review. In short, a fairly strong

[16] H2 argued that predictors conceptualized as risk factors increase risk for alcohol, cigarette, and marijuana initiation and lower age of initiation.
[17] H3 posited that predictors viewed as protective factors decrease risk for alcohol, cigarette, and marijuana initiation and delay age of initiation.

confluence in findings was observed. Of the 66 directional findings subject to validation, full convergence was found for 59 relationships. Hence, with only seven counter findings identified, the general convergence rate for the systematic review cross-validation was 89%. This overarching finding underscores the internal, external, statistical conclusion, and construct validity of those 2004 PPAAUS results that could be cross-validated. It also is worthwhile to note that the bulk of the seven counter findings were insignificant at the .05 level.

Specific convergence rates for H2 and H3 also were impressive. For H2, 96% of the matched directional findings for soft drug initiation were fully consistent, while 79% of the matched directional results for predictors expected to reduce the likelihood of soft drug initiation (H3) fully converged. Discussed in Chapter 7, only two of the 36 primary studies (Hawkins et al., 2002b; Unger & Chen, 1999) examined predictors of time to soft drug initiation. Since both studies utilized data from samples of youth whose biological ages spanned early-late adolescence, the age-graded H2 findings for time to initiation among 6th, 9th, and 12th grade students were not able to be confirmed within this particular cross-validation. Nonetheless, a convergence rate of 92% was obtained for the direction of effects exerted by the H2 variables in predicting time to soft drug initiation among the total sample.

More specific results from the systematic review cross-validation are presented below. The H2 cross-validation findings for soft drug initiation among the total sample and age-graded subsamples are presented first, followed by the H2 cross-validation results for time to initiation among the total sample. Attention then is directed at the H3 cross-validation findings for soft drug initiation among the combined and the age-specific subsamples.

H2 Constructs and Soft Drug Initiation

Total Sample

A total of 21 variables hypothesized to increase risk for soft drug initiation (H2) were examined with total sample data from the 2004 PPAAUS, but were not evaluated in any of the primary studies that investigated predictors of soft drug initiation among samples of similar age youth. Consequently, the directional effects of these predictors could not be cross-validated. These constructs included the bulk of the community domain predictors (*MessMedAlc*, *MessMedMar*, *EasyCig*, and *EasyMar*), all of the school domain variables that were viewed as risk factors (i.e., *Truancy*, *MessTchrAlc*, *MessTchrCig*, *MessTchrMar*, and *Cheat*), two (*MessParAlc* and *MessParMar*) of the three family domain predictors, two (*MessPeerAlc* and *MessPeerMar*) peer variables, and six (*AlcIntent*, *MarIntent*, *AlcEarly*, *CigEarly*, *Steal*, and *ViolBeh*) individual domain constructs. The two control variables (*AlcFreq* and *CigFreq*) that were conceptualized as risk factors also were not examined among early-late adolescents in the primary studies assessed in the systematic review.

Outlined in Table 75, of the H2 results that could be cross-validated, all were confirmed with similar directional findings from the systematic review. In particular, a convergence in findings was observed for the risk capacity of *PeerAlc* in predicting alcohol initiation among the total sample. With respect to cigarette initiation, the risk effects of four variables (*MessParCig*, *PeerCig*,

Table 75.

Systematic Review Cross-Validation Findings: H2 Results for Soft Drug Initiation among the Total Sample [a]

Domain/Predictor	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
	TS [b]	SR, E-L [b]	TS	SR, E-L	TS	SR, E-L
<u>*Community Domain*</u>:						
MessMedCig	---	---	(+)	(?)	***(-)***	N/A
EasyAlc	***(+)***	(?)	(+)	N/A	(-)	N/A
<u>*Family Domain*</u>:						
MessParCig	---	---	(+)	(+,?)	(+)	N/A
<u>*Peer Domain*</u>:						
PeerAlc	(+)	***(+)***	(+)	N/A	(-)	N/A
PeerCig	---	---	(+)	***(+,+,+,+)***	(+)	N/A
PeerMar	---	---	---	---	(+)	***(+)***
MessPeerCig	---	---	(+)	(+,?)	(+)	N/A
<u>*Individual Domain*</u>:						
CigIntent	---	---	(+)	***(+,+)***	(-)	N/A

[a] Bolded/italicized directional effects are significant at the .05 level (or lower); ? = direction of relationship unknown; N/A = predictor not examined in relevant primary studies.
[b] TS = Total Sample from the 2004 PPAAUS; SR = Systematic Review; E-L = Early-Late Adolescence (12-17 years, or 6th-12th grade).

MessPeerCig, and *CigIntent*) were verified. Finally, the sole directional finding (*PeerMar*) that could be cross-validated within the context of marijuana initiation among the total sample was confirmed.

Also depicted in Table 75, the counter findings for the directional effects posed by four H2 variables (*MessMedCig*, *EasyAlc*, *PeerAlc*, and *CigIntent*) were not able to be validated through the systematic review. Detailed in Chapter 9, these constructs exerted protective effects in reducing the likelihood of marijuana initiation among the total sample. However, only one (*MessMedCig*) of these variables exerted significant effects at the .05 level.

Age-Graded Subsamples

Table 76 presents the directional effects for 16 constructs that were viewed as risk factors (H2) for soft drug initiation among 6th, 9th, and 12th grade students and were examined in one or more primary studies using data from samples of early, mid-, or late adolescent youth. The directional effects of 16 other predictors, all viewed as risk factors, could not be cross-validated, since these variables were not examined in the primary studies that were reviewed. These variables included four (*MessMedAlc*, *MessMedCig*, *MessMedMar*, and *EasyMar*) of the six community domain predictors, four (*MessTchrAlc*, *MessTchrCig*, *MessTchrMar*, and *Cheat*) school domain constructs, one (*MessParAlc*) family domain variable, and seven (*AlcInit*, *AlcEarly*, *AlcLate*, *CigEarly*, *CigLate*, *Steal*, and *ViolBeh*) individual domain predictors.

Full convergence. The following 19 positive relationships, which were found with age-graded data from the 2004 PPAAUS, were fully supported with

Table 76.

Systematic Review Cross-Validation Findings: H2 Results for Soft Drug Initiation among Early, Mid-, and Late Adolescents [a]

Domain/Predictor	Alcohol Initiation						Cigarette Initiation						Marijuana Initiation			
	6^{th} [b]	SR,E [b]	9^{th} [b]	SR,M [b]	12^{th} [b]	SR,L [b]	6^{th}	SR,E	9^{th}	SR,M	12^{th}	SR,L	9^{th}	SR,M	12^{th}	SR,L
<u>Community Domain</u>:																
EasyAlc	***(+)***	(?)	(+)	N/A	(+)	N/A	(-)	N/A	(-)	N/A	(+)	N/A	(-)	N/A	(-)	N/A
EasyCig	---	---	---	---	---	---	***(+)***	***(+)***	***(+)***	N/A	(+)	N/A	(+)	N/A	(+)	N/A
<u>School Domain</u>:																
Truancy	(+)	***(+)***	(+)	(?)	***(+)***	N/A	(+)	N/A	(+)	(?)	(+)	N/A	(+)	(?)	(+)	N/A
<u>Family Domain</u>:																
MessParCig	---	---	---	---	---	---	(-)	N/A	(+)	N/A	(+)	(+)	(+)	N/A	(+)	N/A
MessParMar	---	---	---	---	---	---	---	---	---	---	---	---	(+)	***(+, -)***	(+)	N/A
<u>Peer Domain</u>:																
PeerAlc	(+)	***(+,+)*** ***(+,+)***	(+)	***(+)***	(+)	N/A	(-)	N/A	(-)	N/A	(-)	N/A	(+)	N/A	(-)	N/A
PeerCig	---	---	---	---	---	---	(+)	***(+,+,+)***	(+)	***(+)***	(+)	(+)	(+)	N/A	(-)	N/A

(Table 76 continues)

(Table 76 continued)

Domain/Predictor	Alcohol Initiation						Cigarette Initiation						Marijuana Initiation			
	6th	SR,E	9th	SR,M	12th	SR,L	6th	SR,E	9th	SR,M	12th	SR,L	9th	SR,M	12th	SR,L
PeerMar	---	---	---	---	---	---	---	---	---	---	---	---	(-)	***(+)*** (+,-)	(+)	N/A
MessPeerAlc	(+)	(?)	(+)	***(+)***	(+)	N/A	(-)	N/A	(-)	N/A	(-)	N/A	(-)	N/A	***(-)***	N/A
MessPeerCig	---	---	---	---	---	---	***(+)***	N/A	(+)	***(+)***	(+)	(+)	(+)	N/A	(-)	N/A
MessPeerMar	---	---	---	---	---	---	---	---	---	---	---	---	(+)	***(+,+,+)***	***(+)***	N/A
Individual Domain:																
AlcIntent	***(+)***	***(+)***	***(+)***	N/A	***(+)***	N/A	***(-)***	N/A	(-)	N/A	**(-)**	N/A	(-)	N/A	(-)	N/A
CigIntent	---	---	---	---	---	---	***(+)***	***(+,+)***	***(+)***	N/A	***(+)***	N/A	(-)	N/A	***(-)***	N/A
MarIntent	---	---	---	---	---	---	---	---	---	---	---	---	***(+)***	(+, +)	***(+)***	N/A
Select Controls:																
AlcFreq	---	---	---	---	---	---	---	---	(+)	(+)	(+)	***(+)***	***(+)***	(+,+,+)	(+)	N/A
CigFreq	---	---	---	---	---	---	---	---	---	---	---	**---**	***(+)***	***(+,+,+)***	(+)	(?)

[a] Bolded/italicized directional effects are significant at the .05 level (or lower); ? = direction of relationship unknown; N/A = predictor not examined in relevant primary studies.
[b] 6th, 9th, 12th = 6th, 9th, and 12th grade students from the 2004 PPAAUS; SR = Systematic Review; E = Early Adolescence (12-14 years, or 6th-8th grade), M = Mid-Adolescence (14-16 years, or 9th-10th grade), L = Late Adolescence (16-17 years, or 11th-12th grade).

results from the relevant primary studies investigated in the systematic review: alcohol initiation among 6th grade students and *Truancy*, *PeerAlc*, and *AlcIntent*; alcohol initiation among 9th graders and *PeerAlc* and *MessPeerAlc*; cigarette initiation among 6th grade students and *EasyCig*, *PeerCig*, and *CigIntent*; cigarette initiation among 9th grade students and *PeerCig*, *MessPeerCig*, and *AlcFreq*; and cigarette initiation among 12th grade students and *MessParCig*, *PeerCig*, *MessPeerCig*, and *AlcFreq*. The risk capacities of the following variables also were confirmed in predicting marijuana initiation among 9th grade students: *MessPeerMar*, *MarIntent*, *AlcFreq*, and *CigFreq*. Detailed later in this chapter, this confluence in age-graded, directional findings indicate that some of the major threats to the internal, external, statistical conclusion, and construct validity of these particular 2004 PPAAUS results may be deemed less plausible.

Four additional findings from the systematic review are of relevance for some of the H2 age-graded results. First, no primary studies examined *Truancy* within the context of alcohol initiation among late adolescents (Table 76). Kandel et al. (1976) did, however, assess the utility of school truancy in predicting alcohol initiation among mid-late adolescent youth (i.e., high-school students). They found a significant positive relationship between this construct and alcohol initiation among this sample population, which supports the positive association observed among the 12th graders from the 2004 PPAAUS.

Second, *PeerMar* was not investigated in primary studies predicting marijuana initiation among late adolescents. Both Shears et al. (2006) and Kandel and Andrews (1987) found a positive relationship between peer marijuana use and marijuana initiation among early-late and mid-late adolescent youth, however, which supports the positive effects that were observed with the 2004 PAAUUS data from 12th graders.

Third, although primary studies did not investigate *CigIntent* (i.e., intentions to use cigarettes) in predicting cigarette initiation among mid- and late adolescent youth (Table 76), as reported earlier, two previous studies (Chassin et al., 1984; Gritz et al., 2003) did show that among early-late adolescent youth, adolescents who intend to use cigarettes are at an increased risk for initiating this substance. In a related vein, no primary studies that derived data from mid- or late adolescent youth examined the impact of cigarette use intentions on the likelihood of initiating marijuana. Marcos & Bahr (1988) found, however, that among mid-late adolescent youth, adolescents who intended to use cigarettes were at a significantly lower risk for marijuana initiation than their counterparts. This finding is the same as that observed among both 9th and 12th grade students from the 2004 PPAAUS data.

Finally, unlike the present research, none of the primary studies that investigated predictors of marijuana initiation among late adolescents included measures of alcohol use frequency as a possible explanatory factor. Kandel et al. (1976) found that mid-late adolescent youth who engaged in frequent alcohol use were at a significantly higher risk for marijuana initiation. This finding supports the risk effects that *AlcFreq* posed in increasing risk for marijuana initiation among 12th grade students from the 2004 PPAAUS.

Counter findings. Only two directional effects countered the direction of the relationship uncovered using data from the 2004 PPAAUS (Table 76). Both counter findings centered on marijuana initiation among 9th grade students. First, Ellickson et al. (2004) found that parental pro-marijuana norms served as a salient risk factor for marijuana initiation among a sample of 9th grade students. This finding also was observed in the quantitative component of the current research, with *MessParMar* increasing risk for marijuana initiation among 9th grade students. In contrast, Ellickson et al. (2004) also found that among 10th grade students, parental pro-marijuana norms exerted a significant protective effect.

Second, the current research found an inverse relationship between peer marijuana use (*PeerMar*) and marijuana initiation among 9th grade students. This finding is supported by Ellickson et al.'s (2004) research with a sample of 10th grade students. They also found that peer marijuana use constituted a protective factor, by decreasing risk for marijuana initiation. Among their sample of 9th grade students, however, Ellickson et al. (2004) found that peer marijuana use increased risk for marijuana initiation. Walter et al. (1991) reached the same conclusion using data from a sample of 10th grade students.

As discussed in Chapter 9, numerous H2 constructs exerted protective effects, not risk effects as hypothesized. Insignificant protective effects that could not be cross-validated with findings from the systematic review included those posed by the following variables: *EasyAlc* in predicting cigarette initiation among 6th graders, cigarette and marijuana initiation among 9th grade students, and marijuana initiation among 12th graders; *MessParCig* in reducing risk for cigarette initiation among all three subsamples and marijuana initiation among 12th grade students; and *PeerCig* in reducing the likelihood of marijuana initiation among 12th graders. The protective capacity of *MessPeerAlc* in reducing risk for cigarette initiation among 6th, 9th, and 12th grade students, as well as marijuana initiation among 9th and 12th graders, also was unable to be confirmed.

Other H2 counter findings (mostly insignificant) that were unable to be verified through the systematic review cross-validation included the protective capacity of *MessPeerCig* in reducing risk for marijuana initiation among 12th grade students; the protective nature of *AlcIntent* in reducing the likelihood of cigarette and marijuana initiation among 9th and 12th graders, as well as cigarette initiation among 6th grade students; and the protective effects posed by *CigIntent* in reducing risk for marijuana initiation among 9th and 12th grade students.

H2 Constructs and Time to Soft Drug Initiation

Total Sample

Only two primary studies (Hawkins et al., 2002b; Unger & Chen, 1999) examined predictors of time to soft drug initiation, and many known correlates of adolescent drug use were not evaluated in these studies. Therefore, the majority of the 2004 PPAAUS predictors that were viewed as risk factors and were examined within the context of time to soft drug initiation among the total sample could not be cross-validated with findings from the systematic review.

The directional effects stemming from the following risk constructs identified using total sample data from the 2004 PPAAUS could not be confirmed: six (*MessMedAlc*, *MessMedCig*, *MessMedMar*, *EasyAlc*, *EasyCig*, and *EasyMar*) community and five (*Truancy*, *MessTchrAlc*, *MessTchrCig*, *MessTchrMar*, and *Cheat*) school-related predictors, one (*MessParCig*) family domain construct, three (*MessPeerAlc*, *MessPeerCig*, and *MessPeerMar*) peer variables, and six (*AlcIntent*, *MarIntent*, *AlcEarly*, *CigEarly*, *Steal*, and *ViolBeh*) individual domain constructs. The risk effects of two (*AlcFreq* and *CigFreq*) control variables also could not be verified within this particular cross-validation.

Full convergence. Despite the lack of previous research, the risk effects posed by five constructs from the 2004 PPAAUS were confirmed by findings from the systematic review. As Table 77 depicts, the expected relationship between time to alcohol initiation and *MessParAlc* was verified, as were the 2004 PPAAUS findings for the utility of *PeerCig* and *CigIntent* in predicting time to cigarette initiation. All variables served to lower the ages of initiation of these soft drugs. Finally, full convergence was observed with respect to the relationships between time to marijuana initiation and *MessParMar* and *PeerMar*. Both constructs were found to lower age of marijuana initiation.

Counter findings. Only one directional effect identified through the systematic review countered the direction of the relationship uncovered using data from the 2004 PPAAUS. According to Hawkins et al.'s (2002b) study of youth from Seattle, who ranged in age from 10-18 years, a positive association exists between peer alcohol use and time to alcohol initiation. In other words, these researchers found that an increase in peer alcohol use lowered age of

Table 77.

Systematic Review Cross-Validation Findings: H2 Results for Time to Soft Drug Initiation among the Total Sample [a]

Domain/Predictor	Time to Alcohol Initiation		Time to Cigarette Initiation		Time to Marijuana Initiation	
	TS [b]	SR, E-L [b]	TS	SR, E-L	TS	SR, E-L
Family Domain:						
MessParAlc	***(+)***	***(+)***	(+)	N/A	(+)	N/A
MessParMar	---	---	---	---	(+)	(+)
Peer Domain:						
PeerAlc	(-)	***(+)***	(-)	N/A	(-)	N/A
PeerCig	---	---	(+)	***(+,+)***	(-)	N/A
PeerMar	---	---	---	---	(+)	***(+)***
Individual Domain:						
CigIntent	---	---	(+)	***(+)***	(-)	N/A

[a] Bolded/italicized directional effects are significant at the .05 level (or lower); (+) = predictor is associated with a younger age of initiation; N/A = predictor not examined in relevant primary studies.
[b] TS = Total Sample from the 2004 PPAAUS; SR = Systematic Review; E-L = Early-Late Adolescence (12-17 years, or 6th-12th grade).

alcohol initiation. In contrast, the current research found that *PeerAlc* functioned as a protective factor among the total sample, by delaying time to alcohol initiation. However, this finding from the 2004 PPAAUS was insignificant at the .05 level.

Worth noting are the H2 counter findings for the total sample that were not able to be cross-validated with the results from the systematic review (Table 77). Although insignificant at the .05 level, the current research found that *PeerAlc* delayed time to cigarette and marijuana initiation, while *PeerCig* and *CigIntent* delayed time to marijuana initiation.

H3 Constructs and Soft Drug Initiation

Total Sample

Viewed as protective factors and investigated in the context of soft drug initiation among the combined sample, the directional effects of six predictors (*CllgeAsp*, *Academic*, *HiAcadPerf*, *Sports*, *Religious*, and *Social*) could not be cross-validated with findings from the systematic review. The positive (but insignificant) relationships between *Work* and alcohol and marijuana initiation also could not be verified.

The direction of the associations between *AttachSchool* and cigarette initiation, *AttachSchool* and marijuana initiation, and *Work* and cigarette initiation were able to be confirmed. Presented in Table 78, the protective capacity of school attachment (*AttachSchool*) in reducing the likelihood of cigarette and marijuana initiation was verified. Also confirmed was the risk posed by

Table 78.

Systematic Review Cross-Validation Findings: H3 Results for Soft Drug Initiation among the Total Sample [a]

Domain/Predictor	Alcohol Initiation		Cigarette Initiation		Marijuana Initiation	
	TS [b]	SR, E-L [b]	TS	SR, E-L	TS	SR, E-L
<u>*School Domain*</u>:						
AttachSchool	(-)	N/A	(-)	***(-)***	(-)	***(-)***
<u>*Individual Domain*</u>:						
Work	(+)	N/A	(+)	(+)	(+)	N/A

[a] Bolded/italicized directional effects are significant at the .05 level (or lower); N/A = predictor not examined in relevant primary studies.
[b] TS = Total Sample from the 2004 PPAAUS; SR = Systematic Review; E-L = Early-Late Adolescence (12-17 years, or 6th-12th grade).

involvement in work-for-pay activities (*Work*) in increasing the likelihood of cigarette initiation.

Four additional results are worthy of comment, particularly since they support some of the total sample findings for H3. First, the inverse relationship between *AttachSchool* and marijuana initiation among the total sample, which was consistent with the inverse association that Shears et al. (2006) obtained for a sample of early-late adolescent youth, also was confirmed with data from mid-late adolescent youth (Marcos & Bahr, 1988).

Second, no primary studies examined the impact of school performance (i.e., *HiAcadPerf*) on alcohol, cigarette, or marijuana initiation among early-late adolescent youth. However, Kandel et al. (1976) found that among mid-late adolescent youth, poor school performance increased risk for alcohol and marijuana initiation. This finding coalesces with the inverse relationships between high school performance (*HiAcadPerf*) and alcohol and marijuana initiation, both of which were yielded with total sample data from the 2004 PPAAUS.

Third, religiosity was not examined among the primary studies that assessed predictors of soft drug initiation among samples of youth whose ages spanned early-late adolescence. Skinner et al. (1985) did find, however, that high levels of religiousness lowered risk for cigarette initiation among mid-adolescent youth. This finding supports the protective effects that *Religious* exerted in reducing cigarette initiation among the total sample from the 2004 PPAAUS.

Finally, Skinner et al. (1985) also found that for mid-late adolescent youth, a positive relationship existed between involvement in work-for-pay activities and cigarette initiation. This directional relationship coincides with the positive association between *Work* and cigarette initiation found for the 2004 combined sample, as well as the positive relationship that Kandel et al. (2004) obtained for early-late adolescent youth.

Age-Graded Subsamples

The protective capacity of four (*CllgeAsp*, *Academic*, *Sports*, and *Social*) predictors could not be cross-validated with any of the age-graded findings from the systematic review. However, the directional effects for three (*HiAcadPerf*, *Religious*, and *Work*) variables were able to be confirmed.

Full convergence. Outlined in Table 79, four of the seven age-graded directional findings from the 2004 PPAAUS were consistent with those identified through the systematic review. School performance was measured as a protective factor in the current research, with higher values on *HiAcadPerf* indicative of stronger school performance. The primary studies that examined academic achievement measured this construct in terms of poor school performance, a predictor conceptualized to increase risk for soft drug initiation. Taking into account this difference in measurement, the protective capacity of *HiAcadPerf* in reducing the likelihood of cigarette initiation among 6th and 12th grade students, and marijuana initiation among 9th grade students, was confirmed with findings from relevant primary studies. The protective capacity of involvement in religious activities (*Religious*) in reducing the likelihood of alcohol initiation among 6th grade students also was verified.

Table 79.

Systematic Review Cross-Validation Findings: H3 Results for Soft Drug Initiation among Early, Mid-, and Late Adolescents [a]

Domain/Predictor	Alcohol Initiation						Cigarette Initiation						Marijuana Initiation			
	6^{th} [b]	SR, E [b]	9^{th} [b]	SR, M [b]	12^{th} [b]	SR, L [b]	6^{th}	SR, E	9^{th}	SR, M	12^{th}	SR, L	9^{th}	SR, M	12^{th}	SR, L
<u>*School Domain*</u>:																
HiAcadPerf	(+)	---	***(-)***	---	(+)	---	***(-)***	---	(-)	---	***(-)***	---	(-)	---	(-)	---
Poor Academic Perf.		(+, ?)	---	(?)	---	N/A	---	***(+)***	---	N/A	---	(+)	---	***(+,+,+)***	---	(?)
<u>*Individual Domain*</u>:																
Religious	***(-)***	***(-)***	(-)	N/A	(-)	N/A	***(-)***	N/A	(+)	(-)	(-)	N/A	***(-)***	N/A	(-)	N/A
Work	(+)	N/A	(-)	N/A	(+)	N/A	(+)	N/A	(+)	(-)	(+)	N/A	(+)	N/A	(+)	N/A

[a] Bolded/italicized directional effects are significant at the .05 level (or lower); ? = direction of relationship unknown; N/A = predictor not examined in relevant primary studies.

[b] 6^{th}, 9^{th}, 12^{th} = 6^{th}, 9^{th}, and 12^{th} grade students from the 2004 PPAAUS; SR = Systematic Review; E = Early Adolescence (12-14 years, or 6^{th}-8^{th} grade), M = Mid-Adolescence (14-16 years, or 9^{th}-10^{th} grade), L = Late Adolescence (16-17 years, or 11^{th}-12^{th} grade).

Two additional findings provide some insight into several of the age-graded soft drug initiation findings for H3. High attachment to school (*AttachSchool*) was not examined by the primary studies that focused on marijuana initiation among late adolescent youth. However, the negative impact that *AttachSchool* exerted on marijuana initiation among 12th grade students from the 2004 PPAAUS is consistent with Marcos and Bahr's (1988) finding of an inverse relationship between school attachment and marijuana initiation among a sample of mid-late adolescent youth.

The predictive utility of frequent involvement in work-for-pay activities (*Work*) was not evaluated by primary studies that assessed predictors of cigarette initiation among late adolescent youth. However, Skinner and colleagues' (1985) finding of a positive relationship between work and cigarette initiation among their sample of mid-late adolescent youth supports both the risk effects of this construct in predicting cigarette initiation among 12th grade students from the 2004 PPAAUS and the risk capacity that this variable exerted in predicting cigarette initiation among Kandel et al.'s (2004) sample of early-late adolescent youth.

Counter findings. Three age-graded directional effects conflicted with prior findings (Table 79). Discrepancies centered on the direction of the relationships between school performance (*HiAcadPerf*) and alcohol initiation among early adolescents (i.e., 6th grade students), and between cigarette initiation among 9th grade students and involvement in religious (*Religious*) and work-for-pay (*Work*) activities.

In the current research, high academic performance (*HiAcadPerf*) increased risk for alcohol initiation among 6th grade students. Comparatively, Crum and colleagues (2005) found that poor school performance increased risk for alcohol initiation among 8th grade students. Webb et al. (1991) also examined the utility of poor school performance within the context of early adolescent alcohol initiation, but the direction of the insignificant finding was not reported in the publication.

The remaining two conflicting findings centered on the direction of effects posed by *Religious* and *Work* in predicting cigarette initiation among 9th grade students. In the quantitative aspect of the current research, both factors increased risk for initiation, although these associations were insignificant at the .05 level. In Skinner et al.'s (1985) longitudinal study, however, high levels of religiousness and frequent involvement in work-for-pay activities operated in a protective fashion, by reducing the likelihood of cigarette initiation among a sample of junior high-school students.

Finally, it is important to direct attention to the H3 counter findings for seven age-graded relationships (Table 79) that were unable to be verified with results from the systematic review. Although insignificant at the .05 level, *HiAcadPerf* increased risk for alcohol initiation among 12th graders from the 2004 PPAAUS, while *Work* increased the likelihood of alcohol initiation among 6th and 12th grade students. The effects posed by *Work* in increasing risk for cigarette initiation among 6th and 12th graders, and marijuana initiation among 9th and 12th graders, also were unable to be confirmed through the systematic review.

Quantitative Cross-Validation

The internal and external validity of the 2004 PPAAUS findings also were evaluated through the quantitative cross-validation. In particular, the 22 binary logit and Cox regression models that were utilized with the 2004 PPAAUS data were replicated using data from the 2001 PPAAUS sample. Hence, the same strategies (i.e., distal-proximal ordering of ecological domains and backward stepwise procedures) employed with the 2004 PPAAUS data in addressing Research Questions #2-3 (H2-H9), as well as testing Petraitis et al.'s (1995) distal-proximal mediation hypothesis, were used with data from the 2001 PPAAUS. The 2001 PPAAUS findings for alcohol, cigarette, and marijuana initiation and time to initiation among the total sample and the three age-graded subsamples are presented in Tables G8-G29 of Appendix G.

Discussed in Chapter 6, the quantitative cross-validation constituted comparing the 2004 and 2001 PPAAUS findings on two major fronts. First, in providing a broad-based assessment of model fit, each regression model was evaluated in terms of shrinkage. Second, the 2001 and 2004 PPAAUS findings for H2-H9 were compared, along with the results for distal-proximal mediation. Before the cross-validation findings are presented, the validation sample is briefly described below in terms of the rates and ages at which alcohol, cigarette, and marijuana were initiated.

2001 PPAAUS: Select Univariate Findings

Appendix G presents the univariate (Tables G1-G4) and bivariate (Tables G5-G7) results for the 2001 PPAAUS sample. In general, these findings were comparable to the corresponding results yielded from the 2004 PPAAUS sample. In an effort to identify serious collinearity problems, the bivariate correlations between the extraneous measures were examined, along with tolerance statistics and variance inflation factors. Five collinearity issues were identified and resolved before the multivariate models were developed. Specifically, evidence of high collinearity (e.g., low tolerance statistics) precluded the examination of *AlcLate* in the 12th grade cigarette and marijuana initiation and time to initiation models. Due to a zero cell count, *AlcIntent* also was not assessed in relation to time to cigarette initiation among 12th grade students.

The total sample and age-graded univariate findings for soft drug initiation and age of initiation can be summarized as follows. Among the total sample (N = 723), 52% self-reported prior initiation of alcohol use, while 57% and 25% indicated prior initiation of cigarette and marijuana use, respectively. Cigarette initiates (n = 261) began cigarette use at a slightly younger age (13 years), on average, than alcohol initiates (n = 373) began alcohol use (13.33 years). Among marijuana initiates (n = 178), the average age of initiation was roughly 14.5 years.

With respect to age-graded findings, roughly 14% of 6th grade students (n = 236) self-reported prior initiation of alcohol use, while 7% indicated prior initiation of cigarette use. On average, 6th grade alcohol (n = 34) and cigarette (n = 16) initiates indicated beginning use of these drugs at roughly 10 years of age. Among 9th graders (n = 244), 57%, 39%, and 25% indicated prior initiation of alcohol, cigarettes, and marijuana, respectively. The average age of alcohol and cigarette initiation among 9th grade alcohol (n = 136) and cigarette (n = 94) initiates was equivalent (12.4 years), while the average age of marijuana initiation among marijuana users (n = 61) was 13.4 years. Finally, 84%, 62%, and 48% of 12th grade students (n = 243) self-reported prior initiation of alcohol, cigarettes, and marijuana, respectively. On average, 12th grade alcohol (n = 203), cigarette (n= 151), and marijuana (n = 116) initiates reported they began using these drugs at 14.5, 13.6, and 14.7 years of age, respectively.

Shrinkage

The first component of the quantitative cross-validation involved calculating the amount of shrinkage that existed between each 2004 PPAAUS model and corresponding 2001 PPAAUS model. Discussed in Chapter 6, the smaller the shrinkage between corresponding models, the more apt that the 2004 PPAAUS findings are internally valid, and the more likely that the results may be generalized to similar youth from different time periods. The formula ($R^2_{derivation} – R^2_{validation}$) for shrinkage, offered by Collins et al. (1987), was employed for each binary logit regression model, and adapted ($\chi^2_{derivation} – \chi^2_{validation}$) for the Cox regression models. The last column of Table 80 notates the amount of shrinkage that was identified for each of the 22 total sample and age-graded outcomes. A positive value indicates that the data from the 2004 PPAAUS sample did a better job of explaining the given outcome than the data from the 2001 PPAAUS sample.

Two major findings are worth noting. First, on balance, results indicated that more variation in soft drug initiation and time to initiation was accounted for by the 2004 PPAAUS data. In fact, the 2004 PPAAUS data explained roughly 10% more variation in outcomes, on average, than the data from the 2001 PPAAUS sample. In part, some of this explanatory difference may be rooted in the fact that three community domain variables (*EasyAlc*, *EasyCig*, and *EasyMar*) were not included in the 2001 PPAAUS survey and were not examined in any of the 22 regression outcomes. In addition, collinearity issues involving *AlcLate* and *AlcIntent* precluded an examination of these predictors in relation to cigarette and marijuana initiation (and time to initiation) among 12th graders.

Table 80.

Differential Model Fit: 2004 and 2001 PPAAUS

	2004 Model Fit		2001 Model Fit		
Model Type	R^2	χ^2	R^2	χ^2	Shrinkage
		Total Sample Models			
Alcohol Initiation	.483		.532		-.049
Cigarette Initiation	.541		.502		.039
Marijuana Initiation	.506		.516		-.010
Time to Alcohol Initiation		213.512		212.533	.979
Time to Cigarette Initiation		378.217		306.813	71.404
Time to Marijuana Initiation		386.045		322.233	63.812
		Age-Graded Models			
Alcohol Initiation					
Among 6th Graders	.256		.252		.004
Among 9th Graders	.445		.483		-.038
Among 12th Graders	.275		.358		-.083

(Table 80 continues)

(Table 80 continued)

	2004 Model Fit		2001 Model Fit		
Model Type	R^2	χ^2	R^2	χ^2	Shrinkage
Cigarette Initiation					
Among 6th Graders	.338		.227		.111
Among 9th Graders	.575		.475		.100
Among 12th Graders	.500		.458		.042
Marijuana Initiation					
Among 9th Graders	.547		.467		.080
Among 12th Graders	.579		.552		.027
Time to Alcohol Initiation					
Among 6th Graders		85.114		58.616	26.498
Among 9th Graders		80.799		111.100	-30.301
Among 12th Graders		73.920		89.932	-16.012
Time to Cigarette Initiation					
Among 6th Graders		132.339		70.576	61.763
Among 9th Graders		146.793		142.758	4.035
Among 12th Graders		133.967		134.625	-.658
Time to Marijuana Initiation					
Among 9th Graders		161.256		147.314	13.942
Among 12th Graders		197.602		170.159	27.443

Nonetheless, in only seven of the 22 regression models did the 2001 data account for more variation in the outcomes. For three (i.e., marijuana initiation among the total sample, alcohol initiation among 9th graders, and time to cigarette initiation among 12th graders) of these seven outcomes, however, the explanatory power of the 2001 model was only slightly better than that observed in the corresponding 2004 model.

Second, the most comparable models included marijuana initiation and time to alcohol initiation among the total sample, alcohol initiation among 6th graders, and time to cigarette initiation among 12th grade students. In contrast, the largest deviations in model fit were evidenced for time to cigarette and marijuana initiation among the total sample, as well as time to cigarette initiation among 6th grade students.

Taken together, these findings suggest that the amount of explained variation in the outcomes accounted for by the 2001 and 2004 PPAAUS predictors was similar, with a few exceptions. Detailed later in this chapter, since shrinkage did not pose a serious problem, some major threats to the internal and external validity of the 2004 PPAAUS results appear less plausible.

Hypotheses Results

Following the lead of other researchers (Mills & Noyes, 1984), a further comparison of the findings from the 2004 and 2001 PPAAUS was conducted. Specifically, the derivation and validation findings for the directional (H2 and H3) and magnitude (H3, H6, H7, and H9) hypotheses were compared, along with the results for H4, H5, and H8. Discussed in Chapter 6, and detailed later in this chapter, in the event that the bulk of the H2-H9 test results from the 2004 PPAAUS are congruent with those from the 2001 PPAAUS, some of the threats (e.g., third variable explanations and differential time periods) to the internal and external validity of the 2004 PPAAUS findings may be deemed less plausible.

Hypothesis 2

Findings from the 2004 PPAAUS yielded partial support for H2.[18] Discussed earlier, and outlined in Table 81, counter support centered on the protective functions exerted by *AlcIntent*, *CigIntent*, *PeerAlc*, *MessPeerAlc*, *Cheat*, *MessParCig*, and *MessMedAlc*. The bulk of the deviations observed with the 2004 data were found among the 12th grade sample.

Presented in Appendix G (Tables G8-G29), results from the 2001 PPAAUS also yielded partial support for H2; however, more support was garnered with these data than through using the 2004 data. Among the 2001 validation sample, the direction of effects exerted by those predictors that were conceptualized as increasing risk for soft drug initiation and lowering age of initiation met hypothesized expectations, with a single exception (Table 81). Among 9th graders, *AlcIntent* (b = -2.154; p <.05) exhibited a protective capacity in decreasing risk for marijuana initiation.

Although the same counter findings for *AlcIntent* were not evidenced among both the deviation and validation samples, a general confirmation of the protective nature of *AlcIntent* was obtained. This reaffirmation lends some credence to the notion that *AlcIntent* may operate in a protective fashion in decreasing risk for, or delaying time to, cigarette and marijuana initiation.

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[18] Predictors conceptualized as risk factors increase risk for alcohol, cigarette, and marijuana initiation and lower age of initiation.

Table 81.

2004 and 2001 PPAAUS: H2 Counter Findings

	2004 PPAAUS				2001 PPAAUS			
Outcome/Relevant Predictor	TS	6th	9th	12th	TS	6th	9th	12th
Alcohol Initiation								
MessMedAlc				X				
Cigarette Initiation								
AlcIntent				X				
CigIntent		X						
Cheat			X					
Marijuana Initiation								
MessMedAlc				X				
PeerAlc				X				
MessPeerAlc				X				
AlcIntent							X	
CigIntent				X				
Time to Alcohol Initiation								
MessMedAlc				X				
Time to Cigarette Initiation								
MessMedAlc				X				
MessParCig		X						
AlcIntent	X			X				
Time to Marijuana Initiation								
PeerAlc				X				
MessPeerAlc				X				
AlcIntent	X							
CigIntent				X				

Note. TS = Total sample.

Hypothesis 3

H3[19] obtained partial support when it was tested with findings from the 2004 PPAAUS. Shown in Table 82, deviations from H3 centered on *Social*, *Work*, and *Academic*. The majority of the counter findings were observed among the 6th grade subsample.

Table 82.

2004 and 2001 PPAAUS: H3 Counter Findings

	2004 PPAAUS				2001 PPAAUS			
Outcome/Relevant Predictor	TS	6th	9th	12th	TS	6th	9th	12th
Alcohol Initiation								
Social	X							
Work					X			X
Academic		X						
Cigarette Initiation								
Social		X					X	
Work			X					
Marijuana Initiation								
Social			X					
Work	X							
Time to Alcohol Initiation								
Social		X			X			X
Academic		X						
Time to Cigarette Initiation								
Social		X					X	

Note. TS = Total sample.

[19] Predictors conceptualized as protective factors decrease risk for alcohol, cigarette, and marijuana initiation and delay age of initiation.

H3 also was tested with relevant findings from the 2001 PPAAUS (see Appendix G, Tables G8-G29). Similar to the 2004 PPAAUS results, the 2001 validation findings yielded partial support for H3. Although the exact same counter findings for *Academic* evidenced with the 2004 data were not observed with the 2001 validation sample, the risk capacities of *Social* and *Work* were generally reaffirmed. This confirmation lends some credence to the risk effects that these constructs demonstrated in the 2004 PPAAUS data.

Results from the 2001 PPAAUS (Table 82) indicated that frequent involvement in work-for-pay activities (*Work*) increased risk for alcohol initiation among the total sample (b = .186; p <.05) and 12th graders (b = .438; p <.05). With respect to frequent involvement in social activities, *Social* lowered age of alcohol initiation among both the total sample (b = .266; p <.001) and 12th grade students (b = .360; p <.001). *Social* also increased risk for cigarette initiation (b = .637; p <.05) and lowered age of cigarette initiation (b = .278; p <.001) among 9th graders.

Hypothesis 4

Little or no support for H4[20] was yielded with findings from the 2004 PPAAUS. Discussed in Chapter 9, the individual domain predictors made the largest contributions to explaining each total sample and age-graded outcome. For many of these regression models, no peer factors were retained upon entry.

In testing H4 with data from the 2001 PPAAUS, attention was directed at the findings from 16 regression models: alcohol and cigarette initiation and time

[20] Relative to other ecological domains, peer domain predictors explain the largest proportion of variance in alcohol and cigarette initiation and age of initiation.

to initiation among the total sample (see Appendix G, Tables G8-G9 and G11-G12), and alcohol and cigarette initiation and time to initiation among 6th, 9th, and 12th grade students (Tables G14-G19 and G22-G27). Specifically, the percentage decrease in the explained variance in alcohol and cigarette initiation among the total sample, and alcohol and cigarette initiation among 6th, 9th, and 12th graders, was calculated after removing each significant domain in each of the respective final models. The explained variation then was compared. Similarly, the percentage decrease in the final model χ^2 attributed to removing each significant domain also was calculated and compared for time to alcohol and cigarette initiation among the total sample, as well as time to alcohol and cigarette initiation among 6th, 9th, and 12th grade students.

Findings from the 2004 PPAAUS yielded minimal support for H4; however, more support for H4 was garnered with the 2001 data than with the 2004 derivation findings. Similar to the 2004 findings, the results from the 2001 PPAAUS indicated that removing the individual domain was responsible for the largest percentage decrease in the explained variance in alcohol and cigarette initiation (17% and 26% decrease, respectively) and time to alcohol and cigarette initiation (71% and 47% decrease, respectively) among the total sample. The largest percentage decrease in explained variation in alcohol and cigarette initiation and time to initiation among 9th and 12th graders, and cigarette initiation and time to initiation among 6th graders, also was attributed to the individual domain predictors. As was observed with the 2001 findings, many of the 2001 PPAAUS prediction models did not retain the peer domain variables upon entry.

The two 2001 PPAAUS findings that supported H4 (and countered the corresponding 2004 derivation results) centered on alcohol initiation and time to initiation among 6th graders. Among the 2001 6th grade subsample, the peer domain was responsible for the largest percentage decrease (33% and 42%) in respective R^2 and χ^2.

Hypothesis 5

H5[21] garnered partial support with findings from the 2004 PPAAUS. Discussed in Chapter 9, deviation from H5 centered on marijuana initiation and time to initiation among 9th grade students. Among this subsample, removing the control variables was responsible for the largest percentage reduction in R^2 and χ^2 in marijuana initiation and time to marijuana initiation.

In testing H5 with findings from the 2001 PPAAUS, attention turned to six regression models: marijuana initiation and time to marijuana initiation among the total sample (see Appendix G, Tables G10 and G13), and marijuana initiation and time to marijuana initiation among 9th and 12th grade students (Tables G20-G21 and G28-G29). The same strategy used to test H5 with the 2004 data was employed in testing this hypothesis with findings from the validation data. In particular, the percentage decrease in the explained variance in marijuana initiation among the total sample, as well as among 9th and 12th graders, was calculated and compared after removing each significant domain in each of the respective final models. As well, the percentage decrease in the final model χ^2 attributed to removing each significant domain was calculated and compared for

[21] Relative to other ecological domains, individual domain predictors explain the largest proportion of variance in marijuana initiation and age of initiation.

time to marijuana initiation among the total sample, and for time to marijuana initiation among 9th and 12th grade students.

Similar to the 2004 data, the findings from the 2001 PPAAUS yielded partial support for H5. This cross-validation strengthened considerably the validity of the 2004 findings, particularly since the H5 findings from the 2001 data mirrored those obtained using the 2004 results. As was observed with the 2004 data, the 2001 PPAAUS results supported H5 in finding that the individual domain was responsible for the largest percentage decrease (10% and 66%) in R^2 in marijuana initiation among the total sample and 12th grade students, as well as the largest percentage reduction (26% and 43%) in χ^2 for time to marijuana initiation among these respective samples. Similar to that found with the 2004 data, 2001 PPAAUS findings indicated that among 9th grade students, the control variables were responsible for the largest percentage decrease (8.4% and 28.3%) in explained variance in marijuana initiation and time to marijuana initiation.

Hypothesis 6

H6[22] obtained partial support when it was tested with results from the 2004 PPAAUS for marijuana initiation and time to initiation among the total sample and 9th and 12th grade students. The sole deviation from H6 centered on marijuana initiation among 12th graders. Among this subsample, early age of alcohol initiation (*AlcEarly*) was a more highly significant predictor of marijuana initiation than early age of cigarette initiation (*CigEarly*).

[22] Relative to alcohol initiation, cigarette initiation is a stronger predictor of marijuana initiation and age of initiation.

In determining the utility of H6 using the 2001 PPAAUS data, the significance levels and magnitude of effects exerted by *AlcEarly* and *CigEarly* in predicting marijuana initiation and time to initiation among the total sample (see Appendix G, Tables G10 and G13) and 9th (Tables G20 and G28) and 12th (Tables G21 and G29) graders were compared. In short, the 2001 PPAAUS test of H6 garnered minimal support. In fact, with only one validation finding supportive of H6, little semblance in the 2001 and 2004 PPAAUS findings were observed.

Counter to H6, the 2001 PPAAUS findings indicated that early age of alcohol initiation (*AlcEarly*) was a more highly significant predictor of marijuana initiation and time to initiation among the total sample (b = 1.589; p <.001; b = 1.219; p <.001) and 9th grade students (b = 1.779; p <.05; b = 1.252; p <.05), as compared to early age of cigarette initiation (*CigEarly*, b = 1.007; p <.05; b = .543; p <.05; b = 1.134; p <.10; b = .723; p <.20, respectively). In predicting time to marijuana initiation among seniors, the risk effects of *CigEarly* were nullified once the control variables were taken into account. In contrast, *AlcEarly* (b = 1.532; p <.001) was the most highly significant predictor of time to marijuana initiation among this subsample.

The sole finding from the 2001 PPAAUS that supported H6 centered on marijuana initiation among 12th graders. Taking into account the Wald statistics and significance values, *CigEarly* (b = 2.229; p <.001) was more a slightly more highly significant predictor of marijuana initiation among this subsample than *AlcEarly* (b = 2.107; p <.001).

Hypothesis 7

H7 was partially supported with respect to the findings from the 2004 data. This hypothesis posited that community domain predictors explain a larger proportion of variance in alcohol, cigarette, and marijuana initiation and age of initiation among 12^{th} grade students as compared to alcohol and cigarette initiation and age of initiation among 6^{th} and 9^{th} grade students, and marijuana initiation and age of initiation among 9^{th} grade students.

Major deviations centered on alcohol and cigarette initiation among 6^{th} and 12^{th} graders, and time to cigarette initiation among 12^{th} grade students. For example, the amount of variation attributed to the community factors in explaining alcohol initiation among 6^{th} and 12^{th} graders was equivalent, while removing the community factors was responsible for a larger percentage reduction in R^2 for cigarette initiation among 6^{th} grade students than among 12^{th} grade students. Further, no community domain predictors made a significant contribution to explaining time to cigarette initiation among 12^{th} graders, while removing the community factors was responsible for only a 3% reduction in the final model χ^2 for time to cigarette initiation among 9^{th} grade students.

In testing H7 with findings from the 2001 data, attention turned to the age-graded findings for soft drug initiation (see Appendix G, Tables G14-G21) and time to initiation (Tables G22-G29). Outlined in Table 83, little support for H7 was yielded. In general, *MessMedAlc*, *MessMedCig*, and *MessMedMar* were not important predictors of soft drug initiation and time to initiation among all three subsamples. The sole finding that somewhat supported H7 centered on marijuana initiation. However, *MessMedAlc* (b = -1.204; *p* <.10) exerted a direct, protective (rather than risk) effect on marijuana initiation among 12th graders. In contrast, no community domain factors were retained in the 9th grade marijuana initiation model upon entry. This 2001 finding mirrored that found in the corresponding 2004 PPAAUS models. Discussed in Chapter 9, no community variables were related to marijuana initiation among 9th graders, while removing this ecological domain was responsible for a 7% decrease in explained variance in marijuana initiation among 12th grade students.

Table 83.

Hypothesis 7 Findings, 2001 PPAAUS: Percentage Decrease in Final Model R^2/χ^2 Attributed to the Community Domain

	6th Graders		9th Graders		12th Graders	
Outcome	Proportion Decrease	Final Model R^2/χ^2	Proportion Decrease	Final Model R^2/χ^2	Proportion Decrease	Final Model R^2/χ^2
Alcohol Initiation	.000	.252	.000	.483	.000	.358
Cigarette Initiation	.000	.227	.000	.475	.000	.458
Marijuana Initiation	---	---	.000	.467	.013	.552
Time to Alcohol Initiation	.000	58.616	.000	111.100	.000	89.932
Time to Cigarette Initiation	.000	70.576	.000	142.758	.000	134.625
Time to Marijuana Initiation	---	---	.000	147.314	.000	170.159

Note. --- = not assessed in respective model.

Hypothesis 8

H8 argued that parental pro-drug norms is a stronger predictor of alcohol and cigarette initiation and age of initiation among 6th grade students, as compared to 9th or 12th grade students. Only one finding from the 2004 survey provided support for this hypothesis. Discussed in Chapter 9, parental tolerance of cigarette use (*MessParCig*) predicted time to cigarette initiation among 6th grade students, but not 9th or 12th graders. The other 2004 findings for 6th grade students were not supportive. Some 6th grade models never retained *MessParAlc* and *MessParCig* upon entry, while in other models, the effects of these measures were mediated by more proximal variables.

In testing H8 with the 2001 PPAAUS findings for alcohol and cigarette initiation (See Appendix G, Tables G14-G19) and time to initiation (Tables G22-G27) among the three subsamples, the magnitude and corresponding *p*-values of the direct impact of parental tolerance of alcohol (*MessParAlc*) and cigarette (*MessParCig*) use on respective outcomes were examined. In short, the 2001 findings yielded no support for H8. In fact, *MessParAlc* and *MessParCig* had no direct impact on soft drug initiation and time to initiation among 6th grade students.

Hypothesis 9

The 2004 PPAAUS results yielded minimal support for H9.[23] Detailed in Chapter 9, only one of the seven findings was supportive. *PeerAlc* was a stronger predictor of alcohol initiation among 9th than 6th grade students. This variable was not predictive of alcohol initiation among 12th grade students at a conventional level of significance. In terms of the counter findings, *MessPeerCig*

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[23] Peer drug use and pro-drug norms are stronger predictors of alcohol, cigarette, and marijuana initiation and age of initiation among 9th grade students as compared to alcohol and cigarette initiation and age of initiation among 6th and 12th grade students, and marijuana initiation and age of initiation among 12th grade students.

was predictive of cigarette initiation and time to initiation among 6th graders only, while *PeerAlc* was a more salient determinant of time to alcohol initiation among 6th than 9th grade students. Finally, *MessPeerAlc* and *MessPeerMar* were predictive of marijuana initiation among 12th graders only.

H9 also was tested with 2001 findings for soft drug initiation (see Appendix G, Table G14-G21) and time to initiation (Tables G22-G29) among 6th, 9th, and 12th grade students. As shown in Table 84, three of the nine 2001 PPAAUS findings were supportive of H9, thereby lending partial support for this hypothesis.

Supportive findings centered on marijuana initiation and time to initiation. *MessPeerMar* increased risk for marijuana initiation (b = 2.183; p <.001) and elicited earlier ages of marijuana initiation (b= 1.694; p <.001) among 9th grade students. These constructs were not predictive of marijuana initiation and time to initiation among 12th graders. *PeerMar* also was not associated with time to marijuana initiation among seniors. Among 9th graders, however, students who perceived that a large proportion of their peers used marijuana (*PeerMar*, b = .462; p <.05) tended to initiate use at early biological ages.

The 2001 PPAAUS findings that countered H9 dealt specifically with alcohol and cigarette initiation and time to initiation. In general, these results indicated that peer drug use and pro-drug norms were more important determinants of alcohol and cigarette initiation and time to initiation among 6th than 9th or 12th grade students.

Table 84.

Hypothesis 9 Findings, 2001 PPAAUS: Direct Effects of Peer Soft Drug Use and Pro-Drug Norms on Respective Outcomes

	6th Graders		9th Graders		12th Graders	
Outcome/Predictor	B	*p*-value (<)	B	*p*-value (<)	B	*p*-value (<)
Alcohol Initiation						
PeerAlc	1.481	.001	1.457	.001	NS	NS
MessPeerAlc	1.368	.001	NS	NS	NS	NS
Cigarette Initiation						
MessPeerCig	2.318	.05	NS	NS	NS	NS
Marijuana Initiation						
MessPeerMar	---	---	2.183	.001	NS	NS
Time to Alcohol Initiation						
PeerAlc	1.068	.001	.463	.001	NS	NS
MessPeerAlc	1.058	.05	NS	NS	NS	NS
Time to Cigarette Initiation						
MessPeerCig	1.778	.05	NS	NS	NS	NS
Time to Marijuana Initiation						
PeerMar	---	---	.462	.05	NS	NS
MessPeerMar	---	---	1.694	.001	NS	NS

Note. --- = Not assessed in respective model.

Several findings revealed in the 2004 PPAAUS models were not found in the 2001 data. For example, 2004 results indicated that *MessPeerAlc* was important in reducing the risk for marijuana initiation among 12th graders only. This finding was not evidenced in the 2001 results. *MessPeerMar* also was predictive of marijuana initiation among 12th graders who completed the 2004

PPAAUS, but this construct was not predictive of marijuana initiation among seniors who completed the 2001 PPAAUS.

Distal-Proximal Mediation

Parallel to the 2004 PPAAUS findings, all of the validation models yielded some degree of support for Petraitis et al.'s (1995) distal-proximal mediation hypothesis (see Appendix G, Tables G8-G29). The findings from the formal evaluation of the direct and indirect effects exerted by the 2001 PPAAUS predictors are presented in Table 85. In short, not only were the derivation and validation samples strikingly similar in terms of the proportion of indirect effects that were exerted and the patterns identified across domains, but results from both samples provided considerable support for Petraitis et al.'s contention. Four major findings are worth discussing.

First, on balance, 58% of the 2001 community, school, family, and peer variables that initially were retained in the 22 regression models were reduced to insignificance in the final models. This finding is similar to that found with the 2004 PPAAUS data (where 61% of these constructs were reduced to insignificance). Further validation findings indicated that in only eight (i.e., alcohol initiation and time to initiation among the total sample and 12th graders; marijuana initiation among 9th and 12th graders; time to cigarette initiation among 9th graders; and time to marijuana initiation among 9th graders) of the 22 models analyzed were the majority of effects direct in nature. This result also complements the derivation finding (i.e., six of 22 models).

Table 85.

Proportion of Extraneous Effects as Indirect, 2001 PPAAUS: Total Sample and Age-Graded Models

	Proportion of Ecological Domain Effects as Indirect				
Sample/Model Type	Community	School	Family	Peer	Mean Proportion
	Total Sample Models				
Alcohol Initiation	1.00	.60	0.00	0.00	.40
Cigarette Initiation	1.00	1.00	1.00	1.00	1.00
Marijuana Initiation	1.00	.80	1.00	0.00	.70
Time to Alcohol Initiation	---	.60	0.00	1.00	.40
Time to Cigarette Initiation	1.00	1.00	1.00	1.00	1.00
Time to Marijuana Initiation	1.00	1.00	1.00	.66	.92
	Age-Graded Models				
Alcohol Initiation					
Among 6th Graders	1.00	.66	1.00	0.00	.67
Among 9th Graders	---	.66	1.00	.50	.54
Among 12th Graders	---	.66	0.00	---	.17
Cigarette Initiation					
Among 6th Graders	1.00	.50	1.00	0.00	.63
Among 9th Graders	---	.50	1.00	1.00	.63
Among 12th Graders	---	1.00	1.00	1.00	.75
Marijuana Initiation					
Among 9th Graders	---	1.00	---	.50	.38
Among 12th Graders	.50	.33	0.00	1.00	.46
Time to Alcohol Initiation					
Among 6th Graders	1.00	1.00	1.00	0.00	.75
Among 9th Graders	---	.66	1.00	.50	.54

(Table 85 continues)

(Table 85 continued)

	Proportion of Ecological Domain Effects as Indirect				
Sample/Model Type	Community	School	Family	Peer	Mean Proportion
Among 12th Graders	---	.33	0.00	1.00	.33
Time to Cigarette Initiation					
Among 6th Graders	1.00	1.00	1.00	0.00	.75
Among 9th Graders	---	.33	0.00	1.00	.33
Among 12th Graders	---	1.00	---	1.00	.50
Time to Marijuana Initiation					
Among 9th Graders	---	.50	---	0.00	.13
Among 12th Graders	1.00	.33	1.00	1.00	.83
Column averages	.48	.71	.59	.55	.58

Note. --- = No predictors in the ecological domain made a significant contribution to explaining the given outcome.

Second, the 2004 and 2001 PPAAUS differed in terms of the ecological domain that was most apt to have been fully mediated. The 2001 PPAAUS findings indicated that, on average, the school domain predictors were most apt to have been fully mediated by more proximal (family, peer, and individual) variables. In contrast, findings from the 2004 data indicated that the family domain constructs were most apt to have been fully mediated by more proximal predictors, although the school domain variables did come in a close second.

Third, the derivation and validation results were remarkably similar in terms of the average effects wielded by the community domain. Discussed in Chapter 9, findings from the 2004 PPAAUS data indicated that, on average, 50%

of the community domain effects were indirect in nature. This compares to the average of 48% that was found with the validation data.

Finally, a more fine-grained analysis of distal-proximal effects revealed that the findings from both the derivation and validation samples were fairly similar in terms of the types of outcomes and population targets that yielded low and high levels of support for Petraitis et al.'s (1995) contention. With respect to low levels of support, findings from the both the derivation and validation sample indicated that predictors of time to alcohol initiation among the total sample and 12^{th} graders, alcohol initiation among 12^{th} graders, and time to cigarette initiation among 9^{th} grade students tended to exert direct effects. In contrast, predictors of alcohol and cigarette initiation and time to initiation among 6^{th} grade students tended to exert more indirect effects.

Validity of the 2004 PPAAUS Results

As the presentation of the dual cross-validation results revealed, a fairly strong confluence in findings was obtained. Not only were many of the 2004 PPAAUS findings for H2-H9 confirmed in full or part, but the evaluation of shrinkage and the comparison of findings for distal-proximal mediation provided further evidence of the stability of the 2004 PPAAUS results.

The dual cross-validation findings have many implications for the validity of the 2004 PPAAUS findings. Discussed in Chapters 5 and 6, major threats to the internal validity of the 2004 results center on selection bias, history, and causal ordering, while plausible threats to the construct validity of the findings include invalid or unreliable measures; various types of reporting bias (e.g., forward telescoping, over- and under-reporting, recall decay, and heaping); the use of self-report data; and lengthy recall periods. An incongruence between the characteristics of the 2004 PPAAUS sample and 6th, 9th, and 12th grade students from other places, times, and settings constitutes a salient threat to the external validity of the results. Finally, with respect to statistical conclusion validity, low statistical power (often a product of a small sample) and the use of inappropriate statistical techniques constitute areas of concern.

The following discussion provides an indepth and careful consideration of the validity of the 2004 PPAAUS results. Attention is primarily directed at the conflicting findings from the dual cross-validation, and the implications that these results pose for the validity of the 2004 survey findings. Where appropriate, the implications that select congruent results pose for the validity of the 2004 findings also are discussed.

Hypotheses 2 and 3

Verification was obtained for nearly all of the 2004 findings for H2 and H3 that could be assessed, including those directional findings that were subjected to dual confirmation. Not only did the majority of the H2 and H3 directional effects from the 2001 PPAAUS converge with those from the 2004 PPAAUS, but full convergence was established for 59 of the 66 directional findings from the 2004 survey that were assessed in the systematic review cross-validation. With only seven counter findings identified in this cross-validation, a general convergence rate of 89% was obtained. This strong degree of congruence underscores the internal, external, construct, and statistical conclusion validity of much of the 2004 results for H2 and H3.

Hypothesis 2

Systematic review cross-validation. Although the majority of the dual cross-validation findings for H2 and H3 converged, a comparison of the H2 and H3 results from the 2004 PPAAUS with relevant findings from the systematic review revealed seven counter results. In an effort to explain these disparate findings, as well as gauge the plausibility of threats to the validity of relevant 2004 PPAAUS results, the conflicting findings from the systematic review cross-validation were compared to the relevant results from the 2001 PPAAUS. The characteristics of those primary studies that obtained conflicting results also were compared to those from the current research. The triangulation of findings is presented in Table 86, while Table 87 introduces the major differences in study characteristics that were identified.

Four discrepant findings for H2 were identified through the systematic review cross-validation. Three centered on the direction of the relationship between parental tolerance of marijuana use (*MessParMar*) and marijuana initiation among 9th grade students, and between peer marijuana use (*PeerMar*) and marijuana initiation among 9th graders. Although prior research provides mixed results for these relationships, the corresponding 2004 findings may not be entirely accurate. The final conflicting finding involved the direction of the relationship between peer alcohol use (*PeerAlc*) and time to alcohol initiation among the total sample.

Table 86.

Triangulation of the H2 and H3 Counter Findings from the Systematic Review Cross-Validation

Hypothesis/Predictor/Outcome	Predictor Type, 2004 PPAAUS	Predictor Type, Primary Studies	Predictor Type, 2001 PPAAUS
H2: *MessParMar* and marijuana initiation among 9th graders	Risk Factor	▪ Risk Factor among 9th grade students, Ellickson et al. (2004) ▪ Protective Factor among 10th grade students, Ellickson et al. (2004)	Risk Factor
H2: *PeerAlc* and time to alcohol initiation among the total sample	Protective Factor	▪ Risk Factor among 10-18 year olds, Hawkins et al. (2002b)	Risk Factor
H2: *PeerMar* and marijuana initiation among 9th graders	Protective Factor	▪ Protective Factor among 10th grade students, Ellickson et al. (2004) ▪ Risk Factor among 9th grade students, Ellickson et al. (2004) ▪ Risk Factor among 10th grade students, Walter et al. (1991)	Risk Factor
H3: *HiAcadPerf* and alcohol initiation among 6th graders	Risk Factor	▪ Poor school performance as Risk Factor among 8th grade students, Crum et al. (2005)	Protective Factor
H3: *Religious* and cigarette initiation among 9th graders	Risk Factor	▪ Protective Factor among junior high-school students, Skinner et al. (1985)	Protective Factor
H3: *Work* and cigarette initiation among 9th graders	Risk Factor	▪ Protective Factor among junior high-school students, Skinner et al. (1985)	Risk Factor

Table 87.

Possible Explanations for Conflicting Findings: Major Differences between the 2004 PPAAUS and Relevant Primary Studies

Study Elements	2004 PPAAUS	Crum et al. (2005)	Ellickson et al. (2004)	Hawkins et al. (2002b)	Skinner et al. (1985)	Walter et al. (1991)
Age/Grade-Level of Sample:	6th, 9th, 12th grade; Total sample	8th grade	7th, 8th, 9th, 10th grade	10-18 years (majority teens)	Junior-high; High-school; Total sample	10th grade
Gender and Race of Sample:	52% Male; 86% White	Gender unknown; Majority Black	52% Male; 34% Minority	55% Male; 46% White	47% Male; Race unknown	44% Male; 63% White
Geographic Location of Sample:	Rural	Urban	Urban, Rural, Sub	Urban	Rural	Urban, Sub
Time Period of Data Collection:	2004	1992-1993	Mid-late1980s	1985-1993	1980-1982	Unknown
Efforts to Reduce Under/Over-Reporting:	No	Yes, COC	Yes, Saliva pipe	No	Yes, Saliva pipe	No
Analytic Techniques:						
Initiation…	Binary Logit	SEM	Binary Logit	N/A	Discr. Fun. Anal.	Binary Logit
Time to Initiation…	Cox Regression	N/A	N/A	Cox Regression	N/A	N/A
Adequate Consideration of Third Variables:	Yes	No	Yes	No	No	Yes

Parental tolerance of marijuana use (*MessParMar*) increased risk for marijuana initiation among 9th grade students from the 2004 PPAAUS. Comparatively, Ellickson et al. (2004) found that parental tolerance of marijuana use increased risk for marijuana initiation among 9th grade students, but decreased risk for marijuana initiation among 10th grade students. The H2 finding from the 2001 PPAAUS for the directional association between *MessParMar* and marijuana initiation among 9th grade students supports the risk effects evidenced with the 2004 PPAAUS data (Table 86). Taking into account this supportive finding, while considering several major research design differences between the PPAAUS research and Ellickson et al.'s (2004) study (see Table 87, e.g., differences in the geographic locations and demographics of the samples), it is probable that, at a minimum, Ellickson and colleagues' counter finding may be the product, in part, of an examination of data from youth who differ in geography and demographics. The 2004 PPAAUS data and Ellickson and colleagues data also were collected in different time periods.

The second counter age-graded finding also centered on marijuana initiation among 9th grade students. In the current research, *PeerMar* exerted a protective effect, by reducing the likelihood of marijuana initiation among this subsample. Even though Ellickson et al.'s (2004) findings for this relationship among 10th grade students supports that which was yielded in the current research, their second finding, obtained with data from 9th grade students, and Walter et al.'s (1991) finding, generated with data from a sample of 10th grade students, as well as the 2001 PPAAUS result, indicates that peer marijuana use increases risk for marijuana initiation among mid-adolescent youth.

Major differences in study characteristics predominantly reside in the different time periods that data were collected (Table 87). This incongruence, coupled with the possibility of differential age effects, constitute the only plausible explanations for the differences in findings that were observed. For example, both of the primary studies, as well as the 2001 and 2004 PPAAUS, considered a plethora of predictors in binary logit regression models (Table 87), so it is unlikely that the discrepant findings are due to this type of selection bias. Even though studies differed in respective efforts to reduce under- and over-reporting, and the geographic locations of the respective samples differed, these discrepancies do not appear to explain the incongruent findings. Moreover, since both marijuana initiation and *PeerMar* were measured in the same manner in the 2001 and 2004 PPAAUS, the directional difference in the effects of *PeerMar* obtained with these data are not due to differences in measurement.

Finally, the sole H2 counter finding identified through the systematic review for time to soft drug initiation among the total sample centered on the directional effects posed by *PeerAlc*. The current research found that peer alcohol use served as a protective factor, by delaying age of alcohol initiation among the total sample. In contrast, Hawkins et al. (2002b) found that this construct served as a risk factor, in lowering age of alcohol initiation among a sample of youth whose ages ranged from 10 to 18 years. Although the biological ages of Hawkins and colleagues' sample do not correspond well with the ages of the adolescents who comprised the 2004 total sample (Table 87), the biological ages of the students from the 2001 total sample do. Among this sample, *PeerAlc* demonstrated risk effects in lowering age of alcohol initiation.

Since both the 2001 finding and that obtained by Hawkins and colleagues coalesce, it is possible that the differential finding yielded with the 2004 data may be confounded by some validity threats. All three findings were obtained using the same analytic technique (Cox regression), and the sizes of the respective samples were adequate, given the number of predictors that each study investigated. Hence, differential results likely are not the result of threats to the statistical conclusion validity of the 2004 finding. Although Hawkins and associates did not consider a number of important factors known to predict time to alcohol initiation, the 2001 PPAAUS did account for an adequate number of third variables. Virtually the same factors that were examined with the 2001 data were investigated with the 2004 data, so it also is unlikely that differences in the directional findings obtained by these surveys were due to this type of selection bias threat.

Since the 2004 PPAAUS and Hawkins et al.'s (2002b) study differed considerably on several major features (Table 87), an explanation for the protective capacity of *PeerAlc* found with the 2004 data may lie predominantly in differences between the 2004 and 2001 PPAAUS. Discussed in Chapter 6, the only major differences between these two surveys are the time periods at which each were administered, and cohort effects, for which the existence of is not fully clear. Hence, the only practical explanation for the differential finding obtained with the 2004 data is the existence of secular or cohort effects, both of which constitute plausible threats to the 2004 result.

Quantitative cross-validation. Within the quantitative cross-validation, all of the directional effects exerted by the H2 variables met hypothesized expectations, with a single exception. In contrast to the risk effects posed by *AlcIntent* in predicting marijuana initiation among 9th graders from the 2004 sample, *AlcIntent* served a protective function, by decreasing risk for marijuana initiation among 9th graders from the validation sample.

Although this 2001 finding provides additional confirmation of the protective nature of *AlcIntent* in decreasing risk for, or delaying time to, cigarette and marijuana initiation, further insight with regard to marijuana initiation among 9th grade students could not yielded from the systematic review cross-validation, since no primary studies investigated this variable within the context of marijuana initiation. Due to time period differences in the collection of 2001 and 2004 PPAAUS data, as well as the possibility of differential age and cohort effects for these two samples of 9th graders, either of the PPAAUS directional findings may be valid. Further research on this relationship would be helpful in affirming the validity of the 2004 finding.

Hypothesis 3

Systematic review cross-validation. The bulk of the seven counter findings identified through the systematic review cross-validation centered on the direction of the age-graded relationships between soft drug initiation and variables viewed as protective factors. Even though this disjuncture is largely responsible for the convergence rate of 79%, obtained with respect to soft drug initiation and the H3 constructs, the fact that the majority of the directional findings for H3 converged offers some additional evidence to support the validity of the 2004 findings for this hypothesis.

Disconcordant findings centered on the direction of the relationships between high academic performance (*HiAcadPerf*) and alcohol initiation among 6th grade students, and between cigarette initiation among 9th graders and involvement in religious (*Religious*) and work-for-pay (*Work*) activities. The current research found that these H3 constructs exerted risk effects (although insignificant at conventional levels of significance), while Crum et al. (2005) and Skinner et al. (1985) demonstrated the protective functions of these variables.

Additional insight concerning the directional impact of these constructs may be gleaned from findings from the 2001 data (Table 86). The protective effects that *HiAcadPerf* and *Religious* demonstrated in research by Crum et al. (2005) and Skinner et al. (1985) also was obtained with the 2001 data. With respect to the directional effects exerted by *Work*, however, the 2001 and 2004 findings converged. The congruence in this latter finding not only offers some evidence for the validity of this 2004 result, but the PPAAUS research and that conducted by Skinner et al. (1985) differ on a number of important methodological and analytic features (Table 87).

It is not clear why the current research obtained counter findings for *HiAcadPerf* and *Religious*, although it is possible that differential age, secular, or cohort effects contributed to these 2004 results. Only through further research can these 2004 findings be affirmed.

Quantitative cross-validation. In general, the 2004 and 2001 findings for H3 converged, with both surveys yielding partial support for this hypothesis. The major H3 counter finding revealed through the quantitative cross-validation centered on the directional effects posed by frequent involvement in extra-curricular, academic activities (*Academic*).

In the 2004 PPAAUS, *Academic* increased risk for alcohol initiation and lowered time to initiation among 6th grade students. In contrast, this construct served a protective function with the 2001 PPAAUS data, by reducing the risk for these outcomes among 6th graders. Further insight into the directional effect of this construct could not be yielded from the systematic review, since no primary studies investigated the utility of this variable.

It is unlikely that the opposite findings for the directional effects posed by *Academic* on alcohol initiation and time to initiation among 6th graders were the result of various threats to the internal and construct validity of the 2004 results. Moreover, although the 2004 and 2001 regression models differed in terms of specification (e.g., the effects of soft drug availability were evaluated in the 2004 regression models, but not in the 2001 models), it is unlikely that differential model specification had an impact on the directional effects of *Academic*. Given the three-year time interval between survey administrations, however, sample differences in the impact of *Academic* may be due to secular or cohort effects, both of which constitute threats to the external validity of the 2004 findings.

Hypotheses 4-9

Aside from comparing the 2004 and 2001 findings for H2 and H3, the quantitative cross-validation evaluated the convergence in 2001 and 2004 findings on three other fronts: H4-H9, model fit, and distal-proximal mediation. Before attention is directed toward the implications that the conflicting H4-H9 findings from the quantitative cross-validation have for the validity of the relevant 2004 results, it is important to provide a general explanation for why some of the findings from the 2004 PPAAUS did not support H4-H9.

H4-H9 (and H2-H3) were developed with insight from extant comprehensive reviews of the literature. In turn, these reviews of the extant literature presented findings from studies that developed main effects models in evaluating the direct impact that predictors posed for soft drug initiation and time to initiation. In contrast, all 22 regression models developed in the current research took advantage of the backward stepwise regression technique, a procedure that enabled both direct and indirect effects to be considered.

Since the current research employed a different model-building technique than prior studies, some of the incongruence between the 2004 findings and H4-H9 does not necessarily call into question the validity of those conflicting 2004 results (or the findings of the comprehensive reviews). To further confirm the validity of the 2004 PPAAUS results for H4-H9, it is recommended that future research replicate the regression models that were developed, including the manner in which predictors were organized into blocks and the backward stepwise procedure that was used to arrive at the results.

Hypothesis 4

More support for H4 was garnered with the 2001 data than with data from the 2004 PPAAUS, although findings from both surveys did generally converge in providing little support for this hypothesis. In general, the 2001 and 2004 PPAAUS found that the individual domain made the largest contributions to explaining each combined and age-graded outcome. A convergence in results also was observed with respect to the explanatory importance of the peer domain. For many of the 2001 and 2004 regression models, no peer factors were retained upon entry.

While the findings from the 2004 data indicated that the individual domain made the largest contributions to explaining all outcomes, two 2001 PPAAUS results elicited support for H4. Among the 2001 6th grade subsample, the peer domain was responsible for the largest explained variation in alcohol initiation and time to initiation.

There are three possible reasons why these incongruent findings were obtained. The 2004 H4 results for the efficacy of peer factors in predicting alcohol initiation and time to initiation among 6th graders may be valid, thereby lending credence to the possibility of differential cohort effects. On the other hand, the corresponding 2001 validation results may be accurate, which also lends credence to the notion of differential cohort effects. Finally, both the 2001 and 2004 results for H4 may be valid.

Two major explanations may be offered to support the possibility that both the 2001 and 2004 findings are accurate. First, differential cohort effects, a plausible threat to the external validity of both the 2001 and 2004 results, may lie at the heart of the discrepancy in findings. Specifically, compared to the other cohort, one 6th grade cohort may hold distinctively different perceptions of the alcohol-related behavior and norms that their peers demonstrate.

Second, it is possible that no cohort effects were operating. In other words, the saliency of 6th grade students' alcohol-related perceptions of their peers in impacting the likelihood and age of alcohol initiation may have changed over the three-year time interval that elapsed between survey administrations. Due to the inability to rule out the plausibility that cohort effects impacted the results, the 2004 findings for H4 (concerning alcohol initiation and time to initiation among 6th graders) should be interpreted with caution and affirmed by future research.

Hypothesis 5

Similar to the 2004 results, the findings from the 2001 sample yielded partial support for H5. This convergence strengthened considerably the internal, external, and construct validity of the 2004 results, particularly since the H5 findings from the 2001 sample mirrored those from the 2004 sample. Specifically, findings from both samples supported H5 in finding that the individual domain was responsible for the largest explained variation in marijuana initiation among the total sample and 12th graders, as well as for time to marijuana initiation among these respective samples. Data from both samples also indicated that among 9th grade students, the control variables were responsible for the largest explained variation in marijuana initiation and time to initiation.

Hypothesis 6

Little semblance in the derivation and validation results were evidenced for H6. In fact, while only one deviation from H6 was obtained using the 2004 data, only one finding from the 2001 sample supported this hypothesis. Interestingly, this deviation from and support of H6 involved the same outcome (marijuana initiation) and grade-level (12^{th} grade). Among this 2004 subsample, early age of alcohol initiation (*AlcEarly*) was a more highly significant predictor of marijuana initiation than early age of cigarette initiation (*CigEarly*), a finding that counters H6. Comparatively, the antithetic result was obtained for 12^{th} grades from the 2001 sample, with *CigEarly* found to be a more highly significant predictor of marijuana initiation than *AlcEarly*.

A limited number of explanations may be offered for these incongruent findings, particularly since the percentage of 12^{th} graders from each survey who reported alcohol, cigarette, and marijuana initiation were comparable, and both of these subsamples self-reported equivalent average ages of soft drug initiation. For example, 12^{th} graders from the 2001 PPAAUS indicated 14.5 years, on average, as the biological age at which they initiated alcohol use, while seniors from the 2004 PPAAUS reported initiating alcohol use at 14.58 years of age. With respect to age of cigarette initiation, 12^{th} graders from the 2001 sample who initiated cigarette use reported beginning the use of this drug at 13.6 years, on average, compared to an average age of 13.8 years among while 12^{th} grade cigarette initiates from the 2004 sample. Even the average ages of marijuana initiation among 12th grade marijuana initiates were equivalent (14.7 years among the 2001 subsample and 14.5 years among the 2004 subsample).

Since the average age of alcohol and cigarette initiation were equivalent across survey years, subsample differences in the magnitude of the effects posed by early alcohol versus early cigarette initiation in predicting marijuana initiation do not appear to be due to subsample differences in the average ages of alcohol and cigarette initiation. Three possible explanations involving subsample differences may be offered, however, in providing some insight into the conflicting findings that were revealed.

One potential explanation for the incongruent findings centers on the extent to which the marijuana initiates from these subsamples were involved in alcohol and cigarette use between the time these substances were initiated and the PPAAUS was completed. Although H1 (i.e., alcohol>cigarette>marijuana initiation) was supported with total sample data from the 2004 PPAAUS, including data from seniors, and the frequencies with which 12th graders used alcohol and cigarettes were controlled in the corresponding marijuana initiation and time to initiation models, the proportion of the 12th grade subsample from the 2004 PPAAUS initiating marijuana use may have differed from their 2001 PPAAUS counterparts in terms of the quantity of alcohol and cigarettes they consumed. While there is no reason to suggest that one subsample of 12th graders consumed more alcohol or smoked more cigarettes than the other subsample, the quantity of use of these soft drugs was not controlled in the corresponding regression models. Hence, although an unlikely scenario, this explanation remains possible.

Second, descriptive data from both surveys suggest that some degree of forward telescoping on the part of older adolescents, particularly seniors, was operating. For example, compared to 6th grade alcohol initiates from the 2001 PPAAUS, who self-reported 10 years as the average age at which they began alcohol use, on average, 9th and 12th grade alcohol initiates from the 2001 sample self-reported beginning alcohol use at 12.4 and 14.5 years, respectively. Concerning findings from the 2004 PPAAUS, on average, 6th grade alcohol initiates indicated beginning alcohol use at 9.9 years of age. This compares to an average age of 12.8 years among 9th grade alcohol initiates and 14.5 years among 12th grade alcohol initiates from the 2004 PPAAUS.

Although there is no practical way to determine whether differential forward telescoping did exist, if there were considerable differences in forward telescoping between the 2004 and 2001 12th grade subsamples, it is possible that this threat to the construct validity of the findings from both samples played a role in artificially inflating or attenuating the relationships between marijuana initiation among 12th graders and age of alcohol and cigarette initiation. Hence, one or more artificial suppressions or inflations in the regression coefficients for these relationships could have produced the conflicting findings that were observed.

Finally, it is possible that the difference in the magnitude of effects exerted by *AlcEarly* and *CigEarly* in predicting marijuana initiation and time to initiation among 12th graders from the 2001 and 2004 PPAAUS may be due, in part, to the slight disjuncture in model specification. Detailed in Chapter 10, as well as earlier in this chapter, the effects of three variables (e.g., *EasyAlc*, *EasyCig*, and *EasyMar*) were not considered in the 2001 PPAAUS regression models, including the models predicting marijuana initiation and time to initiation among 12th graders. Moreover, collinearity issues precluded an examination of the utility of *AlcLate* and *AlcIntent* in predicting marijuana initiation and time to initiation among seniors from the 2001 PPAAUS. In contrast, the effects of all of these extraneous factors were taken into account in the corresponding 2004 models.

Together, the significance of numerous extraneous-outcome and extraneous-extraneous bivariate correlations from the 2004 PPAAUS provide some evidence to support the notion that a congruence in findings may have been yielded, had the impact of the same independent variables that were taken into account in the 2004 models been accounted for in the 2001 models. The majority of the extraneous measures that were not examined in the 2001 12th grade regression models were significantly associated with both marijuana initiation and age of initiation among the 2004 total sample, as well as early age of alcohol and cigarette initiation. In particular, the bivariate relationships between marijuana initiation among the total sample and *EasyAlc* (r = .285), *EasyCig* (r = .358), and *EasyMar* (r = .491) were significant at the .001 level, as was the bivariate relationship between *EasyCig* and age of marijuana initiation among the total sample (r = .206). Also significant were the 2004 bivariate relationships between marijuana initiation among the total sample and *AlcIntent*

(r = .275, p <.001), and between age of marijuana initiation among the total sample and *AlcLate* (r = .466, p <.001) and *AlcIntent* (r = .201, p <.05).

Further, bivariate findings from the 2004 PPAAUS also revealed that several of the extraneous measures that were not examined in the 12th grade marijuana initiation and time to initiation models were significantly associated with one another. For example, 2004 bivariate correlations significant at the .05 level included the associations between *EasyAlc* and *AlcInit* (r = .480), *EasyCig* and *AlcInit* (r = .475), *EasyMar* and *AlcInit* (r = .453), *EasyMar* and *CigEarly* (r = .411), *AlcIntent* and *AlcEarly* (r = .442), and *AlcLate* and *CigEarly* (r = .533).

Hypothesis 7

More support for H7 was yielded by the 2004 data than the 2001 data. In fact, *MessMedAlc*, *MessMedCig*, and *MessMedMar* were not important predictors of soft drug initiation or time to initiation among all three subsamples from the 2001 PPAAUS. In contrast, major deviations obtained with the 2004 data centered on alcohol and cigarette initiation among 6th and 12th grade students, and time to cigarette initiation among 12th graders. The sole finding from the 2001 PPAAUS that somewhat supported H7 centered on marijuana initiation. Removing the community domain factors was responsible for a larger percentage decrease in R^2 for this outcome among 12th grade students than 6th or 9th grade students.

Although the H7 cross-validation did not fare particularly well, with some major discrepancies revealed, it is important to note that this incongruence also may be due to the fact that *EasyAlc*, *EasyCig*, and *EasyMar* were not assessed in the 2001 PPAAUS models. Discussed in Chapter 6, as well as earlier in this chapter, the 2001 PPAAUS did not include measures of students' perceived ease in accessing alcohol, cigarettes, and marijuana.

Given that these three constructs were assessed in the 2004 PPAAUS models, and they were fairly important predictors of both soft drug initiation and time to initiation, it is reasonable to assume that the 2001 findings for H7 were impacted by model misspecification (e.g., not accounting for the same third variables as those taken into account in the 2004 models). In other words, had the effects of *EasyAlc*, *EasyCig*, and *EasyMar* been taken into account in the 2001 prediction models, it is likely that the 2001 findings for H7 would have been more consistent with the 2004 findings. Since model misspecification constitutes a plausible threat to the internal validity of the 2001 findings, the internal validity of the 2004 results for H7 should not be discounted. In order to more fully determine the extent to which the 2004 findings for H7 are valid, however, future research should take strides to incorporate into prediction models the six community domain constructs that were examined in the 2004 models.

Hypothesis 8

The findings from both the derivation and validation subsamples converged in providing little or no support for H8. Parental tolerance of alcohol (*MessParAlc*) and cigarette (*MessParCig*) use had no direct impact on alcohol and cigarette initiation and time to initiation among 6^{th} graders from the 2001 PPAAUS, while one finding from the 2004 PPAAUS supported H8. Specifically, parental tolerance of cigarette use (*MessParCig*) predicted time to cigarette initiation among 6th grade students, but not 9th or 12th graders.

This overarching convergence in findings indicates that the point of disjuncture in results rests more with the PPAAUS findings and results from comprehensive reviews of past research (upon which H8 was established) than between the 2001 and 2004 PPAAUS findings. The broader issue of the incongruence between select findings from the current research and that of prior research is addressed in the following chapter. With respect to H8, however, the convergence in the 2001 and 2004 results offers some evidence for the internal validity of the 2004 findings.

On the other hand, the internal and external validity of the 2004 findings for H8 may be limited, since this sample (as well as the 2001 sample) was exposed to drug prevention programming within the community in which these youth resided. Discussed in Chapter 6, history effects pose a plausible threat to the internal and external validity of both the 2001 and 2004 PPAAUS results, although this threat constitutes a more salient concern for the 2001 findings. Since the late 1990s, the community from which both samples were derived has participated in the CTC prevention strategy. Parental tolerance of adolescent problem behaviors, including drug use, constitutes one of five risk factors that the community has made explicit strides to reduce.

Given that the findings from one study (Myers & Arter, 2005a) indicated that the CTC prevention strategy may have been helpful in reducing adolescent soft drug use between 1998 and 2001, it is possible that the 2001 PPAAUS findings for the magnitude of the relationships between *MessParCig* and time to cigarette initiation among 6th, 9th, and 12th grade students are more conservative than those yielded with data from the 2004 sample. In other words, as a result of the prevention efforts that were directed at parents' attitudes toward drug use (including cigarette use) between 1998 and 2001, youth from the 2001 PPAAUS (who were at risk for cigarette initiation between 1998 and 2001) may have delayed cigarette initiation until they were older. Since there is no published research on the impact of the CTC prevention strategy on levels of risk, protection, and adolescent drug use between 2001 and 2004, it is unclear whether these prevention initiatives had the same impact on the soft drug behavior of adolescents from the 2004 PPAAUS.

Although the quantitative cross-validation was not able to fully assess the impact of history on the findings from both surveys, the null findings for H8 obtained with the 2001 data provide some support for the notion that the H8 relationships yielded with data from the 2001 sample may have been attenuated, and to a larger degree than those obtained from the 2004 derivation sample. Since both samples were subjected to some level of drug prevention programming, the external validity of the H8 findings is limited to similar youth. In turn, these findings may not be generalizable to similar-age students who have not been exposed to community and school-level drug prevention efforts.

Hypothesis 9

Discussed earlier, findings from both the 2001 and 2004 surveys evidenced little support for H9. Only one of the seven findings from the 2004

PPAAUS was supportive, while three of nine findings from the 2001 survey provided support for H9. Moreover, the sole supportive age-graded finding yielded by the 2004 data was not obtained with age-graded data from the 2001 survey. In particular, findings from the derivation subsamples indicated that *PeerAlc* was a stronger predictor of alcohol initiation among 9th graders than 6th and 12th graders. Comparatively, *PeerAlc* exerted a slightly stronger impact on alcohol initiation among 6th grade students than 9th grade students in the 2001 PPAAUS.

Although other discrepant results were uncovered in this particular cross-validation, the internal and external validity of some of the 2004 PPAAUS findings related to H9 were strengthened, since several of the 2001 PPAAUS findings reinforced the corresponding results that were obtained from the 2004 data. For example, *PeerAlc* was predictive of alcohol initiation among both the 2001 and 2004 6th and 9th grade subsamples. Results from both surveys also converged in finding that *PeerAlc* is not an important predictor of alcohol initiation among 12th grade students. Further, consistent with corresponding model findings from the 2004 PPAAUS, the 2001 data revealed that peer soft drug use and pro-drug norms tended to be more important predictors of alcohol and cigarette initiation and time to initiation among 6th grade students than 9th and 12th graders.

Two major issues related to the H9 findings are worth discussing. First, similar to the incongruent findings yielded for H8, the general lack of convergence in the 2001 and 2004 results for H9 may be a product, in part, of differential history effects (i.e., differential exposure to CTC prevention programming). Discussed earlier, history effects constitute a plausible threat to the internal validity of both the 2001 and 2004 results.

Second, by developing regression models that enabled both the direct and indirect effects of variables to be considered, the current research subjected H9 (and other main effects hypotheses) to rigorous testing. Since this hypothesis was established with insight from extant reviews of primary studies that only examined the main effects of these variables (see Chapter 4), it is not surprising that both the 2001 and 2004 converged in providing little support for this hypothesis. As data from both surveys demonstrated, the majority of effects exerted by peer drug use and prodrug norms on outcomes were indirect. While the implications of these findings for etiology and future research are discussed in the following chapter, it is important to mention here that a more conclusive determination of the validity of the 2004 PPAAUS findings for H9 will need to await the findings from further research that develops regression models capable of modeling both direct and indirect effects.

Model Fit and Distal-Proximal Mediation

Aside from comparing the 2004 and 2001 findings for H2-H9, the quantitative cross-validation involved comparing the fit of the 22 regression models that were developed with 2001 and 2004 data. An additional concern centered on the degree to which the 2001 results for Petraitis et al.'s (1995) distal-proximal mediation hypothesis converged with those from the 2004 PPAAUS. In short, the findings from both supplemental analyses provide additional evidence for the internal and external validity of the 2004 PPAAUS results.

Model Fit

Shrinkage results indicated that the amount of explained variation in the outcomes accounted for by the 2001 and 2004 predictors was similar, with a few exceptions. Discussed earlier, some of the observed incongruence in model fit may be due to the fact that three community domain variables (*EasyAlc*, *EasyCig*, and *EasyMar*) were not examined in any of the 22 regression models developed with 2001 data. As well, collinearity issues involving *AlcLate* and *AlcIntent* precluded an examination of these variables in relation to cigarette and marijuana initiation (and time to initiation) among 12th grade students from the 2001 PPAAUS.

Taking into account these differences in model development, the slight inconsistencies that were observed in this particular cross-validation likely were due to model misspecification (e.g., dissimilarities in accounting for third variables), a plausible threat to the internal validity of the 2001 results. It does not appear likely that the minor incongruence that was observed is due to differential cohort or secular effects that may have occurred during the course of the three-year time interval between survey administrations. Consequently, although shrinkage did not pose a serious problem, some of the differences in model fit appear more rooted in the disjuncture between the specification of the 2001 and 2004 regression models than in the impact that any differential cohort and secular effects may have had on the external validity of the 2004 results.

Distal-Proximal Mediation

Additional evidence for the internal and external validity of the 2004 results centers on the cross-validation findings for Petraitis et al.'s (1995) distal-proximal mediation hypothesis. Discussed earlier, not only did both surveys yield considerable support for this hypothesis, but the results from the 2001 and 2004 total samples and age-graded subsamples were strikingly similar in terms of the proportion of indirect effects that were exerted and the patterns identified across ecological domains. Findings also converged in terms of the types of outcomes and population targets that yielded low and high levels of support.

Only two threats constitute a salient concern for the internal validity of the 2004 results for distal-proximal mediation: causal ordering and history. Similar to the 2004 findings for H2-H9, causal ordering constitutes a plausible threat, since both the 2001 and 2004 data were cross-sectional in nature, and the temporal ordering of constructs was not able to be fully verified. History effects also pose a plausible threat to the internal validity of the 2004 and 2001 findings for distal-proximal mediation. Since the validity of the 2004 results were not confirmed through the systematic review cross-validation, it is not clear how generalizable the 2004 distal-proximal findings are to 6th, 9th, and 12th grade students who have not been exposed to drug prevention initiatives.

The stability in the 2001 and 2004 findings for distal-proximal mediation, however, does underscore the external validity of the 2004 results. Any changes in secular or age effects that may have occurred during the three-year time interval between survey administrations does not appear to have played a major role in impacting the degree of support for Petraitis et al.'s (1995) hypothesis. Discussed earlier, it is possible that differential secular, age, or cohort effects may have impacted the direction (H3) and magnitude (H4) of some of the regression coefficients obtained using the 2004 data. This difference in the likely impact that these threats posed for the 2004 results is not surprising, given that testing Petraitis and colleagues' distal-proximal mediation hypothesis involved assessing blocks of predictors, while the bulk of H2-H9 constituted a more fine-grained evaluation of the direction (H2 and H3) and magnitude (H3, H6, H7, and H9) of specific regression coefficients.

Major Threats to the Validity of the 2004 Results

Taking together the specific counter findings from the dual cross-validation, it is possible to draw some general conclusions regarding those threats to the validity of the 2004 results that do not pose a serious concern, and those that remain plausible. With respect to the former issue, threats that appear to have been tempered center on statistical conclusion validity and certain aspects of construct validity. In contrast, several threats to the internal, external, and construct validity of the 2004 findings, such as causal ordering, history, and the use of self-report data (among others), remain plausible.

Tempered Threats

Given that primary researchers measured extraneous variables in multiple and divergent ways, and arrived at findings using different types of analytic techniques, the systematic review cross-validation allowed for a rigorous verification of the 2004 findings for H2 and H3. The near full convergence in these respective findings not only virtually nullified the two major threats (i.e., inappropriate statistical techniques and low statistical power) to the statistical conclusion validity of the 2004 results, but this strong congruence in the results tempered the threat that any invalid or unreliable measurement of the 2004 constructs posed for the construct validity of the 2004 findings.

Under-reporting, another threat to the construct validity of the 2004 (and 2001) results, also appears to have been minimized. In response to a PPAAUS item, which asked students how confident they were that no one at their school could identify any student's questionnaire (see Appendices A and E), students reported a fairly high degree of confidence. In particular, although 11.7% (n = 88) of the 2004 sample did not respond to this survey item, 55.2% (n = 416) of the sample expressed confidence in the anonymity of students' survey responses. This compares to 9.3% (n = 70) of the sample who reported ill confidence in the anonymity of survey responses.

Interestingly, the degree of confidence that students from the 2001 sample placed in the anonymity of survey responses virtually mirrored that yielded from the 2004 sample. Not only did 99.4% (n = 719) of the sample respond to the survey item, but 68% (n = 492) of the 2001 sample indicated that they were reasonably sure that no one at their school could identify students' questionnaires. This compares to 9.3% (n = 67) of the sample who expressed ill confidence.

Finally, although still a concern, the use of self-report data does not appear to have had a considerable impact on the 2004 findings. Not only did the bulk of the findings from the systematic review cross-validation for H2 and H3 yield considerable support for respective 2004 results, but several of the primary studies whose findings were supportive obtained data from multiple sources, such as parents, peers, teachers, and official records.

Plausible Threats

Internal validity. Taken together, although the dual cross-validation findings generally support the internal validity of the 2004 results, the existence of several threats necessitates that these findings be interpreted with some caution. Major threats to the internal validity of the results include inadequate control of third variables, a drawback endemic to all etiological drug research; history effects stemming from the drug prevention initiatives that were employed in the community from which the 2004 sample was derived; and causal ordering, a limitation inherent to all cross-sectional drug research.

Although the current research provided a fairly comprehensive examination of a plethora of explanatory factors, not all third variables (e.g., personality factors) were taken into account in analyses. As well, the findings are based upon data generated from students who were present in school on the day of survey administration, and who voluntarily agreed to complete the survey. Hence, data were not gathered from adolescents who were absent on the day of survey administration, had dropped out of school prior to data collection, or who were not present in the public school for other reasons (e.g., home-schooled, placed in custodial care). Discussed in Chapter 6, although a comparison of survey response and school enrollment rates indicated that nearly all students enrolled in 6th, 9th, and 12th grade participated in the 2004 PPAAUS, and low numbers of youth in the school district were home-schooled or placed in custodial care, levels of soft drug initiation and explanatory factors may be conservative. Hence, it is possible that some of the regression coefficients are slightly attenuated. Taking these plausible circumstances into account, some care should be taken in interpreting and generalizing the 2004 PPAAUS results to same-age youth.

External validity. Touched upon throughout the previous discussion, several of the conflicting findings that were identified through the quantitative cross-validation appear to be the result of secular, cohort, or age effects. Although it appears that the bulk of the 2004 findings for H2 and H3 were not impacted by these factors, some of the conflicting age-graded findings for H4-H9 may have been impacted by these types of time period effects. Hence, it is likely that secular, cohort, and age effects, coupled with the plausibility of differential history effects, constitute salient threats to the external validity of some of the 2004 results.

Construct validity. With respect to construct validity, the discussion of the conflicting findings from the dual cross-validation underscored three threats that remain plausible: forward telescoping, lengthy period of recall, and recall decay. Forward telescoping and lengthy period of recall remain plausible threats to the construct validity of the 2004 (and 2001) findings for predictors of time to soft drug initiation, particularly among older adolescents.

Indications of forward telescoping emerged when the descriptive findings for time to soft drug initiation among the age-graded subsamples were assessed. For example, compared to 6^{th} grade students from the 2001 PPAAUS, who self-reported 10 years as the average age at which they began alcohol use, on average, 9^{th} and 12^{th} graders from the 2001 sample self-reported beginning alcohol use at 12.4 and 14.5 years, respectively. This same pattern of self-reported ages of initiation was observed for cigarette and marijuana initiation among 9^{th} and 12^{th} grade students from the validation sample, as well as the ages of soft drug initiation reported by 6^{th}, 9^{th}, and 12^{th} grade students from the 2004 PPAAUS.

The impact that forward telescoping and lengthy recall periods had on the relationships between predictors and time to soft drug initiation were not able to be adequately assessed within the systematic review cross-validation. Not only were the majority of the variables examined within the context of the current research not evaluated in the two primary studies (Hawkins et al., 2002b; Unger & Chen, 1999) that examined predictors of time to soft drug initiation, but these two studies assessed time to soft drug initiation among samples of youth who ranged in age from early-late adolescence.

As a precursor to forward telescoping, and a consequence of lengthy recall period, recall decay also remains a plausible threat to the construct validity of the 2004 findings for predictors of time to soft drug initiation. Due to a longer recall period on the part of 12^{th} grade students, compared to 6^{th} and 9^{th} grade students, it is likely that recall decay and forward telescoping constitute more serious threats to the construct validity of the results for predictors of time to soft drug initiation among 12th graders than 6th or 9th graders.

Conclusion

The purpose of this chapter was to present the dual cross-validation results. In providing a rigorous assessment of the internal, external, statistical conclusion, and construct validity of the 2004 PPAAUS findings, the multivariate results were compared to the findings from a systematic review of the extant literature and the multivariate findings from the 2001 PPAAUS.

The systematic review cross-validation provided for a broad-based assessment of the internal and external validity of the 2004 findings for H2 and H3. Supplemental attention also was directed at assessing plausible threats to the construct and statistical conclusion validity of these results. The quantitative cross-validation constituted a more fine-grained evaluation of the internal and external validity of the 2004 findings. Through this assessment, the 2004 PPAAUS results for H2-H9, as well as Petraitis et al.'s (1985) distal-proximal mediation hypothesis, were compared to the corresponding findings from the 2001 PPAAUS. Further, in an effort to evaluate the fit of the 2004 prediction models, shrinkage estimates for each of the 22 regression models were obtained.

In general, this comprehensive dual cross-validation yielded considerable support for a majority of the 2004 findings. Nearly all of the H2 and H3 findings from the 2004 PPAAUS converged with those from both the systematic review cross-validation and the quantitative cross-validation. In general, the majority of the quantitative cross-validation results for H4-H9 also converged. Data from both PPAAUS surveys provided partial and complementary support for H4-H5, although the validation sample garnered less support for H6-H8 than did the derivation sample. In contrast, the 2001 PPAAUS yielded more support for H9 than did the 2001 PPAAUS. Finally, the quantitative cross-validation evidenced a strong convergence in the 2001 and 2004 results for model fit and distal-proximal mediation.

Identifying the various inconsistencies between the 2004 results and findings from the systematic review and the 2001 PPAAUS provided a means by which the validity of the 2004 findings could be assessed. Threats to the statistical conclusion validity of the 2004 results appear to have been minimized. It also appears that the threats that invalid or unreliable measurement, underreporting, and the use of self-report data pose for the construct validity of the results are tempered.

Since several threats do remain plausible, however, the 2004 findings should be interpreted and generalized with some caution. Major threats to the internal validity of the 2004 results include inadequate account of all third variables, history, and causal ordering. The threat posed by causal ordering is minimized, though, given the fairly strong convergence in findings yielded from the systematic review cross-validation. Discussed earlier, the majority of the primary studies included in the review arrived at results using a longitudinal, prospective research design. Some of the 2004 findings also may have been impacted by secular, age, and cohort effects, three types of time period effects that constitute plausible threats to the external validity of the results. Finally, with respect to construct validity, the impact of forward telescoping, lengthy period of recall, and recall decay on findings, particularly those pertaining to time to soft drug initiation among 12th grade students, also remain plausible.

Taking several interrelated approaches, the following chapter puts the findings of the current research into perspective, by placing the results within the larger context of prevention science. Discussed are the multiple contributions that the research makes to the knowledge base, as well as the resultant implications that the findings have for traditional and developmental theory, etiological research, and primary drug prevention policy. The study limitations also are discussed.

CHAPTER 11

DISCUSSION AND CONCLUSIONS

Due to the potential negative consequences that adolescent drug use poses for physical, mental, and cognitive health (Cha et al., 2006; OAS, 2002a; White & Swartzwelder, 2005), as well as for social and economic well-being (Hingson & Kenkel, 2004; Midanik & Greenfield, 2000; ONDCP, 2004), an extensive amount of research has been conducted on the issue (see, e.g., Allen et al., 2003; Hastings et al., 2005; Schaps et al., 1981). In recent years, increasing etiological research has been directed at predictors of patterned soft drug use among youth, as well as abuse and addition, rather than risk and protective factors for initial soft drug initiation among this population (see, e.g., Austin, Macgowan, & Wagner, 2005; DiFranza et al., 2007; Masterman, Hons, & Kelly, 2003; Norman & Turner, 1993; Windle et al., 2008; Zucker, Donovan, Masten, Mattson, & Moss, 2008).

This disjuncture in focus may be rooted in a combination of changing social factors. Some of these factors include an increase in the social acceptance of alcohol and marijuana use by many American adults (see, e.g., Johnson, 1996; Kessler, 2005, Kleiman, 1998; Ripple & Zigler, 2003; Toomey & Wagenaar, 1999); an increase in both apathetic responses to, and a tolerance of, health-risk behaviors among juveniles on the part of parents, the media, and society in general (see, e.g., AMA, 2005; Grube, 2004; OAS, 2002b; Hwang, 1995; Office of Substance Abuse Information and Resource Center, 2003; Santa Cruz County Board of Supervisors, 2002; Solmitz, 2000; Wrenn, 2006); a decrease in attention to the primary prevention of alcohol and cigarette use, in favor of an emphasis on secondary and tertiary drug prevention programming (Pentz, 1996); and an increase in society's acceptance of drug policies founded on harm reduction (MacCoun & Reuter, 2001; Osher, 2005).

However, the fact that regular use and the abuse of (and dependence on) soft drugs is not possible without first initiating these substances justifies the continued study of predictors of alcohol, cigarette, and marijuana initiation among adolescents (Gfroerer & Epstein, 1999; Grant & Dawson, 1998; Robins, 1980). Indeed, as aptly noted by leading researchers in the field of prevention science (see, e.g., Hawkins et al., 1992a; Kandel, 2002; Jenson, 2004; Robins & Przybeck, 1990; Wills et al., 2004), identifying these explanatory factors constitutes an important first step in preventing (or reducing) adolescent soft drug initiation and the multiple negative outcomes that can result. It is within this framework of primary prevention that the current research was established.

In providing a comprehensive investigation of alcohol, cigarette, and marijuana initiation among adolescents, the overarching goals of the research were to build upon extant research findings, address important gaps in the literature, and contribute to the prevention science knowledge base. A four-fold purpose founded the research: 1) provide a systematic review of the soft drug initiation literature; 2) test a modified version of Kandel's (2002) drug sequencing hypothesis; 3) determine if predictors of soft drug initiation differ in kind or marked saliency by stage of adolescent development and drug type; and 4) examine the timing of soft drug initiation by stage of adolescent development and drug type, in terms of predictors that distinguish early versus later ages of initiation. Supplemental attention also was directed at evaluating the utility of Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

Using psychosocial data from a rural sample of 6th, 9th, and 12th grade students who completed the 2004 PPAAUS, a total of nine hypotheses were tested. In an effort to gauge the validity of the findings for H2-H9 and distal-proximal mediation, a dual cross-validation scheme was employed. The systematic review cross-validation involved comparing the quantitative findings for H2-H3 to those yielded from the systematic review of the literature. Through the quantitative cross-validation, the 2004 PPAAUS findings for H2-H9 and distal-proximal mediation were compared to those from the 2001 PPAAUS.

In the sections that follow, the more general contributions of the research are framed in terms of the literature gaps that the study addressed. Key findings then are discussed within the broader context of traditional and developmental theory, the etiological research base, and adolescent drug prevention programming and policy. In the end, the limitations of the research are presented, and recommendations for future research are offered.

General Contributions of the Research

Systematic Review

The first line of inquiry involved conducting a systematic review of the extant literature on soft drug initiation and time to initiation among adolescents. An assessment of extant reviews published between the 1970s and the 2000s revealed that the majority have not focused on (nor attended to) issues that are empirically important to the field of prevention science today (e.g., drug-specific and age-specific risk factors). The current study addressed this inadequacy by using Research Questions #2 and #3 as a guiding framework for systematically assessing the directional findings from 36 empirical studies that met the inclusion criteria set forth in Chapter 5.

Conducting this systematic review not only updated the prevention science knowledge base concerning age- and drug-specific risk and protective factors for soft drug initiation and time to initiation, but through the process of the investigation, a number of important gaps in the empirical literature were identified. Presented later in this chapter, voids worthy of redress are framed in terms of suggestions for future research.

Sequencing

In addressing Research Question #1, a modified version of Kandel's (2002) drug sequencing hypothesis (H1) was tested. In particular, adolescent initiation of alcohol, cigarette, and marijuana use was examined in terms of the degree to which each constitute a distinct stage that together captures a latent continuum of drug involvement. In testing H1 (alcohol>cigarette>marijuana initiation), the current study made three general contributions to the literature.

First, there is some empirical ambiguity with respect to the sequencing of alcohol and cigarette initiation. Some studies have found that cigarette initiation precedes alcohol initiation, while other research obtained the opposite results (see, e.g., Brook, 1993; Donovan & Jessor, 1983; Huba et al., 1981). The current study aimed to provide some clarity on this issue.

Second, through a review of 30 prior investigations of soft drug sequencing, it became evident that the bulk of the extant research that used Guttman scalogram analysis did not ensure the temporal ordering of polydrug initiation. Discussed in Chapter 4, Guttman scalogram analysis only infers temporal ordering in drug initiation sequences. Since the element of time clearly is evident in Kandel's hypothesis, the current research addressed this literature gap, by utilizing age of initiation data in testing H1.

Finally, due to little or no research on the issue, the current study also considered whether differential measurement has an impact on the fit of a given soft drug sequence. Specifically, in addition to testing H1 with age of initiation data, dichotomous initiation data also were used in testing H1. The fit of these respective scales then were compared.

Prediction

In addressing Research Questions #2 and #3, the third and fourth lines of inquiry were informed by two traditional theories (social control theory and social learning theory) and one developmental theory (SDM). The third area of inquiry centered on assessing predictors of soft drug initiation among the total sample and three age-graded subsamples (6^{th}, 9^{th}, and 12^{th} grade students). Aside from testing H2-H9, efforts were directed at determining whether predictors are drug- and age-specific.

Few or no prior cross-sectional studies have simultaneously compared risk factors by drug type and biological age. More often than not, either composite measures of soft drug initiation have been used (Chassin, 1984);

results have not been compared by drug type when examining the age-varying nature of predictors (Allen et al., 2003; Glynn, 1981); or comparisons have made across soft drugs, but age effects have been controlled (see Allen et al., 2003; Glynn, 1981; Roosa, 2000; Vitaro et al., 2004).

Since research is mixed with regard to common versus drug-specific risk factors, this aspect of the study contributed to the knowledge base by providing some insight into the degree to which equifinality exists and the common influence hypothesis holds for the initiation of different types of soft drugs. With respect to age-specific predictors, far less research has examined the age-graded nature of relationships between predictors and soft drug initiation than has assessed determinants of initiation with data from samples of youth who vary on age. In fact, other than expressing the need for further research, few of the extant literature reviews examined were able to offer substantive direction, insight, or expectations that could be used to found the basis of H2-H6. Hence, testing these hypotheses with the age-graded subsamples constituted an attempt to fill this glaring gap in the literature.

The final major area of inquiry centered on examining soft drug initiation in terms of the timing at which it occurs. Similar to the third line of inquiry, H2-H9 were tested, and consideration was paid to the drug- and age-specific nature of predictors. Due to the limited attention that past research has paid to time to soft drug initiation, particularly with respect to the age-graded nature of predictors, the current study approached this aspect of the research from a more exploratory standpoint.

Addressing Explicit Research Calls

Brought to the forefront in Chapter 4, adolescent drug researchers have called for the rectification of a number of important gaps in the prediction literature. In answering these respective research calls, the prediction component of the current research made multiple contributions to the prevention science knowledge base.

In investigating a plethora of factors from all five ecological domains of influence (Donovan, 2004; USDHHS, 2007), the current research took a comprehensive approach to predicting soft drug initiation among rural youth, an understudied population (Burt et al., 2000; Farrington & Coid, 2003). As was identified through the discussion of the prediction literature (Chapter 4), as well as the findings from the systematic review (Chapter 7), not many empirical studies have provided a comprehensive examination of predictors of alcohol, cigarette, and marijuana initiation and time to initiation among adolescents.

The current study also answered research calls for more investigations on the following fronts: determinants of age of soft drug initiation, particularly early age of initiation (Donovan, 2004; Hawkins et al., 2002b; Petraitis et al., 1998); drug-specific risk factors for alcohol, cigarette, and marijuana initiation (Flay & Petraitis, 1991; Kandel, 2002); the direct (Farrington & Coid, 2003) and mediating (Donovan, 2004) effects of risk and protective factors; and the importance that community drug norms, the mass media, and school teachers have for influencing the soft drug initiation behavior of youth (see, e.g., Allen et al., 2003; Donovan, 2004; Petraitis et al., 1998; Wakefield et al., 2003; Wills et al., 1996).

Finally, the current study answered Petraitis et al.'s (1998) research call, by systematically grounding the research in theoretically-derived hypotheses.

Distal-Proximal Mediation

Petraitis et al. (1995) contend that in main effects models, risk factors conceptualized as distal in nature (e.g., school and community domain risk factors) may be less salient predictors of outcomes than more proximal risk factors (e.g., family, peer, and individual domain risk factors), because the effects of distal predictors likely are mediated by the more proximal influences. Although advances have been made in recent years, on balance, much more research has examined the direct effects of risk and protective factors than has investigated the mediating role that risk and protective factors have for reducing the relationships between other predictors and soft drug initiation (Petraitis et al., 1998). Even less research has determined if (and how) mediating processes vary by biological age and drug type (Dishion et al., 1999; Wills et al., 1996).

Given that little or no prior research has provided an explicit test of distal-proximal mediation, the current study contributed to the existing knowledge base by addressing this literature gap. Reinforced by the 2001 PPAAUS results, the findings provide strong evidence for Petraitis and colleagues' contention, while complementing the results from past investigations of the differential saliency of ecological domains and specific mediation pathways (see, e.g., Bauman & Ennett, 1996; Biddle et al., 1980; Conrad et al., 1992; Derzon & Lipsey, 1999c; Dewey, 1999; Donovan, 2004; Engels & Bot, 2006; Mounts, 2004). Moreover, the supportive results further underscore the need for theory to account for causal chains and the distal and proximal risk and protective factors to which these mediation pathways are comprised, the direct effects and mediating capacity of peer and individual domain variables, and the drug- and age-specific nature of mediating processes.

Theoretical and Empirical Implications

The specific implications for theory and the attendant etiological knowledge base reside in the key findings for H1-H9, age- and drug-specific risk factors, and specific aspects of the distal-proximal mediation results. Differences found in predictors of soft drug initiation versus time to initiation also have theoretical and empirical value.

Stage Theory

Sequencing

The supportive findings for H1 speak to several theoretical and empirical issues. On the theoretical front, the results provide evidence for the internal validity of Kandel's sequencing proposition, in finding that the initiation of legal drug use tends to occur prior to marijuana initiation. Further, the H1 findings converged with those from four longitudinal, prospective studies (Andrews et al., 1991; Ellickson et al., 1992; Hawkins et al., 2002b; Kandel, 1975b), in indicating that alcohol initiation typically occurs prior to cigarette initiation for most adolescents who initiate legal drug use. In turn, cigarette initiation tends to occur prior to marijuana initiation among those adolescents who initiate marijuana use. Since these results are based upon soft drug initiation data from a sample of rural youth, and most investigations of the utility of Kandel's sequencing hypothesis utilized data from suburban and urban adolescents (see Chapter 4), the H1 findings also provide evidence for the external validity of this proposition.

On the empirical front, the results from the supplemental Guttman analysis also supported H1; however, the values for CR and CS were substantially higher than those for the initial Guttman scale developed with age of soft drug initiation data. When the age of initiation data were utilized, the fit of the Guttman scale was acceptable; however, due to a larger number of errors, the fit of this scale was not as strong as that of the Guttman scale developed with the dichotomous initiation data. Since the temporal ordering inferred by a Guttman scale only implies, but does not necessarily prove, that temporal ordering in the initiation of multiple drugs exists, using the age of initiation data provided for a more stringent test of H1.

The differing CR and CS coefficients that were produced when using dichotomous versus age of initiation data calls into question the use of the former in Guttman scalogram analysis, while underscoring the impact that measurement can have on the scalability of the drug sequence being investigated. Given that nearly all previous tests of Kandel's (2002) drug sequencing hypothesis used dichotomous initiation data in modeling drug sequences, the differential scale fit obtained suggests that Kandel's proposed drug sequence may not fit a valid Guttman scale quite to the extent it is assumed in the literature.

Prediction

Discussed in Chapter 3, the specific risk factor and common factor hypotheses have been put forth in an attempt to explain both the ordering of alcohol, cigarettes, and marijuana initiation (H1) and progression along the sequence. Empirical support for the prediction component of stage theory may be found in findings that attribute the initiation of alcohol, cigarettes, and marijuana use to unique (or drug-specific) risk factors (Kandel, 2002), while support for the common factor hypothesis lies in research that demonstrates the universality (but differential saliency) of risk factors for the initiation of all three soft drugs (Jessor, 1992). Although full tests of either hypothesis require longitudinal data, the current research provided a partial assessment of the validity of these hypotheses, by comparing risk factors for soft drug initiation by biological age and drug type. In a further attempt to yield some insight into the association between alcohol, cigarette, and marijuana initiation (H1), H6[24] was tested.

With the exception of marijuana initiation among 12th grade students, H6 yielded full support. Although the findings from the 2001 PPAAUS did not fully converge with those from the current research, as discussed in Chapter 10, it is likely that the disjuncture in results is rooted in differential model specification. The supportive findings are reinforced, however, by the favorable results from research that has tested this hypothesis while holding biological age constant (see, e.g., D'Amico & McCarthy, 2006; Duncan & Duncan, 1994; Duncan et al., 1998; Ellickson et al., 1992; Flay et al., 1998; Kandel et al., 1992; Yamaguchi & Kandel, 1984b).

[24] Relative to alcohol initiation, cigarette initiation is a stronger predictor of marijuana initiation and age of initiation.

Hence, taking into account the generally supportive findings for H6, while considering the magnitude and drug-specific effects that frequent alcohol and cigarette use demonstrated in the multivariate models, it was possible to gain further insight into the existence of H1. In general, early age of alcohol initiation and frequent alcohol use were salient predictors of cigarette initiation and time to initiation, while both frequent cigarette use and early age of cigarette initiation were predictive of marijuana initiation and time to initiation. Although frequent alcohol use also was a somewhat important determinant of marijuana initiation and time to initiation, this variable exerted weaker effects on marijuana initiation and time to initiation than frequent cigarette use. Early age of alcohol initiation was not an important determinant of marijuana initiation outcomes.

This set of findings reinforces the important role of cigarette initiation in the H1 sequence, while underscoring the utility of alcohol initiation (particularly early age of initiation) and frequent alcohol use for explaining progression to cigarette initiation, and the importance of cigarette initiation (especially early age of initiation) and frequent cigarette use for explaining progression from cigarette to marijuana initiation. Beyond this insight, however, these findings provide limited explanation for the ordering and hierarchical properties of soft drugs in the H1 sequence. Compared to the alcohol and cigarette frequency of use measures, alcohol and cigarette age of initiation consistently were stronger predictors of the initiation of the substance in the next stage of the H1 sequence. Although age of initiation (and to a lesser extent, frequency of use) tends to constitute a “red flag” or risk marker for future antisocial or problem behavior (Kraemer, Kazdin, Offord, Kessler, Jensen, & Kupfer, 1997), this variable hints at, but does not reveal, changes in environmental and intrapersonal factors that influence adolescent initiation of one soft drug over another.

Drug-specific factors. The cross-sectional nature of the data precluded an investigation of changes in extraneous factors over time in predicting progression in the H1 sequence. However, the multivariate findings are reinforced by the longitudinal results from Hawkins et al. (2002b) in providing more evidence for the specific risk factor hypothesis than the common factor hypothesis. Perhaps more importantly, the results from the current research and Hawkins and colleagues' study converge in pinpointing the nature and saliency of many of these drug-specific factors.

Counter to research results on age-graded differences in the saliency of social control and social learning variables (see, e.g., Allen et al., 2003; Beal et al., 2001; Dewey, 1999; Donovan, 2004; Urberg et al., 1991), no one social control, social learning, or SDM variable was a universal predictor of soft drug initiation or time to initiation among the total sample and the three age-graded subsamples. Among the total sample, however, three variables were found to exert nearly universal effects on alcohol, cigarette, and marijuana initiation and time to initiation: strong attachment to school (*AttachSchool*), frequent involvement in religious activities (*Religious*), and frequent cheating on schoolwork (*Cheat*). Aside from these few exceptions, predictors were found to differ more in kind by drug type than saliency.

Four major direct effects findings from the total sample models are particularly instructive in demonstrating the drug-specific nature of many of the social control, social learning, and SDM constructs. These results also provide some insight into several characteristics that distinguished the alcohol, cigarette, and marijuana initiates. First, compared to cigarette and marijuana initiates from the total sample, alcohol initiates tended to engage in more problem behaviors, particularly truancy (*Truancy*), cheating on schoolwork (*Cheat*), and stealing (*Steal*). Frequent involvement in social activities (*Social*) and peer alcohol use (*PeerAlc*) also were predictive of alcohol initiation among the total sample, but not cigarette or marijuana initiation.

Second, the only substantive commonality between alcohol and marijuana initiates from the total sample was low involvement in religious activities (*Religious*). High levels of this social control (Hirschi, 1969) and SDM variable served as a salient protective factor for the initiation of both soft drugs. This construct also delayed time to alcohol initiation, but not time to marijuana initiation.

Third, many more SDM, social learning, and social control constructs were associated with alcohol initiation than cigarette initiation. In fact, the only direct determinants of cigarette initiation among the total sample included three (*AlcEarly*, *CigIntent*, *AlcIntent*) individual domain variables, and one (*HiAcadPerf*) school bonding variable. Earning good grades in school was important for reducing risk and delaying age of cigarette initiation, but school grades had no direct bearing on risk for alcohol and marijuana initiation, or the ages at which these substances were initiated.

Finally, compared to alcohol and cigarette initiates, the behavior of marijuana initiates in the total sample was predominantly influenced by the drug-related norms espoused by important others. Unexpectedly, tolerant media norms about cigarette use and tolerant alcohol norms espoused by peers served important protective functions in reducing risk for marijuana initiation. These findings are discussed further in a following section.

Consistent with results from a number of longitudinal studies (Hawkins et al., 2002b; Kandel et al., 1978; Kosterman et al., 2000), the most convincing support for Kandel's (2002) notion of drug-specific risk factors resides in two types of interrelated findings from the current research: 1) drug-specific differences in the frequencies of responses to survey items that measured media, teacher, parent, and peer tolerance of alcohol, cigarettes, and marijuana use; the degree of willingness to use these respective drugs; peer use of these substances; and the availability of these drugs; and 2) the significance and magnitude of effects that these variables exerted in the multivariate models.

Briefly noted in Chapter 8, the ordering of the soft drugs in the H1 sequence mirrored the differences in the degree to which the total sample perceived that: 1) each drug was easy to access (*EasyAlc*, *EasyCig*, *EasyMar*); 2) the media (*MessMedAlc*, *MessMedCig*, *MessMedMar*), teachers (*MessTchrAlc*, *MessTchrCig*, *MessTchrMar*), parents (*MessParAlc*, *MessParCig*, *MessParMar*), and peers (*MessPeerAlc*, *MessPeerCig*, *MessPeerMar*) were tolerant of the use of each substance; and 3) peers were using these drugs (*PeerAlc*, *PeerCig*, *PeerMar*). The ordering of these substances also paralleled differences in students' intentions (*AlcIntent*, *CigIntent*, *MarIntent*) to use these respective drugs. In particular, on average, the total sample perceived that teachers, parents, peers, and the media were more tolerant of alcohol use than cigarette or marijuana use, while students reported that these agents of social learning and control were more tolerant of cigarette use than marijuana use. Students also felt that marijuana was more difficult to access than alcohol and cigarettes, more peers drank alcohol than smoked cigarettes or marijuana, and fewer peers smoked marijuana than cigarettes. Students also were more willing to use alcohol than cigarettes or marijuana, but were less willing to use marijuana than cigarettes.

Similar descriptive findings also were found by Hawkins and colleagues (2002b), in their longitudinal study of soft drug sequencing and the impact of parental, peer, and adolescents' soft drug norms on time to alcohol, cigarette, and marijuana initiation. Not only do their sequencing results converge with the findings from the current study in providing support for H1, but their descriptive results indicate that the ordering of soft drugs mirrors the perceptions that parents, peers, and adolescents, themselves, hold concerning the acceptability of these respective substances.

Many of the drug-specific findings from the total sample models also coalesce with Hawkins and colleagues' multivariate results. Both the current research and Hawkins et al.'s study found that the impact of parental and peer soft drug norms tends to be drug-specific, by only predicting initiation and time to initiation of the soft drug to which these norms correspond. In going beyond the scope of Hawkins and colleagues' research, by investigating adolescents' soft drug intentions and perceived soft drug availability, as well as peer alcohol, cigarette, and marijuana use, the current research found that these variables also exerted drug-specific effects in the total sample. For example, *AlcIntent* had an important impact on the initiation and age of initiation of alcohol and cigarette use among the total sample, but not marijuana initiation or time to initiation.

Considering the general convergence in Hawkins et al.'s (2002) results and these key findings from the current research, it appears reasonable to conclude that the ordering of alcohol, cigarette, and marijuana initiation in the H1 sequence may be explained by differences in broader social norms (e.g., norms espoused by the media) concerning the acceptability of each drug. The ordering also may be a consequence of parallel differences in the proscriptive drug-specific norms to which adolescents are exposed in their immediate social environment (e.g., home, school, peers, and the local community). In moving beyond the scope of Hawkins et al.'s research, the current study makes an additional contribution to the literature base in underscoring the possibility that the ordering of soft drugs in the H1 sequence also may be an artifact of the drug-specific nature of peer alcohol, cigarette, and marijuana use and adolescents' soft drug intentions, as was identified in both the total sample and age-graded models. The dual risk and protective nature of these drug-related social learning variables are addressed below, within the context of social learning theory.

Drug- and age-specific factors. The drug-specific nature of the variables discussed above was identified in the total sample models. In reinforcing many of the total sample findings, additional evidence for the specific risk factor hypothesis was yielded from the age-graded soft drug initiation and time to initiation models. As was found in the total sample models, the age-graded findings revealed that across soft drugs, outcomes, and grade-level, the soft drug intention variables continued to exert strong drug-specific effects. In fact, regardless of grade-level, the soft drug intention variables generally constituted the most salient predictors of soft drug initiation and time to initiation. Many of the social learning, SDM, and social control variables that exerted drug-specific effects in the total sample models also continued to exert similar effects in the age-graded models. Further, decomposing the total sample by grade-level enabled a number of age- and drug-specific factors to be revealed.

The subsample findings for alcohol initiation are particularly insightful in this regard. The individual domain factors (i.e., *Religious*, *Steal*, *ViolBeh*) played a more important role in alcohol initiation among 6th graders than 9th and 12th graders. In contrast, peer alcohol use (*PeerAlc*) exerted a larger impact among 9th than 6th grade students, while no peer variables were associated with alcohol initiation among 12th grade students. With respect to the family domain, parental alcohol norms (*MessParAlc*) only were important for 9th graders. While school-related risk and protective factors were moderate predictors of alcohol initiation among 9th grade students, school factors played a minute role in alcohol initiation and time to initiation among early and late adolescents. Finally, the community domain predictors had no impact on alcohol initiation or time to initiation among 9th grade students. In contrast, easy access to alcohol (*EasyAlc*) constituted a risk factor for the alcohol initiation behavior of 6th graders, while media tolerance of alcohol use (*MessMedAlc*) was predictive of alcohol initiation among seniors.

While these alcohol-related findings constitute a sampling of the many age- and drug-specific predictors that were identified in the current research, this illustration, coupled with the drug- and age-specific results for cigarette and marijuana initiation and time to initiation, and the potential age-factor interaction effects that were observed, further underscores the level of support that was yielded for Kandel's (2002) specific risk factor hypothesis.

Social Control Theory

Within the context of the current research, the utility of social control theory (Hirschi, 1969) lies in the findings for the direction (H3) and nature of the effects exerted by the eight (*CllgeAsp*, *AttachSchool*, *Academic*, *HiAcadPerf*, *Sports*, *Work*, *Social*, *Religious*) bonding variables that were investigated. In short, considering the results, as well as the support yielded for several aspects of social learning theory and SDM, the findings converged with those from past research (Akers & Cochran, 1985; Johnson, 1984; Krohn & Massey, 1980) in underscoring the important, but partial explanation of soft drug initiation that Hirschi's original statement of social control articulates. Aside from calling into question the generality of the theory, the results suggest that the strengths of social control theory may be rooted more in the directional propositions that

Hirschi (1969) set forth than the direct effects that the elements of the social bond are purported to exert.

Directional Effects Propositions

With few exceptions, the protective effects (H3) posed by the eight social bonding variables coincided with the directional effects propositions set forth in social control theory (Hirschi, 1969). However, more support was yielded for the bonding elements of attachment, commitment, and belief, than the element of involvement. Among the total sample, for example, attachment (*AttachSchool*), commitment (*CllgeAsp*, *HiAcadPerf*), and belief (*Religious*) variables generally served important protective functions in reducing risk for soft drug initiation and delaying time to initiation. Reinforced by findings from past reviews of the research (Meschke & Patterson, 2003; Swadi, 1999), as well as relevant results from the systematic review, the protective effects exerted by the school bonding variables underscore the importance that increasing school attachment and commitment has for reducing risk and delaying soft drug initiation.

Consistent with past research on delinquency (see, e.g., Agnew, 1991a; Hawden, 1999; Hindelang, 1973; Rankin, 1976; Wiatrowski et al., 1981), the protective effects exerted by the involvement variables (*Work*, *Social*, and to a lesser extent, *Academic*) yielded considerably less support for the directional propositions set forth in the theory than the protective impact of the other bonding variables discussed above. Worthy of comment are the counter findings for the risk effects exerted by *Work* and *Social*.

Parallel to a number of findings from past research on the impact that adolescent involvement in work has on delinquency and alcohol and marijuana use (Agnew, 1986; Heimer, 1995; Ploeger, 1997; Wright, Cullen & Williams, 1997; Wright, Cullen, & Williams, 2002), and reinforced by relevant H3 results from the 2001 PPAAUS, the current research found that involvement in work-for-pay (*Work*) activities tends to increase risk and lower age of soft drug initiation. Ploeger's (1997) longitudinal findings based on data from the National Youth Survey are particularly insightful. After controlling for prior delinquency, employment (measured in terms of work-for-pay status in the past year) was found to increase risk for delinquency and drug use, by increasing adolescents' exposure to delinquent peers.

Other research indicates that extensive involvement in the labor market on the part of adolescents leads to reduced involvement and commitment to school, decreased school performance, and a reduction in time spent with family, all of which increase risk for delinquent behaviors, including drug use (see, e.g., Bachman & Schulenberg, 1993; Greenberger & Steinberg, 1986; Steinberg & Dornbusch, 1991). Considering this body of research, it is reasonable to assume that any one of these mediating processes may more fully explain the risk effects that *Work* tended to exert in the current study.

Reinforced by results from the 2001 PPAAUS, the findings from the current research also contributed to the prevention science knowledge by underscoring the age- and drug-invariant risk effects that involvement in social and entertainment activities (*Social*) exert on soft drug initiation and time to initiation. As evidenced through the systematic review, and an assessment of prior reviews of the literature, little or no prior research had investigated the directional effects posed by this bonding (and SDM) construct. In the current study, frequent involvement in social and entertainment activities not only increased risk for alcohol initiation among the total sample, and cigarette and marijuana initiation among 9th grade students, but *Social* served to lower ages of alcohol and cigarette initiation among 6th graders, as well as lower age of alcohol initiation among 12th grade students. To the extent that the mediating processes that prior research evidenced for *Work* also apply to *Social*, the risk effects exerted by involvement in social activities may be explained by any number of changes that such involvement incurs for levels of school and familial bonding, differential association, and differential reinforcement.

Direct Effect Propositions

Some of the specific findings for distal-proximal mediation have implications for the direct effects propositions set forth by Hirschi (1969). Discussed in Chapter 2, Hirschi hypothesizes that a direct relationship exists between each element of the social bond and delinquency. Reinforced by the results from the 2001 PPAAUS, the findings from the current research level a heavy discount against this proposition. The social bonding variables tended to exert indirect effects on outcomes, particularly the attachment and commitment constructs. For example, when school attachment (*AttachSchool*) exerted important protective effects, the respective impacts on all outcomes were indirect, with the exception of time to cigarette initiation among 6th grade students (where the impact was direct). Further, when *CllgeAsp*, a measure of school commitment, demonstrated important protective effects, the impact of these effects tended to be mediated by more proximal factors. Of the distal-proximal mediation findings that did support Hirschi's (1969) direct effects propositions, the bulk centered on cigarette initiation and time to alcohol initiation among 12th grade students, and time to cigarette initiation among 6th graders. In these models, *HiAcadPerf*, *CllgeAsp*, and *AttachSchool* did exert direct effects.

Drug- and Age-Specific Predictors

Finally, the age- and drug-specific nature of the school bonding variables calls into question the generality of social control theory, while offering some support for the age-graded models detailed in SDM. In short, the importance of school bonding in reducing risk for soft drug initiation and delaying time to initiation generally waned as grade-level increased. In exerting mostly direct effects, the school bonding variables constituted important protective factors for soft drug initiation and time to initiation among 6th grade students. Among 9th and 12th grade students, however, the school bonding variables served as relatively less important determinants of outcomes, although strong bonding served more important protective functions among 9th than 12th grade students.

The age-graded nature of the school bonding variables became more clear-cut once the impact of these constructs on the soft drug initiation behavior of seniors was examined. Generally reinforced by the results from the 2001 PPAAUS, levels of school attachment (*AttachSchool*, *HiAcadPerf*), commitment (*CllgeAsp*), and involvement (*Academic*) among 12th graders had no significant bearing on risk for alcohol and marijuana initiation, or the ages at which marijuana use was initiated.

Social Learning Theory

The bulk of the variables examined in the current research coincide with social learning theory's (Akers, 1977) constructs of differential association (e.g., peer soft drug use) and differential reinforcement (e.g., soft drug tolerance and intentions to use soft drugs). Taking into account the findings for H2, H4, H8, and H9, coupled with the age-graded nature of some of the social learning constructs and the utility of the social control and unique SDM variables that were investigated, the results reinforce those from past research (see, e.g., Akers & Cochran, 1985; Brown et al., 2005; Derzon & Lipsey, 1999a, 1999c; Donovan, 2004; Hwang & Akers, 2003; Kandel & Davies, 1991; Winfree et al., 1989). This underscores the limited, but important, utility that social learning theory (Akers, 1977) has for explaining soft drug initiation among youth.

In short, more support again was yielded for the directional propositions outlined by the theory than the magnitude hypotheses that were tested. Moreover, in underscoring the validity of SDM, the age-specific nature of some of the social learning variables calls into question the purported generality of social learning theory. Further, not only did peer soft drug use and the prodrug norms espoused by the media, parents, and peers tend to exert drug-specific effects, but the direction of these effects often countered the directional expectations outlined in the theory.

Drug-Specific Directional Effects

Given that the H2 counter findings centered on the protective effects exerted by six of the 18 social learning variables, [25] partial support was yielded for the directional propositions outlined in social learning theory. These counter results constitute one of the key findings of the research, particularly since the unexpected directional effects were exerted by drug-related variables (i.e., *MessMedAlc*, *MessMedCig*, *MessParCig*, *MessPeerAlc*, *PeerAlc*, *AlcIntent*, and *CigIntent*) whose risk and protective impacts were contingent upon the soft drug in question. As a parallel finding, a couple of other drug-related variables (e.g., *EasyAlc*) also exerted drug-specific, protective effects.

Taken together, these findings suggest that, instead of operating in a generic fashion, these drug-related predictors may demonstrate dual functions, whereby they increase risk for (and lower age of) the initiation of the soft drug to which they correspond, but exert protective effects in reducing the likelihood (and delaying the age) of other soft drug initiation. Interestingly, these variables tended to exert protective effects for the initiation and time to initiation of the next soft drug in the H1 sequence. For example, *AlcIntent* served as a salient risk factor for alcohol initiation among the total sample and 12th grade students, but demonstrated important protective functions in reducing risk for cigarette initiation and lowering age of initiation among these same samples. Further, *CigIntent* reduced risk and delayed age of marijuana initiation among the 12th grade

[25] Three variables (*AlcIntent*, *CigIntent*, *MarIntent*) served as proxy measures for perceived rewards stemming from the initiation of the soft drug to which the drug-related intention referred, 12 variables (*MessMedAlc*, *MessMedCig*, *MessMedMar, MessTchrAlc*, *MessTchrCig*, *MessTchrMar, MessParAlc*, *MessParCig*, *MessParMar, MessPeerAlc*, *MessPeerCig*, *MessPeerMar*) constituted measures of differential reinforcement, and three variables (*PeerAlc*, *PeerCig*, and *PeerMar*) were designed to capture differential association.

subsample, just as media tolerance of alcohol (*MessMedAlc*) and cigarette (*MessMedCig*) use constituted important protective factors for marijuana initiation among the total sample and seniors.

The results from relevant primary studies (e.g., Ellickson et al., 2004; Hawkins et al., 2002b; Walter et al., 1991) and extant reviews of the literature did not offer much in the way of insight into the dual risk and protective nature of these social learning variables. Little or no research has investigated the impact of these drug-related variables on the initiation of other soft drugs. Discussed in Chapter 10, although these same counter findings were not found using the 2001 PPAAUS data, a general confirmation of the drug-specific, risk and protective nature of *AlcIntent* was obtained, thereby providing additional evidence for the ability of this construct (and others) to demonstrate dual effects.

Theoretical insight. Traditional and developmental theories of delinquency and drug use do not provide an explanation for the dual directional effects exerted by these variables, or the magnitude of effects exerted by the soft drug intention constructs. The theory of planned behavior (TPB, Ajzen, 1991) does inform some of these findings, however. Specifically, although TPB does not directly explain the dual risk and protective nature of the variables, the theory does provide some insight into the salient effects exerted by the soft drug intention variables, and to a lesser extent, the drug-specific effects that these and the other drug-related variables tended to exert.

In short, TPB takes a distal-proximal mediation approach to explaining behavior, through the use of social influence, attitudinal, and behavioral intention variables. The theory argues that intention (e.g., *AlcIntent*) is the strongest predictor of behavior (e.g., alcohol initiation), because it is the most proximal cause of behavior. In turn, behavioral intention is determined by three conceptually independent factors: the individual's attitude toward the specific behavior, the individual's subjective norms, and perceived behavioral control (Ajzen, 1991). With respect to the first factor, TPB holds that only specific attitudes or beliefs toward the behavior under consideration predict the behavior in question. For example, according to the theory, an adolescent's tolerance of alcohol use will not predict marijuana initiation, just as a youth's favorable attitude toward cigarette smoking will not predict alcohol initiation.

Subjective norms are the beliefs that the individual holds concerning how important others view the behavior in question (e.g., perceived parental and peer drug norms), and the individual's perception of what behaviors (e.g., soft drug use) are typical for others like themselves (e.g., other adolescents). Finally, perceived behavioral control refers to individuals' perceptions of their ability to perform the behavior, given the availability (*EasyAlc*, *EasyCig*, *EasyMar*), opportunity, resources, and tools that exist to complete the behavior (Ajzen, 1991). In instances where behavior is not fully under volitional control, both perceived behavioral control and intention will determine whether the behavior is initiated. In contrast, TPB argues that when an individual has full control over whether a behavior will be initiated, behavioral intention constitutes the sole proximate determinant. As a general rule, the theory posits that the more favorable the attitude and the subjective norm, and the greater the perceived control, the stronger an individual's intention is to engage in the given behavior. In turn, the stronger the intention to engage in the given behavior, the higher the risk for behavioral initiation.

In order to accurately predict behavior, TPB argues that several conditions must be met. One of these conditions, that "measures of intention and perceived behavioral control must correspond or be compatible with the behavior that is to be predicted" (Ajzen, 1991, p. 185), speaks to the drug-specific effects that the alcohol (*AlcIntent*) and cigarette (*CigIntent*) intention variables exerted. The theory's mechanized view of intention does not provide insight, however, into the dual risk and protective nature of these constructs. Since intentions and perceptions of control (e.g., perceived availability and opportunity) must be assessed in relation to the particular behavior of interest, it is not surprising that the soft drug intention variables and the proxy measures of perceived soft drug availability (*EasyAlc* and *EasyCig*) tended to exert drug-specific effects.

The front-end component of TPB provides some insight into the drug-specific effects exerted by the prodrug norm variables, although, the theory again does not provide an explanation for the dual risk and protective nature of these constructs. The theory holds that social influences (e.g., drug use and prodrug norms) shape behavioral intention (and subsequently, behavior) indirectly, by their effects on the attitudes, subjective norms, and perceived behavioral control of the individual. These more distal variables are hypothesized to not have consistent effects on different behaviors, however. In particular, TPB posits that since behaviors vary in "action, target, context, and time elements" (Ajzen &

Fishbein, 1980, p. 85), some of these environmental influences may be related to one behavior, but not another, even if the behaviors appear similar. Hence, according to the theory, tolerant alcohol norms espoused by the media (*MessMedAlc*) or peers (*MessPeerAlc*) should predict alcohol initiation (through *AlcIntent*), but not cigarette or marijuana initiation. Some of the drug-specific findings for these drug-related social learning variables provide some support for this aspect of the theory.

In sum, the H2 counter findings yielded in the current research need replication, particularly since these results may have relevance for explaining the hierarchical nature and progression in the H1 sequence. Although TPB does not explain the H1 sequence, the theory compensates for some of the explanatory drawbacks of traditional and developmental theory, by offering a partial explanation for the dual risk and protective nature of the social learning variables that countered H2 and providing insight into the strong effects exerted by the soft drug intention variables.

Magnitude of Effects

In terms of the magnitude of effects exerted by the social learning variables, several types of findings offer general support for the theory, while other results conflict with some of Akers' (1977, 1992) propositions. In terms of support, the results are consistent with those from past research (see, e.g., Agnew, 1991a; Akers & Cochran, 1985; Bahr et al., 1995; Dembo et al., 1986; Matsueda, 1982; Mears & Field, 2002) in finding that the social learning variables generally exerted stronger risk effects on outcomes than the risk and protective effects exerted by the bonding and unique SDM constructs (e.g., involvement in problem behaviors). However, once a closer assessment of the social learning constructs was conducted, and the findings from H4 and H5 were considered, it became apparent that some of the results from the current research countered two of Akers' (1992) propositions concerning the magnitude of effects exerted by domain-specific variables.

Importance of parents, peers, and school. Akers (1992) argues that the social learning influences of parents, peers, and agents of social learning found in schools (e.g., teachers) are the most important for predicting adolescent behavior. As discussed above, the soft drug intentions of youth (*AlcIntent*, *CigIntent*, *MarIntent*) constituted the most consistent and salient predictors of soft drug initiation and time to initiation in the research, a finding that counters this proposition. Across samples and outcomes, the soft drug norm variables generally exerted the second strongest effects, particularly peer tolerance of soft drug use, and to a lesser extent, the prodrug norms espoused by the media. While, at times, peer soft drug use exerted powerful effects, the impact of these differential association measures were inconsistent.

The prodrug norms espoused by parents also were not very important predictors in the total sample models. Further, regardless of biological age, the prodrug norms espoused by teachers were among the less important predictors of soft drug initiation and time to initiation. Due to the paucity of research on teachers' drug norms, further empirical insight into these particular findings could not be yielded.

Importance of peer influence. Akers argues that peer factors exert the strongest effects on adolescent behavior. The findings for H4, H5, and H9 have implications for the validity of this hypothesis. H4 was developed with insight from several meta-analytic studies and reviews of the literature (Allen et al., 2003; Derzon & Lipsey, 1999a; Glynn, 1981; Swadi, 1999) that support Akers' contention, particularly in terms of alcohol and cigarette initiation. The bulk of prior research indicates that peer factors explain a larger proportion of variance in alcohol and cigarette initiation than variables from other ecological domains.

Counter to Akers' proposition, however, there is some empirical evidence to suggest that individual domain predictors explain a larger proportion of variation in marijuana initiation than predictors from other ecological domains (H5, see, e.g., Andrews et al., 1991; D'Amico & McCarthy, 2006; Derzon & Lipsey, 1999c; Yamaguchi & Kandel, 1984b). Although H4 and H5 were tested with total sample and age-graded data, the age-graded hypothesis tests were viewed as more exploratory in nature, since a paucity of age-graded findings speak to these propositions.

The individual domain predictors generally were responsible for explaining the largest variation in alcohol, cigarette, and marijuana initiation and time to initiation. Hence, H4 received little or no support, while H5 obtained considerable, but partial support. Considering the findings for these hypotheses, the replication of the H5 results with data from the validation samples, along with the H5 support yielded by past research that held biological age constant (e.g., Derzon & Lipsey, 1999c; Yamaguchi & Kandel, 1984b), it appears reasonable to tentatively conclude that the generality of Akers' contention concerning the predominant influence of peer drug use and prodrug norms may be limited, particularly with regard to marijuana initiation and time to initiation.

It is important to highlight, however, that the counter support obtained for H4 may be partly rooted in cohort effects and differences in model development. With respect to the first issue, findings from the 2001 PPAAUS indicated that the peer domain was responsible for the largest explained variation in alcohol initiation and time to initiation among 6th grade students. As discussed in Chapter 10, due to the inability to rule out the plausibility that cohort effects impacted the results from the current research, the null H4 findings that were obtained for alcohol initiation and time to initiation among 6th graders should be interpreted with caution and affirmed through future research.

Differences in model development between the current study and past research also may explain some of the counter findings for H4. The current research examined the utility of distal-proximal mediation within the same regression models that were used to test H4 (and the other hypotheses). In contrast, the bulk of prior research has investigated the impact of peer factors in main effects models. Due to these differences in model development, the validity of the conflicting findings for H4 need to be further substantiated using the same distal-proximal approach that the current research took for model development.

Social learning theory argues that mid- and late adolescent youth are impacted more by peer norms and modeling than early adolescent youth (Akers & Lee, 1996). However, the theory does not articulate whether the impact of peer influence on mid- and late adolescent behavior differs in magnitude. Hence, H9 was rooted in extant research findings that identify mid-adolescence as a time period in which the impact of peer factors on soft drug initiation is strongest, particularly the risk effects posed by peer drug use (see, e.g., Beal et al., 2001; Donovan, 2004; Li et al., 2002b; Urberg et al, 1991).

The differences in model development (i.e., main effects analysis versus distal-proximal mediation analysis) between past research and the current study also may explain the limited support for H9 that was obtained. With few exceptions, the findings from the current research and the 2001 PPAAUS converged in underscoring the importance that peer factors play in the soft drug initiation behavior of early adolescents. Discussed in Chapter 10, although the sole supportive age-graded finding (i.e., *PeerAlc* was a stronger predictor of alcohol initiation among 9th than 6th grade students) yielded in the current research was not obtained with the 2001 data, the internal and external validity of some of the 2004 findings for H9 were strengthened. Several of the validation results reinforced the corresponding results that were obtained in the current research.

Nonetheless, the results from the current research counter Akers' (1977) contention that peer influence is strongest during mid-adolescence. Instead of operating in a curvilinear fashion, peer influence was strongest during early adolescence and weakest during late adolescence. Similar to the findings for H4, a more conclusive determination of the results for H9 will need to await the results from studies that develop regression models in the same manner as the current research.

Peer drug use versus prodrug norms. Four major findings involving peer soft drug use, and the relative importance of this behavioral modeling variable and peer prodrug norms, have implications for social learning theory, as well as past and future research. Taken together, these results underscore the need for social learning theory to better account for the age- and drug-specific effects of peer soft drug use and prodrug norms, and age-contingent mediation processes.

First, by and large, findings from both the total sample and the age-graded subsamples indicated that peer soft drug use was a less important predictor of soft drug initiation and time to initiation than peer soft drug norms. The most glaring disjuncture resided in the findings for predictors of marijuana initiation, and to a lesser extent, time to marijuana initiation. Not only was peer alcohol, cigarette, and marijuana use not predictive of marijuana initiation among the total sample, but peer soft drug use exerted insignificant effects on marijuana initiation among 9th and 12th grade students.

The general lack of importance that peer soft drug use posed for explaining soft drug initiation and time to initiation constitutes an unexpected finding. Discussed in Chapter 4, there is a strong research consensus that indicts peer drug use as one of the most salient predictors of adolescent deviant behavior, including soft drug initiation (e.g., Huizinga et al., 1991; Loeber & Stouthamer-Loeber, 1986). In the face of such strong counter evidence, it is tempting to discount the results from the current research; however, an automatic discount may be unwarranted, particularly when considering the unique manner in which the regression models were developed. In contrast to many prior research studies (see, e.g., Ellickson et al., 2001; Lanza-Kaduce et al., 1984; Reifman et al., 1998; Stumphauzer, 1983; Winfree & Bernat, 1998), which examined the impact of peer drug use in main effects models, the current research took a distal-proximal approach to developing models. Hence, there is a possibility that the lack of support for the direct effects posed by peer soft drug use may lie in the distal-proximal approach that was taken. Until further research replicates this approach, this counter finding should not be discounted.

Second, both peer soft drug use and prodrug norms tended to exert drug-specific effects on outcomes. For instance, peer alcohol use (*PeerAlc*) predicted alcohol initiation among the total sample and 6th and 9th grade students, but this construct was not a significant predictor of cigarette initiation among these subsamples. Peer tolerance of alcohol use (*MessPeerAlc*) had no significant bearing on cigarette initiation or time to initiation among the total sample or 6th and 9th grade students. Further, peer tolerance of cigarette use (*MessPeerCig*) was not predictive of marijuana initiation or time to initiation among the total sample or 9th grade students.

Third, the differential impact of peer soft drug use and prodrug norms tended to vary by the biological age of the subsamples. Touched upon earlier, 6th grade students were impacted to a larger degree by peer drug use and prodrug norms than 9th or 12th graders. In contrast, the soft drug initiation behavior of 9th graders was impacted to a larger degree by peer soft drug use than peer prodrug norms. Finally, as discussed above, the marijuana initiation behavior of 12th graders tended to be influenced by peer drug norms, while peer soft drug use had no impact on the soft drug initiation behavior of this subsample.

Finally, although replication with longitudinal data is needed, it is noteworthy to point out the disjuncture in the direct and indirect effects that peer soft drug use and prodrug norms tended to exert. Nearly all of the peer soft drug use measures that entered the models exerted direct effects, while peer prodrug norms tended to exert indirect effects. The former finding is consistent with that found in longitudinal research (see, e.g., Elliott et al., 1985; Huizinga et al., 1991; Loeber & Stouthamer-Loeber, 1986; Matsueda, 1982). However, since there is a paucity of extant research on the relative differences in the direct and indirect effects that peer norms and behavior exert on soft drug initiation, not much insight into this pattern was able to be yielded from prior work.

In a related vein, the age-graded findings for peer factors speak to both the complexity of age-contingent direct and indirect relationships and the potential need for social learning theory to account for these intricate processes. With respect to peer prodrug norms, for instance, peer tolerance of marijuana use (*MessPeerMar*) exerted direct effects on marijuana initiation among 12th graders, but indirect effects (through the individual domain) on marijuana initiation among 9th grade students. Further, peer tolerance of cigarette use (*MessPeerCig*) exerted indirect effects (through the individual domain) on cigarette initiation among 9th and 12th grade students, but direct effects on cigarette initiation among 6th graders.

Parental Drug Norms

Discussed in Chapter 4, prior research supports Akers' (Akers & Lee, 1996) contention concerning the age-graded impact of parental influence. In particular, studies have found that the impact of parental drug use and prodrug norms on soft drug initiation is strongest during early adolescence (Biddle et al., 1980), weakest during mid-adolescence, and possibly as strong (or stronger) during late as opposed to early adolescence (Huba & Bentler, 1980; Kandel & Wu, 1995). Based on these findings, it was anticipated that parental pro-drug norms would constitute more salient predictors of alcohol and cigarette initiation and time to initiation among 6^{th} grade students than 9^{th} or 12^{th} grade students (H8).

The findings from the current research converged with those from the 2001 PPAAUS in providing little support for H8. Only one finding from the current research was supportive; parental tolerance of cigarette use (*MessParCig*) predicted time to cigarette initiation among 6^{th} grade students, but not 9^{th} or 12^{th} graders. Some of the 6^{th} grade models never retained *MessParAlc* and *MessParCig* upon entry, while in other models, the effects of these measures were mediated by more proximal variables.

Discussed in Chapter 10, although differences in the H8 findings from the current research and that of past research may be rooted in analytic differences (i.e., indirect and direct effects models versus direct effects models), historical effects (i.e., drug prevention programming) constitute a plausible threat to the internal and external validity of the findings for H8. Consequently, insight into the validity of Akers' proposition was not able to be yielded. Moreover, these particular hypothesis results should be interpreted with some caution, at least until further research using samples of youth not exposed to drug prevention programming can replicate the results.

SDM

The directional effects (H2 and H3) posed by unique SDM constructs, as well as the findings for H7, have direct implications for the validity of SDM. In addition, although the research was based on cross-sectional data, some of the distal-proximal mediation findings offer some insight into the validity of SDM's direct and indirect effects propositions. On balance, the relevant findings generally support the theory's basic propositions. More support was yielded for the directional effects exerted by the unique SDM constructs that were investigated than was garnered for the community influence hypothesis (H7). Several distal-proximal mediation findings also counter some of the mediation pathways that the theory outlines.

Detailed in Chapter 3, SDM draws heavily on the explanatory constructs outlined in social learning theory, particularly the constructs of differential association (e.g., peer drug use) and differential reinforcement (e.g., prodrug norms of important others). In explaining both prosocial and antisocial behavior, SDM also utilizes the four elements of Hirschi's (1969) social bond. The bond is, however, conceptualized solely in terms of attachment and commitment, and different causal processes are articulated. The support and counter support yielded for social control theory and social learning theory, in terms of the directional effects (H2 and H3) exerted by the attendant variables that were investigated, applies equally to the relevant directional propositions outlined in SDM.

The risk effects (H2) posed by several unique SDM constructs also were investigated. These variables centered on involvement in antisocial behaviors, including truancy (*Truancy*), cheating on schoolwork (*Cheat*), engaging in (or threatening) violence (*ViolBeh*), and stealing (*Steal*). With only one counter finding obtained (i.e., *Cheat*, in predicting cigarette initiation among 9th graders), considerable support was yielded for the risk capacity of these variables.

Influence of Community Factors

Other than the supportive findings produced by Werner (1994), little etiological attention has been directed toward evaluating the utility of SDM's community influence hypothesis (Catalano & Hawkins, 1996). This proposition holds that community-level risk factors are more influential in shaping the behavior of older youth than younger adolescents (H7). In yielding partial support for H7, the findings from the current research question the degree to which this proposition is valid. Although supportive age-graded findings were yielded for both marijuana outcomes, as well as time to alcohol initiation, the hypothesis did not hold for alcohol and cigarette initiation, or time to cigarette initiation. For these outcomes, the community domain predictors were responsible for an equivalent amount of explained variation in alcohol initiation among 6th and 12th grade students, and a larger amount of explained variation in cigarette initiation among 6th graders than 12th graders. In the 12th grade time to cigarette initiation model, no community domain predictors exerted significant impacts.

Due to a lack of extant research on the validity of the community influence hypothesis, insight from prior work was not able to be obtained. Insight into the validity of the results also was not garnered from the H7 cross-validation, since model misspecification on the part of the 2001 data (see Chapter 10) likely resulted in the lack of convergence in the derivation and validation findings. Future research needs to affirm the H7 findings using the same six community domain factors that were employed in the current research. Until this research is conducted, it appears reasonable to conclude that the generality of SDM's community influence hypothesis may be limited to certain types of drugs, such as marijuana.

Direct and Indirect Effects

Although longitudinal replication is needed, several of the distal-proximal mediation results are relevant to SDM. In particular, some of the direct and indirect effects exerted by SDM constructs in predicting soft drug initiation provide some insight into the possible strengths and weaknesses of the theory.

Soft drug intentions. Detailed in Chapter 6, the soft drug intention variables (*AlcIntent*, *CigIntent*, *MarIntent*) constituted proxy measures of perceived rewards for soft drug initiation. Although it is unclear to what extent these respective intention variables captured drug-specific rewards for initiation, it appears reasonable to assume that students who reported a willingness to use alcohol, cigarettes, or marijuana, believed that more rewards than punishments stem from initiating these respective drugs.

SDM does not explain the drug-specific directional effects that the soft drug intention variables tended to exert; however, the direct and powerful effects of these constructs are consistent with the theory's expectations. According to SDM, three constructs are hypothesized to exert direct risk effects on soft drug initiation: 1) belief in antisocial behavior; 2) attachment and commitment to antisocial others and activities; and 3) perceived rewards for problem behaviors and interactions with antisocial others (Catalano & Hawkins, 1996). Hence, although TPB provides a better explanation for the magnitude of effects exerted by the soft drug intention variables, SDM's proposition also received some support.

Involvement in problem behaviors. As discussed in Chapter 3, SDM's general model argues that involvement in problem behavior does not exert a direct impact on antisocial behavior, including soft drug initiation. Counter to this proposition, *Cheat* exerted direct risk effects on alcohol initiation. Frequent involvement in stealing (*Steal*) and truancy (*Truancy*) also exerted direct risk effects on alcohol initiation among the total sample.

Prodrug norms. The theory also argues that prodrug norms espoused by important others exert indirect effects on soft drug initiation by shaping perceived rewards for soft drug initiation. In turn, these rewards are hypothesized to strengthen youths' bonding to antisocial others and activities. The increase in antisocial bonding increases risk for soft drug initiation directly, as well as indirectly through antisocial beliefs.

Although this mediation pathway was not tested, some insight into the utility of this hypothesis was able to be evaluated, in terms of the indirect effects that prodrug norms were found to exert. With three exceptions, this hypothesis was generally supported, particularly with respect to alcohol and cigarette initiation among the total sample. Counter support centered on the direct effects exerted by *MessMedCig*, *MessPeerMar*, and *MessPeerAlc* on marijuana initiation. Again, due to the cross-sectional nature of the data, these findings need to be affirmed through a prospective research design.

Outcome-Specific Predictors

Little or no prior research has taken a systematic and comprehensive approach to investigating differences between predictors of alcohol, cigarette, and marijuana initiation and time to initiation. Hence, one of the major contributions of the current study resides in the key findings for outcome-specific predictors. In short, although predictors exerted slightly more direct effects on soft drug initiation than time to soft drug initiation, the major substantive difference resided in the types of factors predictive of each outcome.

With respect to the direct effects exerted by variables in the total sample models, the largest differences between factors predictive of initiation versus time to initiation centered on alcohol. Although *Steal*, *Religious*, and *AlcIntent* were predictive of both alcohol initiation and time to initiation, *Social*, *PeerAlc*, *Truancy*, and *Cheat* predicted alcohol initiation, but not the ages at which alcohol was initiated. Further, *ViolBeh* and *MessParAlc* were predictive of time to alcohol initiation (by lowering age of initiation), but the impact of *MessParAlc* on alcohol initiation was mediated by more proximal factors. *ViolBeh* had no significant bearing on whether youth initiated alcohol use. Finally, the effects exerted by pro-alcohol norms espoused by the media (*MessMedAlc*), teachers (*MessTchrAlc*), parents (*MessParAlc*), and peers (*MessPeerAlc*) were mediated by more proximal factors. However, these social learning constructs had no significant bearing on the ages at which alcohol was initiated.

Outcome-specific differences in direct effects also were found for cigarettes and marijuana. *ViolBeh* and *MessMedAlc* were predictive of the ages at which youth initiated cigarette use, whether cigarette initiation occurred. With respect to marijuana, media tolerance of cigarette use (*MessMedCig*) constituted a salient protective factor for marijuana initiation among the total sample, by delaying the ages at which this substance was initiated. In contrast, this variable did not even enter the marijuana initiation model.

Several general patterns concerning prodrug norms also emerged, once the direct and mediated effects of these social learning variables were compared by respective initiation and time to initiation outcomes. Concerning the total sample findings, peer prodrug norms tended to impact marijuana initiation directly, while the bulk of the effects of peer prodrug norms on time to marijuana initiation were mediated by the individual domain predictors. In contrast, the impact of prodrug norms espoused by important others on cigarette initiation and time to initiation tended to be mediated by more proximal factors. Finally, compared to alcohol initiation, the ages at which alcohol is initiated was not impacted in any substantive way by the prodrug norms that others espouse, with the exception of parental tolerance of alcohol use (*MessParAlc*), which exerted direct effects.

Although this set of findings needs to be affirmed, the results underscore the need for further comparative studies, and an increase in research on age of soft drug initiation. Taking into account the fact that the largest dissimilarities were observed for alcohol, and alcohol remains the most widely used soft drug among adolescents (Johnston et al., 2007), it also may be worthwhile to conduct explicit research on the differences between predictors of the initiation and age of initiation of this substance.

Policy and Programmatic Implications

The overarching purpose of all social research is to inform social policies that enhance and improve the human condition (Wagenaar, 1993). Adolescent soft drug research has no practical meaning if it does not aid in reinforcing, formulating, or modifying existing adolescent drug policy and prevention programs (Davies, Nutley, & Smith, 2000; Petrosino, Boruch, Soydan, Duggan, & Sanchez-Meca, 2001). Due to the scope of the current research, and the multiple findings that were yielded, resulting policy and programmatic implications are numerous. In general, the key findings speak to three overarching issues, all of which center on empirically-verified elements of effective primary prevention programs: targeting malleable factors, tailoring program components, and administering peer-led prevention programs.

Targeting Malleable Factors

Watzlawick, Weakland, and Fisch (1974) trace ineffective education and treatment policy to three specific sources of error. Two sources are as relevant for the fields of education, mental health, and drug treatment as they are for primary drug prevention: action that is taken, but is directed toward changing the unchangeable; and action that is necessary, but is not taken.

Although advancements in preventing soft drug initiation among youth have been made, more short- than long-term reductions in incidence rates have been observed (see, e.g., Komro & Toomey, 2002; Murray, Pirie, Luepker, & Pallonen, 1989; Schaps, Churgin, Palley, Takata, & Cohen, 1980). As primary prevention implementation and outcome research has indicated, ineffective efforts to prevent or delay soft drug initiation among youth may lie partly in the types of factors that respective programs target for change (Tobler, 1986). Since not all risk and protective factors for soft drug initiation are malleable, focusing efforts on changing static risk factors (e.g., stable personality traits and states) will prove futile (Climent, de Aragon, & Plutchnik, 1990).

The current research identified a host of malleable predictors of soft drug initiation and time to initiation that are legitimate targets for change. Within the community, school, family, and peer domains of influence, for example, total sample findings generally indicate that primary prevention programs that seek to bolster media, parent, and peer intolerance of soft drug use, while increasing school bonding, reducing soft drug use among parents and peers, and limiting access to soft drugs, can be effective in reducing risk for (and delaying ages of) soft drug initiation among adolescents.

The findings from several comprehensive reviews (Black, Tobler, & Sciacca, 1998; Tobler, 1992; Tobler, Roona, Ochshorn, Marshall, Streke, & Stackpole, 2000) of adolescent drug prevention programs reinforce the findings from the current research and attendant prevention implications, in suggesting that targeting many of the malleable risk and protective factors identified in the current research can produce beneficial results. For instance, meta-analytic studies by Tobler (1992, 1996; Tobler et al., 2000) consistently have found that the most effective primary prevention programs are those that alter many of the theoretical constructs shown in the current study to predict soft drug initiation and time to initiation.

The utility of the Seattle Social Development Project (and subsidiary programs like Skills, Opportunities, and Recognition [SOAR]) and CTC is particularly instructive. Founded on SDM, both antisocial behavior prevention initiatives take a comprehensive approach to preventing soft drug initiation by targeting salient risk and protective factors found in all five ecological domains of influence. Many of the predictors targeted for change by CTC were examined in the current research (e.g., parental and peer drug norms, peer soft drug use, availability of soft drugs, school bonding, and involvement in prosocial activities). Findings from both implementation and outcome evaluations of these initiatives yield a considerable amount of support for SDM's general model and a few of the age-graded models (see, e.g., Catalano et al., 2005; Hawkins, Doueck, &

Lishner, 1988; Hawkins et al., 1992b; Hawkins et al., 2001; Hawkins, Kosterman, Catalano, Hill, & Abbott, 2005). Further, within the past 10 years, the Seattle Social Development Project has been deemed a "model" (U.S. Department of Health and Human Services, 2001) and "promising" (Center for the Study and Prevention of Violence, 1999) program, capable of reducing soft drug initiation and several of the problem behaviors (e.g., stealing and truancy) to which initiation is associated (Schinke, 2002; U.S. Department of Education, 2002).

Many primary prevention programs target adolescent drug norms, as well as drug-related intentions (Tobler, 1992), which was one of the most important proximal factors identified in the current study. Research is not clear in indicating whether changes in drug-related expectancies (a social learning construct) and intentions follow changes in drug norms and beliefs, or if drug-related expectancies operate independently of drug use intentions (Black et al., 1998). However, a growing research base does indicate that once youth are provided information showing the number of adolescents and adults actually engaged in drug use, their positive expectancies concerning use decrease (perhaps due to lowered expectations for conformity), while their intentions to use drugs become tempered (see, e.g., Beck & Treiman, 1996; Black et al., 1998; Hansen, 1993; Linkenbach & Perkins, 2003; Thombs, Wolcott, & Farkash, 1997; Tobler, 1986). Compared to programs delivered by teachers and researchers, Black and colleagues' (1998) meta-analysis of 120 adolescent drug prevention programs found that this effect on alcohol use intentions was more pronounced in peer-led programs that emphasized dispelling the myth that all students drink alcohol.

Tailoring Program Components

Other drug prevention implications that stem from the current research coincide with the second source of error that Watzlawick et al. (1974) trace to ineffective policy: action that is necessary, but it not taken. As various standards of effective adolescent drug prevention programs predicate, in order for programs to effectively prevent or delay drug initiation among youth, the content of such should be “broadly based” and comprehensive. Multiple ecological domains of influence need to be targeted (Nation et al., 2003; Weissberg, Kumpfer, & Seligman, 2003). Programs also need to be appropriate for the developmental stage and biological age of the target audience.

Not only did the current study reveal that a number of important risk and protective factors emanate from each of the five ecological domains of influence, but the effects exerted by predictors tended to differ in magnitude or significance by grade-level, with far more age-specific factors identified than universal predictors. Further, had the total sample not been decomposed into the three subsamples of youth, whose biological ages were relatively homogeneous, a number of important age-specific risk factors would have remained masked in the total sample findings. Translated into practice, this masking of age-specific effects can potentially lead to mis-intervention for one or more subsamples of youth (Huba & Bentler, 1980). These general findings, coupled with the results from meta-analytic studies of drug prevention programs (see, e.g., Tobler, 1986, 1992; Tobler et al., 2000) that locate program effectiveness in developmentally-appropriate components, underscore the need for primary prevention programs to continue tailoring their components to the grade-level or biological age of the participants.

The prevention implications that stem from H1, H6, and the drug-specific findings extend beyond some of the more standard recommendations for primary prevention that have been offered (Dryfoos, 1996; Nation et al., 2003; Tobler, 1986). Specifically, these findings direct attention toward the need to tailor programs according to the type of soft drug use the prevention initiative intends to prevent or delay. Given that school-based drug prevention budgets have become constrained in recent years (see, e.g., Carnevale Associates, 2006, 2007; Drug Strategies, 1999; Pentz, 1996), it appears reasonable, from both an economic and empirical perspective, that a potentially promising way to prevent or delay adolescent involvement in drug use may be to focus the bulk of attention on preventing (or delaying) alcohol initiation, particularly that which occurs during early adolescence. Given the typical ordering of involvement in alcohol, cigarette, and marijuana use, primary prevention programs that take this approach may produce reductions in rates of alcohol incidence, while indirectly working toward preventing (or delaying) cigarette and marijuana initiation.

Typical primary prevention programs target the initiation of all drugs, with sometimes unique attention directed at legal drug use (Botvin, Scheier, & Griffin, 2002; Montoya, Atkinson, & McFaden, 2003). There is a growing research base to suggest, however, that a more refined focus on preventing or delaying alcohol initiation, particular among early adolescents, may prove beneficial. For example, in directing efforts to prevent alcohol initiation, the comprehensive school-based drug prevention program "Life Skills Training" has been shown to reduce alcohol incidence rates among 7th, 8th, and 9th grade students (Botvin, Baker, Renick, Filazzola, & Botvin, 1984) and prevent and delay cigarette, marijuana, and hard drug initiation for up to three years later (see, e.g., Botvin, Baker, Dusenbury, Botvin, & Diaz, 1995; Botvin, Baker, Dusenbury, Tortu, & Botvin, 1990; Botvin et al., 2000). Etiological research also suggests that directing prevention efforts toward targeting risk factors for alcohol initiation, particularly during the elementary school years, may be an effective strategy for preventing progression in the soft drug sequence (Hawkins et al., 2002b; Kandel, 2002; Kandel et al., 1992; Pentz & Li, 2002; Welte & Barnes, 1985).

Perhaps due to the fact that primary prevention programs often target the initiation of all drugs, only three drug-specific primary prevention studies have been conducted to date. In general, the findings provide some support for explicitly targeting efforts at preventing or delaying alcohol initiation. An evaluation of a smoking prevention program, the Minnesota Heart Health Program, produced favorable results, with reductions in cigarette initiation evidenced, followed by smaller reductions in rates of alcohol initiation (Perry, Kelder, Murray, & Klepp, 1992). Two longitudinal outcome studies from Project Northland indicated that the program was moderately effective in reducing alcohol initiation, followed by cigarette and tobacco initiation (Perry et al., 1996; Perry et al., 2002). Another outcome evaluation of an alcohol prevention study evidenced reductions in binge drinking, followed by a reduction in rates of marijuana initiation (Cheadle, Pearson, Wagner, Psaty, Diehr, & Koepsell, 1995).

Although limited, the findings from this body of research lend some empirical support for directing prevention efforts explicitly at alcohol initiation. Since the current study's results indicated that predictors of alcohol initiation among 6th, 9th, and 12th grade students differ more in kind than in salience, prevention efforts geared toward preventing the initiation of alcohol may need to be tailored according to the most important age-specific predictors. For example, in the current research, predictors of alcohol initiation among 6th grade students exerted more direct than indirect effects. This suggests that for early adolescent youth, a focus on manipulating the risk and protective factors that exert salient, direct effects (e.g., *Academic*, *Religious*, *PeerAlc*, *AlcIntent*, *Steal*, *ViolBeh*) on alcohol initiation may prove more beneficial than trying to address the multiple mediating relationships between distal and proximal predictors and alcohol initiation.

As another example, every predictor of alcohol initiation (*AlcIntent*, *Truancy*, *Steal*, *MessMedAlc*) among 12th grade students exerted direct effects. What these results suggest is that primary prevention initiatives aimed at preventing or delaying alcohol initiation among late adolescents need to take an explicit direct approach to reducing problem behaviors, while also changing pro-alcohol norms, which may lie at the heart of intentions to use alcohol.

Peer-Led Interventions

With few exceptions, peer soft drug use and pro-drug norms constituted important risk factors for soft drug initiation and time to initiation among youth in the study, particularly for the initiation of the corresponding soft drug. Further, the distal-proximal mediation results indicated that peer factors exert both direct and indirect effects on outcomes, with the individual domain serving as an important mediating force. Moreover, the direct effects exerted by numerous school and community domain predictors were nullified by the peer domain. Taken together, these findings support the continued use of peer-led prevention interventions that are designed to impact drug-related attitudes, norms, and intentions.

Numerous process and outcome evaluations (see, e.g., Botvin, Baker, Filazolla, & Botvin, 1990; Luepker, Johnson, Murray, & Pechacek, 1983; Murray, Davis-Hearn, Goldman, Pirie, & Luepker, 1988) have shown that peer-led drug prevention programs are more effective in decreasing rates of alcohol, cigarette, and marijuana incidence, as well as altering drug-related beliefs, attitudes, and intentions, than teacher- and expert-led interventions. Tobler's (1986) meta-analysis of 143 adolescent drug prevention programs found that compared to other strategies, peer-led programs produced the highest effect sizes for all categories of drug use, including alcohol, cigarettes, and marijuana.

Botvin and colleagues' (1990) evaluation of a 20-session drug abuse prevention program directed at 1,311 7th grade students converges with other research in supporting the use of peer-led booster interventions (see, e.g., Luepker et al., 1983; Murray et al., 1988; Murray, Pirie, Luepker, & Pallonen, 1989; Murray, Richards, Luepker, & Johnson, 1987). Compared to time-limited peer-led and teacher-led interventions, the peer-led intervention coupled with a booster session employed in 8th grade was more effective in reducing rates of alcohol, cigarette, and marijuana incidence and prevalence (Botvin et al., 1990).

In further support of peer-led primary prevention programs, several process evaluations have found these programs to be effective in improving adolescents' knowledge about the prevalence and negative effects of soft drug use (Botvin et al., 1990; Tobler, 1986, 1992), as well as reducing intentions to smoke cigarettes (Clarke, MacPherson, Holmes, & Jones, 1986).

Study Limits

The current research made a number of contributions to the prevention science knowledge base, and on several interrelated fronts. However, since several issues pose limitations, the findings should be interpreted with some care.

Validity of the Results

As part of the study, a dual cross-validation was employed, in an effort to closely examine and rule out plausible threats to the internal, external, construct, and statistical conclusion validity of the findings. Not only were many of the findings for H2-H9 confirmed in full or part, but the evaluation of shrinkage and the comparison of results for distal-proximal mediation provided further evidence of the stability of the findings.

The near full convergence in the dual cross-validation findings for H2 and H3 virtually nullified the two major threats (i.e., inappropriate statistical techniques and low statistical power) to the statistical conclusion validity of the current results. This congruence also tempered the threat that any invalid or unreliable measurement of the 2004 PPAAUS variables posed for the construct validity of the findings. Under-reporting and the use of self-report data, threats to the construct validity of the results, also appear to have been minimized. Not only did the bulk of the findings from the dual cross-validation for H2 and H3 yield considerable support for the H2 and H3 findings in the current study, but several of the primary studies (whose results were supportive) obtained data from multiple sources, such as parents, peers, teachers, and official records.

The inherent limitations of cross-sectional data are recognized. Although the findings are suggestive, further research clearly is necessary to determine the extent to which the results can be replicated with longitudinal data. However, the threat that causal ordering poses for the internal validity of the results does not appear to constitute a serious problem. Through the systematic review cross-validation, a strong degree of convergence in H2 and H3 was obtained. Further, more than 60% of the primary studies that were investigated utilized a longitudinal research design.

Several plausible threats do pose limitations that should be considered when interpreting and generalizing the findings. Major threats to the internal validity of the results include the inadequate control of third variables and historical effects stemming from the drug prevention initiatives that were employed in the community from which the 2004 sample was derived. It is important to note that since the PPAAUS did not include items concerning personality traits or states, the current research was unable to account for these intrapersonal factors. As past reviews of the literature and the current systematic review revealed, several constitutional variables constitute risk factors for soft drug initiation, including impulsivity, sensation-seeking, alienation, depression, and low self-esteem (see, e.g., Conrad et al., 1992; Derzon & Lipsey, 1999a, 1999c; Donovan, 2004).

Discussed in Chapter 10, several of the conflicting findings that were identified through the quantitative cross-validation appear to be the result of secular, cohort, or age effects. Although it appears that the bulk of the H2 and H3 findings from the current research were not impacted by these time period efforts, some of the conflicting age-graded findings for H4-H9 may have been impacted by one or more of these factors. In consequence, secular, cohort, and age effects, coupled with the plausibility of differential history effects, constitute salient threats to the external validity of some of the findings.

Finally, three threats to the construct validity of the results remain plausible. In particular, recall decay, forward telescoping, and lengthy period of recall all remain plausible threats to the construct validity of the results for predictors of time to soft drug initiation, particularly among 12th grade students.

Peer Drug Use Measures

As evidenced in the research, and reinforced by past findings, one of the more salient predictors of soft drug initiation is peer drug use, a proxy measure of delinquent peer association (see, e.g., Huizinga et al., 1991; Loeber & Stouthamer-Loeber, 1986). Discussed in Chapter 4, researchers acknowledge the existence and importance of adolescent-peer homophily. However, there is debate in the literature concerning why this relationship exists and which of these factors temporally precedes and influences the other. Consequently, it is unclear which theory (social control or social learning theory) and attendant assumption of human nature provides the most adequate explanation for this relationship. Instead of causing adolescent drug initiation as Akers (1977) purports, delinquent peer association (i.e., peer drug use) may be a consequence of adolescent drug initiation.

The significant relationships found between peer soft drug use and adolescent soft drug initiation may have originated from either selective peer association on the part of adolescents themselves or peer socialization factors. Research on developmental pathways leading to delinquency does suggest, however, that selective association may be more operable after a given deviant behavior is initiated (Elliott et al., 1985). Specifically, studies have found that the selection of more serious delinquent peers occurs prior to further entrenchment and involvement in more serious delinquent behaviors (see Warr, 2002).

Taking this insight into consideration, along with the support yielded for H1 and the average ages of soft drug initiation among the age-graded subsamples, it is possible that the direct relationships found between peer alcohol use and adolescent alcohol initiation may have resulted more from selective peer association on the part of adolescents themselves. In contrast, it is possible that the salient relationships found between peer marijuana use and adolescent marijuana initiation, particularly among 9^{th} and 12^{th} grade students, may be rooted more in peer socialization factors than in selection processes.

Although the inability to disentangle “feathering” from “flocking” poses more of a limitation for theory than for prevention initiatives at the practical-level, the issue of peer-adolescent homophily is relevant to the current research in two ways. Although the H2 directional findings for peer soft drug use generally converged with those from the systematic review, the cross-sectional nature of the 2004 PPAAUS data precluded a definitive determination of causal ordering between adolescent soft drug initiation and peer soft drug use. This inability to fully verify the temporal ordering of these variables, coupled with the use of self-report data, prevented selection and socialization effects from being adequately assessed and disentangled.

Mentioned above, third variable explanations for the salient relationships found for peer soft drug use and the soft drug initiation among youth also were not taken into account in the research. Since it was not possible to control for any stable personality traits (e.g., low self-control, sensation-seeking, and impulsivity), the degree to which the self-report responses for peer soft drug use were impacted by selective “flocking” (social control perspective) could not be assessed.

Recommendations for Further Research

Quite a number of suggestions for future research stem from the key findings of the current study. Aside from the recommendations put forth above, perhaps the most important suggestion is to replicate the findings from the current research by developing models that are capable of identifying both direct and indirect effects. In testing the utility of Petraitis et al.’s (1995) distal-proximal mediation hypothesis, the current research subjected H4-H9 to rigorous testing, by enabling both the direct and indirect effects of variables to be considered. Discussed earlier, it is not surprising that the findings from the 2004 and 2001

PPAAUS tended to converge in providing partial or little support for H4, H5, and H7-H9. These hypotheses were established with insight from extant reviews of prior research that only examined the main effects of these variables (see Chapter 4), and the current study yielded strong support for distal-proximal mediation. In order to properly replicate the findings, research should employ the distal-proximal scheme that was utilized.

Sequencing Research

Three major recommendations for further study stem from the two H1 tests that were conducted. The differential scale fit that was obtained should be replicated, particularly since the current investigation constituted the first known explicit comparison of measurement strategies (i.e., dichotomous initiation versus age of initiation data). Comparisons of Guttman scale fit based upon different measurement strategies, including the use of more refined age of initiation measures (e.g., calendar year and month), also may advance knowledge.

Echoing Kandel's (1980) suggestion, further prospective investigations of soft drug sequencing also are needed, particularly among rural youth. Finally, future cross-sectional work on soft drug sequencing, particularly studies utilizing Guttman scalogram analysis, should incorporate a measure of time into the initiation measures.

Prediction Research

Methodological and Reporting Issues

The systematic review results speak to a number of important reporting and methodological issues that could be addressed by future prediction research.

A very basic issue centers on information reporting. A broad assessment of the primary studies revealed that 28% of this body of research failed to include descriptive information concerning one or more demographic characteristics (i.e., age, race, and gender) of the sample. Another 28% did not report the direction of insignificant effects. This information helps provide context to and facilitate a greater understanding of published findings. In addition, researchers who conduct systematic reviews rely on this data in drawing conclusions about the internal and external validity of the results, as well as the direction of effects exerted by insignificant variables.

The prevention science knowledge base also could benefit from an increase in cross-validations of research results. Only 6% of primary studies compared respective research findings to those obtained from a similar, but distinct, sample. There also is a need for more consistent efforts in reducing inflated or attenuated regression coefficients, as well as obtaining data from referent sources, such as parent and peers. Roughly 45% of the primary studies did not take explicit strides in reducing under- and over-reporting, and 92% of the studies relied solely on self-report data.

Thorough and Sound Investigations

It is suggested that a more consistent effort be made to study adolescent soft drug initiation from a comprehensive and theoretically-guided, developmental perspective. Although the current research sought to address this gap, a more sustained effort is needed. Only 31% of the primary studies included in the systematic review provided for a comprehensive examination of predictors emanating from multiple ecological domains of influence. Further, only 50% investigated major factors that have been empirically-verified to increase and decrease risk for soft drug initiation.

Multiple Effects of Predictors

The findings underscore the importance of investigating the multiple ways (e.g., direct, mediating, and moderating effects) in which predictors can operate. Although a growing body of research has provided some strong evidence for the magnitude and direction of the direct impact that predictors have on soft drug initiation, further attention is needed.

It also appears that the prevention science knowledge base can be enhanced by addressing theoretical and empirical questions that center on mediation processes. Although the supportive findings for distal-proximal mediation need further affirmation, the results provide strong evidence for the validity of Petraitis et al.'s (1995) proposition. Moreover, these findings, coupled with the age-specific predictors identified in the current study, underscore the need for researchers to move beyond solely testing traditional explanations of soft drug initiation among adolescents. As it stands, developmental theory more appropriately accounts for the distal and proximal nature of risk and protective factors.

The directional findings for the drug-specific and mediating capacity of soft drug intentions (*AlcIntent*, *CigIntent*, and *MarIntent*) and drug tolerance variables (e.g., *MessMedAlc*, *EasyAlc*) need further confirmation. The paucity of comprehensive research on this issue impeded efforts to draw upon extant empirical studies in providing insight into the drug-specific differences in directional effects that these variables exerted. Research on the utility of TBP for explaining the salience of soft drug intentions in predicting soft drug initiation also would be helpful.

Finally, the continued examination of age-specific predictors of soft drug initiation and time to initiation also is needed, particularly age-factor interaction effects. As Huba and Bentler (1980) aptly point out, prevention researchers who do not examine the interactive impact of age on relationships run the risk of committing Type I or II errors for different age groups contained within total samples. If false positives and negatives are translated into prevention practice, these empirical errors can hinder successful efforts to prevent soft drug initiation among particular stages of adolescence.

By and large, most of the potential age-factor interactions that were suggested dealt with alcohol initiation, and to a lesser extent, social learning variables (*MessMedAlc*, *MessParAlc*, *MessParCig*, and *PeerAlc*). Since it was beyond the scope of the study to assess the statistical significance of the age-factor interaction effects that were identified, it is suggested that further research substantiate the importance of these relationships. Compared to the other age-factor interactions that were indicated, the directional effects exerted by differential reinforcement constructs (i.e., parental and media tolerance of alcohol and cigarettes) appear to be particularly susceptible to maturational changes on the part of youth.

Time to Soft Drug Initiation

As was identified through the systematic review, a paucity of research has investigated predictors of time to soft drug initiation among adolescents. One goal of the current research was to address this void; however, further confirmation of the findings is needed, particularly with respect to the results for the 12th grade subsample.

In an effort to reduce the impact that forward telescoping and lengthy period of recall pose for the construct validity of findings, it also is suggested that future research measure age of initiation in a more fine-grained fashion. Utilizing methods that bring about better recall also may be beneficial. For example, instead of asking youth the biological year in which a given drug was initiated (as was done in the PPAAUS), it may be worthwhile for researchers to use the timeline follow-back method (Sobel & Sobel, 1992), while asking respondents to report the calendar year and month that the drug in question was initiated.

The research suggestions provided above stem predominantly from the prediction component of the study. Given that specific research gaps were identified through the systematic review, a number of other recommendations for prediction research are in order. Detailed in Table 88, all of these voids are worthy of redress.

Community Domain Predictors

Three major research gaps coincide with the community domain predictors. More research is needed on community determinants of time to soft

Table 88.

Major Research Gaps identified through the Systematic Review

Void Type	Community Domain	School Domain	Family Domain	Peer Domain	Individual Domain
Domain-Specific Issues:	▪ Generic nature of soft drug use/norms ▪ Community characteristics	▪ Teachers drug use/norms, including generic nature ▪ School drug policies	▪ Generic nature of soft drug use/norms	▪ Generic nature of soft drug use/norms	▪ Generic nature of drug-related intentions ▪ Other soft drug initiation
Initiation:	Alcohol	Alcohol	Alcohol Cigarettes Marijuana	Alcohol Marijuana	Alcohol Cigarettes
Time to Initiation:	Alcohol Cigarettes Marijuana	Alcohol Cigarettes Marijuana	Alcohol Cigarettes Marijuana	Alcohol Cigarettes Marijuana	Alcohol Cigarettes Marijuana
Age-Specific Initiation:	Mid-Adolescence Late Adolescence	Late Adolescence	Late Adolescence	Late Adolescence	Late Adolescence
Age-Specific Time to Initiation:	Early Adolescence Mid-Adolescence Late Adolescence	Early Adolescence Mid-Adolescence Late Adolescence	Early Adolescence Mid-Adolescence Late Adolescence	Early Adolescence Mid-Adolescence Late Adolescence	Early Adolescence Mid-Adolescence Late Adolescence

drug initiation. The systematic review was unable to identify any published studies that investigated the utility of these distal factors.

It also is suggested that further research consider the cross-cutting nature of adult alcohol, cigarette, and marijuana use and norms on the initiation of other soft drugs. Based upon the findings from the primary studies that were examined, not only is more research on alcohol, cigarette, and marijuana availability needed, but the generic nature of these factors in predicting all three types of soft drugs also is warranted.

Finally, more research takes explicit strides in comparing the impact of community influences and characteristics on the soft drug initiation behavior of early, mid-, and late adolescent youth. As identified through the systematic review, none of the primary studies derived community-related data from distinct samples of late adolescent youth. Until more research takes these explicit strides, the validity of Hawkins' (Catalano & Hawkins, 1996) community influence hypothesis (and H7 findings from the current study) can not be fully evaluated and affirmed.

School Domain Predictors

Due to a paucity of past studies on school domain predictors of time to alcohol, cigarette, and marijuana initiation, it is recommended that an increase in research be conducted in this area. Not one of the two primary studies (Hawkins et al., 2002b; Unger & Chen, 1999) that investigated time to soft drug initiation examined the utility of school domain factors. As well, little attention has been directed toward evaluating the efficacy of numerous school-related variables for predicting soft drug initiation (Table 88).

Family Domain Predictors

Additional assessments are needed on the impact that parental and sibling alcohol, cigarette, and marijuana use and norms have on both the initiation and time to initiation of other soft drugs (Table 88). Empirical verification of Hawkins et al. (2002b) and Unger and Chen's (1999) family-related findings for time to soft drug initiation also would be helpful. Warranted are expanded investigations of the elements of the social bond, and the effects that parental and sibling alcohol and marijuana use, and parental and sibling cigarette norms, pose in lowering age of soft drug initiation.

Peer Domain Predictors

Suggestions for future research on peer domain determinants of soft drug initiation center on the following issues (Table 88): the cross-cutting nature of peer alcohol, cigarette, and marijuana use and norms for predicting the initiation of other soft drugs; the utility of peer delinquency; and the degree to which the predictive utility of peer differential association is of value. It also would be informative if more studies investigated the impact of the peer-adolescent bond (e.g., attachment and involvement) on soft drug initiation and time to initiation, particularly with respect to initiation among late adolescents.

Individual Domain Predictors

Parallel to several of the suggestions offered above, the prevention science field could benefit from prediction research that addresses the following issues related to individual domain predictors (Table 88): the generic nature of drug-related predictors, including low alcohol, cigarette, and marijuana refusal self-efficacy, intentions to use alcohol, cigarettes, and marijuana, and norms associated with these types of substances; predictors of time to soft drug initiation; the utility of cigarette initiation for predicting alcohol and marijuana initiation; and the importance of marijuana initiation for predicting alcohol and cigarette initiation. As well, none of the primary studies assessed in the systematic review directed attention toward the efficacy of alcohol initiation as a determinant of cigarette initiation. Such investigations would provide further insight into the validity of Kandel’s (2002) stage theory. Finally, additional research is needed on the impact of involvement in work-for-pay activities and the associated impact that such involvement has on the likelihood of initiating alcohol and marijuana use.

Conclusion

The current study provided one of the most comprehensive cross-sectional investigations to date of predictors of alcohol, cigarette, and marijuana initiation and time to initiation. Although much etiological work remains, the findings contribute to the prevention science knowledge base on a number of diverse fronts. Nearly all nine hypotheses obtained some degree of support. A plethora of drug- and age-specific predictors also were identified, thereby lending more support for the specific risk factor hypothesis than the common factor hypothesis. The results also provide some convincing evidence for the validity of

Kandel's (2002) drug sequencing hypothesis and Petraitis et al.'s (1995) distal-proximal mediation hypothesis.

Although founded on cross-sectional data, the research yielded some support for several of the direct and indirect effects propositions detailed in social learning theory, the social development model, and, to a lesser extent, the theory of planned behavior. Nearly all of the directional effects exerted by the social control variables met hypothesized expectations; however, the findings call into question some of the direct effect hypotheses put forth by the theory. The results also underscore some possible limits of SDM.

The validity of the results was evaluated, by subjecting the results for H2-H9 and distal-proximal mediation to a rigorous dual cross-validation procedure. The relatively strong degree of convergence that was obtained served to bolster the reliability and validity of many of the research findings. These results hopefully can be used to inform future policy and program efforts in the area of adolescent drug use and prevention.

REFERENCES

Abadinsky, H. (2001). *Drugs: An introduction*. Belmont, CA: Wadsworth/Thomson Learning.

Adler, I., & Kandel, D. B. (1983). Risk periods for drug involvement in adolescence in France and in Israel: Application of survival analysis to cross-sectional data. *Social Forces*, *62*, 375-397.

Agnew, R. (1985). Social control theory and delinquency: A longitudinal test. *Criminology*, *23*, 47-61.

Agnew, R. (1986). Work and delinquency among juveniles attending school. *Journal of Crime and Justice*, *9*, 19-41.

Agnew, R. (1991a). The interactive effects of peer variables on delinquency. *Criminology*, *29*, 47-72.

Agnew, R. (1991b). A longitudinal test of social control theory and delinquency. *Journal of Research on Crime and Delinquency*, *28*, 126-156.

Agnew, R. (1993). Why do they do it? An examination of the intervening mechanisms between social control variables and delinquency. *Journal of Research on Crime and Delinquency*, *30*, 245-266.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, *50*, 179-211.

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs, NJ: Prentice Hall.

Akers, R. L. (1977). *Deviant behavior: A social learning approach* (2nd ed.). Belmont, CA: Wadsworth.

Akers, R. L. (1984). Delinquent behavior, drugs, and alcohol: What is the relationship? *Today's Delinquent*, *3*, 19-47.

Akers, R. L. (1985). *Deviant behavior: A social learning approach* (3rd ed.). Belmont, CA: Wadsworth.

Akers, R. L. (1990). Rational choice, deterrence, and social learning in criminology: The path not taken. *Journal of Criminal Law and Criminology*, *81*, 653-676.

Akers, R. L. (1992). *Drugs, alcohol, and society: Social structure, process, and policy*. Belmont, CA: Wadsworth.

Akers, R. L. (1994). *Criminological theories: Introduction and evaluation*. Los Angeles, Roxbury.

Akers, R. L. (1998). *Social learning and social structure: A general theory of crime and deviance*. Boston, MA: Northeastern University Press.

Akers, R. L., & Cochran, J. K. (1985). Adolescent marijuana use: A test of three theories of deviant behavior. *Deviant Behavior*, *6*, 323-346.

Akers, R. L., & Jensen, G. F. (Eds.) (2003). *Social learning and the explanation of crime: A guide for the new century* (Advances in Criminological Theory, Vol. 11). New Brunswick, NJ: Transaction.

Akers, R. L., & Jensen, G. F. (2006). Empirical status of social learning theory: Past, present, and future. In F. T. Cullen, J. Wright, & K. Blevens (Eds.), *Taking stock: The status of criminological theory* (Advances in Criminological Theory, Vol. 15, pp. 37-76). New Brunswick, NJ: Transaction.

Akers, R. L., Krohn, M. D., Lanza-Kaduce, L., & Radosevich, M. (1979). Social learning and deviant behavior: A specific test of a general theory. *American Sociological Review*, *44*, 636-655.

Akers, R. L., & LaGreca, A. J. (1991). Alcohol use among the elderly: Social learning, community context, and life events. In D. J. Pittman & H. R. White (Eds.), *Society, culture, and drinking patterns reexamined* (pp. 242-262). New Brunswick, NJ: Rutgers Center of Alcohol Studies.

Akers, R. L., LaGreca, A. J., Cochran, J., & Sellers, C. (1989). Social learning theory and alcohol behavior among the elderly. *Sociology Quarterly*, *30*, 625-638.

Akers, R. L., & Lee, G. (1996). A longitudinal test of social learning theory: Adolescent smoking. *Journal of Drug Education*, *26*, 317-344.

Akers, R. L., Skinner, W. F., Krohn, M. D., & Lauer, R. M. (1987). Recent trends in teenage tobacco use: Findings from a five-year longitudinal study. *Sociology and Social Research*, *71*, 110-114.

ATTTB, Alcohol and Tobacco Tax and Trade Bureau. (2006). *History of alcohol*. Retrieved June 9, 2006, from http://www.ttb.gov/alcohol/history.htm

Allen, M., Donohue, W. A., Griffin, A., Ryan, D., & Mitchell Turner, M. M. (2003). Comparing the influence of parents and peers on the choice to use drugs: A meta-analytic summary of the literature. *Criminal Justice and Behavior*, *30*, 163-186.

Allison, P. D. (1984). *Event history analysis: Regression for longitudinal event data*. (Quantitative Applications in the Social Sciences Series No. 07-046). Beverly Hills, CA: Sage.

American Medical Association. (2005). *Adults most common source of alcohol for teens, according to poll of teens 13-18*. Retrieved July 9, 2007, from http://www.ama-assn.org/ama/pub/category/15414.html

Amey, C. H., & Albrecht, S. L. (1998). Race and ethnic differences in adolescent drug use: The impact of family structure and the quantity and quality of parental interaction. *Journal of Drug Issues*, *28*, 283-299.

Andrews, J. A., Hops, H., Ary, D., Lichtenstein, E., & Tildesley, E. (1991). The construction, validation and use of a Guttman scale of adolescent substance use: An investigation of family relationships. *Journal of Drug Issues*, *21*, 557-572.

Andrews, J. A., Tildesley, E., Hops, H., Duncan, S. C., & Severson, H. H. (2003). Elementary school age children's future intentions and use of substances. *Journal of Clinical Child and Adolescent Psychology*, *32*, 556-567.

Andrews, K. H., & Kandel, B. D. (1979). Attitude and behavior: A specification of the contingent consistency hypothesis. *American Sociological Review*, *44*, 298-310.

Aquilino, W. S. (1997). Privacy effects on self-reported drug use: Interactions with survey mode and respondent characteristics. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 383-415). Rockville, MD: National Institutes of Health.

Arthur, M. W., Hawkins, J. D., Pollard, J. A., Catalano, R. F., & Baglioni, A. J. (2002). Measuring risk and protective factors for substance use, delinquency, and other adolescent problem behaviors: The Communities that Care Youth Survey. *Evaluation Review*, *26*, 575-601.

Ary, D. V., Duncan, T. E., Duncan, S. C., & Hops, H. (1999). Adolescent problem behavior: The influence of parents and peers. *Behaviour Research and Therapy*, *37*, 217-230.

Austin, A. M., Macgowan, M. J., & Wagner, E. J. (2005). Effective family-based interventions for adolescents with substance use problems: A systematic review. *Research on Social Work Practice*, *15*, 67-83.

Babbie, E. (2004). *The practice of social research* (10^{th} ed.). Belmont, CA: Wadsworth/Thomson Learning.

Babor, T., Caetano, R., Casswell, S., Edwards, G., Giesbrecht, N., Graham, K. (2004). Comparing the developmental trajectories of marijuana use of African American and Caucasian adolescents: Patterns, antecedents, and consequences. *Experimental and Clinical Psychopharmacology*, *12*, 47-56.

Babor, T., Caetano, R., Casswell, S., Edwards, G., Giesbrecht, N., & Graham, K. et al. (2003). *Alcohol: No ordinary commodity*. New York: Oxford University Press.

Bachman, J. G., Johnston, L. D., & O'Malley, P. M. (1981). Smoking, drinking and drug use among American high school students: Correlates and trends, 1975-1979. *American Journal of Public Health*, *71*, 59-69.

Bachman, J. G., Johnston, L. D., O'Malley, P. M., & Humphrey, R. H. (1988). Explaining the recent decline in marijuana use: Differentiating the effects of perceived risks, disapproval, and general lifestyle factors. *Journal of Health and Social Behavior*, *29*, 92-112.

Bachman, J. G., & Schulenberg, J. (1993). How part-time work intensity relates to drug use, problem behavior, time use, and satisfaction among high school seniors: Are these consequences or merely correlates? *Developmental Psychology*, *29*, 220-235.

Bachman, R., & O'Malley, P. M. (1981). When four months equal a year: Inconsistencies in students reports of drug use. *Public Opinion Quarterly*, *45*, 536-548.

Bahr, S. J., Marcos, A. C., & Maughan, S. L. (1995). Family, educational and peer influences on the alcohol use of female and male adolescents. *Journal of Studies on Alcohol*, *56*, 457-469.

Bailey, S. L., & Hubbard, R. L. (1990). Developmental variation in the context of marijuana initiation among adolescents. *Journal of Health and Social Behavior*, *31*, 58-70.

Bandura, A. (1977). *Social learning theory*. Englewood Cliff, NJ: Prentice Hall.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173-1182.

Barnes, G. M., & Welte, J. W. (1986). Patterns and predictors of alcohol use among 7th-12th grade students in New York State. *Journal of Studies on Alcohol*, *47*, 53-62.

Barrera, N., Biglan, A., Ary, D., & Li, F. (2001). Replication of a problem behavior model with American-Indian, Hispanic, and Caucasian youth. *Journal of Early Adolescence*, *21*, 133-158.

Bauman, K. E., Botvin, G., Botvin, E. M., & Baker, E. (1992). Normative expectations and the behavior of significant others: An integration of traditions in research on adolescents' cigarette smoking. *Psychological Reports*, *71*, 568-570.

Bauman, K. E., & Ennett, S. T. (1994). Peer influence on adolescent drug use. *American Psychologist*, *49*, 820-822.

Bauman, K. E., & Ennett, S. T. (1996). On the importance of peer influence for adolescent drug use: Commonly neglected considerations. *Addiction*, *91*, 185-198.

Bauman, K. E., & Fisher, L. A. (1986). On the measurement of friend behavior in research on friend influence and selection: Findings from longitudinal studies of adolescent smoking and drinking. *Journal of Youth and Adolescence*, *15*, 345-353.

Baumrind, D. (1990). Familial antecedents of adolescent drug abuse: A developmental perspective. In. C. L. Jones & R. J. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (pp. 13-44). Rockville, MD: National Institute on Drug Abuse.

Baumrind, D., & Moselle, K. A. (1985). A developmental perspective on adolescent drug abuse. *Alcohol and Substance Abuse in Adolescence*, *4*, 41-67.

Beal, A. C., Ausiello, J. & Perrin, J. M. (2001). Social influences on health risk behaviors among minority school students. *Journal of Adolescent Health*, *28*, 474-480.

Beck, K. H., & Treiman, K. A. (1996). The relationship of social context of drinking, perceived social norms, and parental influence to various drinking patterns of adolescents. *Addictive Behaviors*, *21*, 633-644.

Benda, B. B. (1994). Testing competing theoretical concepts: Adolescent alcohol consumption. *Deviant Behavior*, *15*, 375-396.

Bentler, P. M. (1992). Etiologies and consequences of adolescent drug use: Implications for prevention. *Journal of Addictive Diseases*, *11*, 47-61.

Berndt, T. J. (1979). Developmental changes in conformity to peers and parents. *Developmental Psychology*, *15*, 608-616.

Biddle, B. J., Bank, B. J., & Marlin, M. M. (1980). Parent and peer influences on adolescents. *Social Forces*, *58*, 1057-1079.

Biederman, J., Faraone, S. V., Monuteaux, M. C., & Feighner, J. A. (2000). Patterns of alcohol and drug use in adolescents can be predicted by parental substance use disorders. *Pediatrics*, *106*, 792-797.

Black, D. R., Tobler, N.S., & Sciacca, J. P. (1998). Peer helping/involvement: An efficacious way to meet the challenge of reducing alcohol, tobacco, and other drug use among youth? *Journal of School Health*, *68*, 87-93.

Blankmeyer, M., Flannery, D. J., & Vazonyi, A. (2002). The role of aggression and social competence in children's perceptions of the child-teacher relationship. *Psychology in the Schools*, *39*, 293-304.

Blot, W. J. (1992). Alcohol and cancer. *Cancer Research Supplement*, *52*, 2119s-2121s.

Borio, G. (2005). *The tobacco timeline*. Retrieved July 9, 2006, from http://www.tobacco.org/resources/history/Tobacco_History.html

Botvin, G. J. (1999). Adolescent drug prevention: Current findings and future directions. In M. D. Glantz & G. R. Hartel (Eds.), *Drug abuse: Origins and interventions* (pp. 285-308). Washington, DC: American Psychological Association.

Botvin, G. J., Baker, E., Dusenbury, L., Botvin, E. M., & Diaz, T. (1995). Long term follow-up results of a randomized drug abuse prevention trial in a White middle-class population. *Journal of the American Medical Association*, *273*, 1106-1112.

Botvin, G. J., Baker, E., Dusenbury, L., Tortu, S., & Botvin, E. M. (1990). Preventing adolescent drug abuse through a multimodal cognitive-behavioral approach: Results of a three-year study. *Journal of Consulting and Clinical Psychology*, *58*, 437-446.

Botvin, G. J., Baker, E., Filazolla, A. D., & Botvin, E. M. (1990). A cognitive behavioral approach to substance abuse prevention: One-year follow-up. *Addictive Behaviors*, *15*, 47-63.

Botvin, G. J., Baker, E., Renick, N. L., Filazzola, A. D., & Botvin, E. M. (1984). A cognitive-behavioral approach to substance abuse prevention. *Addictive Behaviors*, *9*, 137-147.

Botvin, G. J., Griffin, K. W., Diaz, T., Scheier, L. M., Williams, C., & Epstein, J. A. (2000). Preventing illicit drug use in adolescents: Long-term follow-up data from a randomized control trial of a school population. *Addictive Behavior*, *25*, 774-796.

Botvin, G. J., Scheier, L. M., & Griffin, K. W. (2002). Preventing the onset and developmental progression of adolescent drug use: Implications for the Gateway Hypothesis. In D. B. Kandel (Ed.), *Stages and pathways of drug involvement: Examining the Gateway Hypothesis* (p. 115-138). New York: Cambridge University Press.

Breslau, N., & Peterson, E. L. (1996). Smoking cessation in young adults: Age at initiation of cigarette smoking and other suspected influences. *American Journal of Public Health*, *86*, 214-220.

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Cambridge, MA: Harvard University Press.

Bronfenbrenner, U. (1989). Ecological systems theory. In R. Vasta (Ed.), *Annals of child development* (Vol. 6, pp. 187-251). Greenwich, CT: JAI Press.

Bronfenbrenner, U., Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bio-ecological model. *Psychological Review*, *101*, 568-586.

Brook, J. S. (1993). Interactional theory: Its utility in explaining drug use behavior among African-American and Puerto Rican youth. In M. R. De La Rosa & J. R. Adrados (Eds.), *Drug abuse among minority youth: Methodological issues and recent research advances* (NIDA Monograph No. 30, pp. 79-101). Rockville, MD: National Institute on Drug Abuse.

Brook, J. S., & Brook, D. W. (1990). The psychosocial etiology of adolescent drug use: A family interactional approach. *General, Social and General Psychology Monographs*, *116*, 111-264.

Brook, J. S., Brook, D. W., Arencibia-Mireles, O., Richter, L., & Whiteman, M. (2001). Risk factors for adolescent marijuana use across cultures and across time. *The Journal of Genetic Psychology*, *162*, 357-374.

Brook, J. S., Gordon, A. S., Whiteman, M., & Cohen, P. (1986). Some models and mechanisms for explaining the impact of maternal and adolescent characteristics on adolescent stage of drug use. *Developmental Psychology*, *22*, 460-467.

Brook, J. S., Gordon, A. S., Whiteman, M., & Cohen, P. (1989). Changes in drug involvement: A longitudinal study of childhood and adolescent determinants. *Psychological Reports*, *65*, 707-726.

Brook, J. S., Lukoff, I. F., & Whiteman, M. (1980). Initiation into adolescent marijuana use. *The Journal of Genetic Psychology*, *137*, 133-142.

Brook, J. S., Whiteman, M., & Gordon, A. S. (1983). Stages of drug use in adolescence: Personality, peer, and family correlates. *Developmental Psychology*, *19*, 269-277.

Brook, J. S., Whiteman, M., Gordon, A. S., Nomura, C., & Brook, D. W. (1986). Onset of adolescent drinking: A longitudinal study of intrapersonal and interpersonal antecedents. *Advances in Alcohol and Substance Abuse*, *5*, 91-110.

Brown, E. C., Catalano, R. F., Fleming, C. B., Haggerty, K. P., Abbott, R. D., Cortes, R. R., et al. (2005). Mediator effects in the social development model: An examination of constituent theories. *Criminal Behavior and Mental Health*, *15*, 221-235.

Brown, J. D., & Hudson, T. (2002). *Criterion-based referenced language testing*. New York: Cambridge University Press.

Brown, S. A., Tapert, S. F., Graholm, E., & Delis, D. C. (2000). Neurocognitive functioning of adolescents: Effects of protracted alcohol use. *Alcoholism: Clinical and Experimental Research*, *24*, 164-171.

Browning, K., Thornberry, T., & Porter, P. (1999). *Highlights of findings from the Rochester youth development study* (OJJDP Fact Sheet No. 99103). Washington, DC: U.S. Department of Justice: Office of Justice Programs.

Brunswick, A. F., & Boyle, J. M. (1979). Patterns of drug involvement: Developmental and secular influences on age of initiation. *Youth and Society*, *11*, 139-162.

Bryant, A. L., Schulenberg, J., Bachman, J. G., O'Malley, P. M., & Johnston, L. D. (2000). *Acting out and lighting up: Understanding the links among school misbehavior, academic achievement, and cigarette use* (MTF Occasional Paper No. 46). Ann Arbor, MI: University of Michigan, Institute for Social Research.

Burgess, R., & Akers, R. L. (1966). A differential-association reinforcement theory of criminal behavior. *Social Problems*, *14*, 128-147.

Burkett, S. R., & Warren, B. O. (1987). Religiosity, peer influence, and adolescent marijuana use: A panel study of underlying causal structures. *Criminology*, *25*, 109-131.

Burt, R. D., Dinh, K. T., & Peterson, A. V. (2000). Predicting adolescent smoking: A prospective study of personality variables. *Preventive Medicine*, *30*, 115-125.

Buttkus, C. (2002). *Communities that Care- Squamish: Annual summary report*. Squamish, British Columbia, Canada: Communities that Care- Squamish Advisory Board and Prevention Team.

Carnevale Associates. (2006). *Federal commitment to school-based drug prevention diminishing*. Accessed June 18, 2008, from http://www.carnevaleassociates.com/School_Prevention_Brief.pdf

Carnevale Associates. (2007). *FY 2008 Federal Drug Budget: Prevention funding continues to decline*. Accessed June 18, 2008, from http://www.carnevaleassociates.com/Federal_Drug_Budget_FY08.pdf

Catalano, R. F., Haggerty, K. P., Oesterle, S., Fleming, C. B., & Hawkins, J. D. (2004). The importance of bonding to school for healthy development: Findings from the Social Development Research Group. *Journal of School Health*, *74*, 252-261.

Catalano, R. F., & Hawkins, J. D. (1996). The social development model: A theory of antisocial behavior. In J. D. Hawkins (Ed.), *Delinquency and crime: Current theories* (pp. 149-197). New York: Cambridge University Press.

Catalano, R. F., Park, J., Harachi, T. W., Haggerty, K. P., Abbott, R. D., & Hawkins, J. D. (2005). Mediating the effects of poverty, gender, individual characteristics, and external constraints on antisocial behavior: A test of the social development model and implications for developmental life-course theory. In D. P. Farrington (Ed.), *Advances in criminological theory: Integrated developmental and life-course theories of offending* (Vol. 14, pp. 93-123).New Brunswick, NJ: Transaction Publishers.

Centers for Disease Control and Prevention. (2002). *Cigarette smoking* related mortality. TIPS, Tobacco information and prevention source. Retrieved June 1, 2006, from http://www.cdc.gov/tobacco.htm

Center for the Study and Prevention of Violence. (1999). *Seattle Social Development Project* (Blueprints Promising Programs FS No. BPP17). Boulder, CO: University of Colorado at Boulder, Author.

Cernkovich, S. A., & Giordano, P. C. (1992). School bonding, race, and delinquency. *Criminology*, *30*, 261-291.

Cha, Y. M., White, A. M., Kuhn, C. M., Wilson, W. A., & Swartzwelder, H. S. (2006). Differential effects of delta9-THC on learning behavior in adolescent and adult rats. *Pharmacology, Biochemistry, and Behavior*, *83*, 448-455.

Champion, D. J. (2000). *Research methods for criminal justice and criminology* (2^{nd} ed.) Upper Saddle River, NJ: Prentice Hall.

Charlesworth, A., & Glantz, S. A. (2005). Smoking in movies increases adolescent smoking: A review. *Pediatrics*, *116*, 1516-1528.

Chassin, L. (1984). Adolescent substance use and abuse. In P. Karoly & J. J. Steffen (Eds.), *Adolescent behavior disorders: Foundations and contemporary concerns* (pp. 99-152). Lexington, MA: D. C. Heath and Company.

Chassin, L., Pillow, D. R., Curran, P. J., & Molina, B. S. (1993). Relation of parental alcoholism to early adolescent substance use: A test of three mediating mechanisms. *Journal of Abnormal Psychology*, *102*, 3-19.

Chassin, L., Presson, C., Bensenberg, M., Corty, E., Olshavsky, R., & Sherman, S. J. (1981). Predicting adolescents' intentions to smoke cigarettes. *Journal of Health and Social Behavior*, *22*, 445-455.

Chassin, L., Presson, C. C., Sherman, S. J., Corty, E., & Olshavsky, R. W. (1984). Predicting the onset of cigarette smoking in adolescents: A longitudinal study. *Journal of Applied Social Psychology*, *14*, 224-243.

Chassin, L., Presson, C. C., Sherman, S. J., Montello, D., & McGrew, J. (1986). Changes in peer and parent influence during adolescence: Longitudinal versus cross-sectional perspectives on smoking initiation. *Developmental Psychology*, *22*, 327-334.

Cheadle, A., Pearson, D., Wagner, E., Psaty, B. M., Diehr, P., & Koepsell, T. (1995). A community-based approach to preventing alcohol use among adolescents on an American Indian reservation. *Public Health Reports*, *110*, 439-447.

Chen, K., & Kandel, D. B. (1995). The natural history of drug use from adolescence to the mid-thirties in a general population sample. *American Journal of Public Health*, *85*, 41-47.

Cieply, M. (2007, May 11). Puffing away that PG rating. *New York Times*, pp. C1, C4.

City-Data.com. (2007). Indiana, Pennsylvania. Retrieved June 20, 2007, from http://www.city-data.com/city/Indiana-Pennsylvania.htm

Clarke, J. H., MacPherson, B., Holmes, D. R., & Jones, R. (1986). Reducing adolescent smoking: A comparison of peer-led, teacher-led, and expert interventions. *Journal of School Health*, *56*, 102-106.

Clayton, R. R. (1992). Transitions in drug use: Risk and protective factors. In M. Glantz & R. Pickens (Eds.), *Vulnerability to drug abuse* (pp.15-51). Washington, DC: American Psychological Association.

Clayton, R. R., & Ritter, C. (1985). The epidemiology of alcohol and drug abuse among adolescents. *Alcohol and Substance Abuse in Adolescence*, *27*, 69-97.

Clayton, R. R., Leukefeld, C. G., Donohew, L., Bardo, M., & Harrington, N. G. (1995). Risk and protective factors: A brief review. *Drugs and Society*, *8*, 7-14.

Climent, C. E., de Aragon, L. V., & Plutchik, R. (1990). Prediction of risk for drug use in high school students. *International Journal of the Addictions*, *25*, 545-556.

Clogg, C. C., & Arminger, G. (1993). On strategy for methodological analysis. *Sociological Methodology*, *23*, 57-74.

Clogg, C. C., Petkova, E., & Haritou, A. (1995). Symposium on applied regression: Statistical methods for comparing regression coefficients between models. *American Journal of Sociology*, *100*, 1261-1293.

Clogg, C. C., & Sawyer, D. O. (1981). A comparison of alternative models for analyzing the scalability of response patterns. *Sociological Methodology*, *12*, 240-280.

Cohen, A. (1991). Dummy variables in stepwise regression. *American Statistical Association*, *45*, 226-228.

Cohen, D. A., Richardson, J., & LaBree, L. (1994). Parenting behaviors and the onset of smoking and alcohol use: A longitudinal study. *Pediatrics*, *94*, 368-375.

Cohen, J. (1977). Sources of peer group homogeneity. *Sociology of Education*, *50*, 227-241.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cohen, J. (1992). A power primer. *American Psychologist*, *112*, 155-159.

Coie, J. D., Watt, N. F., West, S. G., Hawkins, J. D., Asarnow, J. R., Markman, H. J. et al. (1993). The science of prevention: A conceptual framework and some directions for a national research program. *American Psychologist*, *48*, 1013-1022.

Colder, C. R., & Stice, E. (1998). A longitudinal study of the interactive effects of impulsivity and anger on adolescent problem behavior. *Journal of Youth and Adolescence*, *27*, 255-274.

Collins, L. M., Sussman, S., Rauch, J. M., Dent, C. W., Johnson, A., Hansen, W. B. et al., (1987). Psychosocial predictors of young adolescent cigarette smoking: A sixteen-month, three-wave longitudinal study. *Journal of Applied Social Psychology*, *17*, 554-573.

Communities that Care. (2004). *CTC Leith launches its action plan* (CTC Newsletter, Issue No. 9). London, England: Author.

Communities that Care. (2006). *Building safer communities where children and young people are valued*: *Edinburgh risk and protection audit report*. Glasglow, Scotland: Edinburgh Council.

Conger, R. D. (1976). Social control and social learning models of delinquency: A synthesis. *Criminology*, *14*, 17-40.

Conger, R. D., & Simons, R. L. (1995). Life-course contingencies in the development of adolescent antisocial behavior: A matching law approach. In T. P. Thornberry (Ed.), *Developmental theories of crime and delinquency* (pp. 55-100). New Brunswick, NJ: Transaction.

Conrad, K. M., Flay, B. R., & Hill, D. (1992). Why children start smoking cigarettes: Predictors of onset. *British Journal of Addiction*, *87*, 1711-1724.

Cook, D. J., Mulrow, C. D., & Haynes, R. B. (1997). Systematic reviews: Synthesis of best evidence for clinical decisions. *Annals of Internal Medicine*, *12*, 376-380.

Cook, T. D., & Leviton, L. C. (1980). Reviewing the literature: A comparison of traditional methods with meta-analysis. *Journal of Personality*, *48*, 449-472.

Cook, P. J., & Tauchen, G. (1984). The effects of minimum drinking age legislation on youthful auto fatalities, 1970-1977. *Journal of Legal Studies*, *15*, 159-162.

Coombs, R., & Landsverk, J. (1988). Parenting styles and substance use during childhood and adolescence. *Journal of Marriage and Family*, *50*, 473-482.

Cooper, H. M. (1982). Scientific guidelines for conducting integrative research reviews. Review of Educational Research, 52, 291-302.

Cooper, H. M., & Arkin, R. M. (1981). On quantitative reviewing. *Journal of Personality*, *49*, 225-230.

Cooper, H. M., & Rosenthal, R. (1980). Statistical versus traditional procedures for summarizing research findings. *Psychological Bulletin*, *87*, 442-449.

Costello, B. J., & Vowell, P. R. (1999). Testing control theory and differential association: A reanalysis of the Richmond youth project data. *Criminology*, *37*, 815-842.

Costello, E.J., Erkanli, A., Federman, E., & Angold, A. (1999). Development of psychiatric comorbidity with substance abuse in adolescents: Effects of timing and sex. *Journal of Clinical Child Psychology*, *28*, 298-311.

Crews, F. T., Braun, C. J., Hoplight, B., Switzer, R. C., & Knapp, D. J. (2000). Binge ethanol consumption causes differential brain damage in young adolescent rats compared with adult rats. *Alcoholism: Clinical and Experimental Research*, *24*, 1712-1723.

Crum, R. M., Storr, C. L., & Anthony, J. C. (2005). Are educational aspirations associated with the risk of alcohol use and alcohol use-related problems among adolescents? *Substance Use and Misuse*, *40*, 151-169.

D’Amico, E. J., Ellickson, P. L., Wagner, E. F., Turrisi, R., Fromme, K., Ghosh Dastidar, B., et al. (2005). Developmental considerations for substance use interventions for middle school through college. *Alcohol Clinical and Experimental Research*, *29*, 474-483.

D'Amico, E. J., & McCarthy, D. M. (2006). Escalation and initiation of younger adolescents' substance use: The impact of perceived peer use. *Journal of Adolescent Health*, *39*, 481-487.

Dalton, M. A., Bernhardt, A. M., Gibson, J. J., Sargent, J. D., Beach, M. L., Adachi-Mejia, A. M. et al. (2005). Use of cigarettes and alcohol by preschoolers while role-playing as adults: "Honey, have some smokes." *Archives of Pediatrics and Adolescent Medicine*, *159*, 854-859.

Danzinger, S. K. (1995). Family life and teenage pregnancy in the inner-city: Experiences of African-American youth. *Children and Youth Services Review*, *17*, 183-202.

Davies, H. T., Nutley, S. M., & Smith, P. C. (2000). *What works? Evidence-based policy and practice in public services*. Bristol, England: The Policy Press.

Dawson, D. A. (2000).The link between family history and early onset alcoholism: Earlier initiation of drinking or more rapid development of dependence? *Journal of Studies on Alcohol*, *61*, 637-646.

Dawson, D. A., & Grant, B. F. (1998). Family history of alcoholism and gender: Their combined effects on DSM-IV alcohol dependence and major depression. *Journal of Studies on Alcohol*, 59, 97-106.

DeBellis, M. D., & Clark, D. B. (2000). Hippocampal volume in adolescent-onset alcohol use disorders. *American Journal of Psychiatry*, *157*, 737-744.

Dembo, R., Grandon, G., La Voie, L., Schmeidler, J., & Burgos, W. (1986). Parents and drug revisited: Some further evidence in support of social learning theory. *Criminology*, *24*, 85-104.

Dembo, R., Schmeidler, J., Taylor, R., & Burgos, W. (1982). Supports for, and consequences of, early drug involvement among inner city junior high school youths living in three neighborhood settings. *Journal of Drug Education*, *12*, 191-209.

Derzon, J. H., & Lipsey, M. W. (1999a). Predicting tobacco use to age 18: A synthesis of longitudinal research. *Addiction*, *94*, 995-1006.

Derzon, J. H., & Lipsey, M. W. (1999b). A synthesis of the relationship of marijuana use with delinquent and problem behaviors. *School Psychology International*, *20*, 57-68.

Derzon, J. H., & Lipsey, M .W. (1999c). What good predictors of marijuana use are good for: A synthesis of research. *School Psychology International*, *20*, 69-85.

DeVellis, R. F. (2003). *Scale development: Theory and applications*. Thousand Oaks, CA: Sage.

Dewey, J. D. (1999). Reviewing the relationship between school factors and substance use for elementary, middle, and high school students. *The Journal of Primary Prevention*, *19*, 177-225.

Dielman, T. E., Butchart, A. T., Shope, J. T., & Miller, M. (1991). Environmental correlates of adolescent use and misuse: Implications for prevention programs. *International Journal of the Addictions*, *25*, 855-880.

Dielman, T. E., Butchart, A. T., & Shope, J. T. (1993). Structural equation model tests of patterns of family interaction, peer alcohol use, and intrapersonal predictors of alcohol use and misuse. *Journal of Drug Education*, *23*, 273-316.

DiFranza, J. R., Savageau, J. A., Fletcher, K., O'Loughlin, J., Pbert, L., Ockene, J. K., et al. (2007). Symptoms of tobacco dependence after brief intermittent use: The development and assessment of nicotine dependence in youth-2 study. *Archives of Pediatrics and Adolescent Medicine*, *161*, 704-710.

DiFranza, J. R., Wellman, R. J., Sargent, J. D., Weitzman, M., Hipple, B. J., & Winickoff, J. P. (2006). Tobacco promotion and the initiation of tobacco use: Assessing evidence for causality. *Pediatrics*, *117*, 1237-1248.

Dishion, T. J., Capaldi, D., Spracklen, K. M., & Li, F. (1995). Peer ecology of male adolescent drug use. *Development and Psychopathology*, *7*, 803-824.

Dishion, T. J., Capaldi, D. M., & Yoerger, K. (1999). Middle childhood antecedents to progressions in male adolescent substance use: An ecological analysis of risk and protection. *Journal of Adolescent Research*, *14*, 175-205.

Dishion, T. J., Capaldi, D., Spracklen, K. M., & Li, F. (1995). Peer ecology of male adolescent drug use. *Development and Psychopathology*, *7*, 803-824.

Dishion, T. J., & Loeber, R. (1985). Male adolescent marijuana and alcohol use: The role of parents and peers revisited. *American Journal of Drug and Alcohol Abuse*, *11*, 11-25.

Dishion, T. J., McCord, J., & Poulin, F. (1999). When interventions harm: Peer groups and problem behavior. *American Psychologist*, *54*, 755-764.

Dittus, P., & Jaccard, J. (2000). Adolescents' perceptions of maternal disapproval of sex: Relationship to sexual outcomes. *Journal of Adolescent Health*, *26*, 268-278.

Dombrink, J. (1993). Victimless crimes and the culture wars of the 1990s. *Journal of Contemporary Criminal Justice*, *9*, 30-40.

Donnermeyer, J. F. (1992). The use of alcohol, marijuana, and hard drugs by rural adolescents: A review of recent research. *Drugs and Society*, *7*, 31-75.

Donnermeyer, J. F. (1993). Rural youth usage of alcohol, marijuana, and "hard" drugs. *International Journal of the Addictions*, *28*, 249-255.

Donovan, J. E. (2004). Adolescent alcohol initiation: A review of psychosocial risk factors. *Journal of Adolescent Health*, *35*, 529e527-529e518.

Donovan, J. E. (2007). Really underage drinkers: The epidemiology of children's alcohol use in the United States. *Prevention Science*, *8*, 192-205.

Donovan, J. E., & Jessor, R. (1983). Problem drinking and the dimension of involvement with drugs: A Guttman scalogram analysis of adolescent drug use. *American Journal of Public Health*, *73*, 543-552.

Dorsett, D., & Webster, J .T. (1983). Guidelines for variable selection problems when dummy variables are used. *The American Statistician*, *37*, 337-339.

DEA, Drug Enforcement Agency. (2006). *Controlled Substances Act*. Retrieved June 12, 2006, from http://www.usdoj.gov/dea/agency/csa.htm

Drug Strategies. (1999). *Making the grade: A guide to school drug prevention programs*. Washington, DC: Author.

Dryfoos, J .G. (1996). Adolescents at risk: Shaping programs to fit the need. *Journal of Negro Education*, 65, 5-18.

Dull, R. T. (1984). An empirical examination of the social bond theory of drug use. *International Journal of the Addictions*, *19*, 265-286.

Duncan, S. C., Duncan, T. E., & Hops, H. (1998). Progressions of alcohol, cigarette, and marijuana use in adolescence. *Journal of Behavioral Medicine*, *21*, 375-388.

Duncan, T. E., Tildesley, E., Duncan, S. C., & Hops, H. (1995). The consistency of family and peer influences on the development of substance use in adolescence. *Addiction*, *90*, 1647-1660.

Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Orlando, FL: Harcourt Brace Jovanovich.

Edwards, A. (1948). On Guttman's scale analysis. *Educational and Psychological Measurement*, *8*, 313-318.

Ellickson, P. L., D'Amico, E., Collins, R., & Klein, D. (2005). Marijuana use and later problems: When frequency of recent use explains age of initiation effects (and when it does not). *Substance Use and Misuse*, *40*, 343-359.

Ellickson, P. L., Hays, R. D., & Bell, R. M. (1992). Stepping through the drug use sequence: Longitudinal scalogram analysis of initiation and regular use. *Journal of Abnormal Psychology*, *101*, 441-451.

Ellickson, P. L., Tucker, J., Klein, D., & McGuigan, K. (2001). Prospective risk factors for alcohol misuse in late adolescence. *Journal of Studies on Alcohol*, *62*, 773-782.

Ellickson, P. L., Tucker, J. S., Klein, D. J., & Saner, H. (2004). Antecedents and outcomes of marijuana use initiation during adolescence. *Preventive Medicine*, *39*, 976-984.

Elliott, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use*. Beverly Hills, CA: Sage.

Elliott, D. C., Huizinga, D., & Menard, S. (1989). *Multiple problem behaviors: Delinquency, substance use, and mental health problems*. New York: Springer-Verlag.

Empey, L. T., & Lubeck, S. G. (1971). *Explaining delinquency*. Lexington, MA: Heath.

Engels, R. C., & Bot, S. M. (2006). Social influences on adolescent substance use. In D. DeRidder & J. De Wit (Eds.), *New perspectives on health behavior: The role of self-regulation* (pp. 71-96). Hoboken, NJ: Wiley.

Ennett, S. T., & Bauman, K .E. (1994). The contribution of influence and selection to adolescent peer group homogeneity: The case of adolescent cigarette smoking. *Journal of Personality and Social Psychology*, *67*, 653-663.

Ennett, S. T., Bauman, K. E., Foshee, V. A., Pemberton, M., & Hicks, K. A. (2001). Parent-child communication about adolescent tobacco and alcohol use: What do parents say and does it affect youth behavior? *Journal of Marriage and Family*, *63*, 48-62.

Ennett, S. T., Bauman, K. E., Hussong, A., Faris, R., Foshee, V. A., Cai, L., et al. (2006). The peer context of adolescent substance use: Findings from social network analysis. *Journal of Research on Adolescence*, *16*, 159-186.

Ensminger, M. E., Brown, C. H., & Kellam, S. G. (1982). Sex differences in antecedents of substance use among adolescents. *Journal of Social Issues*, *38*, 25-42.

Epstein, J. A., Botvin, G. J., Baker, E., & Diaz, T. (1999). Impact of social influences and problem behavior on alcohol use among inner-city Hispanic and black adolescents. *Journal of Studies on Alcohol*, *60*, 595-604.

Epstein, J. A., Botvin, G. J., Diaz, T., Toth, V., & Schinke, S. P. (1995). Social and personal factors in marijuana use and intentions to use drugs among inner city minority youth. *Developmental and Behavioral Pediatrics*, *16*, 14-20.

Farrell, A. D. (1994). Structural equation modeling with longitudinal data: Strategies for examining group differences and reciprocal relationships. *Journal of Consulting and Clinical Psychology*, *62*, 477-487.

Farrington, D. P. (1986). Age and crime. In M. Tonry & N. Morris (Eds.), *Crime and justice: An annual review of research* (Vol. 7, pp. 189-250). Chicago: University of Chicago.

Farrington, D. P. (2003). Developmental and life-course criminology: Key theoretical and empirical issues- The 2002 Sutherland Award Address. *Criminology*, *41*, 221-255.

Farrington, D. P. (Ed.) (2005). *Integrated developmental and life-course theories of offending* (Advances in Criminological Theory, Vol. 14). New Brunswick, NJ: Transaction.

Farrington, D. P., & Coid, J. W. (2003). Conclusions and the way forward. In D. P. Farrington & J. W. Coid (Eds.), *Early prevention of adult antisocial behavior* (pp. 356-369). New York: Cambridge University Press.

Farrington, D. P., & Weisburd, D. L. (2007). The Campbell Collaboration Crime and Justice Group. *The Criminologist*, *32*, 3-5.

Farrington, D. P., & Welsh, B. C. (2001). Systematic reviews and cost-benefit analyses of correctional interventions. *Prison Journal*, *81*, 339-359.

Federman, E. B., Costello, E. J., Angold, A., Farmer, E. M., & Erkanli, A. (1997). Development of substance use and psychiatric comorbidity in an epidemiologic study of white and American Indiana young adolescents: The Great Smoky Mountains Study. *Drug and Alcohol Dependence*, *44*, 69-78.

Feldman, K. A. (1971). Using the work of others: Some observations on reviewing and interpreting. *Sociology of Education*, *44*, 86-102.

Fisher, D. G., MacKinnon, D. P., Anglin, M. D., & Thompson, J. P. (1987). Parental influences on substance use: Gender differences and stage theory. *Journal of Drug Education*, *17*, 69-86.

Fisher, L. A., & Bauman, K. E. (1988). Influence and selection in the friend adolescent relationship: Findings from studies of adolescent smoking and drinking. *Journal of Applied Social Psychology*, *18*, 289-314.

Flack, V. F., & Chang, P. C. (1987). Frequency of selecting noise variables in subset regression analysis: A simulation study. *The American Statistician*, *41*, 84-86.

Flay, B. R., Hu, F. B., & Richardson, J. (1998). Psychosocial predictors of different stages of cigarette smoking among high school students. *Preventive Medicine*, *27*, A9-A18.

Flay, B. R., Hu, F. B., Siddiqui, O., Day, L. E., Hedeker, D., Petraitis, J., et al. (1994). Differential influence of parental smoking and friends smoking on adolescent initiation and escalation of smoking. *Journal of Health and Social Behavior*, *35*, 248-265.

Flay, B. R., & Petraitis, J. (1991). Methodological issues in drug use prevention research: Theoretical foundations. In C. G. Leukefeld & W. J. Bukowski (Eds.), *Drug abuse prevention intervention research: Methodological issues* (pp. 81-109). Rockville, MD: U.S. Department of Health and Human Services.

Flay, B. R., & Petraitis, J. (1994). A theory of triadic influence: A new theory of health behavior with implications for prevention. In G. L. Albrecht (Ed.), *Advances in medical sociology: A reconsideration of health behavior changes models* (Vol. 4, pp. 19-44). Greenwich, CT: JAI Press.

Fleming, J. P., Kellam, S. G., & Brown, C. H. (1982). Early predictors of age at first use of alcohol, marijuana, and cigarettes. *Drug and Alcohol Dependence*, *9*, 285-303.

Fleming, R., Leventhal, H., Glynn, K., & Ershler, J. (1989). The role of cigarettes in the initiation and progression of early substance use. *Addictive Behaviors*, *14*, 261-272.

Flewelling, R. L., & Bauman, K. E. (1990). Family structure as a predictor of initial substance use and sexual intercourse in early adolescence. *Journal of Marriage and Family*, *52*, 171-181.

Forslund, M. A., & Gustafsen, T. J. (1970). Influences of peers and parents and sex differences in drinking by high-school students. *Quarterly Journal of Studies on Alcohol*, *31*, 868-875.

Foshee, V., & Bauman, K. E. (1992). Parental and peer characteristics as modifiers of the bond-behavior relationship: An elaboration of control theory. *Journal of Health and Social Behavior*, *33*, 66-76.

Fraser, M. W. (1996). Cognitive problem-solving and aggressive behavior among children. *Families in Society*, *77*, 19-32.

George, D., & Mallery, P. (2006). *SPSS for Windows step by step: A simple guide and reference, 13.0 update* (6th ed.). New York: Allyn and Bacon.

Gfroerer, J. C., & Epstein, J. A. (1998). Marijuana initiates and their impact on future drug abuse treatment need. *Drug and Alcohol Dependence*, *54*, 229-237.

Gfroerer, J. C., Wu, L. T., & Penne, M. A. (2002). *Initiation of marijuana use: Trends, patterns and implications* (Report No. SMA 02-3711). Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Office of Applied Studies.

Giedd (2004). Structural magnetic resonance imaging of the adolescent brain. *Annals of the New York Academy of Science*, *1021*, 77-85.

Gillmore, M. R., Catalano, R. F., Morrison, D. M., Wells, E. A., Iritani, B., & Hawkins, J. D. (1990). Racial differences in acceptability and availability of drugs and early initiation of substance use. *American Journal of Alcohol Abuse*, *16*, 185-206.

Ginsberg, I. J., & Greenley, J. R. (1978). Competing theories of marijuana use: A longitudinal study. *Journal of Health and Social Behavior*, *19*, 22-34.

Glantz, M. (1992). A developmental psychopathology model of drug abuse vulnerability. In M. Glantz & R. Pickens (Eds.), *Vulnerability to drug abuse* (pp. 389-418). Washington, DC: American Psychological Association.

Glass, G. V. (1977). Integrating findings: The meta-analysis of research. *Review of Research in Education*, *5*, 351-379.

Glynn, T. J. (1981). From family to peers: A review of transitions of influence among drug-using youth. *Journal of Youth and Adolescence*, *10*, 363-383.

Golub, A., & Johnson, B. D. (2001). Variation in youthful risks of progression from alcohol and tobacco to marijuana and to hard drugs across generations. *American Journal of Public Health*, *91*, 225-232.

Golub, A., & Johnson, D. B. (2002). Substance use progression and hard drug use in inner-city New York. In D. B. Kandel (Ed.), *Stages and pathways of drug involvement: Examining the Gateway Hypothesis* (pp. 90-112). New York: Cambridge University Press.

Golub, A., Johnson, B. D., & Labouvie, E. (2000a). On correcting bias in self reports of age at first substance use with repeated cross-section analysis. *Journal of Quantitative Criminology*, *16*, 45-68.

Golub, A., Labouvie, E., & Johnson, B. D. (2000b). Response reliability and the study of adolescent substance use progression. *Journal of Drug Issues*, *30*, 103-118.

Goodenough, W. H. (1944). A technique for scale analysis. *Educational and Psychological Measurement*, 4, 179-190.

Gorman, D. M. (1996). Etiological theories and the primary prevention of drug use. *Journal of Drug Issues*, *26*, 505-516.

Gorsuch, R. L. & Butler, M. C. (1976). Initial drug abuse: A review of predisposing social psychological factors. *Psychological Bulletin*, *83*, 120-137.

Gottfredson, D. C. (2001). *Schools and delinquency*. New York: Cambridge University Press.

Gottfredson, M. & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.

Gould, L. C., Berberian, R. M., Kasl, S. V., Thompson, W. D., & Kleber, H. D. (1977). Sequential patterns of multiple-drug use among high school students. *Archives of General Psychiatry*, *34*, 216-222.

Graham, S. (1995). Narrative versus meta-analytic reviews of race differences in motivation: A comment on Cooper and Door. *Review of Educational Research*, *65*, 509-514.

Grant, B. F., & Dawson, D. A. (1997). Age of onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Study. *Journal of Substance Abuse*, *9*, 103-110.

Grant, B. F., & Dawson, D. A. (1998). Age of onset of drug use and its association with DSM-IV drug abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse*, *10*, 163-173.

Gray, J. P. (2001). *Why our drug laws have failed and what we can do about it: A judicial indictment of the war on drugs*. Philadelphia, PA: Temple University Press.

Green, S. B. (1991). How many subjects does it take to do a regression analysis? *Multivariate Behavioral Research*, *26*, 499-510.

Greenberger, E., & Steinberg, L. (1986). *When teenagers work: The psychological and social costs of adolescent employment*. New York: Basic.

Griesler, P. C., & Kandel, D. B. (1998). Ethnic differences in correlates of adolescent cigarette smoking. *Journal of Adolescent Health*, *23*, 167-180.

Grinspoon, L. (1971). *Marijuana reconsidered*. Cambridge, MA: Harvard University Press.

Gritz, E. R., Prokhorov, A. V., Hudmon, K. S., Chamberlain, R. M., Taylor, W. C., DiClimente, C. C., et al. (1998). Cigarette smoking in a multiethnic population of youth: Methods and baseline findings. *Preventive Medicine*, *27*, 365-384.

Gritz, E. R., Prokhorov, A. V., Hudmon, K. S., Jones, M. M., Rosenblum, C., Chang, C. C., et al. (2003). Predictors of susceptibility to smoking and ever smoking: A longitudinal study in a triethnic sample of adolescents. *Nicotine and Tobacco Research*, *5*, 493-506.

Grube, J .W. (2004). Alcohol in the media: Drinking portrayals, alcohol advertising, and alcohol consumption among youth. In R. J. Bonnie & M. E. O'Connell (Eds.), *Underage drinking: A collective responsibility* (pp. 597-624). Washington, DC: The National Academies Press.

Gruber, E., DiClemente, R. J., Anderson, M. M., & Lodico, M. (1996). Early drinking onset and its association with alcohol use and problem behavior in late adolescence. *Prevention Medicine*, *25*, 293-300.

Guerra, L. M., Romano, P. S., Samuels, S. J., & Kass, P. H. (2000). Ethnic differences in adolescent substance initiation sequences. *Archives of Pediatrics and Adolescent Medicine*, *154*, 1089-1095.

Guo, J., Hill, K. G., Hawkins, J. D., Catalano, R. F., & Abbott, R. D. (2002). A developmental analysis of sociodemographic, family, and peer effects on adolescent illicit drug initiation. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 838-845.

Guttman, L. L. (1944). A basis for scaling qualitative data. *American Sociological Review*, *9*, 139-150.

Guttman, L. L. (1950). The basis for scalogram analysis. In S. A. Stouffer, L. Guttman, E. A. Suchman, P. F. Lazersfield, S. A. Star, & J. A. Clausen (Eds.), *Measurement and prediction* (pp. 60-90). Princeton, NJ: Princeton University Press.

Halebsky, M. A. (1987). Adolescent alcohol and substance use: Parent and peer effects. *Adolescence*, *22*, 961-967.

Halfors, D., Vevea, J. L., Iritani, B., Cho, H., Khatapoush, S., & Saxe, L. (2002). Truancy, grade point average, and sexual activity: A meta-analysis of risk indicators for youth substance use. *Journal of School Health*, *72*, 205-211.

Hamburg, B. A., Kraemer, H. C., & Jahnke, W. (1975). A hierarchy of drug use in adolescence: Behavioral and attitudinal correlates of substantial drug use. *American Journal of Psychiatry*, *132*, 1155-1163.

Hansen, W. B. (1993). School-based alcohol prevention programs. *Alcohol Research and Health*, *17*, 54-60.

Hansen, W. B., & Collins, L. M. (1994). Seven ways to increase power without increasing N. In L. M. Collins & L. A. Seitz (Eds.), *Advances in data analysis for prevention intervention research* (pp. 184-195). Rockville, MD: National Institutes of Health.

Hansen, W. B., Miller, T. W., & Leukefeld, C. G. (1995). Prevention research recommendations: Scientific integration in the 90s. *Drugs and Society*, *8*, 161-167.

Harrell, A. V. (1997). The validity of self-reported drug use data: The accuracy of responses on confidential self-administered answer sheets. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (pp. 37-58, NIDA Research Monograph 167, NIH Report No. 97-4147). Rockville, MD: National Institutes of Health.

Harrison, L. D. (1995). The validity of self-reported data on drug use. *Journal of* Drug Issues, *25*, 91-111.

Harrison, L. D. (1997). The validity of self-reported drug use in survey research: An overview and critique of research methods. In L. D. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 17-36). Rockville, MD: National Institutes of Health.

Harrison, L. D. (2001). Understanding the differences in youth drug prevalence rates produced by the MTF, NHSDA, and YRBS studies. *Journal of Drug Issues*, *31*, 665-694.

Harrison, L., & Hughes, A. (1997). Introduction- The validity of self-reported drug use: Improving the accuracy of survey estimates. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 1-15). Rockville, MD: National Institutes of Health.

Hastings, G., Anderson, S., Cooke, E., & Gordon, R. (2005). Alcohol marketing and young people's drinking: A review of the research. *Journal of Public Health Policy*, *26*, 296-311.

Hauser-Cram, P. (1983). Some cautions in synthesizing research studies. *Educational Evaluation and Policy Analysis*, *5*, 155-162.

Hawden, J. E. (1999). Daily routines and crime: Using routine activities as measures of Hirschi's involvement. *Youth and Society*, *30*, 395-415.

Hawkins, J. D., Catalano, R. F., & Arthur, M. W. (2002a). Promoting science based prevention in communities. *Addictive Behaviors*, *27*, 951-976.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992a). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin*, *112*, 64-105.

Hawkins, J. D., Catalano, R. F., Morrison, D. M., O'Donnell, J., Abbott, R. D., & Day, L. E. (1992b). The Seattle Social Development Project: Effects of the first four years on protective factors and problem behaviors. In J. McCord & R. E. Tremblay (Eds.), *Preventing antisocial behavior: Interventions from birth through adolescence* (pp. 139-161). New York: Guilford Press.

Hawkins, J. D., Doueck, H. J., & Lishner, D. M. (1988). Changing teaching practices in mainstream classrooms to improve bonding and behavior of low achievers. *American Education Research Journal*, *25*, 31-50.

Hawkins, J. D., Graham, J. W., Maguin, E., Abbott, R., Hill, K. G., & Catalano, R. F. (1997). Exploring the effects of age of alcohol use initiation and psychosocial risk factors on subsequent alcohol misuse. *Journal of Studies on Alcohol*, *58*, 280-290.

Hawkins, J. D., Guo, J., Hill, K. G., Battin-Pearson, S., & Abbott, R. D. (2001). Long-term effects of the Seattle Social Development intervention on school bonding trajectories. *Applied Developmental Science*, *5*, 225-236.

Hawkins, J. D., Hill, K. G., Guo, J., & Battin-Pearson, S. R. (2002b). Substance use norms and transitions in substance use: Implications for the Gateway Hypothesis. In D. B. Kandel (Ed.), *Stages and pathways for drug involvement: Examining the Gateway Hypothesis* (pp. 42-64). New York: Cambridge University Press.

Hawkins, J. D., Kosterman, R., Catalano, R. F., Hill, K. G., & Abbott, R. D. (2005). Promoting positive adult functioning through social development interventions in childhood: Long-term effects from the Seattle Social Development Project. *Archives of Pediatric and Adolescent Medicine*, *159*, 25-31.

Hawkins, J. D., & Weis, J. G. (1985). The social development model: An integrated approach to delinquency prevention. *Journal of Primary Prevention*, *6*, 73-97.

Heimer, K. (1995). Gender, race, and the pathways to delinquency: An interactionist explanation. In J. Hagan and R. D. Peterson (Eds.), *Crime and inequality* (pp. 140-173). Stanford, CA: Stanford University Press.

Hill, K. G., White, H. R., Chung, I.-J., Hawkins, J. D., & Catalano, R. F. (2000a). Early adult outcomes of adolescent binge drinking: person- and variable-centered analyses of binge drinking. *Alcoholism: Clinical and Experimental Research*, *24*, 892-901.

Hill, S. Y., Shen, S., Lowers, L., & Locke, J. (2000b). Factors predicting the onset of adolescent drinking in families at high risk for developing alcoholism. *Biological Psychiatry*, *48*, 265-275.

Hindelang, M. (1973). Causes of delinquency: A partial replication and extension. *Social Problems*, *20*, 471-487.

Hindelang, M. J., Hirschi, T., & Weis, J. G. (1981). *Measuring delinquency*. Beverly Hills, CA: Sage.

Hingson, R. W., Heeren, T., & Winter, M. R. (2006a). Age at drinking onset and alcohol dependence: Age at onset, duration, and severity. *Archives of Pediatrics and Adolescent Medicine*, *160*, 739-746.

Hingson, R. W., Heeren, T., & Winter, M. R. (2006b). Age of alcohol-dependence onset: Associations with severity of dependence and seeking treatment. *Pediatrics*, *118*, 755-763.

Hingson, R., & Kenkel, D. (2004). Social, health, and economic consequences of underage drinking. In R. J. Bonnie & M .E. O'Connell (Eds.), *Reducing underage drinking: A collective responsibility* (pp. 351-382). Washington, DC: National Academies Press.

Hirschi, T. (1969). *Causes of delinquency*. Los Angeles: University of California Press.

Hops, H., Davis, B., & Levin, L. M. (1999). The development of alcohol and other substance use: A gender study of family and peer context. *Journal of Studies on Alcohol*, *13*, 22-31.

Howell, J. C., & Hawkins, J. D. (1998). Prevention of youth violence. In M. Tonry & M. H. Moore (Eds.), *Youth violence: Crime and justice* (Vol. 24, pp. 263-315). Chicago: University of Chicago.

Hruska, R. (2001). *Public schools, Elementary enrollments: 2000-01*. Retrieved December 17, 2007, from http://www.pde.state.pa.us/ k12statistics/LIB/k12statistics/enroll0001/psEE0001.pdf

Hruska, R. (2003). *Public secondary school dropouts by school, 2001-02*. Retrieved October 17, 2007, from http://pde.state.pa.us/k12statistics/lib/k12statistics/01-02pubdropsbyschool.pdf

Hruska, R. (2004a). *Public schools, Elementary enrollments: 2003-04*. Retrieved December 17, 2007, from http://www.able.state.pa.us/k12statistics/lib/K12statistics/03-04pelenroll.pdf

Hruska, R. (2004b). *Public schools, Secondary enrollments: 2003-04*. Retrieved December 17, 2007, from http://www.able.state.pa.us/k12statistics/lib/k12statistics/03-04psecenr2.pdf

Hruska, R. (2005). *Public secondary school dropouts by school, 2003-04*. Retrieved October 17, 2007, from http://pde/state/pa/us/k12statistics/lib/k12statistics/2003-04DROPOUTLISTINGrev1.pdf

Hser, Y. (1997). Self-reported drug use: Results of selected empirical investigations of validity. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (pp. 320-343, NIDA Research Monograph 167, NIH Report No. 97-4147). Rockville, MD: National Institutes of Health.

Huang, B., Kosterman, R., Catalano, R. F., Hawkins, J. D., & Abbott, R. D. (2001). Modeling mediation in the etiology of violence behavior in adolescence: A test of the Social Development Model. *Criminology*, *39*, 75-108.

Huba, G. J., & Bentler, P. M. (1980). The role of peer and adult models for drug taking at different stages of adolescence. *Journal of Youth and Adolescence*, *9*, 449-465.

Huba, G. J., & Bentler, P. M. (1982). A developmental theory of drug use: Derivation and assessment of a causal modeling approach. In P. B. Baltes & O. B. Brim (Eds.), *Lifespan development and behavior* (Vol. 4, pp. 147-203). New York: Academic Press.

Huba, G. J., Wingard, J. A., & Bentler, P. M. (1981). A comparison of two latent variable causal models for adolescent drug use. *Journal of Personality and Social Psychology*, *40*, 180-193.

Huizinga, D., Esbensen, F. A., & Weiher, A. W. (1991). Are there multiple routes to delinquency? *The Journal of Criminal Law and Criminology*, *82*, 83-118.

Hundleby, J. D., & Mercer, G. W. (1987). Family and friends as social environments and their relationship to young adolescents' use of alcohol, tobacco, and marijuana. *Journal of Marriage and Family*, *49*, 151-164.

Hwang, S., & Akers, R. L. (2003). Substance use by Korean adolescents: A cross-cultural test of social learning, social bonding, and self-control theories. In R. L. Akers & G. F. Jensen (Eds.), *Social learning and the explanation of crime: A guide for the new century* (Advances in Criminological Theory, Vol. 11, pp. 39-64). New Brunswick, NJ: Transaction.

Hwang, Y. G. (1995). Student apathy, lack of self-responsibility and false self-esteem are failing American schools. *Education*, *115*, 484-490.

Imperato, P. J., & Mitchell, G. (1986). Cigarette smoking: A chosen risk. *New York State Journal of Medicine*, *86*, 485-489.

Inaba, D. S., & Cohen, W. E. (2004). *Uppers, downers, and all arounders: Physical and mental effects of psychoactive drugs* (5th ed.). Ashland, Oregon: CNS Productions, Inc.

Indiana Area School District. (2007). General information. Accessed June 20, 2007, from http://www.iasd.cc/GeneralInformation.htm

Jackson, G. B. (1980). Methods for integrative reviews. *Review of Educational Research*, *50*, 438-460.

Jensen, G. F., & Brownfield, D. (1983). Parents and drugs. *Criminology*, *21*, 543-554.

Jenson, J. M. (2004). Risk and protective factors for alcohol and other drug use in childhood and adolescence. In M. W. Fraser (Ed.), *Risk and resilience in childhood: An ecological perspective* (2nd ed., pp. 183-208). Washington, DC: National Association of Social Workers Press.

Jessor, R. (1976). Predicting time of onset of marijuana use: A developmental study of high school youth. *Journal of Consulting and Clinical Psychology*, *44*, 125-134.

Jessor, R. (1978). Psychosocial factors in the patterning of drinking behavior. In] J. Fishman (Ed.), *The bases of addiction* (pp. 67-80). Berlin, Federal Republic of Germany: Dahlem Konferenzen.

Jessor, R. (1992). Risk behavior in adolescence: A psychosocial framework for understanding and action. *Developmental Review*, *12*, 374-390.

Jessor, R., Chase, J. A., & Donovan, J. E. (1980). Psychosocial correlates of marijuana use and problem drinking in a national sample of adolescents. *American Journal of Public Health*, *70*, 604-613.

Jessor, R., & Jessor, S. L. (1975). *Problem behavior and psychosocial development: A longitudinal study of youth*. New York: Academic Press.

Jessor, R., & Jessor, S. L. (1978). Theory testing in longitudinal research on marijuana use. In D. B. Kandel (Ed.), *Longitudinal research on drug use: Empirical findings and methodological issues* (pp. 41-71). New York: John Wiley and Sons.

Jessor, R., & Jessor, S. L. (1980). A social-psychological framework for studying drug use. In D. Lattieri, M. Sayers, & H. Pearson (Eds.), *Theories on drug abuse: Selected contemporary perspectives* (pp. 102-109). Rockville, MD: National Institute on Drug Abuse.

Johnson, E. O., & Schultz, L. (2005). Forward telescoping bias in reported age of onset: An example from cigarette smoking. *International Journal of Methods in Psychiatric Research*, 14, 119-129.

Johnson, L. D. (1996). *Changing trends, patterns, and nature of marijuana use*. Paper presented at the National Conference on Marijuana Use: Prevention, Treatment, and Research, Washington, DC.

Johnson, K. A. (1984). The applicability of social control theory in understanding adolescent alcohol use. *Sociological Spectrum*, *4*, 275-294.

Johnson, K. A. (1986). Informal control networks and adolescent orientations toward alcohol use. *Adolescence*, *21*, 767-784.

Johnson, K. A., Bryant, D. D., Collins, D. A., Noe, T. D., Strader, T. N., & Berbaum, M. (1998). Preventing and reducing alcohol and other drug use among high-risk youths by increasing family resilience. *Social Work*, *43*, 297-308.

Johnson, R. E., Marcos, A. C., & Bahr, S. J. (1987). The role of peers in the complex etiology of adolescent drug use. *Criminology*, *25*, 323-340.

Johnston, L. D. (1985). The etiology and prevention of substance use: What can we learn from recent historical changes? In C. L. Jones & R. J. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (pp. 155-177). Rockville, MD: National Institute on Drug Abuse.

Johnston, L. D., Bachman, J. G., & O'Malley, P. (1992). *Monitoring the future: Questionnaire responses from the nation's high school seniors, 1989*. Ann Arbor, MI: Institute for Social Research.

Johnston, L. D., & O'Malley, P. (1997). The recanting of earlier reported drug use by young adults. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 59-80). Rockville, MD: National Institutes of Health.

Johnston, L. D., O'Malley, P., & Bachman, J. G. (1996). *National survey results on drug use from the Monitoring the Future Study, 1975-1995* (NIH Report No. 96-4139). Rockville, MD: National Institute on Drug Abuse.

Johnston, L. D., O'Malley, P., & Bachman, J. G. (1998). *National survey results on drug use from the Monitoring the Future Study, 1975-1997: Volume I. Secondary school students* (NIH Publication No. 98-4345). Rockville, MD: National Institute on Drug Abuse.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2006). *Monitoring the Future national survey results on drug use, 1975-2005. Volume I: Secondary school students* (NIH Report No. 06-5883). Bethesda, MD: National Institute on Drug Abuse.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2007). *Monitoring the Future national survey results on adolescent drug use: Overview of key findings, 2006* (NIH Report No. 07-6202). Bethesda, MD: National Institute on Drug Abuse.

Joy, J. E., Watson, S. J., & Benson, J. A. (Eds.). (1999). *Marijuana and medicine: Assessing the science base*. Washington, DC: National Academy Press.

J. S. White Surveys. (2004). *Indiana Area School District: Primary Prevention Awareness, Attitude, and Use Survey, Results of the PPAAUS 2004*. State College, PA: Author.

Junger-Tas, J., & Marshall, I. H. (1999). The self-report methodology in crime research. In M. Tonry (Ed.), *Crime and justice: An annual review of research* (Vol. 25, pp. 291-367). Chicago: University of Chicago Press.

Jurgensen, J. (2007, July 20). Drinks, drugs, dysfunction star on summer cable TV. *Wall Street Journal- Eastern Edition*, pp. B1, B6.

Kachigan, S. K. (1986). *Multivariate statistical analysis: A conceptual Introduction* (2nd ed.). New York: Radius Press.

Kandel, D. B. (1973). Adolescent marijuana use: Role of parents and peers. *Science*, *181*, 1067-1070.

Kandel, D. B. (1974). Inter- and intragenerational influences on adolescent marijuana use. *Journal of Social Issues*, *30*, 107-135.

Kandel, D. B. (1975a). The measurement of "ever use" and "frequency quantity" in drug use surveys. In J. Elinson & D. Nurco (Eds.), *Operational definitions in socio-behavioral drug use research 1975* (NIDA Research Monograph No. 2, NTIS Report No. 246-338, pp. 27-35). Rockville, MD: National Institute on Drug Abuse.

Kandel, D. B. (1975b). Stages in adolescent involvement in drug use. *Science*, *190*, 912-914.

Kandel, D. B. (1978a). Convergence in prospective longitudinal surveys of drug use in normal populations. In D. B. Kandel (Ed.), *Longitudinal research on drug use: Empirical findings and methodological issues* (pp. 3-38). New York: John Wiley and Sons.

Kandel, D. B. (1978b). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology*, *84*, 427-436.

Kandel, D. B. (1978c). Similarity in real life adolescent friendship pairs. *Journal of Personality and Social Psychology*, *36*, 306-312.

Kandel, D. B. (1980a). Developmental stages of adolescent drug involvement. In D. Lattieri, M. Sayers, & H. Pearson (Eds.), *Theories on drug abuse: Selected contemporary perspectives* (pp. 120-127). Rockville, MD: National Institute on Drug Abuse.

Kandel, D. B. (1980b). Drug and drinking behavior among youth. *Annual Review of Sociology*, *6*, 235-285.

Kandel, D. B. (1981). Drug use by youth: An overview. In D. L. Lettieri & J. P. Ludford (Eds.), *Drug abuse and the American adolescent* (NIDA Research Monograph 38, pp. 1-24). Rockville, MD: National Institute on Drug Abuse.

Kandel, D. B. (1982). Epidemiological and psychosocial perspectives on adolescent drug use. *Journal of the American Academy of Child and Adolescent Psychiatry*, *21*, 328-347.

Kandel, D. B. (1985). On processes of peer influences in adolescent drug use: A developmental perspective. *Alcohol and Substance Abuse in Adolescence*, *4*, 139-163.

Kandel, D. B. (1989). Issues of sequencing of adolescent drug use and other problem behaviors. In B. Segal (Ed.), *Perspectives on adolescent drug use* (pp. 55-76). New York: Haworth Press.

Kandel, D. B. (1996). The parental and peer contexts of adolescent deviance: An algebra of interpersonal influences. *Journal of Drug Issues*, *26*, 289-315.

Kandel, D. B. (Ed.). (2002). *Stages and pathways of drug involvement: Examining the Gateway Hypothesis*. New York: Cambridge University Press.

Kandel, D. B. (2003). Does marijuana use cause the use of other drugs? *Journal of the American Medical Association*, *289*, 482-483.

Kandel, D. B., & Andrews, K. (1987). Processes of adolescent socialization by parents and peers. *International Journal of the Addictions*, *22*, 319-342.

Kandel, D. B., & Davies, M. (1991). Friendship networks, intimacy, and illicit drug use in young adulthood: A comparison of two competing theories. *Criminology*, *29*, 441-469.

Kandel, D. B., & Davies, M. (1992). The progression to regular marijuana involvement: Phenomenology and risk factors for near-daily use. In M. Glantz & R. Pickens (Eds.), *Vulnerability to drug abuse* (pp. 211-253). Washington, DC: American Psychological Association Press.

Kandel, D. B., Davies, M., Karus, D., & Yamaguchi, K. (1986). The consequences in young adulthood of adolescent drug involvement. *Archives of General Psychiatry*, *43*, 746-754.

Kandel, D. B., & Faust, R. (1975). Sequence and stages in patterns of adolescent drug use. *Archives of General Psychiatry*, *32*, 923-932.

Kandel, D. B., Kessler, R. C., & Margulies, R. Z. (1978). Antecedents of adolescent initiation into stages of drug use: A developmental analysis. In D. B. Kandel (Ed.), *Longitudinal research on drug use: Empirical findings and methodological issues* (pp. 73-99). New York: John Wiley and Sons.

Kandel, D. B., Kiros, G. E., Schaffsan, C., & Hu, M. C. (2004). Racial/ethnic differences in cigarette smoking initiation and progression to daily smoking: A multilevel analysis. *American Journal of Public Health*, *94*, 128-135.

Kandel, D. B., & Lesser, G. S. (1969). Parental and peer influences on educational plans of adolescents. *American Sociological Review*, *34*, 213-223.

Kandel, D. B., & Lesser, G. S. (1973). *Youth in two worlds*. San Francisco: Jossey-Bass.

Kandel, D. B., & Logan, A. (1984). Patterns of drug use from adolescence to young adulthood: I. Periods of risk for initiation, continued use, and discontinuation. *American Journal of Public Health*, *74*, 660-666.

Kandel, D. B., Simcha-Fagan, O., & Davies, M. (1986). Risk factors for delinquency and illicit drug use from adolescent to young adulthood. *Journal of Drug Issues*, *16*, 67-90.

Kandel, D. B., Treiman, D., Faust, R., & Single, E. (1976). Adolescent involvement in legal and illegal drug use: A multiple classification analysis. *Social Forces*, *55*, 438-459.

Kandel, D. B., & Wu, P. (1995). The contributions of mothers and fathers to the intergenerational transmission of cigarette smoking in adolescence. *Journal of Research on Adolescence*, *5*, 225-252.

Kandel, D. B., & Yamaguchi, K. (1985). Developmental patterns of the use of legal, illegal, and medically prescribed psychotropic drugs from adolescence to young adulthood. In C. L. Jones & R. J. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (pp. 193-235). Rockville, MD: National Institute on Drug Abuse.

Kandel, D. B., & Yamaguchi, K. (1993). From beer to crack: Developmental patterns of drug involvement. *American Journal of Public Health*, *83*, 851-855.

Kandel, D. B., & Yamaguchi, K. (2002). Stages of drug involvement in the U.S. population. In D. B. Kandel (Ed.), *Stages and pathways of drug involvement: Examining the Gateway Hypothesis* (pp. 65-89). New York: Cambridge University Press.

Kandel, D. B., Yamaguchi, K., & Chen, K. (1992). Stages of progression in drug involvement from adolescence to adulthood: Further evidence of the Gateway Theory. *Journal of Studies on Alcohol*, *53*, 447-457.

Kaplan, H. (1975). *Self-attitudes and deviant behavior*. Pacific Palisades, CA: Goodyear.

Kaplan, H. B. (1980). Self-esteem and self-derogation theory of drug abuse. In D. Lattieri, M. Sayers, & H. Pearson (Eds.), *Theories on drug abuse: Selected contemporary perspectives* (pp. 128-131). Rockville, MD: National Institute on Drug Abuse.

Kaplan, H. B., Martin, S. S., & Robbins, C. (1982). Application of a general theory of deviant behavior: Self-derogation and adolescent drug use. *Journal of Health and Social Behavior*, *23*, 274-294.

Kaplan, H. B., Martin, S. S., & Robbins, C. (1984). Pathways to adolescent drug use: Self-derogation, peer influence, weakening of social controls, and early substance use. *Journal of Health and Social Behavior*, *25*, 270-289.

Kaprio, J., Hammar, N., Koskenvuo, M., Floderus-Myrhed, B., Langinvainio, H., & Sarna, S. (1982). Cigarette smoking and alcohol use in Finland and Sweden: A cross-national twin study. *International Journal of Epidemiology*, *11*, 378-386.

Kessler, (2005). Alcohol marketing and youth: The challenge for public health. *Journal of Public Health Policy*, *26*, 292-295.

Khoury, E. L. (1998). Are girls different? A developmental perspective on gender differences in risk factors for substance use among adolescents. In W. A. Vega, A. G. Gil, & Associates (Eds.), *Drug use and ethnicity in early adolescence*. New York: Plenum Press.

Killen, J. D., Robinson, T. N., Haydel, K. F., Hayward, C., Wilson, D. M., Hammer, L. D. et al. (1997). Prospective study of risk factors for the initiation of cigarette smoking. *Journal of Consulting and Clinical Psychology*, *65*, 1011-1016.

Kirby, L., & Fraser, M. (1997). Risk and resilience in childhood. In M. Fraser (Ed.), *Risk and resilience in childhood* (pp. 10-33). Washington, DC: National Association of Social Workers.

Kleiman, M. A. (1998). Drugs and drug policy: The case for a slow fix. *Issues in Science and Technology*, *15*, 45-52.

Komro, K., & Toomey, T. L. (2002). Strategies to prevent underage drinking. *Alcohol Research and Health*, *26*, 5-14.

Kosterman, R., Hawkins, J. S., Guo, J., Catalano, R. F., & Abbott, R. D. (2000). The dynamics of alcohol and marijuana initiation: Patterns and predictors of first use in adolescence. *American Journal of Public Health*, *90*, 360-366.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archive of General Psychiatry*, *54*, 337-343.

Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry*, *158*, 848-856.

Kraemer, H. C., & Thiemann, S. (1987). *How many subjects? Statistical power analysis in research*. Newbury Park, CA: Sage.

Krohn, M. D., Lanza-Kaduce, L., & Akers, R. L. (1984). Community context and theories of deviant behavior: An examination of social learning and social bonding theories. *Sociological Quarterly*, *25*, 353-371.

Krohn, M. D., & Lizotte, A. J. (1996). Reciprocal causal relationships among drug use, peers, and beliefs: A five-wave panel model. *Journal of Drug Issues*, *26*, 405-429.

Krohn, M. D., & Massey, J. L. (1980). Social control and delinquent behavior: An examination of the elements of the social bond. *The Sociological Quarterly*, *21*, 529-543.

Krohn, M. D., Massey, J. L., Skinner, W. F., & Lauer, R. M. (1983). Social bonding theory and adolescent cigarette smoking: A longitudinal analysis. *Journal of Health and Social Behavior*, *24*, 337-349.

Krohn, M. D., Skinner, W. F., Massey, J. L., & Akers, R. L. (1985). Social learning theory and adolescent cigarette smoking: A longitudinal study. *Social Problems*, *32*, 455-*473.*

Krohn, M. D., Skinner, W. F., Zielinski, M., & Naughton, M. (1989). Elaborating the relationship between age and adolescent cigarette smoking. *Deviant Behavior*, *10*, 105-129.

Krosnick, J. A., & Judd, C. M. (1982). Transitions in social influence at adolescence: Who induces cigarette smoking? *Developmental Psychology*, *18*, 359-368.

Kumpfer, K. L., & Turner, C. W. (1990). The social ecology model of adolescent substance abuse: Implications for prevention. *International Journal of the Addictions*, *25*, 435-463.

LaGrange, R. L., & White, H. R. (1985). Age differences in delinquency: A test of theory. *Criminology*, *23*, 19-45.

Lanza-Kaduce, L., Akers, R. L., Krohn, M. D., & Radosevich, M. (1984). Cessation of alcohol and drug use among adolescents: A social learning model. *Deviant Behavior*, *5*, 79-96.

Laub, J., & Sampson, R. J. (1988). Unraveling families and delinquency: A reanalysis of the Gluecks data. *Criminology*, *26*, 355-380.

Lawless, J. F. (2003). *Statistical models and methods for lifetime data* (2nd ed.). Hoboken, NJ: John Wiley and Sons.

Leukefeld, C. G., Logan, T. K., Clayton, R. R., Martin, C., Zimmerman, R., Cattarello, A., et al. (1998). Adolescent drug use, delinquency, and other problem behaviors. In T. P. Gullota, G. R. Adams & R. Montemayor (Eds.), *Delinquent violent youths: Theory and intervention* (Vol. 9, pp. 98-218). Newbury, CA: Sage.

Li, C., Pentz, M. A., & Chou, C. P. (2002a). Parental substance use as a modifier of adolescent substance use risk. *Addiction*, *97*, 1537-1550.

Li, F., Barrera, M., Hops, H., & Fisher, K. J. (2002b). The longitudinal influence of peers on the development of alcohol use in late adolescence: A growth mixture analysis. *Journal of Behavioral Medicine*, *25*, 293-315.

Li, T. K., Hewitt, B. G., & Grant, B. F. (2004). Alcohol use disorders and mood disorders: A National Institute on Alcohol Abuse and Alcoholism perspective. *Biological Psychiatry*, *56*, 718-720.

Lieberson, S. (1985). *Making it count: The improvement of social theory and research*. Los Angeles: University of California Press.

Light, R. J., & Pillemer, D. B. (1982). Numbers and narrative: Combining their strengths in research reviews. *Harvard Educational Review*, *52*, 1-26.

Light, R. J., & Smith, P. V. (1971). Accumulating evidence: Procedures for resolving contradictions among different research studies. *Harvard Educational Review*, *41*, 429-471.

Linkenbach, J., & Perkins, H. W. (2003). Misperceptions of peer alcohol norms in a statewide survey of youth adults. In H. W. Perkins (Ed.), *The social norms approach to preventing school and college age substance abuse: A handbook for educators, counselors, and clinicians* (pp. 173-181). San Francisco: Jossey-Bass.

Liska, A. E., Felson, R. B., Chamlin, M., & Baccaglini, W. (1984). Estimating attitude-behavior reciprocal effects with a theoretical specification. *Social Psychology Quarterly*, *47*, 15-23.

Liska, A. E., & Reed, M. D. (1985). Ties to conventional institutions and delinquency: Estimating reciprocal effects. *American Sociological Review*, *50*, 547-560.

Litrownik, A., Elder, J., & Campbell, N. (2000). Evaluation of a tobacco and alcohol use prevention program for Hispanic migrant adolescents: Promoting the protective factor of parent-child communication. *Preventive Medicine*, *31*, 124-133.

Loeber, R. (1988). The natural history of juvenile conduct problems, delinquency, and associated substance use: Evidence for developmental progressions. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in Clinical Child Psychology* (Vol. 11, pp. 73-127). New York: Plenum.

Loeber, R. (1990). Development and risk factors of juvenile antisocial behavior and delinquency. *Clinical Psychology Review*, *10*, 1-41.

Loeber, R., & Stouthamer-Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct problems. In M. Tonry & N. Morris (Eds.), *Crime and justice: An annual review of research* (Vol. 7, pp. 29-149). Chicago: University of Chicago Press.

Luepker, R. V., Johnson, C. A., Murray, D. M., & Pechacek, T. F. (1983). Prevention of cigarette smoking: Three-year follow-up of an education program for youth. *Journal of Behavioral Medicine*, *6*, 53-62.

Luthar, S. S. (1991). Vulnerability and resilience: A study of high-risk adolescents. *Child Development*, *62*, 600-616.

MacCoun, R. J., & Reuter, P. (2001). *Drug war heresies: Learning from other vices, times, and places*. New York: Cambridge University Press.

MacKinnon, D. P. (1994). Analysis of mediating variables in prevention and intervention research. In A. Cazares & L. A. Beatty (Eds.), *Scientific methods of prevention intervention research* (p. 127-148, NIDA Research Monograph 134). Rockville, MD: National Institute on Drug Abuse.

Maddox, S. J., & Printz, R. J. (2003). School bonding in children and adolescents: Conceptualization, assessment, and associated variables. *Clinical Child and Family Psychology Review*, *6*, 31-49.

Magnussen, D., & Bergman, L. R. (1988). Individual and variable-based approaches to longitudinal research on early risk factors. In M. Rutter (Ed.), *Studies on psychosocial risk: The power of longitudinal data* (pp. 45-61). New York: Cambridge University Press.

Magnussen, D., & Bergman, L. R. (1990). A pattern approach to the study of pathways from childhood to adulthood. In L. N. Robins & M. Rutter (Eds.), *Straight and devious pathways from childhood to adulthood* (pp. 101-115). New York: Cambridge University Press.

Magura, S., & Kang, S. Y. (1997). The validity of self-reported cocaine use in two high-risk populations. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 227-246). Rockville, MD: National Institutes of Health.

Maisto, S. A., Connors, G. J., & Allen, J. P. (1995). Contrasting self-report screens for alcohol problems: A review. *Alcoholism: Clinical and Experimental Research*, *19*, 1510-1516.

Marcos, A. C., & Bahr, S. J. (1988). Control theory and adolescent drug use. *Youth and Society*, *19*, 395-425.

Marcos, A. C., & Bahr, S. J. (1995). Drug progression model: A social control test. *International Journal of the Addictions*, *30*, 1383-1405.

Marcos, A. C., Bahr, S. J., & Johnson, R. E. (1986). Testing a bonding/association theory of adolescent drug use. *Social Forces*, *65*, 135-161.

Margulies, R. Z., Kessler, R. C., & Kandel, D. B. (1977). A longitudinal study of onset of drinking among high-school students. *Journal of Studies on Alcohol*, *38*, 897-912.

Magura, S., & Kang, S. Y. (1997). The validity of self-reported cocaine use in two high-risk populations. In L. Harrison & A. Hughes (Eds.), *The validity of self-reported drug use: Improving the accuracy of survey estimates* (NIDA Research Monograph 167, NIH Report No. 97-4147, pp. 227-246). Rockville, MD: National Institutes of Health.

Martin, C. S., Kaczynski, N. A., Maisto, S. A., & Tarter, R. E. (1996). Polydrug use in adolescent drinkers with and without DSM-IV alcohol abuse and dependence. *Alcoholism: Clinical and Experimental Research*, *20*, 1099-1108.

Massey, J. L., & Krohn, M. D. (1986). A longitudinal examination of an integrated social process model of deviant behavior. *Social Forces*, *65*, 106-134.

Masten, A. (1987). Resilience in development: Implications of the study of successful adaptation for developmental psychopathology. In D. Cicchetti (Ed.), *The emergence of a discipline: Rochester symposium on developmental psychopathology* (pp. 261-294). Hillsdale, NJ: Lawrence Erlbaum.

Masterman, P. W., Hons, B. S., & Kelly, A. B. (2003). Reaching adolescents who drink harmfully: Fitting intervention to developmental reality. *Journal of Substance Abuse Treatment*, *24*, 347-355.

Matsueda, R. L. (1982). Testing control theory and differential association: A causal modeling approach. *American Sociological Review*, *47*, 489-504.

Matsueda, R. L., & Heimer, K. (1987). Race, family structure, and delinquency: A test of differential association and social control theories. *American Sociological Review*, *52*, 826-840.

McCormick, J., Rodney, P., & Varcoe, C. (2003). Reinterpretations across studies: An approach to meta-analysis. *Qualitative Health Research*, *13*, 933-944.

McDermott, D. (1984). The relationship of parental drug use and parents' attitude concerning adolescent drug use to adolescent drug use. *Adolescence*, *19*, 89-97.

McGee, Z. T., (1992). Social class differences in parental and peer influence on adolescent drug use. *Deviant Behavior*, *13*, 349-372.

McGloin, J. M., & Pratt, T. C. (2003). Cognitive ability and delinquent behavior among inner-city youth: A life-course analysis of main, mediating, and interaction effects. *International Journal of Offender Therapy and Comparative Criminology*, *47*, 253-271.

McIver, J. P., & Carmines, E. G. (1982). *Unidimensional scaling*. Beverly Hills, CA: Sage.

Mears, D. P., & Field, S. H. (2002). A closer look at the age, peers, and delinquency relationship. *Western Criminology Review*, *4*, 20-29.

Meier, R. F., Burkett, S. R., & Hickman, C. A. (1984). Sanctions, peers and deviance: Preliminary models of a social control process. *Sociological Quarterly*, *25*, 67-82.

Menard, S. (2002). *Applied logistic regression analysis* (2nd ed., Quantitative Applications in the Social Sciences Series No. 07-106). Thousand Oaks, CA: Sage.

Menard, S., Elliott, D. S., & Wofford, S. (1993). Social control theories in developmental perspective. *Studies on Crime and Crime Prevention*, *2*, 69-87.

Menzel, H. (1953). A new coefficient for scalogram analysis. *Public Opinion Quarterly*, *17*, 268-280.

Mertler, C. A., & Vannatta, R. A. (2005). *Advanced and multivariate statistical methods: Practical application and interpretation* (3rd ed.). Glendale, CA: Pyrczak Publishing.

Meschke, L. L., & Peterson, J. M. (2003). Resilience as a theoretical basis for substance abuse prevention. *The Journal of Primary Prevention*, *23*, 483-514.

Meyers, L. S., Gamst, G., & Guarino, A. J. (2006). *Applied multivariate research: Design and interpretation*. Thousand Oaks, CA: Sage.

Midanik, L. T., & Greenfield, T. K. (2000). Trends in social consequences and dependence symptoms in the United States: The National Alcohol Surveys, 1984-1995. *American Journal of Public Health*, *90*, 53-56.

Miller, D. S., & Miller, T. Q. (1997). A test of socioeconomic status as a predictor of initial marijuana use. *Addictive Behaviors*, *22*, 479-489.

Mills, C. J., & Noyes, H. L. (1984). Patterns and correlates of initial and subsequent drug use among adolescents. *Journal of Consulting and Clinical Psychology*, *52*, 231-243.

Minor, W. W. (1984). Neutralization as a hardening process: Considerations in the modeling of change. *Social Forces*, *62*, 995-1019.

Moffitt, T. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Bulletin*, *106*, 674-701.

Montoya, I. D., Atkinson, J.,& McFaden, W. C. (2003). Best characteristics of adolescent gateway drug prevention programs. *Journal of Addictions Nursing*, *14*, 75-83.

Morgan, S. E., Reichert, T., & Harrison, T. R. (2002). *From numbers to words: Reporting statistical results in the social sciences*. Boston: Allyn and Bacon.

Mounts, N. S. (2004). Adolescent perceptions of parental management of peer relationships in an ethnically diverse sample. *Journal of Adolescent Research*, *19*, 446-467.

Mueller, D. (1986). Guttman scaling. In D. Mueller (Ed.), *Measuring social attitudes: A handbook of researchers and practitioners* (pp. 47-51). New York: Teachers College Press.

Mulrow, C. D., & Cook, D. J. (1997). Systematic reviews: Critical links in the great chain of evidence. *Annals of Internal Medicine*, *126*, 389-391.

Murray, D. M., Davis-Hearn, M., Goldman, A. I., Pirie, P., & Luepker, R. V. (1988). Four- and five-year follow-up results from four seventh-grade smoking prevention strategies. *Journal of Behavioral Medicine*, *11*, 395-405.

Murray, D. M., Pirie, P., Luepker, R. V., & Pallonen, U. E. (1989). Five- and six-year follow-up results from four seventh-grade smoking prevention strategies. *Journal of Behavioral Medicine*, *12*, 207-218.

Murray, D. M., Richards, P. S., Luepker, V., & Johnson, C. A. (1987). The prevention of cigarette smoking in children: Two- and three-year follow-up comparisons of four prevention strategies. *Journal of Behavioral Medicine*, *10*, 595-611.

Myers, D. L., & Arter, M. (2005a). Communities that Care ® in practice. *Journal for Juvenile Justice Services*, *20*, 59-77.

Myers, D. L., & Arter, M. (2005b). Evaluating the implementation of Indiana area 'Communities That Care ®.' *Crime Prevention and Community Safety: An International Journal*, *7*, 43-52.

Nation, M., Crusto, C., Wandersman, A., Kumpfer, K. L., Seybolt, D., Morrissey-Kane, E., et al. (2003). What works in prevention: Principles of effective prevention programs. *American Psychologist*, *58*, 449-456.

NHTSA, National Highway Traffic Safety Administration. (2004). *Traffic safety facts 2003 data: Young drivers* (DOT HS Report No. 809774). Washington, DC: U.S. Department of Transportation, National Highway Traffic Safety Administration, National Center for Statistics and Analysis.

NIDA, National Institute on Drug Abuse. (2001). *Nicotine addiction* (Research Report Series, Report No. 01-4342). Bethesda, MD: Author.

Newcomb, M. D., & Bentler, P. M. (1988). *Consequences of adolescent drug use: Impact on the lives of young adults*. Newbury Park, CA: Sage.

Norman, E., & Turner, S. (1993). Adolescent substance abuse prevention programs: Theories, models, and research in the encouraging 80's. *The Journal of Primary Prevention*, *14*, 3-20.

Nunnally, J. C., & Bernstein, I. C. (1994). *Psychometric theory* (3rd ed.). New York: McGraw-Hill.

O'Donnell, J. A., Hawkins, J. D., & Abbott, R. D. (1995). Predicting serious delinquency and substance use among aggressive boys. *Journal of Consulting and Clinical Psychology*, *63*, 529-537.

O'Donnell, J. A., Hawkins, J. D., Catalano, R., Abbott, R., & Day, E. (1995). Preventing school failure, drug use, and delinquency among low-income children: Long-term intervention in elementary schools. *American Journal of Orthopsychiatry*, *65*, 87-100.

Oetting, E. R., & Beauvais, F. (1986). Peer cluster theory: Drugs and the adolescent. *Journal of Counseling and Development*, *65*, 17-22.

Oetting, E. R., & Beauvais, F. (1987). Common elements in youth drug abuse: Peer clusters and other psychosocial factors. *Journal of Drug Issues*, *17*, 133-151.

OAS, Office of Applied Studies. (2001a). *Illicit drug use among youths who used cigarettes and alcohol*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2001b). *Summary of findings from the 2000 National Survey on Drug Abuse*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies (2002a). *Mortality data from Drug Abuse Warning Network, 2001* (Report No. SMA 03-3781). Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies (2002b). *Parental disapproval of youths' substance use* (NHSDA Report No. PHD955). Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2003). *Emergency department trends from the Drug Abuse Warning Network, Final estimates 1995-2002* (Report No. SMA 03-3780). Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2004a). *Alcohol dependence or abuse and age at first use*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2004b). *Overview of findings from the 2003 National Survey on Drug Use and Health* (NSDUH Series H-24, DHHS Report No. SMA 04-3963). Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2005). *Results from the 2004 National Survey on Drug Use and Health: Detailed tables, prevalence estimates, standard errors, P values, and sample sizes*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2006a). *Results from the 2005 National Survey on Drug Use and Health: Detailed tables, prevalence estimates, standard errors, P values, and sample sizes*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Author.

OAS, Office of Applied Studies. (2006b). *Treatment Episode Data Set (TEDS), Highlights- 2005: National admissions to substance abuse treatment services* (DHHS Publication No. SMA 07-4229). Rockville, MD: Substance Abuse and Mental Health Services Administration, Author.

ONDCP, Office of National Drug Control Policy. (2004). *The economic costs of drug abuse in the U.S., 1992-2002* (Report No. 207303). Washington, DC: Executive Office of the President, Author.

Office of Substance Abuse Information and Resource Center. (2003). *Adolescent alcohol and drug use in Maine: Progress and needs*. Augusta, ME: Office of Substance Abuse Information and Resource Center, Department of Behavioral and Developmental Services. Retrieved June 14, 2008, from mainegov-images.informe.org/dhhs/osa/.../osa/2003/adolescentreport.pdf

O'Malley, P., Bachman, J. G., & Johnston, L. D. (1988). *Differentiation of period, age, and cohort effects on drug use: 1976-1986* (Monitoring the Future Occasional Paper 22). Ann Arbor, MI: Institute for Social Research.

Orcott, J. D. (1987). Differential association and marijuana use: A closer look at Sutherland (with a little help from Becker). *Criminology*, *25*, 341-358.

Orgogozo, J. M., Dartiques, J. F., Lafont, S., Letenneur, L., Commenges, D., Salamon, R., et al. (1997). Wine consumption and dementia in the elderly: A prospective community study from the Bordeaux area. *Neurological Review*, *153*, 185-192.

Osher, C. N. (2005). *Denver legalizes marijuana possession: Commonsense for drug policy*. Retrieved June 13, 2006, from http://www.mapinc.org/newscsdo/v05/n1745/a09.html

Oxford, M. L., Harachi, T. W., Catalano, R. F., & Abbott, R. D. (2000). Preadolescent predictors of substance initiation: A test of both the direct and mediated effect of family social control factors on deviant peer associations and substance initiation. *American Journal of Drug and Alcohol Abuse*, *27*, 599-616.

Pacula, R. L., Chriqui, J. F., & King, J. (2004). *Marijuana decriminalization: What does it mean in the United States?* (Working Paper No. 9690). Cambridge, MA: National Bureau of Economic Research.

Paternoster, R. (1988). Examining three-wave deterrence models: A question of temporal order and specification. *Journal of Criminal Law and Criminology*, *79*, 135-179.

Paton, S., Kessler, R., & Kandel, D. B. (1977). Depressive mood and adolescent illicit drug use: A longitudinal analysis. *The Journal of Genetic Psychology*, *131*, 267-289.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist*, *44*, 329-335.

Patterson, G. R., & Dishion, T. J. (1985). Contributions of families and peers to delinquency. *Criminology*, *23*, 63-69.

Patrick, C. H. (1970). *Alcohol, culture, and society*, New York: AMS Press.

Pedhazur, E. J. (1982). *Multiple regression in behavioral research: Explanation and prediction* (2nd ed.). New York: Holt, Rinehart, and Winston.

Penning, M., & Barnes, G. E. (1982). Adolescent marijuana use: A review. *International Journal of the Addictions*, *17*, 749-791.

Pennsylvania Department of Education. (2005a). *Home education in Pennsylvania, 2000-01*. Harrisburg, PA: Author, Division of Data Services. Retrieved October 17, 2007, from http://www.pde.state.pa.us/k12statistics/lib/k12statistics/00-01HomeEdAppendixA.pdf

Pennsylvania Department of Education. (2005b). *Home education in Pennsylvania, 2003-04*. Harrisburg, PA: Author, Division of Data Services. Retrieved October 17, 2007, from http://www.pde.state.pa.us/k12statistics/lib/k12statistics/appxa-0304_10.pdf

Pennsylvania Department of Education. (2007). *Poverty Level by School District*. Retrieved October 17, 2007, from http://www.pde.state.pa.us/early_childhood/cwp/view.asp?a=316&q=125548

Pennsylvania Juvenile Court Judges' Commission. (2005). *Pennsylvania juvenile court dispositions, 2004*. Harrisburg, PA: Author. Retrieved October 17, 2007, from http://www.jcjc.state.pa.us/jcjc/lib/jcjc/statistics/2004/04dispo1of2.pdf

Pentz, M. A. (1996). *Preventing drug abuse through the community: Multi-component programs make the difference*. Paper presented at National Conference on Drug Abuse Prevention Research, Washington, DC.

Pentz, M. A., & Li, C. (2002). The Gateway Theory applied to prevention. In D. B. Kandel (Ed.), *Stages and pathways of drug involvement: Examining the Gateway Hypothesis* (pp. 139-157). New York: Cambridge University Press.

Perry, C. L., Kelder, S. H., Murray, D. M., & Klepp, K. I. (1992). Community-wide smoking prevention: Long-term outcomes of the Minnesota Heart Health Program and the Class of 1989 Study. *American Journal of Public Health*, *82*, 1210-1216.

Perry, C. L., Williams, C. L., Komro, K. A., Veblen-Mortensen, S., Stigler, M. H., Munson, K. A., et al. (2002). Project Northland: Long-term outcomes of community action to reduce adolescent alcohol use. *Health Education Research*, *17*, 117-132.

Perry, C. L., Williams, C. L., Veblen-Mortensen, S., Toomey, T. L., Komro, K. A., Anstine, P. S., et al. (1996). Project Northland: Outcomes of a community-wide alcohol use prevention program during early adolescence. *American Journal of Public Health*, *86*, 956-965.

Peterson, P. L., Hawkins, J. D., Abbott, R. D., & Catalano, R. F. (1994). Disentangling the effects of parental drinking, family management, and parental alcohol norms on current drinking by black and white adolescents. *Journal of Research on Adolescence*, *4*, 203-227.

Peterson, P. L., Hawkins, J. D., & Catalano, R. F. (1992). Evaluating comprehensive community drug risk reduction interventions: Design challenges and recommendations. *Evaluation Review*, *16*, 579-602.

Petraitis, J., Flay, B. R., & Miller, T. Q. (1995). Reviewing theories of adolescent drug use: Organizing pieces in the puzzle. *Psychological Bulletin*, *117*, 67-86.

Petraitis, J., Flay, B. R., Miller, T. Q., Torpy, E. J., & Greiner, B. (1998). Illicit substance use among adolescents: A matrix of prospective predictors. *Substance Use and Misuse*, *33*, 2561-2604.

Petrosino, A., Boruch, R. F., Soyden, H., Duggan, L., & Sanchez-Meca, J. (2001). Meeting the challenges of evidence-based policy: The Campbell Collaboration. *Annals of the American Academy of Political and Social Science*, *578*, 14-34.

Pickles, A., Pickering, K., Taylor, C., Sutton, S., & Yang, S. (2001). Multilevel risk models for retrospective age-of-onset data: School children's first cigarette. *Journal of Adolescent Research*, *16*, 188-204.

Pierce, J. P., Choi, W. S., Gilpin, E. A., Farkas, A. J., & Merritt, R. K. (1996). Validation of susceptibility as a predictor of which adolescents take up smoking in the United States. *Health Psychology*, *15*, 355-361.

Ploeger, M. (1997). Youth employment and delinquency: Reconsidering a problematic relationship. *Criminology*, *35*, 659-676.

Pokorny, S. B., Jason, L. A., & Schoeny, M. E. (2003). The relation of retail tobacco availability to initiation and continued smoking. *Journal of Clinical Child and Adolescent Psychology*, *32*, 193-204.

Pollard, J. A., Hawkins, J. D., & Arthur, M. W. (1999). Risk and protection: Are both necessary to understand diverse behavioral outcomes in adolescence? *Social Work Research*, *23*, 145-158.

Pratt, T. C., & Cullen, F. T. (2000). The empirical status of Gottfredson and Hirschi's General Theory of Crime: A meta-analysis. *Criminology*, *38*, 931-964.

Preiss, R. W., & Allen, M. (1995). Understanding and using meta-analysis. *Evaluation and the Health Professions*, *18*, 315-335.

Ramirez, J. R., Crano, W. D., Quist, R., Burgoon, M., Alvaro, E., & Grandpre, J. (2004). Acculturation, familism, parental monitoring, and knowledge as predictors of marijuana and inhalant use in adolescents. *Psychology of Addictive Behaviors*, *18*, 3-11.

Randall, V. R. (2006). *History of tobacco*. Retrieved June 10, 2006, from http://academic.udayton.edu/health/syllabi/tobacco/history.htm

Randolph, K. A. (2004). The dynamic nature of risk factors for substance use among adolescents. *Journal of Child and Adolescent Substance Abuse*, *13*, 33-47.

Rankin, J. H. (1976). Investigating the interrelations among social control variables and conformity. *The Journal of Criminal Law and Criminology*, *67*, 470-480.

Rankin, J. H. (1980). School factors and delinquency: Interactions by age and sex. *Sociology and Social Research*, *64*, 420-434.

Reidel, M. (2000). Doing research with secondary data. In M. Reidel (Ed.), *Research strategies for secondary data* (pp. 49-73). Thousand Oaks, CA: Sage.

Reifman, A., Barnes, G., Dintcheff, B., Farrell, M., & Uhteg, L. (1998). Parental and peer influences on the onset of heavier drinking among adolescents. *Journal of Studies on Alcohol*, *59*, 311-317.

Resnick, G., & Burt, M. R. (1996). Youth at risk: Definitions and implications for service delivery. *American Journal of Orthopsychiatry*, *66*, 172-188.

Ried, L. D., Martinson, O. B., & Weaver, L. C. (1987). Factors associated with drug use of fifth through eighth grade students. *Journal of Drug Education*, *17*, 149-151.

Ripple, C. H., & Zigler, E. (2003). Research, policy, and the federal role in prevention initiatives for children. *American Psychologist*, *58*, 482-490.

Robertson, E. B., Sloboda, Z., Boyd, G. M., Beatty, L., & Kozel, N. (Eds.). (1997). *Rural substance abuse: State of knowledge and issues* (NIDA Research Monograph 168, NIH Publication No. 97-4177). Rockville, MD: National Institute on Drug Abuse.

Robins, L. N. (1979). Addict careers. In R. L. DuPont, A. Goldstein, & J. O'Donnell (Eds.), *Handbook on drug abuse* (pp. 325-355). Rockville, MD: National Institute on Drug Abuse.

Robins, L. N. (1980). The natural history of drug abuse. In D. J. Lettieri, M. Sayers, & H. W. Pearson (Eds.), *Theories on drug abuse: Selected contemporary perspectives* (pp. 215-224). Rockville, MD: National Institute on Drug Abuse.

Robins, L. N., & Przybeck, T. R. (1990). Age of onset of drug use as a factor in drug and other disorders. In C. L. Jones & R. J. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (pp. 178-192, Monograph No. 56, DHHS Report No. 90-1335). Rockville, MD: National Institute on Drug Abuse.

Robinson, L. A., Klesges, R. C., Zbikowski, S. M., & Galser, R. (1997). Predictors of risk for different stages of adolescent smoking in a biracial sample. *Journal of Consulting and Clinical Psychology*, *65*, 653-662.

Romer, D. (Ed.) (2003). *Reducing adolescent risk: Toward an integrated approach*. Thousand Oaks, CA: Sage.

Roosa, M. W. (2000). Some thoughts about resilience versus positive development, main effects versus interactions, and the value of resilience. *Child Development*, *71*, 567-569.

Rouse, B. A., Kozel, N. J., & Richards, L. G. (1985). *Self-report methods of estimating drug use: Meeting current challenges to validity* (NIDA Research Monograph 57, DHHS Publication NO. ADM 85-1402). Rockville, MD: National Institute on Drug Abuse.

Rubington, E., & Weisberg, M .S. (Eds.). (1989). *The study of social problems*. New York: Oxford University Press.

Rutter, M. (1985). Resilience in the face of adversity: Protective factors and resistance to psychiatric disorder. *British Journal of Psychiatry*, *147*, 598-611.

Sampson, R. J., & Laub, J. H. (1990). Crime and deviance over the life-course: The salience of adult social bonds. *American Sociological Review*, *55*, 609-627.

Sampson, R. J., & Laub, J. H. (1993). *Crime in the making: Pathways and turning points through life*. Cambridge: Harvard University Press.

Santa Cruz County Board of Supervisors. (2002). *Santa Cruz Board of Supervisors' response to the 2001-02 grand jury final report*. Retrieved June 15, 2008, from http://www.co.santa-cruz.ca.us/grandjury/ BS01-02response.htm

Schaps, E., Churgin, S., Palley, C. S., Takata, B., & Cohen, A. Y. (1980). Primary prevention research: A preliminary review of program outcome studies. *International Journal of the Addictions*, *15*, 657-676.

Schaps, E., DiBartolo, R., Moskowitz, J., Palley, C. S., & Churgin, S. (1981). A review of 127 drug abuse prevention program evaluations. *Journal of Drug Issues*, *11*, 17-43.

Scheier, L. M., Newcomb, M. D., & Skager, R. (1994). Risk, protection, and vulnerability to adolescent drug use: Latent-variable models of three age groups. *Journal of Drug Education*, *24*, 49-82.

Schinke, S. (2002). *A practitioner's guide to science-based prevention: A handbook of promising, effective, and model programs*. Washington, DC: Substance Abuse and Mental Health Services Administration.

Schulenberg, J., & Maggs, J. L. (2001). *A developmental perspective on alcohol and other drug use during adolescence and the transition to young adulthood* (Monitoring the Future Occasional Paper No. 51). Ann Arbor, MI: Institute for Social Research.

Schulenberg, J., Maggs, J. L., & Hurrelmann, K. (Eds.) (1997). *Health risks and developmental transitions during adolescence*. New York: Cambridge University Press.

Schulenberg, J. E., Maggs, J. L., & O'Malley, P. M. (2003). How and why the understanding of developmental continuity and discontinuity is important: The sample case of long-term consequences of adolescent substance use. In J. Mortimer & M. J. Shanahan (Eds.), *Handbook of the life course* (pp. 413-436). New York: Kluwer Academic/Plenum.

Schwalbe, C. S., Fraser, M. W., Day, S. H., & Cooley, V. (2006). Classifying juvenile offenders according to risk of recidivism: Predictive validity, race/ethnicity, and gender. *Criminal Justice and Behavior*, *33*, 305-324.

Schwartz, J. E. (1986). A general liability model for categorical data applied to Guttman Scales and current-status data. *Sociological Methodology*, *16*, 79-119.

Sellers, C. S., & Winfree, T. L. (1990). Differential associations and definitions: A panel study of youthful drinking behavior. *International Journal of the Addictions*, *25*, 755-771.

Shadish, W. R., Cook, T. D., & Campbell, D.T. (2002). *Experimental and quasi experimental designs for generalized causal inference*. Boston: Houghton Mifflin Company.

Shaw, C. R., & McKay, H. D. (1969). *Juvenile delinquency and urban areas*. Chicago: University of Chicago Press.

Shears, J., Edwards, R. W., & Stanley, L. R. (2006). School bonding and substance use in rural communities. *Social Work Research*, *30*, 6-18.

Sherman, L. W., Gottfredson, D. C., MacKenzie, D. L., Eck, J. E., Reuter, P., & Bushway, S. D. (1997). *Preventing crime: What works, what doesn't, what's promising*. Washington, DC: National Institute of Justice.

Sieving, R. E., McNeely, C., & Blum, R. W. (2000). Maternal expectations, mother-child connectedness, and adolescent sexual debut. *Archives of Pediatrics and Adolescent Medicine*, *154*, 809-816.

Sieving, R. E., Perry, C. L., & Williams, C. L. (2000). Do friendships change behaviors, or do behaviors change friendships? Examining paths of influence in young adolescent's alcohol use. *Journal of Adolescent Health*, *26*, 27-35.

Simons, R. L., Conger, R. D., & Whitbeck, L. B. (1988). A multistage social learning model of the influences of family and peers upon adolescent substance abuse. *Journal of Drug Issues*, *18*, 293-315.

Simons-Morton, B. G., Crump, A. D., Haynie, D. L., & Saylor, K. E. (1999). Student school-bonding and adolescent problem behavior. *Health Education Research*, *14*, 99-107.

Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence: The impact of pubertal change and school context*. Hawthorne, NY: Aldine de Gruyter.

Single, E., Kandel, D. B., & Faust, R. (1974). Patterns of multiple drug use in high school. *Journal of Health and Social Behavior*, *15*, 344-357.

Skinner, B. F. (1999). *The behavior of organisms: An experimental analysis* (3rd ed.). Cambridge, MA: B. F. Skinner Foundation.

Skinner, W. F., Massey, J. L., Krohn, M. D., & Lauer, R. M. (1985). Social influences and constraints on the initiation and cessation of adolescent tobacco use. *Journal of Behavioral Medicine*, *8*, 353-376.

Smith, R. B. (1968). A discussion of an attempt at constructing reproducible item sets. *Journal of Educational Measurement*, *5*, 55-60.

Smith, G. M., & Fogg, C. P. (1978). Psychological predictors of early use, late use, and nonuse of marijuana among teenage students. In D. B. Kandel (Ed.), *Longitudinal research on drug use* (pp. 101-113). New York: John Wiley and Sons.

Sobel, L. C., & Sobel, M. B. (1992). Timeline follow-back: A technique for assessing self-reported alcohol consumption. In J. P. Allen & R. Z. Litten (Eds.), *Measuring alcohol consumption: Psychosocial and biochemical methods* (pp. 41-72). Totowa, NJ: Humana Press.

Solmitz, D. O. (2000). *The roots of apathy and how schools can reduce apathy*, Retrieved June 15, 2008, from http://www.rem1.org/local_voices/summer2000/education/apathy.htm

Sommers, I., Fagan, J., & Baskin, D. (1994). The influence of acculturation and familism on Puerto Rican delinquency. *Justice Quarterly*, *11*, 207-228.

Spoth, R., & Redmond, C. (1996). Illustrating a framework for rural prevention research. In R. D. Peters & R. J. McMahon (Eds.), *Preventing childhood disorders, substance abuse, and delinquency* (pp. 299-3280. Thousand Oaks, CA: Sage.

Stein, J. A., Newcomb, M. D., & Bentler, P. M. (1987). An eight-year study of multiple influences on drug use and drug use consequences. *Journal of Personal and Social Psychology*, *53*, 1094-1105.

Steinberg, L., & Dornbusch, S. M. (1991). Negative correlates of part-time employment during adolescence: Replication and elaboration. *Developmental Psychology*, *27*, 304-313.

Stookey, J. A., & Baer, M. A. (1976). A critique of Guttman Scaling: With special attention to its application to the study of collegial bodies. *Quality and Quantity*, *10*, 251-260.

Strasburger, V. C. (1995). *Adolescents and the media*. Thousand Oaks, CA: Sage.

Stumphauzer, J. S. (1983). Learning not to drink: Adolescents and abstinence. *Journal of Drug Education*, *13*, 39-48.

Stein, J. A., Newcomb, M. D., & Bentler, P. M. (1987). An 8-year study of multiple influences on drug use and drug use escalation. *Journal of Personality and Social Psychology*, *53*, 1094-1105.

Sutherland, E. H., & Cressey, D. R. (1978). *Criminology* (9th ed.). Philadelphia: Lippincott.

Sutherland, I., & Shepard, J. P. (2001). Social dimensions of adolescent substance use. *Addiction*, *96*, 445-458.

Swadi, H. (1999). Individual risk factors for adolescent substance use. *Drug and Alcohol Dependence*, *55*, 209-224.

Tarter, R. E., & Mezzich, A. C. (1992). Ontogeny of substance abuse: Perspectives and findings. In M. Glantz & R. Pickens (Eds.), *Vulnerability to drug abuse* (pp. 149-177). Washington, DC: American Psychological Association.

Taub, D. E., & Skinner, W. F. (1990). A social bonding-drug progression model of amphetamine use among young women. *American Journal of Drug and Alcohol Abuse*, *16*, 77-95.

Thombs, D. L., Wolcott, B. J., & Farkash, L G. (1997). Social context, perceived norms and drinking behavior among young people. *Journal of Substance Abuse*, *9*, 257-267.

Thompson, A. E., Smith-DiJulio, K., & Matthews, T. (1982). Social control theory: Evaluating a model for the study of adolescent alcohol and drug use. *Youth and Society*, *13*, 303-326.

Thompson ISI ResearchSoft. (2004). *EndNote* (Version 8.0). Berkeley, CA: Author.

Thornberry, T. P. (1987). Toward an interactional theory of delinquency. *Criminology*, *25*, 863-892.

Thornberry, T. P. (2004). Introduction: Some advantages of developmental and life-course perspectives for the study of crime and delinquency. In T. P. Thornberry (Ed.), *Developmental theories of crime and delinquency* (pp. 1-10). New Brunswick, NJ: Transaction.

Thornberry, T. P., Lizotte, A. J., Krohn, M. D., Farnsworth, M., & Jang, S. J. (1991). Testing interactional theory: An examination of reciprocal causal relationships among family, school, and delinquency. *Journal of Criminal Law and Criminology*, *82*, 3-35.

Thornberry, T. P., Lizotte, A. J., Krohn, M. D., Farnsworth, M., & Jang, S. J. (1994). Delinquent peers, beliefs, and delinquent behavior: A longitudinal test of interactional theory. *Criminology*, *32*, 47-84.

Thornburg, H. D. (1982). *Development in adolescence* (2nd ed.). Monterey, CA: Brooks/Cole.

Tilson, E. C., McBride, C. M., Lipkus, I. M., & Catalano, R. F. (2004). Testing the interaction between parent-child relationship factors and parent smoking to predict youth smoking. *Journal of Adolescent Health*, *35*, 182-189.

Tittle, C. R., Burke, M. J., & Jackson, E. (1986). Modeling Sutherland's theory of differential association: Toward an empirical clarification. *Social Forces*, *65*, 405-432.

Tobler, N. (1986). Meta-analysis of 143 adolescent drug prevention programs: Quantitative outcome results of program participants compared to a control or comparison group. *Journal of Drug Issues*, *16*, 537-567.

Tobler, N. (1992). Drug prevention programs can work: Research findings. *Journal of Addictive Diseases*, *11*, 1-28.

Tobler, N., Roona, M., Ochshorn, P., Marshall, D., Streke, A., & Stackpole, F. (2000). School-based adolescent drug prevention programs: 1998 meta-analysis. *Journal of Primary Prevention*, *20*, 275-336.

Toomey, T. L., & Wagenaar, A. C. (1999). Policy options for prevention: The case of alcohol. *Journal of Public Health Policy*, *20*, 192-213.

Toumbourou, J. (2005). *Communities that Care in Australia: The next phase* (Newsletter No. 3). Melbourne, Australia: Centre for Adolescent Health.

Towns, C. (1917). *Habits that handicap: The menace of opium, alcohol, and tobacco, and the remedy*. New York: Century Company.

Tyc, V. L., Hadley, W., Allen, D., Varnell, S., Ey, S., Rai, S. N., et al. (2004). Predictors of smoking intentions and smoking status among nonsmoking and smoking adolescents, *Addictive Behaviors*, *29*, 1143-1147.

Unger, J. B., & Chen, X. (1999). The role of social networks and media receptivity in predicting age of smoking initiation: A proportional hazard model of risk and protective factors. *Addictive Behaviors*, *24*, 371-381.

Urberg, K. A., Degirmencioglu, S. M., & Pilgrim, C. (1997). Close friend and group influence on adolescent cigarette smoking and alcohol use. *Developmental Psychology*, *33*, 834-844.

Urberg, K. A., Cheng, C. H., & Shyu, S. J. (1991). Grade changes in peer influence on adolescent cigarette smoking: A comparison of two measures. *Addictive Behaviors*, *16*, 21-28.

Urberg, K. A., Luo, Q., Pilgrim, C., & Degirmencioglu, S. M., (2003). A two-stage model of peer influence in adolescent substance use: Individual and relationship-specific differences in susceptibility to influence. *Addictive Behaviors*, *28*, 1243-1256.

UWP, The United Way of Pennsylvania. (2006). *Indiana County statistical profile*. Retrieved June 18, 2007, from http://www.ruralpa2.org/county_profiles/cfm

U.S. Department of Education. (2002). *Exemplary and promising safe, disciplined and drug-free schools programs, 2001*. Washington, DC: Author.

U.S. Department of Health and Human Services. (2001). *Youth violence: A report of the Surgeon General*. Washington, DC: U.S. Government Printing Office.

Vakalahi, H. F. (2001). Adolescent substance use and family-based risk and protective factors: A literature review. *Journal of Drug Education*, *31*, 29-46.

van den Bree, M. B., Whitmer, M. D., & Pickworth, W. B. (2004). Predictors of smoking development in a population-based sample of adolescents: A prospective study. *Journal of Adolescent Health*,*35*, 172-181.

VanVoorhis, C. W., & Morgan, B. L. (2007). *Statistical rules of thumb: What we don't want to forget about sample sizes*. Retrieved February 4, 2007, from http://www.psichi.org/pubs/articles/article_182.asp.

Vega, W. A., Zimmerman, R. S., Warheit, G. J., Apospori, E., & Gil, A. G. (1993). Risk factors for early adolescent drug use in four ethnic and racial groups. *American Journal of Public Health*, *83*, 185-189.

Vitaro, F., Wanner, B., Brendgen, M., Gosselin, C., & Gendreau, P. L. (2004). Differential contributions of parents and friends to smoking trajectories during adolescence. *Addictive Behaviors*, *29*, 831-835.

Voss, H. (1964). Differential association and reported delinquent behavior: A replication. *Social Problems*, *12*, 78-85.

Wada, K. (1999). The concept of "gateway drug." *Nihon Arukoru Yakubutsu Igakkai Zasshi*, *34*, 95-106.

Wade, T. J., & Brannigan, A. (1998). The genesis of adolescent risk-taking: Pathways through family, school, and peers. *Canadian Journal of Sociology*, *23*, 1-19.

Wagenaar, A. (1993). Research affects public policy: The case of the legal drinking age in the United States. *Addiction*, *88*, 75S-81S.

Wakefield, M., Flay, B. R., Nichter, M., & Giovino, G. (2003). Role of the media in influencing trajectories of youth smoking. *Addiction*, *98*, 79-103.

Wallace, J. M., Brown, T. N., Bachman, J. G., & LaVeist, T. A. (2003). The influence of race and religion on abstinence from alcohol, cigarettes, and marijuana among adolescents. *Journal of Studies on Alcohol*, *64*, 843-848.

Waller, M. A., Okamoto, S. K., Miles, B. W., & Hurdle, D. E. (2003). Resiliency factors related to substance use/resistance: Perceptions of Native adolescents of the Southwest. *Journal of Sociology and Social Welfare*, *30*, 79-94.

Walter, H., Vaughan, R. D., & Cohall, A. T. (1991). Risk factors for substance use among high school students: Implications for prevention. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 556-562.

Warr, M. (1993a). Age, peers, and delinquency. *Criminology*, *31*, 17-40.

Warr, M. (1993b). Parents, peers, and delinquency. *Social Forces*, *72*, 247-265.

Warr, M. (2002). *Companions in crime: The social aspects of criminal conduct*. Cambridge: Cambridge University Press.

Warr, M., & Stafford, M. (1991). The influence of delinquent peers: What they think or what they do? *Criminology*, *29*, 851-866.

Watson, R. (2005). The gateway to high society. *British Medical Journal*, *331*, 116.

Webb, J. A., Baer, P. E., McLaughlin, R. J., McKelvey, R. S., & Caid, C. D. (1991). Risk factors and their relation to initiation of alcohol use among early adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 563-568.

Weber, M. D., Graham, J. W., Hansen, W. B., Flay, B. R., & Johnson, A. C. (1989). Evidence of two paths of alcohol use onset in adolescents. *Addictive Behaviors*, *14*, 399-408.

Weisburd, D., & Britt, C. (2003). *Statistics in criminal justice* (2nd ed.). Belmont, CA: Wadsworth/Thompson.

Weissberg, R. P., Kumpfer, K. L., & Seligman, M. E. (2003). Prevention that works for children and youth: An introduction. *American Psychologist*, *58*, 425-432.

Welsh, B. C., & Farrington, D. P. (2006a). Evidence-based crime prevention. In B. C. Welsh & D. P. Farrington (Eds.), *Preventing crime: What works for children, offenders, victims, and places* (pp. 1-17). Dordrecht, The Netherlands: Springer.

Welsh, B. C., & Farrington, D. P. (Eds.) (2006b). *Preventing crime: What works for children, offenders, victims, and places*. Dordrecht, The Netherlands: Springer.

Welte, J. W., & Barnes, G. M. (1985). Alcohol: The gateway to other drug use among secondary-school students. *Journal of Youth and Adolescence*, *14*, 487-498.

Werch, C. E., Carlson, J. M., Pappas, D. E., Dunn, M., & Williams, T. (1997). Risk factors related to urban youth stage of alcohol initiation. *American Journal of Health Behavior*, *21*, 377-388.

Werner, E. E. (1994). Overcoming the odds. *Developmental and Behavioral Pediatrics*, *15*, 131-136.

Werner, E. E., & Smith, R. S. (1982). *Vulnerable and invincible: A longitudinal study of resilient children and youth*. New York: Adams, Bannister, and Cox.

White A. M., & Swartzwelder, H. S. (2005). Age-related effects of alcohol memory and memory-related brain function in adolescents and adults. In M. Galanter (Ed.), *Recent developments in alcoholism, Vol. 17: Alcohol problems in adolescents and young adults: Epidemiology, neurobiology, prevention, and treatment* (pp. 161-176). New York: Springer.

White, H. R. (1987). Longitudinal stability and dimensionality structure of problem drinking in adolescence. *Journal of Studies on Alcohol*, *48*, 541-550.

White, H. R., Johnson, V., & Horwitz, A. (1986). An application of three deviance theories to adolescent substance use. *International Journal of the Addictions*, *21*, 347-366.

White, H. R., Pandina, R. J., & LaGrange, R. L. (1987). Longitudinal predictors of serious substance use and delinquency. *Criminology*, *25*, 715-740.

Whitlock, J. L., & Hamilton, S. F. (2003). The role of youth surveys in community youth development initiatives. *Applied Developmental Science*, *7*, 39-51.

Wiatrowski, M. D., & Anderson, K. L. (1987). The dimensionality of the social bond. *Journal of Quantitative Criminology*, *3*, 65-81.

Wiatrowski, M. D., Griswold, D. B., & Roberts, M. K. (1981). Social control theory and delinquency. *American Sociological Review*, *46*, 525-541.

Wiencke, J. K., Thurston, S. W., Kelsey, K. T., Varkonyi, A., & Wain, J. C. (1999). Early age at smoking initiation and tobacco carcinogen DNA damage in the lung. *Journal of the National Cancer Institute*, *97*, 614-619.

Williams, F., & McShane, M. (2004). *Criminological theory* (4^{th} ed.). Upper Saddle River, NJ: Pearson Hall.

Williams, J. H., Ayers, C. D., Abbott, R. D., Hawkins, J. D., & Catalano, R. F. (1996). Structural equivalence of involvement in problem behavior by adolescents across racial groups using multiple groups confirmatory factor analysis. *Social Work Research*, *20*, 168-178.

Williams, J. H., Ayers, C. D., Abbott, R. D., Hawkins, J. D., & Catalano, R. F. (1999). Racial differences in risk factors for delinquency and substance use among adolescents. *Social Work Research*, *23*, 241-257.

Williams, J. H., Van Dorn, R. A., Ayers, C. D., Bright, C. L., Abbott, R. D., & Hawkins, J. D. (2007). Understanding race and gender differences in delinquent acts and alcohol and marijuana use: A developmental analysis of initiation. *Social Work Research*, *31*, 71-81.

Wills, T. A., & Cleary, S. D. (1999). Peer and adolescent substance use among 6^{th}-9^{th} graders: Latent growth analyses of influence versus selection mechanisms. *Health Psychology*, *18*, 453-463.

Wills, T. A., Pierce, J. P., & Evans, R. I. (1996). Large-scale environmental risk factors for substance use. *American Behavioral Scientist*, *39*, 808-823.

Wills, T., Resko, J., Ainette, M., & Mendoza, D. (2004). Smoking onset in adolescence: A person-centered analysis with time-varying predictors. *Health Psychology*, *23*, 158-167.

Wilson, D. B. (2001). Meta-analytic methods for criminology. *Annals of the American Academy of Political and Social Science*, *578*, 71-89.

Windle, M., Spear, L., Fuligni, A. J., Angold, A., Brown, J. D., Pine, D. et al. (2008). Transitions into underage and problem drinking: Developmental processes and mechanisms between 10 and 15 years of age. *Pediatrics*, *121*, S273-S289.

Winfree, L., & Bernat, F. (1998). Social learning, self-control, and substance abuse by eighth grade students: A tale of two cities. *Journal of Drug Issues*, *28*, 539-558.

Winfree, L T., & Griffiths, C. T. (1983). Social learning and marijuana use: A trend study of deviant behavior in a rural middle school. *Rural Sociology*, *48*, 219-239.

Winfree, L. T., Griffiths, C. T., & Sellers, C. S. (1989). Social learning theory, drug use, and American Indian youths: A cross-cultural test. *Justice Quarterly*, *6*, 395-417.

Winters, K. C., Anderson, N., Bengston, P., Stinchfield, R. D., & Latimer, W. W. (2000). Development of a parent questionnaire for the assessment of adolescent drug use. *Journal of Psychoactive Drugs*, *32*, 3-13.

Winters, K. C., Stinchfield, R. D., Henly, G. A., & Schwartz, R. (1990). Validity of adolescent self report of substance involvement. *International Journal of the Addictions*, *25*, 1379-1395.

Wrenn, D. (2006). *Apathetic parents: The primary danger to children*. Retrieved June 15, 2008, from http://www.dondodd.com/wrenn/archives06/100706.html

Wright, J. P., Cullen, F. T., & Williams, N. (1997). Working while in school and delinquent involvement: Implications for social policy. *Crime and Delinquency*, *43*, 203-221.

Wright, J. P., Cullen, F. T., & Williams, N. (2002). The embeddedness of adolescent employment and participation in delinquency: A life-course perspective. *Western Criminology Review*, *4*, 1-19.

Yamaguchi, K., & Kandel, D. B. (1984a). Patterns of drug use from adolescence to young adulthood: II. Sequences of progression. *American Journal of Public Health*, *74*, 668-672.

Yamaguchi, K., & Kandel, D. B. (1984b). Patterns of drug use from adolescence to young adulthood: III. Predictors of progression. *American Journal of Public Health*, *74*, 673-681.

Yoon, Y., Yi, H., & Hilton, M. E. (2005). *Liver cirrhosis mortality in the United States, 1970-2002* (Surveillance Report No. 70). Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism.

Yu, J., & Williford, W. R. (1992). The age of alcohol onset and alcohol, cigarette, and marijuana use patterns: An analysis of drug use progression of young adults in New York State. *International Journal of the Addictions*, *27*, 1313-1323.

Zakhari, S. (1997). Alcohol and the cardiovascular system: Molecular mechanisms for beneficial and harmful action. *Alcohol Health and Research World*, *21*, 21-29.

Zimring, F. E., & Hawkins, G. (1992). *The search for rational drug control*. New York: Cambridge University Press.

Zucker, R. A. (1994). Pathways to alcohol problems: A developmental account of the evidence for contextual contributions to risk. In R. A. Zucker, G. M. Boyd, & J. Howard (Eds.), *The development of alcohol problems* (pp. 255-289). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Zucker, R. A., Donovan, J. E., Masten, A. S., Mattson, M. E., & Moss, H. B. (2008). Early developmental processes and the continuity of risk for underage drinking and problem drinking. *Pediatrics*, *121*, S252-S272.

APPENDIX A

2004 PPAAUS INSTRUMENT

PRIMARY PREVENTION AWARENESS, ATTITUDE AND USE SURVEY

DO NOT PUT YOUR NAME ANYWHERE ON THIS SURVEY

GENERAL DIRECTIONS: Use a No. 2 pencil and fill in the circle that shows your answer. If you want to change your answer, please erase carefully. Please give ONLY ONE answer for each question, unless the directions for the question say "all that apply."

Your gender:
- ○ Male
- ○ Female

Your grade:
⑤ ⑥ ⑦ ⑧ ⑨ ⑩ ⑪ ⑫

Your age:
- ○ 9 or younger
- ○ 10
- ○ 11
- ○ 12
- ○ 13
- ○ 14
- ○ 15
- ○ 16
- ○ 17
- ○ 18
- ○ 19 or older

Your overall grade average:
- ○ Excellent
- ○ Very Good
- ○ Good
- ○ Average
- ○ Below Average
- ○ Poor

Your race or ethnic background:
- ○ White
- ○ Black
- ○ Hispanic
- ○ Mixed race
- ○ Asian or Pacific Islander
- ○ American Indian or Alaskan Native
- ○ Other

After high school, what do you think you will do? (Choose the ONE that is most important to you.)
- ○ Get a job
- ○ Go to college
- ○ Go to technical school
- ○ Join the military
- ○ Get married
- ○ I don't plan to finish high school
- ○ I'm not sure

DIRECTIONS: Below are some things that describe how you feel about school. Please fill in the circle that comes CLOSEST to showing how you feel about each of them. The higher the number, the more positive you are about each one.

School in general	DISLIKE ←	①	②	③	④	⑤	⑥	⑦	→ ENJOY
Teachers	NOT HELPFUL ←	①	②	③	④	⑤	⑥	⑦	→ HELPFUL
Subjects	BORING ←	①	②	③	④	⑤	⑥	⑦	→ INTERESTING
Classmates	UNFRIENDLY ←	①	②	③	④	⑤	⑥	⑦	→ VERY FRIENDLY
What I learn	NOT AT ALL IMPORTANT ←	①	②	③	④	⑤	⑥	⑦	→ VERY IMPORTANT

DIRECTIONS: A few different kinds of activities are listed below. Fill in the circle that comes CLOSEST to showing how often you do each type of activity. If you do several things from the same category, add their times together for your answer.

HOW OFTEN DO YOU TAKE PART IN:

	Never	Before But Not in the Past Year	A Few Times a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Entertainment and social activities (going to movies, on a date, to a concert, to a party, etc.)	○	○	○	○	○	○
Academic activities outside of school (doing homework, school projects, research, reading books, etc.)	○	○	○	○	○	○
Sports and physical activities (team sports, jogging, swimming, dance class, exercise, etc.)	○	○	○	○	○	○
Religious activities (going to services, church/synagogue activities, meetings, etc.)	○	○	○	○	○	○
Work-for-Pay activities (part-time job, babysitting, mowing lawns, chores at home, etc.)	○	○	○	○	○	○

DIRECTIONS: A few drugs are listed below. This set of questions deals with whether or not you are WILLING to try these things or WILLING to use them. These are NOT questions about whether or not you have actually used them. For each one, fill in the circle that comes CLOSEST to showing how you feel right now about using it. The higher the number, the more willing you are to use that substance. Here are some examples:

If you would never use it, mark the ⓪ | If you probably wouldn't use it, mark the ① | If you're not sure whether or not you would use it, mark the ② | If you would like to try it or would like to use it, mark the ③ | If you would use it any chance you got, mark the ④

CIGARETTES	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
ALCOHOL (beer, wine, coolers, hard liquor)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
MARIJUANA (pot, hash, hemp, weed)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how often you do each of the following things.

HOW OFTEN DO YOU:

	Never	Before But Not in the Past Year	A Few Times a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Skip school	○	○	○	○	○	○
Steal something	○	○	○	○	○	○
Cheat on schoolwork	○	○	○	○	○	○
Get drunk	○	○	○	○	○	○
Get high on drugs	○	○	○	○	○	○
Sell drugs	○	○	○	○	○	○
Push others around to get something you want or make them afraid	○	○	○	○	○	○

DIRECTIONS: Below is a list of alcohol, tobacco and other drugs. Please fill in the circle that comes CLOSEST to showing how often you use (or have ever used) each one of these things. REMEMBER THAT YOUR ANSWERS ARE ABSOLUTELY CONFIDENTIAL AND PRIVATE.

	Never Used	Used Before, But Not in the Past Year	Use About Once or Twice a Year	Use About Once or Twice a Month	Use About Once or Twice a Week	Use About Every Day
CIGARETTES	○	○	○	○	○	○
CHEWING TOBACCO, SNUFF	○	○	○	○	○	○
BEER (beer, ale, malt liquor)	○	○	○	○	○	○
WINE (wine, champagne)	○	○	○	○	○	○
COOLERS (wine- or alcohol-based)	○	○	○	○	○	○
LIQUOR (vodka, whiskey, rum, tequila, bourbon, etc.)	○	○	○	○	○	○
MARIJUANA (pot, hash, hemp, weed, smoke, etc.)	○	○	○	○	○	○
INHALANTS (whippets, butane, paint thinner or glue to sniff, etc.)	○	○	○	○	○	○
COCAINE (coke, snow, blow, dust, etc.)	○	○	○	○	○	○
CRACK (rock, fry, etc.)	○	○	○	○	○	○
HEROIN (smack, horse, skag, H, etc.)	○	○	○	○	○	○
HALLUCINOGENS (acid, trip, LSD, tabs, 'shrooms, etc.)	○	○	○	○	○	○
AMACTIN (mac, little feet, etc.)	○	○	○	○	○	○
CRYSTAL METH (ice, crank, speed, etc.)	○	○	○	○	○	○
Ecstasy or other DESIGNER DRUGS such as GHB, Special K, etc.	○	○	○	○	○	○
DOWNERS (tranqs, barbs, sedatives, sleeping pills, reds, etc.)	○	○	○	○	○	○
UPPERS (bennies, diet pills, caffeine pills, meth, speed, etc.)	○	○	○	○	○	○
TRAINING DRUGS (steroids, roids, juice, etc.)	○	○	○	○	○	○
Cough & Cold Medicine - To Get High on Purpose	○	○	○	○	○	○

DIRECTIONS: Below is a list of situations in which you may have been a driver or a passenger. Please fill in the circle that comes CLOSEST to showing how often you do each of these things.

HOW OFTEN DO YOU:

	Never	Before But Not in the Past Year	About Once or Twice a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Ride in a car in which the driver was drinking while driving or drinking shortly before driving?	○	○	○	○	○	○
Ride in a car in which the driver was smoking pot while driving or shortly before driving?	○	○	○	○	○	○
Drive a car while or shortly after drinking? ○ I don't drive	○	○	○	○	○	○
Drive a car while or shortly after smoking pot? ○ I don't drive	○	○	○	○	○	○

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If you have ever used alcohol, cigarettes or marijuana, mark the age at which you first used it.

	Never Used	Age 8	9	10	11	12	13	14	15	16	17	18
ALCOHOL	○	○	○	○	○	○	○	○	○	○	○	○
CIGARETTES	○	○	○	○	○	○	○	○	○	○	○	○
MARIJUANA	○	○	○	○	○	○	○	○	○	○	○	○

If you wanted to get any of the things listed below, how easy do you think it would be to get it?

	Very Difficult	Somewhat Difficult	Somewhat Easy	Very Easy	I'm Not Sure
Alcohol (beer, wine, coolers, or hard liquor)	○	○	○	○	○
Cigarettes	○	○	○	○	○
Marijuana	○	○	○	○	○
A drug like cocaine, LSD, or amphetamines	○	○	○	○	○
A handgun	○	○	○	○	○

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how many times these things have happened to you in the past 12 months at school.

IN THE PAST 12 MONTHS, HOW OFTEN HAVE YOU:

	Never	Once	2 or 3 times	4 or 5 times	6 to 9 times	10 times or more
Been threatened to be hit or beaten up **at school?**	○	○	○	○	○	○
Been attacked and hit by someone or beaten up **at school?**	○	○	○	○	○	○
Been threatened by someone with a weapon **at school?**	○	○	○	○	○	○
Been attacked by someone with a weapon **at school?**	○	○	○	○	○	○

When you smoke, how many cigarettes **per day** do you smoke **(on an average)?**

○ I don't smoke cigarettes
○ Less than 1 cigarette per day
○ 1 cigarette per day
○ 2 to 5 cigarettes per day
○ 6 to 10 cigarettes per day
○ 11 to 20 cigarettes per day
○ More than 20 cigarettes per day

During the past 30 days, how many times did you use marijuana?

○ 0 times
○ 1 or 2 times
○ 3 to 9 times
○ 10 to 19 times
○ 20 times or more

In the past year or so, where did you **drink alcohol? (Fill in ALL the circles that apply.)**

○ Didn't drink
○ In school
○ At home
○ At a friend's home
○ In a car, truck, etc.
○ At a hangout
○ At a fraternity house
○ Before/after a school activity (sports event, dance, etc.)
○ At work
○ College dorm or apartment

From which of these people or places did you get **alcohol? (Fill in ALL the circles that apply.)**

○ Didn't drink
○ Friends
○ Students at school
○ Street friends
○ People at work
○ College students
○ Family members
○ Adults other than family
○ Took from home
○ Bought from a store
○ Stole from a store
○ Stole from someone

In the past year or so, where did you **smoke cigarettes? (Fill in ALL the circles that apply.)**

○ Didn't smoke cigarettes
○ In school
○ At home
○ At a friend's home
○ In a car, truck, etc.
○ At a hangout
○ At a fraternity house
○ Before/after a school activity (sports event, dance, etc.)
○ At work
○ College dorm or apartment

From which of these people or places did you get **cigarettes? (Fill in ALL the circles that apply.)**

○ Didn't smoke cigarettes
○ Friends
○ Students at school
○ Street friends
○ People at work
○ College students
○ Family members
○ Adults other than family
○ Took from home
○ Bought from a store
○ Stole from a store
○ Stole from someone

In the past year or so, where did you **smoke marijuana? (Fill in ALL the circles that apply.)**

○ Didn't smoke marijuana
○ In school
○ At home
○ At a friend's home
○ In a car, truck, etc.
○ At a hangout
○ At a fraternity house
○ Before/after a school activity (sports event, dance, etc.)
○ At work
○ College dorm or apartment

From which of these people or places did you get **marijuana? (Fill in ALL the circles that apply.)**

○ Didn't smoke marijuana
○ Friends
○ Students at school
○ Street friends
○ People at work
○ College students
○ Family members
○ Adults other than family
○ Took from home
○ Stole from someone

The last time you were in a physical fight at school, with whom did you fight?

○ I was never in a physical fight at school
○ A total stranger
○ A friend or someone I know
○ A boyfriend, girlfriend or date
○ A brother, sister, or family member
○ Someone not listed above
○ More than one person

During the past 12 months, how many times were you in a physical fight at school?

○ 0 times
○ 1 time
○ 2 or 3 times
○ 4 or 5 times
○ 6 or 7 times
○ 8 or 9 times
○ 10 or 11 times
○ 12 or more times

During the past 30 days, have you been afraid of being hurt by someone **in your school or on school property?**

○ Yes
○ No

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how many times you have done each of these things in the past 12 months at school.

IN THE PAST 12 MONTHS, HOW OFTEN HAVE YOU:

	Never	Once	2 or 3 times	4 or 5 times	6 to 9 times	10 times or more
Threatened to beat up someone at school?	◯	◯	◯	◯	◯	◯
Hit someone with your fists or beat up someone at school?	◯	◯	◯	◯	◯	◯
Threatened someone with a weapon at school?	◯	◯	◯	◯	◯	◯
Attacked someone with a weapon at school?	◯	◯	◯	◯	◯	◯

How many students in your grade do you think . . .

	None	About 1/4	About 1/2	About 3/4	All
use ALCOHOL?	◯	◯	◯	◯	◯
use MARIJUANA?	◯	◯	◯	◯	◯
smoke CIGARETTES?	◯	◯	◯	◯	◯
use OTHER DRUGS?	◯	◯	◯	◯	◯
get DRUNK?	◯	◯	◯	◯	◯

What message do you get about use of alcohol?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

What message do you get about use of cigarettes?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

What message do you get about use of marijuana?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

During the past 12 months, how many times did you carry a weapon TO SCHOOL?

◯ 0 times
◯ 1 or 2 times
◯ 3 to 9 times
◯ 10 to 19 times
◯ 20 times or more

What **one kind of weapon** do you carry **most often** TO SCHOOL?

◯ I don't carry a weapon to school
◯ A handgun
◯ Other gun, such as a rifle or shotgun
◯ A knife, razor, or box cutter
◯ A club, stick, bat, or pipe
◯ A martial arts weapon
◯ A semi-automatic weapon
◯ Some other weapon

From which of these people or places **have you gotten weapons that you took TO SCHOOL? (Fill in ALL the circles that apply.)**

◯ Didn't carry a weapon
◯ Friends
◯ Students at school
◯ Street friends
◯ People at work
◯ College students
◯ Family members
◯ Adults other than family
◯ Took from home
◯ Bought from a store
◯ Stole from a store
◯ Stole from someone

Why do you carry a weapon TO SCHOOL? **(Fill in ALL the circles that apply.)**

◯ I have never carried a weapon
◯ I was hurt by someone carrying a weapon
◯ I saw someone else hurt by someone carrying a weapon
◯ I was threatened
◯ I'm afraid
◯ My friends carry weapons
◯ For hunting
◯ For protection
◯ For show
◯ For pushing around other people
◯ Other

Are you reasonably sure that no one at your school can identify anyone's questionnaire?

◯ Yes
◯ Not sure
◯ No

When you have finished the survey, please place it face up in front of you, and wait quietly for it to be collected.

APPENDIX B

2004 PPAAUS ADMINISTRATION INSTRUCTIONS AND TEACHER SCRIPT

Instructions for Administering the
PRIMARY PREVENTION AWARENESS, ATTITUDE, AND USE SURVEY

1. Follow the attached script. You may make minor changes to reflect your teaching style, but the order of presentation and the thrust of the text MUST NOT BE CHANGED. This script is designed to eliminate as many confounding factors as possible. Be sure that you do not omit the sentence about the voluntary nature of the study.
2. Always refer to the instrument as a “survey” or “questionnaire.” Do not call it a “test.”
3. Present the script in an unbiased and enthusiastic manner in order to increase the likelihood of accurate reporting.
4. DO NOT WALK AROUND THE ROOM while students are completing the survey. Do not look at any student’s questionnaire. Keep a blank survey at your desk from which to answer any questions from students.
5. Reinforce, when necessary, that all responses are confidential.
6. Do not speculate on how the survey results will be used, and do not encourage the students to do so.
7. While students are completing the survey, handle disruptions and interruptions with minimal response.
    a. When answering a student’s question, be matter-of-fact so that you do not influence his or her response.
    b. “Just give your own opinion,” or “Just think about the question and give your own best answer” are usually sufficient.
    c. It is best to send troublemakers out of the room as soon as you sense that they are not taking the survey seriously.
8. This survey includes a self-reported use question about “AMACTIN,” which is a fabricated word. In PPAAUS, it is a lie-indicator item. It is absolutely essential that you do not tell the students what it is. Our suggested response to requests for a definition of the bogus drug is, “I’m not sure what it is. I’ll try to get more information about it.”
9. Be sure that you have all of the supplies you need before the survey session begins. You will need:
    a. An adequate number of surveys;
    b. Extra pencils and erasers;
    c. An envelope for the completed surveys; and
    d. Your script.
10. Do not collect the completed surveys yourself; choose a student who is liked and trusted by a great majority of the class. The student will collect the surveys, shuffle them, place them in an envelope, and seal it.
11. Collect ALL surveys. Put any blank surveys in the envelope of completed questionnaires. Do not allow any student to leave the room with a questionnaire. Other classes may be taking the survey later in the day.
12. Read the script through a few times before the survey session to familiarize yourself with the procedure and to note any small changes you will make in your presentation.
13. Your school administration will advise you how to return completed surveys to the office. If you would like to address questions or comments to Diagnostics Plus, please given them to your school survey coordinator and ask that they be included with the surveys to be returned.

THANK YOU VERY MUCH FOR YOUR COOPERATION

Teacher Script
Primary Prevention Awareness, Attitude, and Use Survey

Instead of our usual activity today, this class will be filling out a student questionnaire. This is NOT a test, so there are no right or wrong answers. What's RIGHT is what's TRUE for you. Please don't put your name anywhere on the survey. The people in this school district want to know about OVERALL attitudes, not what any ONE student thinks. This is the questionnaire. (*Hold it up for the class to see*.) You'll be reading the questions and marking your answers right on the survey.

Some of the questions on this survey deal with drugs and alcohol. I really want you to feel ABSOLUTELY SURE that no one in this school or this district knows which questionnaire belongs to which student…so I'm going to stay at the front of the room while you're working on t his, and I won't look at anyone's answers. If you don't want to answer a certain question, you don't have to.

When everyone is finished. I'm going to ask someone to collect your questionnaires row by row, to mix them up while they're being collected, and NOT to look at anyone's answers. Then all of the questionnaires will go in this envelope, and it will be sealed. It won't be opened until it's in central Pennsylvania with the people who are doing this research. Does everyone have a pencil and eraser? (*Distribute supplies to those students who need them*.) I'm going to hand out the questionnaires now. Don't begin until I tell you to start. (*Distribute the surveys or ask a student to distribute them. When each student has a survey, continue*.)

Let's read the general directions together, right after the words "DO NOT PUT YOUR NAME ANYWHERE ON THIS QUESTIONNAIRE." "Use a Number Two pencil and fill in the circle that shows your answer. If you want to change your answer, erase carefully. Please give ONLY ONE answer for each question, unless the directions for the question say 'all that apply.'"

Before many sections of the questionnaire, you'll find specific directions. Read them carefully before you go on to answer the questions.

You may begin now. THERE WILL BE NO TALKING UNTIL EVERYONE IS FINISHED. If you have any questions while you're filling in this survey, leave your questionnaire at your seat and come to my desk, and I'll try to help you.

When you've finished answering the questions, put your survey on your desk, and sit QUIETLY until everyone has finished. After all of the surveys have been collected, we can talk about the questionnaire, if you'd like to.

When all students have completed the questionnaire, ask the designated student to collect them. (Choose a student who is liked and trusted by the rest of the class). Be certain that the collector DOES NOT look at anyone's survey, and be certain that ALL questionnaires are returned. Keep one copy for yourself for reference and place all other unused surveys in the collector's envelope.

APPENDIX C

INDEPENDENT AND CONTROL VARIABLES: CODING SCHEME

Table C1.

Block #1: Community Domain Predictors

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors				
MessMedAlc	Perceived media message about alcohol use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use/mixed/no message (1)	Dichotomy
MessMedCig	Perceived media message about cigarette use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use/mixed/no message (1)	Dichotomy
MessMedMar	Perceived media message about marijuana use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use/mixed/no message (1)	Dichotomy
EasyAlc	Ease in getting alcohol	Very difficult (0), Somewhat diff (1), Not sure (2), SW easy (3), Very easy (4)	---	Treat as Continuous
EasyCig	Ease in getting cigarettes	Very difficult (0), Somewhat diff (1), Not sure (2), SW easy (3), Very easy (4)	---	Treat as Continuous
EasyMar	Ease in getting marijuana	Very difficult (0), Somewhat diff (1), Not sure (2), SW easy (3), Very easy (4)	---	Treat as Continuous

--- not applicable.

Table C2.

Block #2: School Domain Predictors

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors				
Truancy	Lifetime freq. skipping school	Never (0), Not in past year (1), Few X yr (2), 1-2X mth (3), 1-2X wk (4), Almost every day (5)	Never (0), Not in past year (1), Few X yr (2), 1-2X mth (3), 1-2X wk/Almost every day (4)	Treat as Continuous
Cheat	Lifetime freq. cheating on schoolwork	Never (0), Before, but not in past year (1), Few X's a year (2), Once/twice a month (3), Once/twice a week (4), Almost every day (5)	Never (0), Not in past year (1), Few X yr (2), 1-2 X mth, 1-2X wk, Almost every day (3)	Treat as Continuous
MessTchrAlc	Perceived teacher message about alcohol use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0), OK to use/mixed message/no message (1)	Dichotomy
MessTchrCig	Perceived teacher message about cigarette use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use/mixed message/no message (1)	Dichotomy
MessTchrMar	Perceived teacher message about marijuana use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use/mixed message/no message (1)	Dichotomy

(Table C2 continues)

(Table C2 continued)

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Protective Factors				
CllgeAsp	Planning on attending college	No (0), Yes (1)	---	Dichotomy
AttachSchool (Index)	Degree to which school is enjoyable, teachers are helpful, subjects are interesting	1 → 7 (negative → positive agreement)	Lowest level of school attachment (3) → Highest level of school attachment (21)	Continuous
Academic	Lifetime freq. involvement in academic activities outside of school	Never (0), Before, not past year (1), Few times year (2), Once/twice month (3), Once/twice week (4), Almost every day (5)	Never, Not in past Yr, Few X yr (0), 1-2X mth (1), 1-2X wk (2), Almost every day (3)	Treat as Continuous
HiAcadPerf	Overall grade average	Poor (0), Below average (1), Average (2), Good (3), Very good (4), Excellent (5)	---	Treat as Continuous

--- not applicable.

Table C3.

Block #3: Family Domain Predictors

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors				
MessParAlc	Perceived parental message about alcohol use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy
MessParCig	Perceived parental message about cigarette use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy
MessParMar	Perceived parental message about marijuana use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy

Table C4.

Block #4: Peer Domain Predictors

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors				
PeerAlc	Perceived # alcohol-using students in same grade	None (0), About ¼ (1), About ½ (2), About ¾ (3), All (4)	---	Treat as Continuous
PeerCig	Perceived # cigarette-using students in same grade	None (0), About ¼ (1), About ½ (2), About ¾ (3), All (4)	---	Treat as Continuous
PeerMar	Perceived # marijuana-using students in same grade	None (0), About ¼ (1), About ½ (2), About ¾ (3), All (4)	---	Treat as Continuous
MessPeerAlc	Perceived peer message about alcohol use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy
MessPeerCig	Perceived peer message about cigarette use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy
MessPeerMar	Perceived peer message about marijuana use	OK to use (1), No message (2), Mixed message (3), Not OK to use (4)	Not OK to use (0) OK to use, mixed message, no message (1)	Dichotomy

--- not applicable.

Table C5.

Block #5: Individual Domain Predictors

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors				
AlcInit	Age of alcohol initiation	Continuous measure, age of initiation	Alcohol abstention (0) Alcohol initiation (1)	Dichotomy
AlcEarly	Age of alcohol initiation	Continuous measure, age of initiation	Alc abstainers/Late alc initiates, 16-18 yrs (0) Early alc initiates, 8-15 yrs (1)	Dichotomy
AlcLate	Age of alcohol initiation	Continuous measure, age of initiation	Alc abstainers/Early alc initiates, 8-15 yrs (0) Late alc initiates, 16-18 yrs (1)	Dichotomy
CigEarly	Age of cigarette initiation	Continuous measure, age of initiation	Cig abstainers/Late cig initiates, 16-18 yrs (0) Early cig initiates, 8-15 yrs (1)	Dichotomy
CigLate	Age of cigarette initiation	Continuous measure, age of initiation	Cig abstainers/Early cig initiates, 8-15 yrs (0) Late cig initiates, 16-18 yrs (1)	Dichotomy

(Table C5 continues)

(Table C5 continued)

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Risk Factors Continued…				
AlcIntent	Degree of willingness to use alcohol	Would Not (0) → Any chance (4)	Would not (0) Probably not, Not sure, Would like to, Any chance (1)	Dichotomy
CigIntent	Degree of willingness to use cigarettes	Would Not (0) → Any chance (4)	Would not (0) Probably not, Not sure, Would like to, Any chance (1)	Dichotomy
MarIntent	Degree of willingness to use marijuana	Would Not (0) → Any chance (4)	Would not (0) Probably not, Not sure, Would like to, Any chance (1)	Dichotomy
Steal	Lifetime frequency of stealing	Never (0), Not in past yr (1), Few X yr (2), 1-2X mth (3), 1-2X wk (4), Almost every day (5)	Never stole (0) Stole (1)	Dichotomy
ViolBeh	At school, past yr. freq. pushing someone around, hitting someone, threatening beat someone	Never (0), 1X (1), 2-3Xs (2), 4-5Xs (3), 6-9Xs (4), 10+ Xs (5)	No violent acts (0) 1 or more violent acts (1)	Dichotomy

(Table C5 continues)

(Table C5 continued)

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Protective Factors				
Sports	Lifetime freq. involvement in sports, physical activity	Never (0), Before, not in past year (1), Few Xs year (2), Once/twice month (3), Once/twice week (4), Almost every day (5)	Never, Not in past yr, Few X yr (0), 1-2X mth (1), 1-2X wk (2), Almost every day (3)	Treat as Continuous
Work	Lifetime freq. work-for-pay	Never (0), Before, not in past year (1), Few Xs year (2), Once/twice month (3), Once/twice week (4), Almost every day (5)	---	Treat as Continuous
Social	Lifetime freq. involvement in social, entertainment activities	Never (0), Before, not in past year (1), Few Xs year (2), Once/twice month (3), Once/twice week (4), Almost every day (5)	---	Treat as Continuous
Religious	Lifetime freq. going to religious services, activities, meetings	Never (0), Before, not in past year (1), Few Xs year (2), Once/twice month (3), Once/twice week (4), Almost every day (5)	---	Treat as Continuous

--- not applicable.

Table C6.

Block #6: Control Variables

Predictor	Original Survey Item	Original Coding	Recode	Variable Type
Gender	Your gender	Male (1), Female (2)	Female (0) Male (1)	Dichotomy
Race	Your race or ethnic background	White (1), Black (2), Hispanic (3), Mixed (4), Asian (5), American Indian (6), Other (7)	Nonwhite (0) White (1)	Dichotomy
Age [a]	Your age	11 years (11) → 19 years or older (19)	11 years (0) → 19 years or older (8)	Continuous
AlcFreq (Index)	Lifetime freq. beer, wine, coolers, liquor use	Abstention (0), Before, not in past year (1), Once/twice year (2), Once/twice month (3), Once/twice week (4), About every day (5)	Abstention (0) → Wide-range/frequent use (13)	Continuous
CigFreq	Lifetime freq. of cigarette use	Abstention (0), Before, not in past year (1), Once/twice year (2), Once/twice month (3), Once/twice week (4), About every day (5)	---	Continuous

[a] Only will be used in total sample models.
--- not applicable.

APPENDIX D

DETAILED ACCOUNT OF GUTTMAN SCALE DEVELOPMENT

The following discussion constitutes a detailed supplement to the explanation of Guttman scaling that was provided in Chapter 6. Additional information concerning Guttman scaling in general, and the analytic steps that were taken in testing H1, are described.

Guttman scaling was initially developed as a way to scale attitudes (Guttman, 1944). This technique can be used to scale behaviors as well. Depending on the goal of the analysis, a latent variable (and its manifest indicators) either is established a priori (as is done in hypothesis testing) or is determined through exploratory analysis (McIver & Carmines, 1982). Concerning confirmatory analysis (i.e., as in the current research), a hypothesis is supported if the response pattern that is proposed is supported by the data. Evidence of support is found in the value of the CR and CS. Once a hypothesis is supported, further empirical analysis is required in order to gain a more in-depth understanding of the latent construct or items to which it is comprised (e.g., predicting positioning on the continuum or why scale items are ordered in a particular manner).

Scale Development and Analysis

Step #1. A Guttman scalogram response matrix was developed following common convention (McIver & Carmines, 1982). Columns constituted drug scale items and rows contained response patterns for each case (i.e., respondent). Initially, cases were ordered numerically starting at the top of the matrix, such that the first row in the matrix constituted scale item responses for Case #1 (followed by Case #2, #3, etc.).

Step #2. The total number of affirmative responses were summed (for each drug scale item) and then divided by the total number of cases. This calculation identified (for each drug item) the proportion of cases who responded affirmatively to initiating (McIver & Carmines, 1982).

Step #3. Both scale items and cases were arranged in order of magnitude, resulting in a hierarchical pattern of responses that resembled a triangle (McIver & Carmines, 1982). Concerning scale items, the drug scale item found to have the largest proportion of affirmative responses constituted the first drug column in the matrix, while the second drug column contained the drug item that had the second largest proportion of responses.

It is worth noting that due to the nature of the hypothesis that was tested, it was expected that this column rotation would not be necessary. Since the hypothesis that was tested posited that alcohol is the first drug that is initiated (followed by cigarettes, and then marijuana), and the underlying continuum of drug involvement constitutes a hierarchy, it was expected that a higher proportion of students self-reported alcohol than cigarette or marijuana initiation, and a larger number of students self-reported cigarette than marijuana initiation (thus exhibiting a hierarchical response pattern). Hence, it was assumed that the initial ordering of drug items in the matrix constituted the correct ordering, with column rotation not necessary.

Using scale scores for each case, response patterns of soft drug initiation also were rearranged (i.e., rank-ordered) according to order of magnitude (McIver & Carmines, 1982). Cases with the highest scale scores (i.e., 3) were positioned at the top of the matrix, while cases with the lowest scale scores (i.e., 0) were positioned at the bottom of the matrix.

Error identification and counting. Discussed in Chapter 6, errors were identified with the use of horizontal lines. The logistics underlying this approach to error identification and counting is synonymous to the widely used Goodenough-Edwards (G-E) method of error-counting (Edwards, 1948). Regardless of whether error is identified and counted using horizontal lines in the response matrix, or is assessed by examining each observed response pattern (as is done using the G-E method), error is identified in terms of “the number of responses departing from the patterns predicted…scores which are inconsistent with the assumption of perfect reproducibility would be scored as the nearest scale type consistent with the notion of the scale” (Edwards, 1948, p. 348).

The only difference between the horizontal line approach and the G-E method is that the latter strategy identifies and counts errors on a case-by-case basis. Since the G-E method is more time-consuming than the horizontal line approach (particularly with large sample sizes), and both methods identify and count the same number of errors, the current research used the horizontal line approach.

As an indicator of cumulation, scale scores are important for identifying errors, since they infer which scale items should have been responded to affirmatively (and in what temporal order). For example, according to the hypothesis that was tested, a scale score of “2” infers that only alcohol and cigarette use have been initiated, and in that respective order (i.e., 1-2-0). An examination of a student’s item responses may indicate that alcohol and marijuana use were initiated (and in that temporal order). If an attempt were made to reproduce the scale item responses that this student provided based solely on knowledge of this respondent’s scale score (i.e., 2), one would make two errors (one for cigarette initiation and one for marijuana initiation).

Evaluating Scale Fit

The CR and CS are two criteria typically used to evaluate the scalability of a set of scale items. The minimal acceptability standards associated with these coefficients (i.e., .90 and .60, respectively) constitute benchmarks for determining the scalability of a set of response patterns. The scalability of the soft drug initiation data was evaluated according these two criteria.

Coefficient of reproducibility. Discussed in Chapter 6, Guttman (1950) developed the CR to estimate the goodness of fit between observed response patterns and the hypothesized response pattern. The less error that exists in observed response patterns, the better the fit of the scale, and the more apt that individual scale items may be predicted accurately. Moreover, the less error that exists, the more likely that scale items are internally consistent and are (together) representative of a unidimensional, latent construct (McIver & Carmines, 1982). In order for a set of items to be considered scalable (i.e., capturing a unidimensional, latent construct), Guttman (1950) established the standard that the error that results from reproducing scale items equals 10% (or less) of the total responses. This constitutes the rationale underlying the minimal acceptability threshold of CR ≥ .90 (Guttman, 1950).

A CR < .90 means that the observed response patterns contain too much error and little confidence can be placed in the accuracy with which scale item responses are predicted by scale scores (Guttman, 1950). It is important to note that a high degree of reproducibility (e.g., CR ≥ .90) does not ensure that a Guttman scale actually measures the latent construct in question. The CR simply is a measure of confidence, with a high (and acceptable) CR indicating that the scale items are internally consistent and are measuring the same thing (Babbie, 2004; Hindelang, Hirschi, & Weis, 1981).

Coefficient of scalability. The CR is a necessary, but insufficient, benchmark for determining scalability, because scale reproducibility can be impacted by the marginal distributions of scale items (Guttman, 1950). Since a Guttman scale is assumed to be cumulative and hierarchical in nature, some scale items will elicit a high level of agreement (e.g., many youth reporting alcohol initiation), while other scale items will elicit a low level of agreement (e.g., not many youth reporting marijuana initiation). This skewness in the marginal distributions of scale items can inflate the CR, since responses to these scale items will tend to be more reproducible (as a result of less error) given knowledge of scale scores than scale items that elicit less extreme responses distributions (Schwartz, 1986; Stookey & Baer, 1976).

The CS provides a “check” against an inflated CR in reflecting the degree to which responses to scale items can be predicted given only knowledge of the marginal frequencies (Smith, 1968). In order to calculate the CS (PI/1-MMR), values for the percentage improvement (PI) and the minimal marginal reproducibility (MMR) first must be obtained. The MMR is simply the sum of the proportion of cases that initiated (p) each scale item [or the proportion of cases that abstained (q) from each scale item, whichever is largest] divided by the number of scale items (k). The PI equates to the difference between the MMR and the CR.

APPENDIX E

2001 PPAAUS INSTRUMENT

PRIMARY PREVENTION AWARENESS, ATTITUDE AND USE SURVEY

DO NOT PUT YOUR NAME ANYWHERE ON THIS SURVEY

GENERAL DIRECTIONS: Use a No. 2 pencil and fill in the circle that shows your answer. If you want to change your answer, please erase carefully. Please give ONLY ONE answer for each question, unless the directions for the question say "all that apply."

Your gender:
- ◯ Male
- ◯ Female

Your grade:
⑤ ⑥ ⑦ ⑧ ⑨ ⑩ ⑪ ⑫

Your age:
- ◯ 9 or younger
- ◯ 10
- ◯ 11
- ◯ 12
- ◯ 13
- ◯ 14
- ◯ 15
- ◯ 16
- ◯ 17
- ◯ 18
- ◯ 19 or older

Your overall grade average:
- ◯ Excellent
- ◯ Very Good
- ◯ Good
- ◯ Average
- ◯ Below Average
- ◯ Poor

Your race or ethnic background:
- ◯ White
- ◯ Black
- ◯ Hispanic
- ◯ Mixed race
- ◯ Asian or Pacific Islander
- ◯ American Indian or Alaskan Native
- ◯ Other

After high school, I think I will: (choose the ONE that is most important to you)
- ◯ Get a job
- ◯ Go to college
- ◯ Go to technical school
- ◯ Join the military
- ◯ Get married
- ◯ I don't plan to finish high school
- ◯ I'm not sure

DIRECTIONS: Below are some things that describe how you feel about school. Please fill in the circle that comes CLOSEST to showing how you feel about each of them. The higher the number, the more positive you are about each one.

School in general	DISLIKE ←	①	②	③	④	⑤	⑥	⑦	→ ENJOY
Teachers	NOT HELPFUL ←	①	②	③	④	⑤	⑥	⑦	→ HELPFUL
Subjects	BORING ←	①	②	③	④	⑤	⑥	⑦	→ INTERESTING
Classmates	UNFRIENDLY ←	①	②	③	④	⑤	⑥	⑦	→ VERY FRIENDLY

DIRECTIONS: A few different kinds of activities are listed below. Fill in the circle that comes CLOSEST to showing how often you do each type of activity. If you do several things from the same category, add their times together for your answer.

HOW OFTEN DO YOU TAKE PART IN:

	Never	Before But Not in the Past Year	A Few Times a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Entertainment and social activities (going to movies, on a date, to a concert, to a party, etc.)	◯	◯	◯	◯	◯	◯
Academic activities (doing homework, school projects, research, reading books, etc.)	◯	◯	◯	◯	◯	◯
Sports and physical activities (team sports, jogging, swimming, dance class, exercise, etc.)	◯	◯	◯	◯	◯	◯
Religious activities (going to services, church/synagogue activities, meetings, etc.)	◯	◯	◯	◯	◯	◯
Work-for-Pay activities (part-time job, babysitting, mowing lawns, chores at home, etc.)	◯	◯	◯	◯	◯	◯

DIRECTIONS: A few drugs are listed below. This set of questions deals with whether or not you are WILLING to try these things or WILLING to use them. These are NOT questions about whether or not you have actually used them. For each one, fill in the circle that comes CLOSEST to showing how you feel right now about using it. The higher the number, the more willing you are to use that substance. Here are some examples:

If you would never use it, mark the ⓪ — If you probably wouldn't use it, mark the ① — If you're not sure whether or not you would use it, mark the ② — If you would like to try it or would like to use it, mark the ③ — If you would use it any chance you got, mark the ④

CIGARETTES	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
ALCOHOL (beer, wine, coolers, "hard" liquor)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
MARIJUANA (pot, hash, hemp, weed)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
COCAINE (coke, snow, blow, dust)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
HALLUCINOGENS (acid, trip, LSD, 'shrooms)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE
INHALANTS (whippets, butane, paint thinner)	NEVER ←	⓪	①	②	③	④	→ ANY CHANCE

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how often you do each of the following things.

HOW OFTEN DO YOU:

	Never	Before But Not in the Past Year	A Few Times a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Skip school	○	○	○	○	○	○
Steal something	○	○	○	○	○	○
Cheat on schoolwork	○	○	○	○	○	○
Get drunk	○	○	○	○	○	○
Get high on drugs	○	○	○	○	○	○
Sell drugs	○	○	○	○	○	○
Push others around to get something you want or make them afraid	○	○	○	○	○	○

DIRECTIONS: Below is a list of alcohol, tobacco and other drugs. Please fill in the circle that comes CLOSEST to showing how often you use (or have ever used) each one of these things. REMEMBER THAT YOUR ANSWERS ARE ABSOLUTELY CONFIDENTIAL AND PRIVATE.

	Never Used	Used Before, But Not in the Past Year	Use About Once or Twice a Year	Use About Once or Twice a Month	Use About Once or Twice a Week	Use About Every Day
CIGARETTES	○	○	○	○	○	○
CHEWING TOBACCO, SNUFF	○	○	○	○	○	○
BEER (beer, ale, malt liquor)	○	○	○	○	○	○
WINE (wine, champagne)	○	○	○	○	○	○
COOLERS (wine- or alcohol-based)	○	○	○	○	○	○
LIQUOR (vodka, whiskey, rum, tequila, bourbon, etc.)	○	○	○	○	○	○
MARIJUANA (pot, hash, hemp, weed, smoke, etc.)	○	○	○	○	○	○
INHALANTS (whippets, butane, paint thinner or glue to sniff, etc.)	○	○	○	○	○	○
COCAINE (coke, snow, blow, dust, etc.)	○	○	○	○	○	○
CRACK (rock, fry, etc.)	○	○	○	○	○	○
HEROIN (smack, horse, skag, H, etc.)	○	○	○	○	○	○
HALLUCINOGENS (acid, trip, LSD, tabs, 'shrooms, etc.)	○	○	○	○	○	○
AMACTIN (mac, little feet, etc.)	○	○	○	○	○	○
CRYSTAL METH (ice, crank, speed, etc.)	○	○	○	○	○	○
DESIGNER DRUGS (Ecstasy, XTC, China White, etc.)	○	○	○	○	○	○
DOWNERS (tranqs, barbs, sedatives, sleeping pills, reds, etc.)	○	○	○	○	○	○
UPPERS (bennies, diet pills, caffeine pills, meth, speed, etc.)	○	○	○	○	○	○
TRAINING DRUGS (steroids, roids, juice, etc.)	○	○	○	○	○	○
Cough & Cold Medicine - To Get High on Purpose	○	○	○	○	○	○

DIRECTIONS: Below is a list of situations in which you may have been a driver or a passenger. Please fill in the circle that comes CLOSEST to showing how often you do each of these things.

HOW OFTEN DO YOU:

	Never	Before But Not in the Past Year	About Once or Twice a Year	About Once or Twice a Month	About Once or Twice a Week	Almost Every Day
Ride in a car in which the driver was drinking while driving or drinking shortly before driving?	○	○	○	○	○	○
Ride in a car in which the driver was smoking pot while driving or shortly before driving?	○	○	○	○	○	○
Ride in a car in which the driver was drinking AND smoking pot?	○	○	○	○	○	○
Drive a car while or shortly after drinking? ○ I don't drive	○	○	○	○	○	○
Drive a car while or shortly after smoking pot? ○ I don't drive	○	○	○	○	○	○
Drive a car while or shortly after drinking AND smoking pot? ○ I don't drive	○	○	○	○	○	○

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If you have ever used alcohol, cigarettes or marijuana, mark the age at which you first used it.

	Never Used	Age 8	9	10	11	12	13	14	15	16	17	18
ALCOHOL	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯
CIGARETTES	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯
MARIJUANA	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯	◯

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how many times these things have happened to you in the past 12 months at school.

IN THE PAST 12 MONTHS, HOW OFTEN HAVE YOU:

	Never	Once	2 or 3 times	4 or 5 times	6 to 9 times	10 times or more
Been threatened to be hit or beaten up at school?	◯	◯	◯	◯	◯	◯
Been attacked and hit by someone or beaten up at school?	◯	◯	◯	◯	◯	◯
Been threatened by someone with a weapon at school?	◯	◯	◯	◯	◯	◯
Been attacked by someone with a weapon at school?	◯	◯	◯	◯	◯	◯

When you smoke, how many cigarettes **per day** do you smoke **(on an average)?**
- ◯ I don't smoke cigarettes
- ◯ Less than 1 cigarette per day
- ◯ 1 cigarette per day
- ◯ 2 to 5 cigarettes per day
- ◯ 6 to 10 cigarettes per day
- ◯ 11 to 20 cigarettes per day
- ◯ More than 20 cigarettes per day

During the past 30 days, how many times did you use marijuana?
- ◯ 0 times
- ◯ 1 or 2 times
- ◯ 3 to 9 times
- ◯ 10 to 19 times
- ◯ 20 times or more

In the past year or so, where did you **drink alcohol? (Fill in ALL the circles that apply.)**
- ◯ Didn't drink
- ◯ In school
- ◯ At parties
- ◯ At home
- ◯ At a friend's home
- ◯ In a car, truck, etc.
- ◯ At a hangout
- ◯ Before/after a school activity (sports event, dance, etc.)
- ◯ At a public place (mall, etc.)
- ◯ At work

From which of these people or places did you get **alcohol? (Fill in ALL the circles that apply.)**
- ◯ Didn't drink
- ◯ Friends
- ◯ Students at school
- ◯ Out-of-school kids
- ◯ People at work
- ◯ College students
- ◯ Family
- ◯ Adults other than family
- ◯ Bought from a store
- ◯ Stole from a store
- ◯ Stole from someone

In the past year or so, where did you **smoke cigarettes? (Fill in ALL the circles that apply.)**
- ◯ Didn't smoke cigarettes
- ◯ In school
- ◯ At parties
- ◯ At home
- ◯ At a friend's home
- ◯ In a car, truck, etc.
- ◯ At a hangout
- ◯ Before/after a school activity (sports event, dance, etc.)
- ◯ At a public place (mall, etc.)
- ◯ At work

From which of these people or places did you get **cigarettes? (Fill in ALL the circles that apply.)**
- ◯ Didn't smoke cigarettes
- ◯ Friends
- ◯ Students at school
- ◯ Out-of-school kids
- ◯ People at work
- ◯ College students
- ◯ Family
- ◯ Adults other than family
- ◯ Vending machine
- ◯ Bought from a store
- ◯ Stole from a store
- ◯ Stole from someone

In the past year or so, where did you **smoke marijuana? (Fill in ALL the circles that apply.)**
- ◯ Didn't smoke marijuana
- ◯ In school
- ◯ At parties
- ◯ At home
- ◯ At a friend's home
- ◯ In a car, truck, etc.
- ◯ At a hangout
- ◯ Before/after a school activity (sports event, dance, etc.)
- ◯ At a public place (mall, etc.)
- ◯ At work

From which of these people or places did you get **marijuana? (Fill in ALL the circles that apply.)**
- ◯ Didn't smoke marijuana
- ◯ Friends
- ◯ Students at school
- ◯ Out-of-school kids
- ◯ People at work
- ◯ College students
- ◯ Family
- ◯ Adults other than family
- ◯ Stole from someone

Smoking marijuana:
- ◯ Speeds up your heart rate
- ◯ Slows down your heart rate
- ◯ Don't know

The last time you were in a physical fight at school, with whom did you fight?
- ◯ I was never in a physical fight at school
- ◯ A total stranger
- ◯ A friend or someone I know
- ◯ A boyfriend, girlfriend or date
- ◯ A brother, sister, or family member
- ◯ Someone not listed above
- ◯ More than one person

During the past 12 months, how many times were you in a physical fight at school?
- ◯ 0 times
- ◯ 1 time
- ◯ 2 or 3 times
- ◯ 4 or 5 times
- ◯ 6 or 7 times
- ◯ 8 or 9 times
- ◯ 10 or 11 times
- ◯ 12 or more times

If someone has just one drink of **alcohol:**
- ◯ It doesn't affect their coordination
- ◯ It affects their coordination
- ◯ Don't know

During the past 30 days, have you been afraid of being hurt by someone **in your school or on school property?**
- ◯ Yes
- ◯ No

During the past 12 months, how many times did you carry a weapon TO SCHOOL?
- ◯ 0 times
- ◯ 1 or 2 times
- ◯ 3 to 9 times
- ◯ 10 to 19 times
- ◯ 20 times or more

DIRECTIONS: Fill in the circle that comes CLOSEST to showing how many times you have done each of these things in the past 12 months at school.

IN THE PAST 12 MONTHS, HOW OFTEN HAVE YOU:

	Never	Once	2 or 3 times	4 or 5 times	6 to 9 times	10 times or more
Threatened to beat up someone at school?	◯	◯	◯	◯	◯	◯
Hit someone with your fists or beat up someone at school?	◯	◯	◯	◯	◯	◯
Threatened someone with a weapon at school?	◯	◯	◯	◯	◯	◯
Attacked someone with a weapon at school?	◯	◯	◯	◯	◯	◯

How many students in your grade do you think . . .

	None	About 1/4	About 1/2	About 3/4	All
use ALCOHOL?	◯	◯	◯	◯	◯
use MARIJUANA?	◯	◯	◯	◯	◯
smoke CIGARETTES?	◯	◯	◯	◯	◯
use OTHER DRUGS?	◯	◯	◯	◯	◯
get DRUNK?	◯	◯	◯	◯	◯

What message do you get about use of alcohol?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

What message do you get about use of cigarettes?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

What message do you get about use of marijuana?

	It's Not OK to Use	Mixed Message	It's OK to Use	No Message Given
From your parents	◯	◯	◯	◯
From your friends	◯	◯	◯	◯
From your teachers	◯	◯	◯	◯
From the media (TV, radio, movies, music, magazines)	◯	◯	◯	◯

What **one kind of weapon** do you carry **most often** TO SCHOOL?

- ◯ I don't carry a weapon to school
- ◯ A handgun
- ◯ Other gun, such as a rifle or shotgun
- ◯ A knife, razor, or box cutter
- ◯ A club, stick, bat, or pipe
- ◯ A martial arts weapon
- ◯ A semi-automatic weapon
- ◯ Some other weapon

From which of these people or places **have you gotten weapons that you took to school? (Fill in ALL the circles that apply.)**

- ◯ Don't carry a weapon
- ◯ Friends
- ◯ Students at school
- ◯ Out-of-school kids
- ◯ People at work
- ◯ College students
- ◯ Family
- ◯ Adults other than family
- ◯ Bought from a store
- ◯ Stole from a store
- ◯ Stole from someone

Why do you carry a weapon to school? **(Fill in ALL the circles that apply.)**

- ◯ I have never carried a weapon
- ◯ I was hurt before by someone carrying a weapon
- ◯ I saw someone else hurt by someone carrying a weapon
- ◯ I was threatened
- ◯ I'm afraid
- ◯ My friends carry weapons
- ◯ For hunting
- ◯ For protection
- ◯ For show
- ◯ For pushing around other people
- ◯ Other

Nicotine is a chemical in **cigarettes.**

- ◯ It makes cigarette smokers want to smoke more
- ◯ It makes cigarette smokers want to quit smoking
- ◯ Don't know

Inhalants:

- ◯ Cause lung damage
- ◯ Don't get into the lungs
- ◯ Don't know

Are you reasonably sure that no one at your school can identify anyone's questionnaire?

- ◯ Yes
- ◯ No
- ◯ Not sure

When you have finished the survey, please place it face up in front of you, and wait quietly for it to be collected.

APPENDIX F

PRIMARY STUDIES FOR SYSTEMATIC REVIEW

(# 01) Amey & Albrecht (1998)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 1,389	*Demographics:*	▪ 72% White, 19% Black, 8% Latino, Gender not reported
Geographic Location of Sample:	▪ Urban, Suburban, Rural	*Location of Data Collection:*	▪ Parents, Homes ▪ Youth, Not reported	*Data Source:*	▪ Self-report surveys, youth; Interviews, parents
Probability Sampling Method:	▪ Yes, Nationally representative	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1992; 10-17 years (majority teens)

Age/grade criterion variable(s) measured: ▪ 1992; 10-17 years (majority teens)

Criterion variable(s) and operationalization:
- Alcohol = Dichotomous initiation
- Cigarettes = Dichotomous initiation
- Marijuana = Dichotomous initiation

Domains and Predictors:

Family: Family structure, Quality of parental relationship, Parental monitoring

Controls: Gender, Race, Age, Residential location, Family income

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit (hierarchical)

Major Limitations:
- Cross-sectional
- Limited number of domains and predictors

(# 02a-c) Bailey & Hubbard (1990)

Design:	▪ Longitudinal	*Sample Size:*	▪ 3,454	*Demographics:*	▪ 51% Male, 70% White, 24% Black
Geographic Location of Sample:	▪ Urban, rural (southeast U.S.)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, County-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Code numbers instead of names	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1985-1988; 6^{th}, 7^{th}, 8^{th} grade

Age/grade criterion variable(s) measured: ▪ 1985-1988; 7^{th} grade (a), 8^{th} grade (b), 9^{th} grade (c)

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation by 7^{th} grade (a), 8^{th} grade (b), 9^{th} grade (c)

Domains and Predictors:

Community: Adult opinions about adol. alc/mar use, Adult intolerance of adol. alc/mar use
School: Grades, +/- school attachment, Comfort among classmates
Family: Importance of family vs. peers, Ability to get along w/ parents, Ability to communicate w/ parents, Importance of harmony w/ parents
Peer: Importance of peers vs. family, Peer communication, Dependence on peers, Peer intolerance of alc/mar use by adol., Peer opinions about alc/mar use, Peer alc/mar use
Individual: Costs/benefits of mar use, Imp. of future goals, Imp. of respect
Controls: Gender, Race

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations: ▪ Parental drug use and drug norms not examined

(# 03) Brook, Whiteman, Gordon, Nomura, & Brook (1986)

Design:	▪ Longitudinal	*Sample Size:*	▪ 318	*Demographics:*	▪ 48% Male, 59% Black
Geographic Location of Sample:	▪ Urban (CT, KS, NJ, NY, OH, SC)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ Unknown	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, COC	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 9th-10th grade   *Age/grade criterion variable(s) measured:* ▪ 11th-12th grade

Criterion variable(s) and operationalization: ▪ Alcohol = 0 (Abstainers), 1 (Experimental Initiates), 2 (Regular Initiates)

Domains and Predictors:
Family: Parental warmth, Parental harmony, Fathers' alcohol use, Siblings' drug use
Peer: Peer deviance, Peer legal drug use
Individual: Orientation to work, Deviance
Controls: Gender, Race, SES

Mediation/Moderation: ▪ Yes, Peer X family factors

Analytic technique(s): ▪ Multinomial discriminant function analysis

Major Limitations:
▪ 24% attrition, Drop-outs lower levels of protection and more involvement in drug use
▪ Parental and peer drug norms not examined

(# 04a-c) Chassin, Presson, Sherman, Corty, & Olshavsky (1984)

Design:	▪ Longitudinal	*Sample Size:*	▪ 1,947	*Demographics:*	▪ 47% Male, Race not reported
Geographic Location of Sample:	▪ Urban, Suburban, Rural (Midwest)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No (School district wide)	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva pipeline	*Cross-Validation:*	▪ Yes

Age/grade predictor variable(s) measured: ▪ 6th-11th grade

Age/grade criterion variable(s) measured: ▪ 7th-8th grade (a), 9th-12th grade (b), 7th-12th grade (c)

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation at 7th-8th grade (a), 9th-12th grade (b), 7th-12th grade (c)

Domains and Predictors:

Community: Adult smoking, Direct experience w/ smoking (articles, ads)
School: Academic strain, Value placed on academic success
Family: Parental smoking norms, Expectations for youths' academic success, Parental support, Strictness, Expectations consistency, Parental smoking, Sibling smoking
Peer: Peer smoking, Best friends' smoking, Peer expectations for youths' academic success, Peer support, Peer-respondent value consistency, Peer smoking norms, Best friend smoking norms, Boy/girlfriend smoking norms
Individual: Smoking intention, Internal & external locus of control, Smoking norms, Tolerance for deviance

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Discriminant function analysis

Major Limitations:
▪ 29% attrition, Drop-outs more prone to deviance
▪ Did not report the direction of NS effects

(# 05) Chassin, Presson, Sherman, Montello, & McGrew (1986)

Design:	▪ Longitudinal	*Sample Size:*	▪ 2,128	*Demographics:*	▪ 96% White, Gender not reported
Geographic Location of Sample:	▪ Urban, Suburban (Midwest)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva pipeline	*Cross-Validation:*	▪ Yes

Age/grade predictor variable(s) measured: ▪ 6^{th} – 11^{th} grade *Age/grade criterion variable(s) measured:* ▪ 7^{th} – 12^{th} grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:

Family: Parental smoking, Parental cigarette norms, Parental social support, Parental strictness, Parental academic expectations for youth

Peer: Peer smoking, Peer cigarette norms, Peer prosocial support, Peer strictness, Peer academic expectations for youth

Individual: School grade, gender (as modifiers)

Mediation/Moderation:

▪ Yes, Gender X parental smoking-- significant for females only; Grade X parental strictness-- among 6^{th}-7^{th} graders, stricter parents associated with increased risk for smoking initiation; among 10^{th}-11^{th} graders, stricter parents associated with decreased risk for smoking initiation

Analytic technique(s): ▪ Binary logit

Major Limitations:

▪ Limited number of domains and predictors
▪ 24% attrition among T1 abstainers; Drop-out abstainers had higher levels of peers who smoked and lower levels of parental expectations for academic/general success
▪ 33% attrition among T1 initiates; Drop-out initiates have higher levels of peers who smoked, lower parental expectations for academic/general success, higher levels of smoking tolerance among parents and peers

(# 06) Crum, Storr, & Anthony (2005)

Design:	▪ Longitudinal	*Sample Size:*	▪ 1,183	*Demographics:*	▪ Majority Black, Gender not reported
Geographic Location of Sample:	▪ Urban (mid-Atlantic region)	*Location of Data Collection:*	▪ Private location in school	*Data Source:*	▪ Interviews
Probability Sampling Method:	▪ No, School district wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, COC	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1992; 7th grade *Age/grade criterion variable(s) measured:* ▪ 1993; 8th grade

Criterion variable(s) and operationalization: ▪ Alcohol = Dichotomous initiation

Domains and Predictors:

Community: Neighborhood disadvantage
School: Academic performance, Post H/S educational plans
Peer: Peer alcohol use
Controls: Age, Gender, Race, Prior alcohol use

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Structural equation modeling

Major Limitations:

▪ Small time interval between T1 and T2
▪ Limited generalizability (majority of sample Black, unknown gender make-up)
▪ Limited number of predictors (Parental drug use and drug norms, Peer drug norms not examined)

(# 07) D'Amico & McCarthy (2006)

Design:	▪ Longitudinal	*Sample Size*:	▪ Alcohol initiation = 824 ▪ Cigarette initiation = 877 ▪ Marijuana initiation = 941	*Demographics*:	▪ 45% Male, 44% White, 26% Latino, 4% Black
Geographic Location of Sample:	▪ Urban (CA)	*Location of Data Collection*:	▪ Classrooms	*Data Source*:	▪ Self-report survey
Probability Sampling Method:	▪ No, School-wide	*Efforts to Reduce Under/Over-Reporting*:	▪ Yes, COC and codes instead of names	*Cross-Validation*:	▪ No

Age/grade predictor variable(s) measured: ▪ Fall of 6^{th}-8^{th} grade

Age/grade criterion variable(s) measured: ▪ Spring of 6^{th}-8^{th} grade

Criterion variable(s) and operationalization:
- Alcohol = Dichotomous initiation
- Cigarettes = Dichotomous initiation
- Marijuana = Dichotomous initiation

Domains and Predictors:

Peer: Peer alcohol use, Peer cigarette use, Peer marijuana use

Individual: Alcohol use, Cigarette use, Marijuana use

Controls: Gender, Race, Grade

Mediation/Moderation:
- No

Analytic technique(s):
- Binary logit

Major Limitations:
- Small time interval between T1 and T2
- 30% exclusion due to inability to match T1 & T2 respondents, Excluded youth were more apt to be soft drug initiates at T1
- Limited number of domains and predictors

(# 08) Duncan, Duncan, & Hops (1998)

Design:	▪ Longitudinal	*Sample Size:*	▪ Alcohol initiation = 115 ▪ Cigarette initiation = 199 ▪ Marijuana initiation = 287	*Demographics:*	▪ Across samples, 46% Male, 96% White
Geographic Location of Sample:	▪ Urban, Suburban, Rural (OR)	*Location of Data Collection:*	▪ Research lab	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, newspapers and fliers	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, COC	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 13 yrs. average (T1) *Age/grade criterion variable(s) measured:* ▪ 14 yrs. average (T2)

Criterion variable(s) and operationalization:
▪ Alcohol = Dichotomous initiation
▪ Cigarettes = Dichotomous initiation
▪ Marijuana = Dichotomous initiation

Domains and Predictors:
Individual: Frequency of alcohol use (T2-T5) for cigarette and marijuana initiation models, Frequency of cigarette use (T2-T5) for alcohol and marijuana initiation models
Controls: Gender, Age

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Latent growth modeling

Major Limitations:
▪ Limited generalizability, majority White sample
▪ Limited number of domains and predictors

(# 09a-d) Ellickson, Tucker, Klein, & Saner (2004)

Design:	▪ Longitudinal, Cross-Sectional	*Sample Size:*	▪ L = 1,358 (8th grade), 1,072 (9th grade), 909 (10th grade) at T2 ▪ C-S = 1,955	*Demographics:*	▪ 52% Male, 34% Minority
Geographic Location of Sample:	▪ Urban, Rural, Suburban (CA & OR)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No (School district wide)	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva pipeline, Codes instead of names	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured:
▪ L = 1985; 7th grade
▪ L = 1986; 8th grade
▪ L = 1987; 9th grade
▪ C-S = 1985; 7th grade

Age/grade criterion variable(s) measured:
▪ L = 1986; 8th grade (a)
▪ L = 1987; 9th grade (b)
▪ L = 1988; 10th grade (c)
▪ C-S = 1985; 7th grade (d)

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation by 8th grade (a), 9th grade (b), 10th grade (c), 7th grade (d)

Domains and Predictors:
Community: Mar offers, Adult mar use
School: Poor grades
Peer: Pro-mar norms, Mar use
Family: Parental pro-mar norms, Mar use by older siblings, Nuclear family, Parental communication about problems
Individual: Mar use intentions, Mar as not harmful, + mar consequences, Mar as non-addictive, Delinquent behavior, Alc use, Cig use, Rebelliousness, Low mar resistance
Controls: Gender, Race, SES, Age relative to cohort

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations: ▪ On average, 20% attrition over waves of longitudinal data collection

(# 10) Ennett, Bauman, Foshee, Pemberton, & Hicks (2001)

Design:	▪ Longitudinal	*Sample Size*:	▪ Alcohol initiation = 195 adolescent-parent dyads ▪ Cigarette initiation = 378 adolescent-parent dyads	*Demographics*:	▪ 51% Male, 80% White
Geographic Location of Sample:	▪ Urban, Rural, Suburban (48 U.S. states)	*Location of Data Collection*:	▪ Homes	*Data Source*:	▪ Telephone interview, youth and parents
Probability Sampling Method:	▪ Yes, Nationally representative	*Efforts to Reduce Under/Over-Reporting*:	▪ No	*Cross-Validation*:	▪ No

Age/grade predictor variable(s) measured: ▪ 12-14 years

Age/grade criterion variable(s) measured: ▪ 13-15 years

Criterion variable(s) and operationalization:
▪ Alcohol = Dichotomous initiation
▪ Cigarettes = Dichotomous initiation

Domains and Predictors:
Family: Parental cig and alc use, Pro-drug norms, Parent-child communication about rules for alc/cig use, consequences of alc/cig use, media portrayals of alc/cig use, Supportiveness, Parental monitoring, Family structure
Controls: Gender, Age, Race, SES

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations:
▪ 19% attrition between T1 and T2
▪ Limited number of domains and predictors

(# 11) Epstein, Botvin, Baker, & Diaz (1999)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 4,847	*Demographics:*	▪ 48% Male, Black & Hispanic
Geographic Location of Sample:	▪ Urban (NYC)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, CO pipeline	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1994; 7th grade

Age/grade criterion variable(s) measured: ▪ 1994; 7th grade

Criterion variable(s) and operationalization: ▪ Alcohol = Dichotomous initiation

Domains and Predictors:

Community: Alc availability, Adult alc use
School: Truancy
Family: Mother's alc use, Fathers' alc use, Sibling alc use
Peer: Peer alc use, Friends' alc use
Individual: Cig use, Mar use, Deviant behavior, Religious service attendance
Controls = Age, Gender, Race, Family structure, SES

Mediation/Moderation: ▪ Yes, Gender X predictors

Analytic technique(s): ▪ Binary logit

Major Limitations:

- Cross-sectional
- Limited generalizability (Minority, urban sample)
- Limited number of predictors (Parental and peer drug norms not examined)
- Did not report the direction of NS effects

(# 12) Epstein, Botvin, Diaz, Toth, & Schinke (1995)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 554	*Demographics:*	▪ 49% Male, 50% Black, 36% Hispanic, 4% White
Geographic Location of Sample:	▪ Urban (NYC)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, CO pipeline	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 7th grade   *Age/grade criterion variable(s) measured:* ▪ 7th grade

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation

Domains and Predictors:

Community: Mar use of person most admired, Adult mar norms
School: Poor academic performance, Teacher social support
Family: Parental tolerance of youth's drug use, 2-parent family, Parental social support
Peer: Peer social support, Peer mar norms, Tolerance of respondent's drug use, Peer mar use
Individual: Mar knowledge, Pro-mar norms, Risk-taking, Self-esteem, Hopelessness, Assertiveness, Decision-making, Life skills, Self-efficacy
Controls = Gender, Age, SES

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations:

▪ Cross-Sectional
▪ Limited generalizability, majority of sample minority
▪ Parental drug use not examined
▪ Did not report the direction of NS effects

(# 13) Flay, Hu, & Richardson (1998)

Design:	▪ Longitudinal	*Sample Size:*	▪ 2,912	*Demographics:*	▪ 45% Male, 40% Hispanic, 29% White, 9% Black
Geographic Location of Sample:	▪ Urban (L.A., San Diego)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1986; 7th grade *Age/grade criterion variable(s) measured:* ▪ 1992; 12th grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Abstainers versus initiates, experimenters, regular users

Domains and Predictors:

School: School grades
Family: Parental smoking, Parental approval of smoking, Family conflict
Peer: Peer cig use, # friend cig initiates, Peer cig approval, Peer-provided cig offers
Individual: Smoking intention, Risk-taking, Refusal skills self-efficacy, Positive cig outcome expectancy, Alc use, Mar use, Gender

Mediation/Moderation: ▪ Gender moderation observed for abstainers versus experimental and regular use

Analytic technique(s): ▪ Multinomial logit

Major Limitations: ▪ More then 50% attrition between T1 and T2

(# 14) Flay, Hu, Siddiqui, Day, Hedeker, Petraitis, Richardson, & Sussman (1994)

Design:	▪ Longitudinal	*Sample Size:*	▪ 1,402	*Demographics:*	▪ 45% Male, 38% White, 30% Hispanic, 12% Black
Geographic Location of Sample:	▪ Urban (L.A., San Diego)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1986; 7th grade *Age/grade criterion variable(s) measured:* ▪ 1987; 8th grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:
Family: Parental smoking, Parental smoking norms
Peer: Peer smoking, Peer smoking norms
Individual: Smoking intention, Refusal self-efficacy, Negative outcome expectation

Mediation/Moderation:
▪ Moderation = Gender and race differences in impact of predictors
▪ Mediation = Smoking intentions mediated relationship between cigarette initiation and (a) peer smoking, (b) parental smoking, (c) parental approval of smoking, (d) refusal self-efficacy

Analytic technique(s): ▪ Structural equation modeling

Major Limitations: ▪ Limited number of domains and predictors

(# 15) Flewelling & Bauman (1990)

Design:	▪ Longitudinal ▪ Cross-Sectional	*Sample Size:*	▪ Long = 659, Alc initiation 802, Cig initiation 1,212, Mar initiation ▪ C-S = 2,062	*Demographics:*	▪ Not reported
Geographic Location of Sample:	▪ Urban (Southeast)	*Location of Data Collection:*	▪ Homes	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ Yes	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1985; 12-14 years

Age/grade criterion variable(s) measured: ▪ Long = 1987; 14-16 years ▪ C-S = 1985; 12-14 years

Criterion variable(s) and operationalization:
- Alcohol = Dichotomous initiation (2 models, c-s and longitudinal)
- Cigarettes = Dichotomous initiation (2 models, c-s and longitudinal)
- Marijuana = Dichotomous initiation (2 models, c-s and longitudinal)

Domains and Predictors: *Family*: Single-parent, Stepparent *Controls* = Gender, Race, Age, Mothers' education (SES proxy)

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations:
- Long = 22% attrition, differences between retained and drop-outs not assessed
- C-S = confounding consequences with causes
- Limited number of domains and predictors
- Generalizability unclear, demographics not reported

(# 16) Foshee & Bauman (1990)

Design:	▪ Longitudinal	*Sample Size:*	▪ 685	*Demographics:*	▪ Not reported
Geographic Location of Sample:	▪ Urban (Metro areas in Southeast)	*Location of Data Collection:*	▪ Homes	*Data Source:*	▪ Self-report surveys, Youth and mothers
Probability Sampling Method:	▪ Yes	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, saliva pipeline	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1985; 12-14 years

Age/grade criterion variable(s) measured: ▪ 1987; 14-16 years

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:
School: Commitment to school & religious activities (combined measure)
Family: Mothers' smoking norms, Mothers' smoking, Fathers smoking, Parental attachment
Peer: Peer smoking
Individual: Belief in conventional rules
Controls: Gender, Race, Age, Family structure, Mothers' education (SES proxy)

Mediation/Moderation:
▪ Yes, Mothers' current smoking X Attachment, X Belief, X Commitment; Fathers' current smoking X Attachment, X Belief, X Commitment

Analytic technique(s):
▪ Binary logit

Major Limitations:
▪ 22% attrition, although no significant differences on levels of variables
▪ Limited/unclear generalizability, Only generalizable to youth living in two-parent households; Demographic make-up of sample unknown

(# 17) Gritz, Prokhorov, Hudmon, Jones, Rosenblum, Chang, Chamberlain, Taylor, Johnston, & de Moor (2003)

Design:	▪ Longitudinal	*Sample Size:*	▪ 659	*Demographics:*	▪ 42% White, 37% Black, 20% Hispanic, Gender not reported
Geographic Location of Sample:	▪ Urban (Houston Galveston, TX)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva pipeline & school absentee follow-up	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 5th, 8th, 12th grade *Age/grade criterion variable(s) measured:* ▪ 6th, 9th, 1-yr post h/s

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation among total sample

Domains and Predictors:

Community: Media smoking messages
School: Academic performance, Detentions & suspensions
Family: Household smokers, Parental cig norms
Peer: Peer cig use, Peer smoking norms
Individual: Depression, Decisional balance, Susceptibility to smoking (intention proxy), Cultural identity
Controls: Grade, Gender, Race, Parents' education, Marital Status

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations:

▪ 18% attrition, drop-outs more apt to have poorer academic performance, be Black, older, and have parents who are divorced
▪ Small time interval between T1 and T2
▪ Did not report the direction of NS effects

(# 18) Hawkins, Hill, Guo, & Battin-Pearson (2002)

Design:	▪ Longitudinal	*Sample Size:*	▪ Alc initiation = 603 ▪ Cig initiation = 703 ▪ Mar initiation = 740	*Demographics:*	▪ 55% Male, 46% White, 24% Black, 21% Asian
Geographic Location of Sample:	▪ Urban (Seattle)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 8 waves, beginning in 1985; 10-16 years (majority teens)

Age/grade criterion variable(s) measured: ▪ 9 waves, ending in 1993; 10-18 years (majority teens)

Criterion variable(s) and operationalization:
- Alcohol = Time to initiation
- Tobacco = Time to initiation
- Marijuana = Time to initiation

Domains and Predictors:

Family: Proactive family management, Parental alcohol norms, Parental tobacco norms, Parental mar norms, Maternal bonding
Peer: Peer alc use, Peer mar use

Individual: Tolerant alc norms, Tolerant tobacco norms, Tolerant mar norms, Alc initiation (for tobacco and mar initiation models), Tobacco initiation (for alc and mar initiation models)
Controls: Gender, Race

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Survival analysis (hierarchical)

Major Limitations: ▪ Limited number of predictors (did not examine peer drug norms or parental soft drug use)

(# 19) Kandel & Andrews (1987)

Design:	▪ Longitudinal	*Sample Size:*	▪ 1,110 adolescent-parent-best friend triads	*Demographics:*	▪ Male, female, Race not reported
Geographic Location of Sample:	▪ Rural, Suburban, Urban (NY State)	*Location of Data Collection:*	▪ Classroom, Youth, Peers; Homes, Parents	*Data Source:*	▪ Self-report survey, Youth, best friends, parents
Probability Sampling Method:	▪ Yes, Two-stage stratified random	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Codes instead of names	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ Fall 1971; 9th-12th gr. *Age/grade criterion variable(s) measured:* ▪ Spring 1972; 9th-12th gr.

Criterion variable(s) and operationalization: ▪ Alcohol = Dichotomous initiation ▪ Marijuana = Dichotomous initiation

Domains and Predictors:
Family: Parental alc use, Parental alc norms, Parental mar norms, Child-parent closeness
Peer: Alc norms, Mar norms, Alc use, Mar use, # alc-using peers, # mar-using peers
Individual: Alc norms, mar norms

Mediation/Moderation: ▪ 6 significant marijuana initiation mediation pathways:
(1) Parental alc use → Perceived # mar-using peers → Mar initiation
(2) Parental alc use → Perceived # mar-using peers → Adolescent anti-mar norms → Mar initiation
(3) Parental anti-mar norms → Perceived parental anti-mar norms → Adolescent anti-mar norms → Mar initiation
(4) Peer anti-mar norms → Adolescent anti-mar norms → Mar initiation
(5) Peer anti-mar norms → Peer mar use → Perceived # mar-using peers → Mar initiation
(6) Peer anti-mar norms → Peer mar use → Perceived # mar-using peers → Adolescent anti-mar norms → Mar initiation

Analytic technique(s): ▪ Path analysis

Major Limitations:
▪ 5-6 month time interval between T1 and T2
▪ Limited number of domains and predictors
▪ Generalizability unclear, Demographic make-up not reported
▪ Did not report the direction of NS effects

(# 20) Kandel, Kiros, Schaffsan, & Hu (2004)

Design:	▪ Longitudinal	*Sample Size*:	▪ 5,347	*Demographics*:	▪ 50% Male, Race not reported
Geographic Location of Sample:	▪ Urban, Rural, Suburban (across U.S.)	*Location of Data Collection*:	▪ School; Homes	*Data Source*:	▪ Self-report surveys & interviews with Youth, Parents, School administrators (Nat'l Long. Study of Adol. Health); Official data
Probability Sampling Method:	▪ Yes, Stratified random	*Efforts to Reduce Under/Over-Reporting*:	▪ No	*Cross-Validation*:	▪ No

Age/grade predictor variable(s) measured: ▪ April-Dec 1995; 7th-12th grade

Age/grade criterion variable(s) measured: ▪ April-Aug 1996; 8th grade – 1year post high-school

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:

Community: Community ban of cigarette vending machines, Prohibition of tobacco marketing, Cigarette state sales tax
School: Positive scholastic attitude, Strictness of school smoking policy, Prevalence of smokers in school, Percentage minorities in school
Family: Quality of parent-child relationship, Parental cigarette use
Peer: Peer cigarette use
Individual: Work-for-pay, Delinquency, Depression
Controls: Gender, Race, Age, Parents' education (SES proxy), Two-parent family

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit

Major Limitations:
▪ Limited number of predictors (Parental and peer drug norms not examined)
▪ Generalizability unclear, Racial make-up not reported

(# 21) Kandel, Treiman, Faust, & Single (1976)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ Alcohol initiation = 1,096 adol-parent-best friend triads ▪ Marijuana initiation = 1,077 adol-parent-best friend triads	*Demographics:*	▪ Males, females, Race not reported
Geographic Location of Sample:	▪ Rural, Urban, Suburban (NY)	*Location of Data Collection:*	▪ Classrooms, Youth and friends ▪ Homes, Parents	*Data Source:*	▪ Self-report survey, Youth, best friends, parents
Probability Sampling Method:	▪ Yes, Two-stage stratified random	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Codes instead of names	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ Fall 1971; 9^{th}-12^{th} grade

Age/grade criterion variable(s) measured: ▪ Fall 1971; 9^{th}-12^{th} grade

Criterion variable(s) and operationalization: ▪ Alcohol = Dichotomous initiation ▪ Marijuana = Dichotomous initiation

Domains and Predictors:

School: Grade average, Educational aspirations, Days absent from school
Family: Closeness to parent, Liquor use, Drug use
Peer: Peer activity, Drug use
Individual: Church attendance, Hard liquor use, Cigarette use, Depression
Controls: Gender, Race, School grade

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Multiple classification analysis

Major Limitations:
▪ Cross-sectional
▪ Limited number of predictors (Peer and parental drug norms not examined)
▪ Generalizability unclear, Race and gender make-up not reported

(# 22) Marcos & Bahr (1988)

Design:	▪ Cross-Sectional	*Sample Size*:	▪ 2,626	*Demographics*:	▪ 47% Male, 82% White
Geographic Location of Sample:	▪ Urban (Metro southwest)	*Location of Data Collection*:	▪ Classrooms	*Data Source*:	▪ Self-report survey
Probability Sampling Method:	▪ Yes, Random sample of required classes	*Efforts to Reduce Under/Over-Reporting*:	▪ No	*Cross-Validation*:	▪ No

Age/grade predictor variable(s) measured: ▪ 1984; 9^{th} – 12^{th} grade   *Age/grade criterion variable(s) measured*: ▪ 1984; 9^{th} – 12^{th} grade

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation

Domains and Predictors: *School*: Educational attachment   *Individual*: Conventional values, Marijuana refusal self-efficacy

Mediation/Moderation: ▪ Yes, Tested a theoretically-derived mediation path model (3 direct effect predictors estimated)

Analytic technique(s): ▪ Path analysis

Major Limitations:
▪ Cross-sectional
▪ Limited number of domains and predictors (Parental and peer drug use and norms not examined)

(# 23) Pokorny, Jason, & Schoeny (2003)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 5,234	*Demographics:*	▪ Not reported
Geographic Location of Sample:	▪ Rural, Suburban, Urban (IL)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey, Official data
Probability Sampling Method:	▪ Yes, Cluster	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ March/April 1999; 6th – 8th grade (July/Aug 1999 for tobacco retail data)

Age/grade criterion variable(s) measured: ▪ March/April 1999; 6th – 8th grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:

Community: Perceived ease in obtaining tobacco products, Ease in buying tobacco; # tobacco retailers sold tobacco illegally, Rate of tobacco retailers, % population at risk for cigarette initiation
Family: Adult household smoker
Peer: Cigarette smoking
Controls: Age, Gender, Race, Age, Median community income (SES proxy)

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Hierarchical linear modeling

Major Limitations:

▪ Cross-sectional
▪ Limited number of predictors (Parental and peer drug norms not examined)
▪ Generalizability unclear, Gender and race demographics not reported
▪ Did not report the direction of NS effects

(# 24) Ramirez, Crano, Quist, Burgoon, Alvaro, & Grandpre (2004)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 1,094	*Demographics:*	▪ 52% Male, 79% White, 29% Hispanic
Geographic Location of Sample:	▪ Rural, Urban (Southwest AZ)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 4^{th} – 12^{th} grade (majority in early-late adolescence)

Age/grade criterion variable(s) measured: ▪ 4^{th} – 12^{th} grade (majority in early-late adolescence)

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation

Domains and Predictors: *Family*: Parental monitoring, Familism; *Individual*: Acculturation, Marijuana knowledge; *Controls*: Gender, Grade-level, Region

Mediation/Moderation: ▪ Yes, Marijuana knowledge X familism

Analytic technique(s): ▪ Binary logit

Major Limitations:
▪ Cross-sectional
▪ Limited number of domains and predictors (Parental and peer drug use and norms not examined)

(# 25) Robinson, Klesges, Zbikowski, & Glaser (1997)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 4,448	*Demographics:*	▪ 50% Male, 81% Black, 17% White
Geographic Location of Sample:	▪ Urban (south)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, codes instead of names	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 7th grade *Age/grade criterion variable(s) measured:* ▪ 7th grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:

Community: Perceived ease in obtaining cigarettes, Cigarettes as too expensive
School: Academic success
Family: Household smoking
Peer: Peer cigarette use, Proportion peers smoke regularly
Individual: Rebelliousness and risk-taking, Positive expectations from smoking, Body mass index, Smoking as way to reduce body weight
Controls: Gender, Race

Mediation/Moderation: ▪ Moderation, Some gender and race differences

Analytic technique(s): ▪ Binary logit

Major Limitations:
▪ Cross-sectional
▪ Teacher-administered surveys
▪ Limited number of domains and predictors (Parental and peer drug norms not examined)

(# 26) Shears, Edwards, & Stanley (2006)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 181,351	*Demographics:*	▪ Male & female, Race not reported
Geographic Location of Sample:	▪ Rural, Urban, Suburban (193 communities)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ Yes	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1996-2000 combined data; 7^{th}-12^{th} grade

Age/grade criterion variable(s) measured: ▪ 1996-2000 combined data; 7^{th}-12^{th} grade

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation

Domains and Predictors:

Community: Community-level measure of SES (reduced lunches)
School: School attachment
Family: Intact family
Peer: Marijuana use
Controls: Gender, Race, Grade-level

Mediation/Moderation: ▪ Yes, level of school attachment differs by grade-level

Analytic technique(s): ▪ Binary logit

Major Limitations:

- Cross-sectional
- Limited number of predictors (Parental and peer drug norms and parental drug use not examined)
- Generalizability unclear, Gender and race demographics not reported

(# 27a-c) Skinner, Massey, Krohn, & Lauer (1985)

Design:	▪ Longitudinal	*Sample Size:*	▪ 426	*Demographics:*	▪ 47% Male, Race not reported, 65% junior high-school students at T2
Geographic Location of Sample:	▪ Rural (Midwest)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva sample	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured:	▪ 1980 (T1), 1981 (T2); 7th-12th grade	*Age/grade criterion variable(s) measured:*	▪ 1982 (T3); 9th- two years post-h/s (majority in high-school)

Criterion variable(s) and operationalization:	▪ Cigarettes = Dichotomous initiation at mid-late adolescence (a), junior high, mid-adolescence (b), senior high, mid-late adolescence (c)
Domains and Predictors:	*School*: Commitment to education, Time spent on homework, Commitment to school activities *Family*: Attachment to father, Attachment to mother, Parental supervision, Parental smoking *Peer*: Attachment to friends, Association with female smoking peers, Association with male smoking peers *Individual*: Work-for-pay, Religiosity, Alcohol/Cigarette norms, Drug use norms, Gen. prosocial beliefs
Mediation/Moderation:	▪ No
Analytic technique(s):	▪ Discriminant function analysis
Major Limitations:	▪ 9% attrition, drop-outs reported higher levels of smoking, lower levels of bonding, higher levels of associations with smoking peers ▪ Limited number of predictors (Parental and peer drug norms not examined) ▪ Generalizability unclear, Racial make-up not reported

(# 28) Smith & Fogg (1978)

Design:	▪ Longitudinal	*Sample Size:*	▪ 651	*Demographics:*	▪ 47% Male, Majority White
Geographic Location of Sample:	▪ Suburban (near Boston)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey, Youth and peers
Probability Sampling Method:	▪ No, School-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1969; 7th-8th grade *Age/grade criterion variable(s) measured:* ▪ 1973; 11th-12th grade

Criterion variable(s) and operationalization: ▪ Marijuana = Dichotomous initiation

Domains and Predictors:

School: Likes school, Grade average
Individual: Views cigarette smokers as unattractive, views cigarette smokers negatively, Feels valued/accepted, Obedient/law-abiding, Works hard, Cigarette use, Feels capable during challenges, Self-sufficient, Ambitious, Sociable, Impulsive

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Discriminant function analysis

Major Limitations:

- Poor measures
- Limited number of domains and predictors
- Did not control for gender, race
- Did not report the direction of NS effects

(# 29) Tilson, McBride, Lipkus, & Catalano (2004)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 428 parent-child dyads	*Demographics:*	▪ 46% Male, 28% Black, 27% Asian, 35% Multi-ethnic
Geographic Location of Sample:	▪ Urban (Seattle)	*Location of Data Collection:*	▪ Classroom, Telephone	*Data Source:*	▪ Self-report survey, Youth; Phone interview, Parents
Probability Sampling Method:	▪ Yes, Stratified	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1994; 6^{th}-8^{th} grade *Age/grade criterion variable(s) measured:* ▪ 1994; 6^{th}-8^{th} grade

Criterion variable(s) and operationalization: ▪ Cigarettes = Dichotomous initiation

Domains and Predictors:

Family: Parent-child connectedness, Parental tobacco norms, Parental cigarette use, Parents' age, Parents' gender, Relation to respondent
Controls: Gender, Race, Age, Family structure, Acculturation, Parents' level of education, Parents' income

Mediation/Moderation:

▪ Moderation = Parental smoking modified association between parent-child connectedness and parental disapproval of smoking. Among youth whose parents did not smoke, those who reported low levels of parent-child connectedness were 2X's more likely to initiate cigarette use than those who reported high levels of connectedness. Among youth whose parents smoked, connectedness was not related to respondent smoking status.

Analytic technique(s): ▪ Binary logit

Major Limitations:

- Cross-Sectional
- Limited generalizability, minority sample
- Limited number of domains and predictors
- 28% dyad sample excluded due to missing data
- Students asked to disclose identity (names, addresses, phone numbers)

(# 30) Unger & Chen (1999)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 10,030	*Demographics:*	▪ 51% Male, 54% White, 28% Hispanic, 6% Black
Geographic Location of Sample:	▪ Urban, Rural, Suburban (CA)	*Location of Data Collection:*	▪ Homes	*Data Source:*	▪ Telephone interviews
Probability Sampling Method:	▪ Yes, Random digit dialing	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1993-1996 combined data; 12-17 years

Age/grade criterion variable(s) measured: ▪ 1993-1996 combined data; 12-17 years

Criterion variable(s) and operationalization: ▪ Cigarettes = Time to cigarette initiation

Domains and Predictors:

Family: Parental smoking, Sibling smoking
Peer: Male smoking friends, Female smoking friends

Individual: Favorite cigarette brand, Ownership of tobacco promotional item, Intention to use a tobacco promotional item
Controls: Age, Year of survey

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Survival analysis

Major Limitations:
▪ Cross-Sectional
▪ Limited number of domains and predictors (Parental and peer drug norms not examined)

(# 31) Urberg, Degirmencioglu, & Pilgrim (1997)

Design:	▪ Longitudinal	*Sample Size:*	▪ Alcohol initiation = 220 ▪ Cigarette initiation = 207	*Demographics:*	▪ 49% Male, 49% White
Geographic Location of Sample:	▪ Suburban (outside Midwestern city)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self report surveys, youth and peers
Probability Sampling Method:	▪ No, School-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ Fall of 6^{th}, 8^{th}, 10^{th} grade

Age/grade criterion variable(s) measured: ▪ Spring of 6^{th}, 8^{th}, 10^{th} grade

Criterion variable(s) and operationalization:
- Alcohol = Dichotomous initiation
- Cigarettes = Dichotomous initiation

Domains and Predictors:

Peer: Peer group use of drugs, Close friend use of drugs
Controls: Gender, Race, Grade-level

Mediation/Moderation:
- No

Analytic technique(s):
- Binary logit

Major Limitations:
- Short time interval between T1 and T2
- Limited number of domains and predictors (did not examine parental or peer drug norms, or parental drug use)

(# 32) Urberg, Luo, Pilgrim, & Degirmencioglu (2003)

Design:	▪ Longitudinal	*Sample Size:*	▪ 757	*Demographics:*	▪ 49% Male, 49% White
Geographic Location of Sample:	▪ Suburban (near Midwestern city)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey, youth and peers
Probability Sampling Method:	▪ No, School-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 7th, 9th, 11th grade *Age/grade criterion variable(s) measured:* ▪ 8th, 10th, 12th grade

Criterion variable(s) and operationalization:
▪ Alcohol = 0 (Abstainers), 1 (Initiates, but not current use), 2 (Current users), 3 (Current users & current drunkenness)
▪ Cigarettes = 0 (Abstainers), 1 (Initiates, but not current use), 2 (Current users)

Domains and Predictors:
School: Value of academics
Family: Value of spending time with parents, Parental monitoring, Relationship with mother
Peer: Positive friendship quality, Peer conflict, Peer acceptance, Peer cig use, Peer alc use
Individual: Sensation-seeking, Self-esteem, Harm from cig use, Harm from alc use
Controls: Gender, Race, Grade-level

Mediation/Moderation:
▪ Yes, Moderation observed for cigarette and alcohol initiation
1) Cigarette initiation = Peer friendship quality X Peer cig use, Peer acceptance X Peer cig use
2) Alcohol initiation = Peer friendship quality X Peer alcohol use, Peer conflict X Peer alcohol use

Analytic technique(s):
▪ Hierarchical regression

Major Limitations:
▪ Sample drop-outs had higher levels of alcohol and cigarette use, perceived less harm in using alcohol and cigarettes, lower levels of parental monitoring, valued academics to smaller degree
▪ Limited number of predictors (parental and peer drug norms and parental drug use not examined)
▪ Did not report the direction of NS effects

(# 33) Walter, Vaughan, & Cohall (1991)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 167 (Norm models), 181 (Belief models), 171 (Stress models), 165 (Disaffiliation models)	*Demographics:*	▪ Across samples, 44% Male, 63% White
Geographic Location of Sample:	▪ Urban, Suburban (NYC, Rockland County, NY)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School selected according to criteria	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 10^{th} grade *Age/grade criterion variable(s) measured:* ▪ 10^{th} grade

Criterion variable(s) and operationalization:
▪ Alcohol = Dichotomous initiation
▪ Cigarettes = Dichotomous initiation
▪ Marijuana = Dichotomous initiation

Domains and Predictors: *School*: Poor grades, Absences, Failures, Suspensions *Family*: Parental support, Parental alc, cig, mar norms
Peer: Peer alc, cig, mar norms, # peers use alc, cig, mar, Same-grade peer alc, cig, mar use
Individual: Alc, cig, mar norms, Alc, cig, mar refusal self-efficacy, Alc, cig, mar use risks, Strainful life events, Anxiety, Depression
Controls: Gender, Age, Area of residence

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Binary logit (stepwise)

Major Limitations:
▪ Cross-sectional
▪ 52% participation rate in urban sample
▪ Small sample size given number of predictors
▪ Did not report the direction of NS effects

(# 34) Webb, Baer, McLaughlin, McKelvey, & Caid (1991)

Design:	▪ Longitudinal	*Sample Size:*	▪ 114	*Demographics:*	▪ 33% Male, 94% White
Geographic Location of Sample:	▪ Suburban (near Houston, TX)	*Location of Data Collection:*	▪ Classroom	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, COC	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 7th grade   *Age/grade criterion variable(s) measured:* ▪ 8th grade

Criterion variable(s) and operationalization: ▪ Alcohol = 0 (Abstainers), 1 (Experimenters), 2 (Users)

Domains and Predictors:

School: Grade average
Family: Parental alcohol use, Parent-adolescent relationship, Parent-adolescent conflict
Peer: Peer alcohol use, Peer pro-alcohol norms
Individual: Deviance tolerance, Deviant behavior, Sensation-seeking, Self-derogation, Emotional maladjustment, Religious commitment
Controls: Gender

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Discriminant function analysis

Major Limitations:
▪ Small sample size
▪ Limited generalizability, majority White sample
▪ 44% attrition between T1 and T2
▪ Did not report the direction of NS effects

(# 35) Werch, Carlson, Pappas, Dunn, & Williams (1997)

Design:	▪ Cross-Sectional	*Sample Size:*	▪ 211	*Demographics:*	▪ Male & female, 85% Black
Geographic Location of Sample:	▪ Urban (Jacksonville, FL)	*Location of Data Collection:*	▪ School	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ Yes, Saliva pipeline	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ 1995; 6^{th} grade

Age/grade criterion variable(s) measured: ▪ 1995; 6^{th} grade

Criterion variable(s) and operationalization: ▪ Alcohol = 1 (Abstainers), 2 (Contemplators- Abstainers thinking of initiating), 3 (Preparers- Abstainers planning to initiate), 4 (Initiates within past 6 mths), 5 (Initiate Maintainers- Alcohol users for 6+ mths)

Domains and Predictors:

Community: Adult alcohol use, Alcohol cues
Family: Parental alcohol expectations
Peer: Peer alcohol use, Peer alcohol expectations
Individual: Alcohol intentions, Susceptibility to alcohol use, Alcohol benefits, Alcohol expectancies, Resistance self-efficacy, Self-control practices, Motivation

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Canonical discriminant analysis

Major Limitations:

▪ Cross-Sectional
▪ Small sample size
▪ Limited generalizability, Majority of sample low-income, minority; gender make-up not clear

(# 36) Williams, Van Dorn, Ayers, Bright, Abbott, & Hawkins (2007)

Design:	▪ Longitudinal	*Sample Size:*	▪ 588	*Demographics:*	▪ 51% Male, 64% White
Geographic Location of Sample:	▪ Urban (Seattle)	*Location of Data Collection:*	▪ Classrooms	*Data Source:*	▪ Self-report survey
Probability Sampling Method:	▪ No, School district-wide	*Efforts to Reduce Under/Over-Reporting:*	▪ No	*Cross-Validation:*	▪ No

Age/grade predictor variable(s) measured: ▪ Fall 1987-Fall 1992; 7^{th}-12^{th} grade

Age/grade criterion variable(s) measured: ▪ Fall 1987-Fall 1992; 7^{th}-12^{th} grade

Criterion variable(s) and operationalization:
- ▪ Alcohol = Dichotomous initiation
- ▪ Marijuana = Dichotomous initiation

Domains and Predictors:

Family: Clarity of family rules, Parental supervision
Peer: Delinquent friends
Controls: Gender, Race, SES

Mediation/Moderation: ▪ No

Analytic technique(s): ▪ Contemporary log-log modeling

Major Limitations: ▪ Limited number of domains and predictors (Parental and peer drug use and norms, individual drug norms, problem behaviors not examined)

APPENDIX G

2001 PPAAUS Univariate, Bivariate, and Multivariate Results

Table G1.

2001 PPAAUS, Dependent Measures: Descriptives for Total Sample

Variable	Min	Max	Mean	SD
Alcohol Initiation	0	1	.515	.500
Cigarette Initiation	0	1	.361	.450
Marijuana Initiation	0	1	.246	.431
Age of Alcohol Initiation [a]	8	18	13.332	2.395
Age of Cigarette Initiation [b]	8	18	12.996	2.131
Age of Marijuana Initiation [c]	8	18	14.241	1.595

Note. Dichotomous initiation measures include all respondents, N = 723.
[a] Only includes alcohol initiates, valid n = 373
[b] Only includes cigarette initiates, valid n = 261
[c] Only includes marijuana initiates, valid n = 178

Table G2.

2001 PPAAUS, Dependent Measures: Descriptives for Age-Graded Subsamples

Variable	Min	Max	Mean	SD
6th Grade (n = 236)				
Alcohol Initiation	0	1	.144	.351
Cigarette Initiation	0	1	.067	.251
Age of Alcohol Initiation [a]	8	13	10.029	1.507
Age of Cigarette Initiation [b]	8	12	10.250	1.290
9th Grade (n = 244)				
Alcohol Initiation	0	1	.557	.497

(Table G2 continues)

(Table G2 continued)

Variable	Min	Max	Mean	SD
Cigarette Initiation	0	1	.385	.487
Marijuana Initiation	0	1	.250	.433
Age of Alcohol Initiation [c]	8	15	12.441	1.832
Age of Cigarette Initiation [d]	8	15	12.351	1.521
Age of Marijuana Initiation [e]	11	15	13.377	1.082
12th Grade (n = 243)				
Alcohol Initiation	0	1	.835	.371
Cigarette Initiation	0	1	.621	.486
Marijuana Initiation	0	1	.477	.500
Age of Alcohol Initiation [f]	8	18	14.482	2.061
Age of Cigarette Initiation [g]	8	18	13.688	2.173
Age of Marijuana Initiation [h]	11	18	14.715	1.624

Note. Dichotomous initiation measures derived from initiation data from all respondents in each age-graded subsample.
[a] Only includes alcohol initiates, valid n = 34
[b] Only includes cigarette initiates, valid n = 16
[c] Only includes alcohol initiates, valid n = 136
[d] Only includes cigarette initiates, valid n = 94
[e] Only includes marijuana initiates, valid n = 61
[f] Only includes alcohol initiates, valid n = 203
[g] Only includes cigarette initiates, valid n = 151
[h] Only includes marijuana initiates, valid n = 116

Table G3.

2001 PPAAUS, Dichotomous Predictors/Controls: Percentage Frequencies (N = 723)

Variable	0	1	Variable	0	1
			Community Domain		
MessMedAlc 0 = Not OK to use 1 = OK to use, mixed message, no message	.212	.788	MessMedMar 0 = Not OK to use 1 = OK to use, mixed message, no message	.560	.440

(Table G3 continues)

(Table G3 continued)

Variable	0	1	Variable	0	1
MessMedCig 0 = Not OK to use 1 = OK to use, mixed message, no message	.317	.683			
			School Domain		
MessTchrAlc 0 = Not OK to use 1 = OK to use, mixed message, no message	.815	.185	MessTchrMar 0 = Not OK to use 1 = OK to use, mixed message, no message	.893	.107
MessTchrCig 0 = Not OK to use, 1 = OK to use, mixed message, no message	.840	.160	CllgeAsp 0 = No 1 = Yes	.192	.808
			Family Domain		
MessParAlc 0 = Not OK to use, 1 = OK to use, mixed message, no message	.729	.271	MessParMar 0 = Not OK to use, 1 = OK to use, mixed message, no message	.914	.531
MessParCig 0 = Not OK to use, 1 = OK to use, mixed message, no message	.866	.134			
			Peer Domain		
MessPeerAlc 0 = Not OK to use, 1 = OK to use, mixed message, no message	.268	.732	MessPeerMar 0 = Not OK to use, 1 = OK to use, mixed message, no message	.469	.531
MessPeerCig 0 = Not OK to use, 1 = OK to use, mixed message, no message	.398	.602			
			Individual Domain		
AlcInit 0 = Alcohol abstention 1 = Alcohol initiation	.484	.516	AlcIntent 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.317	.683
AlcEarly 0 = Alcohol abstainers, Late alcohol initiates (16-18 yrs) 1 = Early alcohol initiates (8-15 yrs)	.578	.422	CigIntent 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.609	.391
AlcLate 0 = Alcohol abstainers, Early alcohol initiates (8-15 yrs) 1 = Late alcohol initiates (16-18 yrs)	.906	.094	MarIntent 0 = Would Not 1 = Probably not, Not sure, Would like to, Any chance	.690	.310

(Table G3 continues)

(Table G3 continued)

Variable	0	1	Variable	0	1
CigEarly 0 = Cigarette abstainers, Late cigarette initiates (16-18 yrs) 1 = Early cigarette initiates (8-15 yrs)	.690	.310	Steal 0 = Never stole before 1 = Stole before	.692	.371
CigLate 0 = Cigarette abstainers, Early cigarette initiates (8-15 yrs) 1 = Late cigarette initiates (16-18 yrs)	.949	.051	ViolBeh 0 = No violent acts 1 = 1 or more violent acts	.657	.343
Control Variables					
Gender 0 = Female 1 = Male	.479	.521	Race 0 = Nonwhite 1 = White	.072	.928

Table G4.

2001 PPAAUS, Ordinal/Continuous Predictors and Controls: Descriptives (N = 723)

Variable	Min	Max	Mean	SD
School Domain				
Truancy	0	4	.666	1.004
Cheat	0	3	1.431	1.230
AttachSchool	3	21	13.723	4.095
Academic	0	3	2.467	.894
HiAcadPerf	0	5	3.493	1.076
Peer Domain				
PeerAlc	0	4	1.722	1.169
PeerCig	0	4	1.630	1.087
PeerMar	0	4	1.077	.942

(Table G4 continues)

(Table G4 continued)

Variable	Min	Max	Mean	SD
Individual Domain				
Sports	0	3	2.239	.958
Religious	0	5	2.874	1.434
Work	0	5	3.615	1.356
Social	0	5	3.734	1.034
Controls				
Age	11	19	15	2.487
AlcFreq	0	13	3.994	4.239
CigFreq	0	5	1.071	1.720

Table G5.

2001 PPAAUS, Bivariate Correlations, Dichotomous Initiation [a]

Variable	LogitAlc	LogitCig	LogitMar	Variable	LogitAlc	LogitCig	LogitMar
			Community Domain				
MessMedAlc	.189**	.150**	.100**	MessMedMar	---	---	.153**
MessMedCig	---	.097**	.044				
			School Domain				
MessTchrAlc	.213**	.183**	.223**	AttachSchool	-.340**	-.281**	-.303**
MessTchrCig	---	.189**	.226**	Academic	-.199**	-.219**	-.295**
MessTchrMar	---	---	.271**	HiAcadPerf	-.211**	-.225**	-.176**
Cheat	.445**	.359**	.348**	CllgeAsp	-.065	-.043	-.055
Truancy	.403**	.387**	.385**				
			Family Domain				
MessParAlc	.304**	.189**	.208**	MessParMar	---	---	.215**
MessParCig	---	.236**	.218**				
			Peer Domain				
PeerAlc	.610**	.455**	.411**	MessPeerAlc	.481**	.351**	.324**

(Table G5 continues)

(Table G5 continued)

Variable	LogitAlc	LogitCig	LogitMar	Variable	LogitAlc	LogitCig	LogitMar
PeerCig	---	.401**	.362**	MessPeerCig	---	.382**	.367**
PeerMar	---	---	.451**	MessPeerMar	---	---	.460**
				Individual Domain			
AlcInit	---	.619**	---	AlcIntent	.614**	.431**	.361**
AlcEarly	---	.559**	.539**	CigIntent	---	.613**	.456**
AlcLate	---	.113**	.003	MarIntent	---	---	.631**
CigEarly	---	---	.596**	Work	.140**	.143**	.067
CigLate	---	---	.100**	Religious	-.203**	-.221**	-.277**
ViolBeh	.210**	.155**	.155**	Sports	-.050	-.064	-.092*
Steal	.325**	.312**	.286**	Social	.297**	.285**	.283**
				Control Variables			
Gender	.075*	.017	.085*	AlcFreq	---	.636**	.640**
Race	.062	.020	-.002	CigFreq	---	---	.643**
Age	.550**	.450**	.428**				

[a] N = 723; * $p < .05$; ** $p < .01$; --- signifies those relationships that were not examined for the respective soft drug in question.

Table G6.

2001 PPAAUS, Bivariate Correlations, Age of Initiation [a]

Variable	AgeAlc1[b]	AgeCig1[c]	AgeMar1[d]	Variable	AgeAlc1	AgeCig1	AgeMar1
			Community Domain				
MessMedAlc	-.020	-.076	-.172*	MessMedMar	---	---	-.005
MessMedCig	---	-.108	-.062				
			School Domain				
MessTchrAlc	.084	.065	.078	AttachSchool	.030	.138**	.167*
MessTchrCig	---	.001	.013	Academic	.046	.179**	.089
MessTchrMar	---	---	.058	HiAcadPerf	.075	.199**	.214**
Cheat	.095	.034	.122	CllgeAsp	.185*	.214**	.159*
Truancy	.053	-.005	-.092				
			Family Domain				
MessParAlc	-.052	-.028	-.019	MessParMar	---	---	-.002
MessParCig	---	-.184**	-.155*				
			Peer Domain				
PeerAlc	.332**	.151*	.050	MessPeerAlc	.106*	-.015	.102

(Table G6 continues)

(Table G6 continued)

Variable	AgeAlc	AgeCig	AgeMar	Variable	AgeAlc	AgeCig	AgeMar
PeerCig	---	.047	-.069	MessPeerCig	---	-.036	.021
PeerMar	---	---	-.088	MessPeerMar	---	---	.069
			Individual Domain				
AlcInit	---	.124*	---	MarIntent	---	---	.053
AlcEarly	---	-.376**	-.483**	ViolBeh	-.175**	-.209**	-.172*
AlcLate	---	.539**	.527**	Steal	-.195**	-.165**	.005
CigEarly	---	---	.322**	Religious	.004	.142*	.230**
CigLate	---	---	.397**	Sports	-.057	-.064	.043
AlcIntent	.034	.016	.023	Social	.092	.092	.032
CigIntent	---	.039	-.039	Work	.112*	.103	.110
			Control Variables				
Gender	-.093	-.150*	-.119	AlcFreq	---	.004	-.008
Race	.204**	.197**	.124	CigFreq	---	---	-.199**
Age	.592**	.425**	.399**				

[a] * p <.05; ** p <.01; --- signifies those relationships that were not examined for the respective soft drug in question.
[b] Only includes alcohol initiates, valid n = 373
[c] Only includes cigarette initiates, valid n = 261
[d] Only includes marijuana initiates, valid n = 178

Table G7.

2001 PPAAUS, Predictors and Control Variables: Bivariate Correlations ± .400 (N = 723)

Variable		*r*	Variable	*r*
		Community Domain		
MessMedAlc & …	MessMedCig	.579	MessMedMar	.405
		School Domain		
Truancy & …	AlcInit	.403	CigFreq	.402
	AlcFreq	.488		
MessTchrAlc & …	MessTchrCig	.557	MessTchrMar	.505
MessTchrCig & …	MessTchrMar	.533		
Cheat & …	AlcInit	.455	AlcFreq	.445
	Age	.421		
		Peer Domain		
PeerAlc & …	Cheat	.495	AlcEarly	.464
	PeerCig	.747	CigEarly	.403
	PeerMar	.775	AlcIntent	.456
	MessPeerAlc	.484	Age	.752
	MessPeerMar	.400	AlcFreq	.592
	AlcInit	.610		
PeerCig & …	Cheat	.413	AlcInit	.501
	PeerAlc	.747	Age	.651
	PeerMar	.730	AlcFreq	.456
	MessPeerAlc	.400		
PeerMar & …	PeerAlc	.775	Age	.706
	MessPeerMar	.404	AlcFreq	.525

(Table G7 continues)

(Table G7 continued)

Variable		*r*	Variable	*r*
		Peer Domain (continued)		
PeerMar & …	AlcInit	.514	CigFreq	.420
MessPeerAlc & …	MessPeerCig	.680	AlcInit	.481
	MessPeerMar	.613	Age	.444
	AlcIntent	.440	AlcFreq	.450
MessPeerCig & …	MessPeerMar	.640	AlcFreq	.416
MessPeerMar & …	MarIntent	.450	Age	.414
	AlcInit	.432	AlcFreq	.462
		Individual Domain		
AlcEarly & …	CigEarly	.615	AlcFreq	.675
	AlcIntent	.503	CigFreq	.492
	MarIntent	.451		
AlcLate & …	CigLate	.484		
CigEarly & …	AlcInit	.541	AlcFreq	.571
	CigIntent	.541	CigFreq	.699
	MarIntent	.483		
AlcIntent & …	AlcInit	.614	Cheat	.403
	MarIntent	.424	AlcFreq	.555
CigIntent & …	AlcInit	.437	AlcFreq	.479
	MarIntent	.492	CigFreq	.659
MarIntent & …	AlcInit	.481	CigFreq	.522
	AlcFreq	.582		

(Table G7 continues)

(Table G7 continued)

Variable		*r*	Variable	*r*
		Control Variables		
Age & …	AlcInit	.550	CigFreq	.410
	AlcFreq	.532		
AlcFreq & …	AlcInit	.772	CigFreq	.621
CigFreq & …	AlcInit	.531		

Table G8.

2001 PPAAUS: Predictors of Alcohol Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.954*** (.191)	2.597	.542** (.228)	1.719	.438* (.233)	1.550	---	---	---	---	---	---
Truancy			.640*** (.111)	1.897	.628*** (.112)	1.873	.497*** (.126)	1.644	.298** (.135)	1.347	.246* (.137)	1.279
MessTchrAlc			.608** (.249)	1.837	---	---	---	---	---	---	---	---
Cheat			.597*** (.079)	1.817	.608*** (.081)	1.836	.256*** (.094)	1.292	---	---	---	---
AttachSchool			-.093*** (.026)	.911	-.089*** (.026)	.915	-.052* (.030)	.950	---	---	---	---
HiAcadPerf			-.230** (.093)	.795	-.242** (.095)	.785	-.300*** (.109)	.741	-.277** (.121)	.758	-.288** (.122)	.749
MessParAlc					1.134*** (.220)	3.108	.795*** (.245)	2.215	.468* (.273)	1.598	.493* (.276)	1.636
PeerAlc							1.006*** (.115)	2.735	1.137*** (.136)	3.118	.885*** (.171)	2.423
MessPeerAlc							1.308*** (.297)	3.698	.912*** (.322)	2.489	.770** (.327)	2.161

(Table G8 continues)

(Table G8 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									2.733*** (.344)	15.373	2.747*** (.347)	15.603
Steal									.668** (.261)	1.950	.728*** (.266)	2.072
ViolBeh									.624** (.274)	1.866	.682** (.277)	1.979
Work									.187** (.090)	1.206	.186** (.091)	1.204
Social									.207* (.123)	1.230	.203* (.123)	1.225
Age											.172** (.072)	1.188
-2LL	975.327		737.005		715.108		571.120		457.929		452.139	
Cox/Snell R^2	.036		.306		.327		.449		.529		.532	

[a] N = 723; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G9.

2001 PPAAUS: Predictors of Cigarette Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.840*** (.212)	2.316	.434* (.240)	1.544	---	---	---	---	---	---	---	---
Truancy			.540*** (.093)	1.716	.524*** (.094)	1.689	.399*** (.098)	1.490	.245*** (.116)	1.278	---	---
Cheat			.433*** (.079)	1.542	.455*** (.079)	1.576	.257*** (.088)	1.293	---	---	---	---
MessTchrAlc			.515** (.223)	1.673	.491** (.224)	1.634	---	---	---	---	---	---
AttachSchool			-.050** (.025)	.951	-.048* (.025)	.954	---	---	---	---	---	---
HiAcadPerf			-.303*** (.091)	.739	-.261*** (.091)	.770	-.316*** (.093)	.729	---	---	---	---
MessParCig					.879*** (.265)	2.408	.543* (.279)	1.720	---	---	---	---
PeerAlc							.650*** (.104)	1.915	.572*** (.127)	1.773	---	---
MessPeerCig							1.161*** (.226)	3.193	.511* (.274)	1.666	---	---

(Table G9 continues)

(Table G9 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcEarly									1.421*** (.258)	4.141	1.401*** (.288)	4.059
CigIntent									2.435*** (.244)	11.421	2.525*** (.252)	12.493
AlcIntent									.795** (.381)	2.214	---	---
Steal									.458* (.245)	1.582	.523** (.253)	1.686
Work									.196* (.088)	1.216	---	---
Age											.346*** (.061)	1.414
AlcFreq											.147*** (.039)	1.158
-2LL	928.505***		759.243***		751.470***		670.961***		469.395***		442.116***	
Cox/Snell R^2	.023		.227		.236		.316		.482		.502	

[a] N = 723; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G10.

2001 PPAAUS: Predictors of Marijuana Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.715*** (.175)	2.044	.384* (.213)	1.468	---	---	---	---	---	---	---	---
Truancy			.513*** (.099)	1.670	.472*** (.100)	1.603	.287*** (.110)	1.333	.281** (.135)	1.325	---	---
Cheat			.477*** (.094)	1.612	.475*** (.095)	1.608	.349*** (.105)	1.418	---	---	---	---
MessTchrMar			.977*** (.299)	2.657	1.005*** (.295)	2.731	.626** (.309)	1.870	.886** (.396)	2.426	.893** (.412)	2.441
AttachSchool			-.073*** (.027)	.929	-.072*** (.028)	.931	---	---	---	---	---	---
Academic			-.440*** (.112)	.644	-.444*** (.112)	.641	-.474*** (.125)	.623	---	---	---	---
MessParMar					.779** (.343)	2.178	---	---	---	---	---	---
MessParAlc					.511** (.227)	1.667	.507** (.237)	1.660	---	---	---	---

(Table G10 continues)

(Table G10 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
PeerMar							.757*** (.141)	2.132	.592*** (.194)	1.808	.365* (.218)	1.440
MessPeerMar							1.773*** (.368)	5.891	1.322*** (.429)	3.749	.893* (.465)	2.444
MessPeerCig							.950*** (.356)	2.586	.845* (.442)	2.327	.861* (.480)	2.364
CigEarly									1.555*** (.324)	4.736	1.007** (.394)	2.737
AlcEarly									1.356*** (.366)	3.880	1.589*** (.434)	4.900
MarIntent									2.117*** (.303)	8.306	2.048*** (.350)	7.752
Sports									-.324** (.155)	.723	---	---
Social									.416** (.188)	1.516	---	---
Gender											.567* (.324)	1.764
Age											.406*** (.106)	1.501

(Table G10 continues)

(Table G10 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcFreq											.110* (.048)	1.117
CigFreq											.290*** (.100)	1.336
-2LL	790.121***		606.886***		595.437***		485.099***		316.666***		282.423***	
Cox/Snell R^2	.023		.242		.254		.359		.492		.516	

[a] N = 723; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G11.

2001 PPAAUS: Predictors of Time to Alcohol Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.152*** (.050)	1.164	.136*** (.051)	1.146	.128*** (.051)	1.137	---	---	---	---

(Table G11 continues)

(Table G11 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Cheat			.172***	1.188	.148***	1.160	.108**	1.114	---	---	---	---
			(.049)		(.049)		(.050)					
AttachSchool			-.040***	.961	-.038***	.963	-.028*	.972	-.025*	.975	---	---
			(.014)		(.014)		(.015)		(.014)			
CllgeAsp			-.258*	.772	-.236*	.790	-.245*	.783	-.239*	.788	-.255*	.775
			(.134)		(.133)		(.134)		(.137)		(.137)	
HiAcadPerf			-.101*	.904	-.113**	.893	-.118**	.889	-.101*	.904	-.148***	.862
			(.056)		(.056)		(.056)		(.056)		(.054)	
MessParAlc					.416***	1.516	.326***	1.385	.234***	7.136	.335***	1.397
					(.108)		(.108)		(.107)		(.108)	
MessPeerAlc							.881***	2.413	---	---	---	---
							(.226)					
AlcIntent									1.965***	7.136	2.263***	9.610
									(.269)		(.272)	
Steal									.417***	1.518	.440***	1.553
									(.108)		(.108)	
Social									.171***	1.186	.266***	1.304
									(.061)		(.062)	
Race											-.606***	.545
											(.229)	

(Table G11 continues)

(Table G11 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Age											-.207*** (.032)	.813
-2LL	4301.795		4228.821		4214.399		4195.705		4092.234		4047.508	
Chi-Square	0.000		74.702***		90.264***		99.606***		171.949***		212.533***	

[a] N = 723; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G12.

2001 PPAAUS: Predictors of Time to Cigarette Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.341* (.184)	1.406	---	---	---	---	---	---	---	---	---	---
Truancy			.192*** (.056)	1.211	.164*** (.057)	1.179	.127** (.056)	1.136	---	---	---	---
Cheat			.173*** (.058)	1.188	.172*** (.058)	1.188	.117** (.059)	1.124	---	---	---	---

(Table G12 continues)

(Table G12 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AttachSchool			-.036** (.017)	.965	-.033** (.017)	.967	---	---	---	---	---	---
HiAcadPerf			-.171*** (.061)	.843	-.135** (.062)	.874	-.187** (.060)	.830	---	---	---	---
MessParCig					.500*** (.154)	1.649	.313** (.155)	1.367	---	---	---	---
PeerAlc							.158** (.080)	1.171	---	---	---	---
MessPeerCig							.847*** (.183)	2.333	---	---	---	---
AlcEarly									1.125*** (.168)	3.081	1.018*** (.176)	2.768
CigIntent									1.442*** (.159)	4.230	1.415*** (.163)	4.115
AlcIntent									.799*** (.300)	2.224	.707** (.313)	2.027
Steal									.213* (.129)	1.237	---	---
Age											-.081* (.041)	.923

(Table G12 continues)

(Table G12 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcFreq											.050** (.021)	1.051
-2LL	3110.305		3045.459		3035.652		3005.334		2808.813		2804.448	
Chi-Square	3.454*		72.129***		87.947***		108.752***		303.230***		306.813***	

[a] N = 723; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G13.

2001 PPAAUS: Predictors of Time to Marijuana Initiation, Total Sample [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.472*** (.152)	1.603	.367** (.153)	1.444	.292* (.158)	1.339	---	---	---	---	---	---
Truancy			.247*** (.072)	1.280	.249*** (.071)	1.283	.216*** (.070)	1.241	.131* (.070)	1.140	---	---
Cheat			.176** (.077)	1.193	.164** (.077)	1.178	---	---	---	---	---	---

(Table G13 continues)

(Table G13 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AttachSchool			-.051** (.020)	.951	-.049** (.020)	.952	-.035* (.020)	.965	---	---	---	---
Academic			-.293*** (.076)	.746	-.280*** (.077)	.755	-.266*** (.077)	.798	---	---	---	---
MessParMar					.501** (.198)	1.650	---	---	---	---	---	---
PeerMar							.202** (.095)	1.224	---	---	---	---
MessPeerMar							1.429*** (.322)	4.176	.535 (.334)	1.708	---	---
MessPeerCig							.678** (.288)	1.970	.566** (.283)	1.762	.480* (.283)	1.616
CigEarly									.670*** (.227)	1.954	.543** (.237)	1.721
AlcEarly									1.286*** (.276)	3.620	1.219*** (.290)	3.383
MarIntent									1.234*** (.220)	3.434	1.267*** (.219)	3.549
CigIntent									.340* (.203)	1.405	---	---

(Table G13 continues)

(Table G13 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Religious									-.154*** (.052)	.857	-.143*** (.053)	.867
Work									-.116** (.059)	.891	-.122** (.058)	.885
Gender											.281* (.160)	1.325
Race											-.768** (.309)	.464
CigFreq											.153*** (.050)	1.166
AlcFreq											.066** (.027)	1.068
-2LL	2086.881		2009.141		2003.322		1953.004		1788.952		1769.509	
Chi-Square	9.754***		97.330***		106.468***		131.830***		294.057***		322.233***	

[a] N = 723; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G14.

2001 PPAAUS: Predictors of Alcohol Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.949** (.448)	2.582	.979** (.475)	2.663	---	---	---	---	---	---	---	---
Truancy			.680** (.287)	1.974	.648** (.283)	1.911	.787*** (.294)	2.198	.541* (.309)	1.717	.541* (.309)	1.717
AttachSchool			-.137*** (.050)	.872	-.155** (.051)	.891	---	---	---	---	---	---
Cheat			.415* (.215)	1.514	.416* (.219)	1.511	---	---	---	---	---	---
MessParAlc					1.213*** (.453)	3.362	---	---	---	---	---	---
PeerAlc							1.562*** (.339)	4.768	1.481*** (.373)	4.399	1.481*** (.373)	4.399
MessPeerAlc							1.553*** (.473)	4.726	1.368*** (.499)	3.927	1.368*** (.499)	3.927

(Table G14 continues)

(Table G14 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcIntent									2.369*** (.659)	10.692	2.369*** (.659)	10.692
-2LL	189.486***		166.850***		164.883***		125.935***		125.935***		125.935***	
Cox/Snell R^2	.021		.111		.118		.189		.252		.252	

[a] N = 236; * p <.10; ** p <.05; *** p <.01; standard error in parentheses.

Table G15.

2001 PPAAUS: Predictors of Alcohol Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Cheat			.415*** (.118)	1.514	.395*** (.119)	1.485	.259** (.132)	1.296	---	---	---	---
AttachSchool			-.106** (.043)	.899	-.088** (.044)	.916	---	---	---	---	---	---

(Table G15 continues)

(Table G15 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
HiAcadPerf			-.435*** (.159)	.647	-.503*** (.167)	.605	-.665*** (.172)	.514	-.426** (.201)	.653	-.426** (.201)	.653
MessParAlc					.850** (.345)	2.340	.628* (.371)	1.873	---	---	---	---
PeerAlc							1.047*** (.224)	2.848	1.457*** (.292)	4.295	1.457*** (.292)	4.295
MessPeerAlc							1.451*** (.454)	4.269	---	---	---	---
AlcIntent									2.682*** (.513)	14.615	2.682*** (.513)	14.615
Steal									1.527*** (.403)	4.603	1.527*** (.403)	4.603
ViolBeh									1.680*** (.464)	5.363	1.680*** (.464)	5.363
-2LL	335.036		288.560***		282.220***		241.822***		174.192***		174.192***	
Cox/Snell R^2	.000		.173		.195		.318		.483		.483	

[a] N = 244; * *p* <.10; ** *p* <.05; *** *p* <.01; standard error in parentheses.

Table G16.

2001 PPAAUS: Predictors of Alcohol Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.635*** (.206)	1.887	.621*** (.214)	1.860	.621*** (.214)	1.860	---	---	---	---
Cheat			.435** (.169)	1.545	.391** (.178)	1.479	.391** (.178)	1.479	---	---	---	---
HiAcadPerf			-.354** (.172)	.702	-.348* (.185)	.706	-.348* (.185)	.706	-.689*** (.263)	.502	-.689*** (.263)	.502
MessParAlc					1.583*** (.517)	4.869	1.583*** (.517)	4.869	1.649** (.653)	5.202	1.649** (.653)	5.202
AlcIntent									4.388*** (.791)	80.454	4.388*** (.791)	80.454
Work									.438** (.187)	1.550	.438** (.187)	1.550
Social									1.122*** (.302)	3.070	1.122*** (.302)	3.070
-2LL	217.356		184.917***		172.946***		172.946***		109.829***		109.829***	
Cox/Snell R^2	.000		.125		.167		.167		.358		.358	

[a] N = 243; * *p* <.10; ** *p* <.05; *** *p* <.01; standard error in parentheses.

Table G17.

2001 PPAAUS: Predictors of Cigarette Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	1.004* (.655)	.655	1.192* (.341)	3.294	---	---	---	---	---	---	---	---
Truancy			1.127*** (.341)	3.087	1.162*** (.342)	3.198	1.265*** (.379)	3.544	---	---	---	---
HiAcadPerf			-.719*** (.275)	.487	-.725*** (.278)	.485	-.650** (.292)	.522	-.757** (.327)	.469	-.789** (.359)	.454
MessParCig					2.318*** (.684)	10.155	1.497** (.716)	4.470	---	---	---	---
MessPeerCig							2.327*** (.835)	10.248	2.330** (.911)	10.278	2.318** (.934)	10.159
AlcInit									1.865*** (.678)	6.457	1.877** (.732)	6.531
CigIntent									1.868*** (.700)	6.472	1.730** (.729)	5.639
Steal									1.870*** (.708)	6.491	2.666*** (.926)	14.386

(Table G17 continues)

(Table G17 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Race											-2.812** (1.124)	.060
-2LL	114.231***		96.646***		90.164***		79.531***		62.918***		56.150***	
Cox/Snell R^2	.012		.083		.108		.147		.205		.227	

[a] N = 236; * *p* <.10; ** *p* <.05; *** *p* <.01; standard error in parentheses.

Table G18.

2001 PPAAUS: Predictors of Cigarette Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.486*** (.170)	1.625	.453*** (.174)	1.574	.454*** (.173)	1.575	.451** (.209)	1.570	.451** (.209)	1.570
Cheat			.338** (.130)	1.402	.341** (.132)	1.407	.279** (.136)	1.321	---	---	---	---
AttachSchool			-.087* (.045)	.917	-.082* (.046)	.921	-.080* (.046)	.923	---	---	---	---

(Table G18 continues)

(Table G18 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
HiAcadPerf			-.548*** (.170)	.578	-.520*** (.190)	.595	-.494*** (.173)	.610	-.536** (.212)	.585	-.536** (.212)	.585
MessParCig					.991** (.489)	2.693	---	---	---	---	---	---
PeerCig							.423** (.207)	1.527	---	---	---	---
MessPeerCig							.707** (.337)	2.028	---	---	---	---
AlcEarly									1.958*** (.457)	7.088	1.958*** (.457)	7.088
CigIntent									2.397*** (.395)	10.988	2.397*** (.395)	10.988
Social									.637** (.253)	1.890	.637** (.253)	1.890
-2LL	325.288		263.360***		259.153***		252.728***		168.044***		168.044***	
Cox/Snell R^2	.000		.224		.237		.257		.475		.475	
% Correct	.615		.709		.725		.758		.857		.857	

[a] N = 244; * *p* <.10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G19.

2001 PPAAUS: Predictors of Cigarette Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.282** (.126)	1.326	.264** (.126)	1.303	.227* (.130)	1.255	---	---	---	---
Academic			-.312* (.162)	.732	-.321** (.163)	.726	-.272* (.166)	.762	---	---	---	---
MessParAlc					.480* (.285)	1.615	---	---	---	---	---	---
MessPeerCig							1.252*** (.354)	3.498	---	---	---	---
AlcEarly									1.793*** (.389)	6.007	1.434*** (.446)	4.196
CigIntent									2.920*** (.401)	18.534	2.925*** (.425)	18.635
AlcIntent									1.382* (.731)	3.981	---	---
Race											1.895** (.834)	6.654

(Table G19 continues)

(Table G19 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcFreq											.143** (.061)	1.154
-2LL	322.400		309.742***		306.872***		296.654***		180.220***		173.578***	
Cox/Snell R^2	.000		.051		.062		.101		.443		.458	

[a] N = 243; * *p* <.10; ** *p* <.05; *** *p* <.01; standard error in parentheses.

Table G20.

2001 PPAAUS: Predictors of Marijuana Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AttachSchool			-.175*** (.045)	.839	-.175*** (.045)	.839	-121** (.049)	.886	---	---	---	---
Academic			-.328* (.177)	.720	-.328* (.177)	.720	-.358* (.194)	.699	---	---	---	---
Cheat			.358** (.147)	1.430	.358** (.147)	1.430	.311* (.168)	1.365	---	---	---	---

(Table G20 continues)

(Table G20 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
PeerMar							.883*** (.277)	2.417	.687* (.377)	1.987	---	---
MessPeerMar							2.814*** (.630)	16.683	2.072*** (.710)	7.942	2.183*** (.785)	8.875
CigEarly									2.026*** (.484)	7.587	1.134* (.638)	3.107
AlcEarly									2.192*** (.807)	8.956	1.779** (.908)	5.926
MarIntent									1.775*** (.540)	5.899	1.249** (.587)	3.488
AlcIntent									-1.676* (.989)	.187	-2.154** (.989)	.116
CigFreq											.478*** (.177)	1.613
AlcFreq											.176** (.076)	1.193
-2LL	274.420		234.567***		234.567***		182.859***		134.615***		120.785***	
Cox/Snell R^2	.000		.151		.151		.313		.436		.467	

[a] N = 244; * p <.10; ** p <.05; *** p <.01; standard error in parentheses.

Table G21.

2001 PPAAUS: Predictors of Marijuana Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	1.183*** (.284)	3.263	.926*** (.338)	2.524	.876** (.343)	2.402	.648* (.365)	1.912	---	---	---	---
MessMedAlc	-.740* (.415)	.477	-1.017** (.456)	.362	-.983** (.468)	.374	-1.041** (.489)	.353	-1.204* (.643)	.300	-1.204* (.643)	.300
Truancy			.566*** (.139)	1.761	.562*** (.141)	1.754	.530*** (.146)	1.699	.522*** (.200)	1.685	.522*** (.200)	1.685
MessTchrMar			.859** (.394)	2.361	.720* (.400)	2.054	.695* (.417)	2.004	1.044* (.561)	2.842	1.044* (.561)	2.842
Academic			-.661*** (.183)	.516	-.663*** (.185)	.515	-.631*** (.189)	.532	---	---	---	---
MessParMar					1.151** (.495)	3.161	.948* (.503)	2.580	1.903** (.787)	6.706	1.903** (.787)	6.706
MessPeerMar							1.028** (.475)	2.794	---	---	---	---
MessPeerCig							1.103** (.487)	3.014	---	---	---	---
CigEarly									2.229*** (.613)	9.293	2.229*** (.613)	9.293

(Table G21 continues)

(Table G21 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigLate									1.671** (.681)	5.319	1.671** (.681)	5.319
AlcEarly									2.107*** (.579)	8.227	2.107*** (.579)	8.227
MarIntent									2.769*** (.471)	15.940	2.769*** (.471)	15.940
-2LL	318.000***		269.310***		263.377***		247.879***		141.275***		141.275***	
Cox/Snell R^2	.073		.241		.259		.305		.552		.552	

[a] N = 243; * *p* <.10; ** *p* <.05; *** *p* <.01; standard error in parentheses.

Table G22.

2001 PPAAUS: Predictors of Time to Alcohol Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.894** (.425)	2.445	.852** (.425)	2.345	---	---	---	---	---	---	---	---
Truancy			.461** (.223)	1.586	.484** (.216)	1.622	.466** (.206)	1.594	---	---	---	---
Cheat			.385** (.184)	1.469	.369** (.186)	1.446	---	---	---	---	---	---
AttachSchool			-.166*** (.041)	.891	-.102** (.043)	.903	-.091* (.047)	.913	---	---	---	---
MessParAlc					.926** (.366)	2.526	---	---	---	---	---	---
PeerAlc							1.074*** (.253)	2.926	1.068*** (.265)	2.910	1.068*** (.265)	2.910
MessPeerAlc							1.217*** (.406)	3.376	1.058** (.408)	2.880	1.058** (.408)	2.880
AlcIntent									2.169*** (.611)	8.747	2.169*** (.611)	8.747
-2LL	353.989		332.117		331.003		311.556		298.899		298.899	
Chi-Square	4.730**		32.394***		35.680***		54.403***		58.616***		58.616***	

[a] N = 236; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G23.

2001 PPAAUS: Predictors of Time to Alcohol Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Cheat			.293*** (.077)	1.341	.255*** (.079)	1.291	.158** (.080)	1.171	---	---	---	---
CllgeAsp			-.538** (.219)	.584	-.543** (.219)	.581	-.496** (.223)	.609	-.502** (.212)	.605	-.489** (.213)	.613
HiAcadPerf			-.298*** (.095)	.742	-.332*** (.094)	.717	-.299*** (.096)	.741	---	---	---	---
MessParAlc					.574*** (.184)	1.775	.392** (.185)	1.479	---	---	---	---
PeerAlc							.469*** (.120)	1.598	.456*** (.119)	1.578	.463*** (.119)	1.589
MessPeerAlc							.993*** (.357)	2.699	---	---	---	---
AlcIntent									1.739*** (.376)	5.690	1.843*** (.380)	6.315
Steal									.647*** (.179)	1.909	.696*** (.182)	2.006
ViolBeh									.502*** (.176)	1.652	.486*** (.177)	1.626

(Table G23 continues)

(Table G23 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Race											-.806** (.360)	.447
-2LL	1418.273		1377.633		1368.467		1337.079		1297.906		1293.776	
Chi-Square	0.000		41.477***		51.432***		77.935***		108.498***		111.100***	

[a] N = 244; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G24.

2001 PPAAUS: Predictors of Time to Alcohol Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.276*** (.064)	1.317	.259*** (.064)	1.295	.247*** (.064)	1.280	.132* (.068)	1.141	.132* (.068)	1.141
MessTchrAlc			.248* (.147)	1.282	---	---	---	---	---	---	---	---
Academic			-.213*** (.076)	.809	-.221*** (.075)	.802	-.241*** (.076)	.786	-.141* (.079)	.869	-.141* (.079)	.869

(Table G24 continues)

(Table G24 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessParAlc					.460*** (.142)	1.584	.388*** (.143)	1.474	.242* (.145)	1.274	.242* (.145)	1.274
MessPeerAlc							.912** (.423)	2.490	---	---	---	---
AlcIntent									2.160*** (.515)	8.674	2.160*** (.515)	8.674
Steal									.396*** (.149)	1.486	.396*** (.149)	1.486
Religious									-.147*** (.049)	.864	-.147*** (.049)	.864
Social									.360*** (.090)	1.434	.360*** (.090)	1.434
-2LL	2015.347		1979.946		1972.469		1966.386		1902.396		1902.396	
Chi-Square	0.000		37.396***		45.777***		49.089***		89.932***		89.932***	

[a] N = 243; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G25.

2001 PPAAUS: Predictors of Time to Cigarette Initiation among 6th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedAlc	.967 (.641)	2.630	1.022 (.641)	2.778	---	---	---	---	---	---	---	---
Truancy			.753** (.296)	2.124	.869*** (.290)	2.385	.854*** (.291)	2.349	---	---	---	---
Cheat			.495** (.252)	1.641	.500* (.267)	1.649	.452* (.245)	1.571	---	---	---	---
HiAcadPerf			-.643*** (.232)	.526	-.640*** (.230)	.527	-.521** (.225)	.594	-.467** (.234)	.627	---	---
MessParCig					1.884*** (.550)	6.580	1.147** (.566)	3.149	---	---	---	---
MessPeerCig							2.085*** (.783)	8.041	1.837** (.778)	6.276	1.778** (.777)	5.920
AlcInit									1.389*** (.512)	4.012	1.585*** (.537)	4.882
CigIntent									1.410** (.609)	4.098	1.383** (.616)	3.986
Steal									1.332** (.559)	3.788	1.635*** (.586)	5.130

(Table G25 continues)

(Table G25 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Gender											1.239** (.614)	3.453
Race											-1.584*** (.594)	.205
-2LL	169.963		150.738		144.986		135.075		123.601		117.875	
Chi-Square	2.463		26.750***		38.393***		46.886***		61.577***		70.576***	

[a] N = 236; * $p < .10$; ** $p < .05$; *** $p < .001$; standard error in parentheses.

Table G26.

2001 PPAAUS: Predictors of Time to Cigarette Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Truancy			.303*** (.097)	1.354	.255** (.101)	1.290	.316*** (.103)	1.371	.210** (.097)	1.234	.225** (.097)	1.253
Cheat			.218** (.099)	1.243	.204** (.100)	1.226	---	---	---	---	---	---

(Table G26 continues)

(Table G26 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
HiAcadPerf			-.458*** (.116)	.633	-.435*** (.117)	.647	-.420*** (.116)	.657	-.211* (.125)	.810	-.231* (.125)	.793
MessParCig					.666** (.257)	1.946	.501* (.265)	1.650	.561** (.268)	1.752	.544** (.268)	1.723
PeerCig							.284** (.138)	1.328	---	---	---	---
MessPeerAlc							.771** (.376)	2.162	---	---	---	---
AlcEarly									1.397*** (.360)	4.041	1.460*** (.364)	4.306
CigIntent									1.370*** (.279)	3.934	1.359*** (.279)	3.894
Social									.262* (.136)	1.299	.278** (.135)	1.320
Race											-.733* (.406)	.481
-2LL	999.556		948.064		942.027		936.068		862.864		860.147	
Chi-Square	0.000		57.865***		69.850***		73.297***		142.047***		142.758***	

[a] N = 244; * p < .10; ** p <.05; *** p <.001; standard error in parentheses.

Table G27.

2001 PPAAUS: Predictors of Time to Cigarette Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Academic			-.289*** (.080)	.749	-.289*** (.080)	.749	-.247*** (.080)	.781	---	---	---	---
MessPeerCig							.903*** (.267)	2.466	---	---	---	---
AlcEarly									1.168*** (.200)	3.217	1.208*** (.200)	3.346
CigIntent									1.282*** (.209)	3.603	1.338*** (.210)	3.811
AlcIntent									1.163* (.600)	3.201	1.045* (.601)	2.842
Race											.897* (.461)	2.453
-2LL	1552.570		1540.665		1540.665		1526.352		1413.410		1408.423	
Chi-Square	0.000		13.421***		13.421***		25.078***		132.217***		134.625***	

[a] N = 243; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G28.

2001 PPAAUS: Predictors of Time to Marijuana Initiation among 9th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AttachSchool			-.146*** (.034)	.864	-.146*** (.034)	.864	-.091*** (.032)	.913	-.061* (.033)	.941	-.078** (.034)	.925
Cheat			.320** (.125)	1.377	.320** (.125)	1.377	.260** (.122)	1.297	---	---	---	---
PeerMar							.551*** (.164)	1.735	.360** (.175)	1.434	.462** (.183)	1.587
MessPeerMar							2.421*** (.601)	11.258	1.551** (.634)	4.716	1.694*** (.604)	5.440
CigEarly									1.297*** (.382)	3.659	.723 (.449)	2.061
AlcEarly									1.361** (.617)	3.900	1.252** (.624)	3.499
MarIntent									.833** (.405)	2.300	---	---
Race											-.890* (.502)	.411
CigFreq											.241*** (.093)	1.272

(Table G28 continues)

(Table G28 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
AlcFreq											.082** (.038)	1.085
-2LL	656.126		622.455		622.455		574.928		530.985		518.422	
Chi-Square	0.000		34.162***		34.162***		71.661***		117.204***		147.314***	

[a] N = 244; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.

Table G29.

2001 PPAAUS: Predictors of Time to Marijuana Initiation among 12th Grade Students [a]

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
MessMedMar	.700*** (.190)	2.013	.417** (.208)	1.517	.355* (.210)	1.426	---	---	---	---	---	---
Truancy			.373*** (.090)	1.452	.383*** (.089)	1.467	.356*** (.090)	1.427	.261*** (.095)	1.298	.230** (.092)	1.259
MessTchrMar			.512** (.222)	1.669	.474** (.224)	1.607	.498** (.208)	1.645	.501** (.205)	1.651	.479** (.204)	1.614

(Table G29 continues)

(Table G29 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
Academic			-.355*** (.090)	.701	-.346*** (.090)	.707	-.308*** (.091)	.735	---	---	---	---
MessParMar					.622*** (.235)	1.863	.556** (.235)	1.743	---	---	---	---
MessPeerMar							.836** (.365)	2.307	---	---	---	---
MessPeerCig							.650* (.359)	1.916	---	---	---	---
CigEarly									.477* (.271)	1.611	---	---
AlcEarly									1.268*** (.320)	3.554	1.532*** (.281)	4.628
MarIntent									1.253*** (.250)	3.501	1.306*** (.248)	3.691
CigIntent									.527** (.251)	1.694	---	---
Religious									-.184*** (.066)	.832	-.150** (.067)	.861
Work									-.152* (.078)	.859	-.140* (.074)	.869

(Table G29 continues)

(Table G29 continued)

Predictor	Block #1		Block #2		Block #3		Block #4		Block #5		Block #6	
	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)	B	Exp(B)
CigFreq											.170*** (.054)	1.185
-2LL	1203.463		1156.198		1149.969		1139.159		1039.091		1040.131	
Chi-Square	14.146***		67.894***		74.842***		78.128***		168.616***		170.159***	

[a] N = 243; * *p* < .10; ** *p* <.05; *** *p* <.001; standard error in parentheses.