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Using Adiposity Change in College Freshman to Examine the Comorbidity of ADHD and Obesity

Katherine L. Ratcliff
Indiana University of Pennsylvania

USING ADIPOSITY CHANGE IN COLLEGE FRESHMAN TO EXAMINE THE COMORBIDITY OF ADHD AND OBESITY

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Katherine L. Ratcliff

Indiana University of Pennsylvania

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Indiana University of Pennsylvania
The School of Graduate Studies and Research
Department of Psychology

We hereby approve the dissertation of

Katherine L. Ratcliff

Candidate for the degree of Doctor of Psychology

October 16, 2008	*(Signature on File)*
_____	_____
	Donald U. Robertson, Ph.D. Professor of Psychology, Advisor
October 16, 2008	*(Signature on File)*
_____	_____
	David J. LaPorte, Ph.D. Professor of Psychology
October 16, 2008	*(Signature on File)*
_____	_____
	William M. Meil, Ph.D. Associate Professor of Psychologist

ACCEPTED

_____ _____

Timothy P. Mack, Ph.D.
Dean
The School of Graduate Studies and Research

Title: Using Adiposity Change in College Freshman to Examine the Comorbidity of ADHD and Obesity

Author: Katherine L. Ratcliff

Dissertation Chair: Dr. Donald U. Robertson

Dissertation Committee Members: Dr. David J. Laporte
Dr. William M. Meil

An increasing number of studies are finding a relationship between obesity and Attention-deficit/Hyperactivity Disorder (ADHD), although little is known about the potential mechanisms underlying this relationship. The present study examines this potential relationship and possible underlying mechanisms of maladaptive eating patterns and impulsivity trait characteristics in a longitudinal analysis of a college-student sample (n = 264). Body fat percentage (BF%) was collected at two time points during the student's first semester at college and eating behavior patterns and ADHD symptomology was assessed by self-report measures. Impulsivity trait characteristics were assessed by both self-report and experimental methods. No significant relationship was found between ADHD symptoms and BF% and the change in BF% across time-points was not significantly related to any other variables studied. Relationships between ADHD symptoms, eating behavior patterns, impulsivity, and BF% were inconsistent. Findings do not support a relationship between obesity and ADHD in a non-clinical population, but highlight the complexity of underlying mechanisms of both these conditions. Implications of utilizing a college-student population and suggestions for future research are discussed.

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CHAPTER I

INTRODUCTION

A small body of evidence has suggested a comorbidity of Attention Deficit/Hyperactivity Disorder (ADHD) and obesity in both childhood and adulthood. This was initially surprising to researchers conceptualizing ADHD children as over-active and therefore expending greater amounts of energy. However, a more in depth examination of these two conditions reveal several underlying similarities in each of their causal mechanisms. These similarities include the increased levels of impulsivity, the role of dopamine, and an abnormal sensitivity to reward and punishment.

The following review of literature will describe both ADHD and obesity and theories and research explaining each, emphasizing those theories which may help to understand the link between these two conditions. The primary focus will be on each of these conditions in adulthood. Additionally, although a majority of obesity research has been conducted using a between group design, support for the use of a within group design will be presented. Specifically, the college student population can be utilized to study the actual process of becoming obese, weight gain.

First, the literature on obesity will define and establish the growing importance of studying this condition, and discuss some of the theories of obesity. Although obesity typically results from either over-eating, under-activity, or both, most attention will be paid to the theories on eating behaviors attributed to obesity. Following the general obesity discussion, the literature on college weight gain specifically will be presented. This will include evidence for increased weight gain during the college freshman year, specifically during the first semester, as well as studies attempting to explain this

phenomenon. This body of literature is much less theoretical than the obesity literature, indicating a need for increased theory-based research to be conducted in this area. The review will then change focus to discuss the other condition of interest, adult Attention Deficit/Hyperactivity Disorder (ADHD), including the DSM-IV definition and the importance of studying this disorder. Several theories of ADHD will then be discussed, including biological, neuropsychological, and cognitive theories. Although most research to date has been on children, support for these theories on adults will be discussed as well as how ADHD is assessed in adulthood.

Following the review on the two conditions of interest, ADHD and obesity, the literature review will explain the specific constructs to be studied, including impulsivity and behavioral inhibition as well as sensitivity to reward and punishment. These constructs have been defined in multiple ways in different bodies of literature. Therefore, an attempt will be made to explain the different ways each has been conceptualized, including how each is measured, and then establish a working definition for the current study. Finally, the evidence of the effect each construct has on both ADHD and obesity will be discussed. This will set the stage for discussing the evidence of the comorbidity between ADHD and obesity and the one study that has tried to explain this comorbidity.

Obesity

Obesity is defined by the U.S. National Institutes of Health (NIH) and the World Health Organization (WHO) as a body mass index (BMI) greater than 30. BMI is calculated based on units in kilograms divided by height in meters squared. These two organizations have issued guidelines, dividing the BMI into four major categories; underweight ($BMI < 18.5$), normal ($18.5 \leq BMI < 25$), overweight ($25 \leq BMI < 30$), and

obese (BMI ≥ 30). Within the obese category are several subcategories, as the health risks sharply increase with BMI after the obesity threshold.

Obesity is considered a general medical condition and is therefore not listed in Diagnostic and Statistical Manual of Mental Disorders-IV, although Binge Eating Disorder is currently included as a diagnosis set for future study. Binge-Eating Disorder is characterized by a) recurrent episodes of binge eating associated with subjective and behavioral indicators of impaired control over, and significant distress about, the binge eating and b) the absence of the regular use of inappropriate compensatory behaviors that are characteristic of Bulimia Nervosa. Individuals with this eating pattern seen in clinical settings have varying degrees of obesity (APA, 2000).

A large body of evidence indicates that higher levels of body weight and fat are associated with increased risks of developing numerous adverse health outcomes and increased mortality. Each year, at least 280,000 deaths among U.S. adults are attributable to obesity. Adverse health affects include coronary disease, hypertension and hyperglycemia, type 2 diabetes mellitus, endometrial and gallbladder cancer, and osteoarthritis. Higher BMI and substantial weight gain during adulthood were also found to be strongly associated with reduced quality of life as measured by daily physical functioning and vitality, a burden of physical pain, and feelings of well-being (Manson, Skerrett, & Willett, 2002). Psychologically, obesity is also a risk factor for decreased self esteem and increased rates of depression (Annis, Cash, & Hrabosky, 2004), poorer psychosocial functioning (Warschburger, 2005), as well as feelings of shame and isolation (Sissem & Heckert, 2004).

Given the numerous adverse effects of obesity, it is of growing concern that the rate of obese adults in the U.S. has increased rapidly in the past several years. An ongoing, state-based, random-digit--dialed telephone survey of the noninstitutionalized, U.S. civilian population aged 18 or older revealed that age-adjusted obesity rates increased from 15.6% in 1995 to 19.8% in 2000 and up to 23.7% in 2005 (Ogden, Carroll, Curtin, McDowell, Tabak, & Flegal, 2006). The same study revealed that, in 2005, among the total U.S. adult population surveyed, 60.5% were overweight, 23.9% were obese, and 3.0% were extremely obese (BMI $\geq$ 40). Obesity prevalence was 24.2% among men and 23.5% among women and ranged from 17.7% among adults aged 18-29 years to 29.5% among adults aged 50-59 years. Among racial/ethnic populations, the greatest obesity prevalence was 33.9% for non-Hispanic blacks.

Weight gain is agreed to be the result of energy imbalance, such that the caloric intake is greater than caloric expenditure, over a long period of time. The cause of energy imbalance for each individual may be due to a combination of several factors including genetics, environmental factors, and individual behaviors including physical activity and eating behaviors (NIM, 2007).

Theories of obesity. There is evidence to suggest that like height, weight is highly heritable trait, as much as 40-70% heritability (Barsh, Farooqi, & O'Rahilly, 2000; Maes, Neale, & Eaves, 1997). However, despite obesity's strong genetic determinants, the genetic composition of the population does not change rapidly. Therefore, the large increase in obesity must reflect major changes in non-genetic factors (Hill & Trowbridge, 1998).

Environmental factors have also been found to be a causal factor of obesity. Individuals in the U.S. are exposed to an environment in which energy-dense foods are widely available, inexpensive, and promoted heavily, while energy-saving devices and other changes in lifestyle increase sedentary behavior. The changes in negative food environment include an exponential increase in fast-food restaurants in the past 20 years as well as a systematic increase in portion sizes (Brownell, 2002). Changes in physical activity environment include increases in energy-saving devices such as the automobile, elevators and escalators, resulting in few people receiving more than minimal activity at work or in day-to-day activities, and the growing popularity of the computer, television, and video games, making sedentary behavior more appealing and engaging (Brownell, 2002). However, environmental changes do not explain why only some persons exposed to these factors experience weight gain whereas others do not. In order to understand this phenomenon, individual differences in behavior must be examined. This includes both physical activity as well as eating behavior.

There are numerous theories that discuss factors related to physical activity, including social-cognitive determinants (Dishman, et al., 2002), environmental factors (Motl & Dishman, 2005), various psychosocial variables (Litwin, 2003; Lewis, Marcus, Pate, & Dunn, 2002), mood (Berger & Motl, 2000), and self-efficacy (Motl, Dishman, & Ward, 2005). However, the current study places a greater emphasis on theories of personal eating behavior. Predominant theories on the causes of individual differences in eating behavior include emotional overeating, externality, restraint eating, and the relatively new idea of eating as an addictive behavior that may account for excess caloric intake.

Emotional overeating, sometimes described in terms of psychosomatic theory (Kaplan & Kaplan, 1957), is the tendency for obese individuals to respond to negative arousal states such as anger, fear, or anxiety by eating excessively. This is often attributed to the confusion between internal arousal states and hunger, possibly due to early learning experiences (Bruch, 1961). Eating as the result of a negative emotional state has been supported in both the psychological and the physiological literature. Studies have shown that the salience of negative mood promoted overeating in obese binge eating females (Chua, Touyz, & Hill, 2004), an association among increased stress and fatty food intake among adolescents (Cartwright, Wardle, Steggles, Simon, Croker, & Jarvis, 2003), and an effect of fear on increased food consumption among obese but not non-obese individuals (Schachter, Goldman, & Gordon, 1968). Physiologically, sweetness, and high fat content in food has been found to improve mood and mitigate effects of stress by enhancing the level of dopamine and serotonin activation (Gibson, 2006).

The externality theory of obesity, formulated by Schachter (1968, 1971), proposes that obese persons, compared to nonobese persons, are more responsive to external cues, such as the presence of food, and less to internal physiological cues associated with hunger. Several studies testing obese versus non-obese rats (Schachter, 1971) as well as human subjects (Schachter, 1971; Rodin, Slochower, & Fleming, 1977; Herman, Olmsted, & Polivy, 1983) have supported this theory.

A theory of restraint eating poses that both external and emotional eating are consequences of intense dieting (Herman & Mack, 1975; Nisbett, 1972). The cognitively mediated effort to combat the urge to eat, or diet, is termed restraint. People vary in the extent to which the exercise restrain, from those who constantly worry about what they

eat and struggle to diet versus unrestrained eaters who eat freely as the desire strikes (Ruderman, 1986). According to Nisbett (1972) each individual has his own, homostatically regulated, range of body weight and those at a high range are under intense social pressure to weigh less, resulting in intense dieting and persistent hunger. This restrictive control can be broken down by disinhibitory processes, such as alcohol or negative emotional state, that lead to loss of contact with internal feeling of hunger and satiety and result in overeating (Herman, Olmsted, & Polivy, 1983). This hypothesis, developed initially to explain Schachter's externality findings, proposes that differences in level of restraint underlie obesity, such that obese persons are more likely to be "high restraint" persons (Herman et al., 1983).

More recent literature on eating behavior suggests that some that for some individuals, food intake may be the result of biological mechanisms resulting in a physiological addiction to food, especially foods high in processed sugar (Wang, Volkow, & Logan, 2001). Adaptation in the same pathways that link sweet and fatty food to improved mood and decreased stress, addressed in the emotional overeating literature, leads to overeating of calorie-dense foods and consequent obesity (Gibson, 2006). This adaptation is proposed to be the result of chronic exposure to such foods and enhanced by inherited sensitivity, called sensitivity to reward (Gibson, 2006; Davis, Sachan, & Berkson, 2004). Sensitivity to Reward is a psychobiological trait rooted in the neurobiology of the mesolimbic dopamine (DA) pathway. Striatal DA plays and important role in appetitive and consummatory motivated behaviors such as eating, drug seeking, and sexual activity, with lower DA availability associated with diminution in motivation (Davis et al., 2004). In addition to the sensitivity of these brain reward regions

correlating significantly with the risk for a variety of drug addictions, sensitivity to reward has also been found to predict overeating and preference for food high in fat and sugar, in turn, predicting higher BMI (Davis, Patte, Levitan, Reid, Tweed, & Curtis, 2007).

College weight gain. The study of obesity and its precipitating factors is difficult because weight gain of humans is usually a very slow process, undetectable by ordinary daily self-observation and is caused by such small changes in energy balance that it is practically undetectable by current technology. One possible model of this small increase in positive energy balance, proposed by Levitsky, Halbmaier, and Mrdjenovic (2004), is the increase in the body weight that is believed to occur in freshman during their first year at college. Since it is at a rate much more rapid than the typical U.S. population this weight gain is easier to measure within a given time period.

College freshman weight gain has been widely examined with mixed results. Graham and Jones (2002) found no significant weight gain at the end of freshman year, but only a small sample was used (*n = 49).* Hodge et al., (1993) also found no difference in average weight between measurements six month apart. More recent studies, with larger samples have consistently found weight gain in college freshman significantly higher than what would be found in the normal population. These studies have found that weight increases for approximately 75% of students between the beginning and end of freshman year, with a statistically significant mean weight increase of 2.5± 5.0 kg (5.5 ± 11.02 lbs) and BMI increased from 22.4± 3.7 to 23.3± 3.8 kg/m^2 (Racette, Deusinger, Strube, Highstein, & Deusinger, 2005). A majority of this weight gain appears to take place during the first semester. Levitsky, Halbmaier, and Mrdjenovic (2004) found a mean,

highly significant, weight gain of 1.9 ± 2.4 kg (4.18± 5.29 lbs) and BMI increased from 20.8±2.1 to 21.5±2.3 kg/m^2 during the first semester. Anderson, Shapiro, and Lundgren (2003) found that the top twenty-five percent of their sample gained over 2.3 kg during the first semester alone.

In examining why college freshman gain weight at a rate greater than the population, studies have found that this weight is not related to changes in exercise or dietary behaviors (Racette et al., 2005) or personal characteristics such as appearance or health evaluation, sexuality, self-esteem, locus of control, or self-monitoring (Hodge et al., 1993). Environmental factors, such as housing were found to impact weight gain. Hovell (1985) reports that female student living in on-campus housing, compared with those living in off-campus housing, were 2.6-5.5 times more likely to gain at least 15% above their ideal weight. Both environmental and behavioral factors, including consuming evening snacks, high-fat, and other 'junk' foods, all-you-can-eat dining halls, recent dieting; and meal frequency significantly predict weight gain variance (Levitsky et al., 2004). When initial body weight is used as a covariate, environmental factors drop out and weight gain is best predicted by junk food and evening snacks, recent dieting, and hours of sleep (Levitsky et al., 2004). No research has been found linking ADHD symptoms to college weight gain, although increased attention is currently being given to the prevalence of ADHD in the college and adults populations.

Attention Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder is defined in the DSM-IV (APA, 2000) as a persistent pattern of inattention and/or hyperactivity-impulsivity, present before age 7, which present in more than one setting and result in significant impairment in

functioning. The disorder is broken down into three subtypes; predominantly inattentive type, including only inattention symptoms; predominantly hyperactive-impulsive type, including only hyperactivity and impulsivity symptoms; and combined type, which includes both inattention and hyperactive and impulsivity symptoms. Combined type is the most common type diagnosed in children and adolescents, although it is still unknown whether this is true for adults. It is possible, although not yet concluded that adult ADHD may also include different factors. For example, Conners, Erhardt, and Epstein (1999) have found that adult self-ratings of symptoms ascribed to adult ADHD load onto four factors; inattention/executive functions/academic problems, hyperactivity/restlessness, impulsivity/emotional lability, and problems with self concept.

Typically, the disorder is first diagnosed in during elementary school years, when school adjustment is compromised. The disorder is relatively stable through early adolescence and, in most individuals, symptoms attenuate during late adolescence and adulthood (APA, 2000). Estimates of the prevalence of adult ADHD vary widely. In longitudinal studies that have followed children diagnosed with ADHD, rates of the adult disorder range from less than five percent to over fifty percent (Weiss & Hechtman, 1993; Claude & Firestone, 1995; Mannuzza et al. 1993; Mannuzza et al., 1998). Differences in prevalence may be due to methods of reporting, as Fischur (1997) found at only 3 percent of 21-year-olds met criteria according to self-report, but 42 percent of the sample met diagnostic criteria when parental reports were used.

In college groups specifically, one study found 2.5 percent of the sample was 1.5 standard deviations above the mean on measures of ADHD symptoms and .5 percent were two standard deviations above the mean (Weyandt, Linterman, & Rice, 1995). A

different study, looking at self reported ADHD symptoms of college students by gender found a prevalence ranging from 0.2% for males having the combined subtype to 2.3% for females meeting criteria for the hyperactive/impulsive subtype (DuPaul, Schaughency, Weyandt, Kiesner, & Stanish, 2001). Summarizing the recent studies of all adult ADHD prevalence research, if one takes an averaged continuation rate of 10 to 30 percent with a childhood prevalence of 3 to 11 percent, adult prevalence would be estimated between .3 and 3.3 percent, which is only slightly lower than estimates from community samples of 2 to five percent (Johnston, 2002).

Late adolescent and adult outcomes of childhood ADHD have been fairly negative outcomes across domains. Compared to controls, late adolescents (18-19 years old) with ADHD completed less formal schooling, achieved lower grades, failed more courses, were more often expelled (Weiss, Hechtman, Perlman, Hopkins & Wener, 1979) and obtained worse scores on standardized achievement tests (Mannuzza, Klein, Bessler, Malloy, & La-Padula, 1993). Cognitive deficits were also noted, including performing worse on test vigilance and visual motor integration (Cohen, Weiss, & Minde, 1972) and attentional processes (Mannussa et al., 1993). However, most of these deficits appeared to normalize by later adulthood (Mannuzza & Klein, 1999). ADHD adolescents, aged 18-19 on average, ADHDs had fewer friends, scored more poorly on social skills and self-esteem scales, and were rated by clinicians as having poorer psychosocial adjustment compared with controls (Weiss et al., 1979). One third of children with ADHD versus one fifth of the controls have an ongoing DSM-III-R diagnosis at adult follow-up in a longitudinal study (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998). Multiple theories attempt to explain these deficits and etiology of ADHD.

Theories of ADHD. Numerous biological, neuropsychological, and cognitive theories have been proposed and have studied the etiology and primary deficits of ADHD. Several of these hypotheses are similar in nature, and could therefore explain a link, to research and theories on the etiology of obesity. These theories include the dopamine hypothesis mediating reward sensitivity, genetic research on the 7-repeat allele of the dopamine-4 receptor gene, the importance of the behavioral inhibition system, and cascading effects of behavioral inhibition on four primary executive functions.

Genetic research on ADHD has estimated the heritability to be about .74 (Goodman & Stevenson, 1989). Additional genetic research, looking at the 7-repeat allele (7R) of the dopamine-4 receptor gene (DRD4), a variant associated with decreased affinity for dopamine, found that those with 7R carriers reported significantly greater inattention as well as significantly higher maximal lifetime BMI scores (Levitan, et al., 2004).

Much of the research on the biological basis of ADHD was partially inspired by the dopamine (DA) hypothesis, which assumes that the primary sites of action of the stimulants are in the dopamine pathways (Costellanos et al., 1996; Levy, 1991). DA has been implicated in brain circuits mediating reward and reinforcement (Castellanos, 1999). Studies generally agree that ADHD patients react to reward in a fundamentally different way than control groups, although how they differentiate from controls is still unclear. Wender (1974) suggested ADHD children have reduced sensitivity to reward based on ADHD children being unable to delay gratification. Barkley (1989) argued that, in addition to reward having less initial value, reinforcement also looses its effect more quickly in ADHD children. To explain why this is the case, Haenlein and Caul (1987)

proposed a theory in which children with ADHD have an elevated reward threshold, which therefore decreases the experienced magnitude of reinforcement. According this theory, stimulant drugs are effective by lowering children's reward threshold, thereby increasing the magnitude of reward, which has been empirically supported (Wilkinson, Kircher, McMahon, & Sloane, 1995). In contrast, Douglas and Parry (1994) suggested children with ADHD have increased sensitivity to reward, resulting in an increased tendency to seek immediate rewards, overreaction to the failure of obtaining rewards, and increased vulnerability to arousing and distracting effects of reward.

A different psychobiological system regulating behavior has also been implicated in ADHD research. This system is the behavioral inhibition system (BIS; Pickering & Gray, 1999), which is hypothesized as a motivational brain system that provides for the cessation of ongoing behavior as well a focusing of attention on relevant environmental cues (Quay, 1997). Quay's (1997) theory proposes that children with ADHD are less sensitive to cues that non-reward and punishment are likely to follow a particular response as a result of under-activity of their BIS.

Currently, a unifying theory of ADHD, proposed by Barkley (1997), is receiving the most attention in the literature. According to a this model, deficient response inhibition is the core deficit in ADHD, which in turn has cascaded effects on four other executive functions (Barkley, 1997). Executive functions are defined as those abilities that are critical for self-regulation and goal directed persistence. The four executive functions proposed to be affected by behavioral inhibition are working memory, self-regulation of affect/motivation/arousal, internalization of speech, and reconstitution. Each of these executive functions then affects the overall control of motor behaviors, fluency

and syntax. In terms of observable behaviors, this relates to inhibiting task-irrelevant responses, executing goal-directed responses, executing novel or complex motor sequences, goal-directed persistence, sensitivity to response feedback, task re-engagement following disruption, and control of behavior by internally represented information. Ultimately, each of these behaviors results from deficits in four key executive functions resulting from behavioral inhibition (Barkley, 1997).

Support of theories in adults. Most empirical studies working to prove and disprove the above theories have been conducted on children. However, recent studies have been giving increased attention to the adult population. Several studies have found differences between ADHD adults and controls across the major domain of executive functions, similar to deficits found in the child population (Hervey, Epstein, & Curry, 2004; Nigg, Stavro, Ettenhofer, Hambrick, Miller, & Henderson, 2005; Fischer, Barkley, Smallish, & Fletcher, 2005; Stavro, Ettenhofer, & Nigg, 2007; Murphy, Barkley, & Bush, 2001). Whether these deficits are attributed to comorbid anxiety and conduct disorders have resulted in mixed findings (Fischer, et al., 2005; Murphy et al., 2001).

Executive functions in adults appear to split into two domains, inattention-disorganization and hyperactivity-impulsivity, providing support for the DSM-IV criteria (APA, 2000) to be used in the adult population (Nigg et al., 2005). However, ADHD subjects appear to have stronger deficits in inattentive-disorganized symptoms (Nigg et al., 2005) and this behavioral domain tends to be more highly related to adaptive functioning (Stavro, et al., 2007) than hyperactivity-impulsivity, which refutes Barkley's (1997) theory that deficits are primarily the result of response inhibition difficulties. However, a meta-analytic review looked at several different executive functioning

domains and findings supported Barkley's theory (Hervey, et al., 2004). Tests in the attention domain and the response inhibition domain has similar weighted mean effected sizes which were slightly higher than tests measuring other executive functions in adults (Hervey, et al., 2004). Additionally, in a young adult population, several measures of behavioral inhibition accounted for a significant proportion of variance in measure of executive functions beyond that accounted for by IQ (Cheung, Mitsis, & Haplerin, 2004), providing further support for this model.

Longitudinal studies have shown that executive functioning deficits persist from childhood through adolescence and into adulthood, especially when there is the presence of current ADHD (Fischer et al., 2005). In terms of the stability of the executive functions, the latent constructs appear to have a degree of stability, but individual measures vary considerably across time, suggesting that multiple measures be used (Wadsworth & Harper, 2007).

Measuring ADHD in adulthood. ADHD is typically diagnosed in childhood, but it can sometimes remain undiagnosed until the person is in adulthood (APA, 2000). In accordance with DSM-IV diagnostic criteria, evidence of the disorder must be able to be traced back to childhood in order for it to be diagnosed at any age (APA, 2000). Therefore, optimal assessment of adult ADHD would include self-report measures, retrospective self-report measures, family history, rating scales, and cognitive evaluation (Wadsworth & Harper, 2007). Comprehensive clinical interviews are often used as well as rating scales such as Connors Adult ADHD Rating Scales (CAARS; 1999), the Current Symptoms Scales by Barkley and Murphy (CSS; 1998), or the Wender Utah Rating Scale (Ward, Wender, & Riemherr, 1998) for example. Assessment of cognitive

inefficiency, including sustained and divided attention, verbal fluency, processing speed, and response inhibition, is also recommended. Additionally, continuous performance tasks (CPTs) are objective cognitive assessments of sustained attention and response control that are useful and recommended as a part of a multi-method assessment for ADHD (Wadsworth & Harper, 2007). CPTs are used to assess both inattention and impulsivity, the latter of which is one of the key constructs in this study.

Impulsivity. Impulsivity is a construct that is often used in the literature yet is poorly defined and conceptualized. Impulsivity is defined both in terms of personality and psychopathology. According to the personality perspective of impulsivity, this trait is not necessarily as negative as it is when defined in terms of psychopathology (Carver, 2005). For example, when manifested as spontaneity, impulsiveness brings a sense of vigor and freedom (Dickman, 1990) or can aid in survival, such as when a threat or an opportunity must be reacted to quickly (Carver, 2005). A detailed explanation of how psychodynamic, trait, temperament, biological, and cognitive models of personality address impulsivity is addressed elsewhere (see Carver, 2005).

Generally, impulsivity can be defined as the tendency to act with little forethought and it can be expressed as rapid, spontaneous, ill-planned, excessive, and potentially maladaptive conduct (Enticott & Ogloff, 2006), however it is much more complicated than this. Researchers studying impulsivity generally use either idiosyncratic definitions to best fit their research question or borrow a definition used by a previous study (Milich & Kramer, 1984). Even the DSM-IV (2000) uses multiple definitions depending on the disorder. Various definitions used in the DSM-IV (2000) include the inability to stop, look, and listen; inability to delay gratification; inability to resist temptation; inability to

inhibit motor movement; poor planning ability; calling out in class; poor time perspective; weak restraints; and poor self control. In terms of the DSM-IV criteria for ADHD, impulsivity criteria cut-offs are combined with hyperactivity criteria and include blurting out answers before the questions have been finished, having trouble waiting one's turn, and often interrupting or intruding others.

Several theorists have described impulsivity as a multidimensional construct (e.g. Enticott& Ogloff, 2006; Dickman, 1990), but the number and names of these constructs has varied widely. One conceptualization is that impulsivity can be divided into functional and dysfunctional impulsivity (Dickman, 1990). In this theory, functional impulsivity represents the tendency to engage in rapid, error-prone information processing when such a strategy is preferred whereas dysfunctional impulsivity refers to the tendency to engage in rapid, error-prone information processing because of an inability to use a slower, more methodical approach. A different conceptualization splits impulsivity into three different factors; motor impulsiveness, non-planning impulsiveness, and attentional impulsiveness (Patton, Stanford, & Barratt, 1995). Each of these is measured by use of survey methodology. Additionally, impulsivity is measured on separate scales of several different personality measures, which are also self-report (Eysenck & Eysenck, 1978, Patrick, Curtin, & Tellegen, 2002). Each of these self-report measures are often described to measuring personality impulsivity (Reynolds, Ortegren, Richards, & Wit, 2006) or cognitive impulsivity (White, Moffitt, Caspi, Bartusch, Needles, & Stouthamer-Loeber, 1994).

In contrast, impulsivity measured by laboratory experiments is often called behavioral impulsivity (Dougherty, et al., 2003; Reynolds, et al., 2006; White et al.,

1994). Many of these laboratory procedures are based on the assumption of impulsivity involving rapid, error-prone behavior (Milich & Kramer, 1984). Among these tasks, the construct of impulsivity can be further divided into "impulsive disinhibition" and "impulsive decision-making" (Raynolds, et al., 2006) or rapid-decision impulsivity and reward-directed impulsivity (Dougherty et al., 2003) or impulsivity of cognitive skills and impulsive motoric behavior (Milich & Kramer, 1984), depending on which tests are analyzed.

Impulsivity has been hypothesized to be caused by the breakdown of self-control mechanisms (Monterosso & Ainslie, 1999), rapid action without forethought, heightened automatic arousal, emphasis on the present, inability to delay gratification and inhibitory dyscontrol (Enticott & Ogloff, 2006), although many arguments for the causal nature of impulsivity tend to be circular. The definition and causal nature of impulsivity varies widely depending on the assessment instruments used to measure this contrast.

Measuring impulsivity. Impulsivity can be measured by several different methods depending on which construct of impulsivity one is working with. Considering impulsivity as a personality trait that people are cognitively aware of, a number of self-report measures are available. One of the most widely used self-report measures (Davis et al., 2006) is the Barratt Impulsivity Scale –11 (Pattton et al., 1995). This is a 31-item, self-report measure that is divided into six primary factors (attention, motor impulsiveness, cognitive complexity, perseverance, cognitive instability, and self control) and three secondary factors (Attentional Impulsiveness, Motor Impulsiveness, and Nonplanning Impulsiveness). The most commonly used personality scale to assess impulsivity is the Impulsivity scale of Eysenck & Essenck, (1978), which is most closely

related to the Motor Impulsiveness scale of the BIS-11 (Patton, et al., 1995). If one is interested in assessing the functional aspects of impulsivity, the Functional and Dysfunction Impulsivity Scales could be administered (Dickman, 1990).

Various laboratory methods are also used to measure impulsivity, the most common of which include the Conner's CPT, Go/No-Go paradigms, and Stop tasks (Lijffijt, Kenemans, Verbaten, & Engeland, 2005; Reynolds et al., 2006). The Conner's CPT (Conners, 2004) is a computerized task in which participants are required to press the spacebar when any letter except for the letter "X" appears on the screen. The percentage of trials when letters other than "X" appear was 90%. Errors of commission occurred when participants pressed the spacebar on trials when the letter "X" was presented. Similarly, the Go/No-Go task presents participants with "correct" and "incorrect" stimuli, typically in the form of numerals (Newman, Widom, & Nathan, 1985). In this particular version of the task, eight numbers are presented, four designated as corrected and four designated as incorrect. They are instructed to respond only to the correct numbers, being rewarded (i.e. paid ten cents) for correct responses and punished (i.e. subtracted ten cents) for incorrect responses. In the Stop Task, participants are instructed to respond to a visual go signal as quickly as possible, but to withhold this response when an auditory stop signal is present (Logan, Schachar, & Tannock, 1997). The stop signal is presented on 25% of the trails at varying delays following the go signal. The delay to the stop signal is varied systematically across trials according to the participant's performance until the participant inhibits his or her responses on 50% of trials. The stop reaction time can be inferred from the delay by subtracting the final mean delay at which the tone is presented from the mean go reaction time. Longer stop reaction

times are taken to indicate more impulsive responding(Logan, Schachar, & Tannock, 1997).

Additional measures have included time perception tasks, Stroop Test errors, Trail Making Test (Forms A and B), Balloon Analogue Risk Task (BART), and Delay of Gratification Tasks or Delay-Discounting tasks (White, et al., 1994). Time perception is measured by using both time estimation and time production tasks (White, et al., 1994). In time estimation, the stopwatch is run for six consecutive intervals of 2, 4, 12, 5, 45, and 60 seconds, and the subject is asked after each interval to estimate how many seconds had passed. In time production, the subject was asked to signal when he or she thought the previously defined intervals had passed. These two measures are found to be highly reliable and correlated are therefore combined into a single index called time perception. The BART (Lejuex et al., 2002) is a risk taking task in which participants are directed to "pump up" a balloon presented on a screen by clicking a computer mouse. For each pump, and counter increases by a certain amount of money. Participants may transfer the money in the counter to 'bank' at any time, but this also terminated the trail. After an unpredictable number of 'pumps' the balloon will 'explode' resulting in a loss of all the money not yet transferred to the bank. Participants who emit more pumps before banking are considered more impulsive. One version of a delay of gratification task is the Delay-Discounting task (Richards et al., 1999 as cited in Reynolds et al., 2006). In this computerized version, during a series of choice trails, participants are offered the choice between $10 available after a delay or a smaller amount available immediately. The amount of immediate money is incrementally raised until the participant chooses it as

equally often the delayed reward. The greater discounting by delay is considered to be more impulsive.

One study examined the relationships among selected self-report and behavioral measures of impulsivity including the BIS-11, the impulsiveness scale from the Eysenck Personality Scales (I7; Eysenck, Pearson, Easting, & Allsopp, 1985) among the "personality measures" and the stop-task, go/no-go task, delay-discounting task, and the balloon analogue risk task among the "behavioral" measures (Logan, Schachar, & Tannock, 1997). Results indicated that self-report measures tend to correlate amongst themselves but not with behavioral measures, suggesting different aspects of impulsivity being measured by self-report versus behavioral methods. These results have been replicated in one study unable to significantly correlate the BIS-11 with a number of laboratory measures used (Cheung, Mitsis, & Malperin, 2004). An additional study found that eleven different measures revealed two factors. One factor consisted of self-reported and observer reported impulsivity measures whereas the second factor consisted of experimental measures such as the Trail Making Test, Stroop errors, time perception, CPT and Delay of Gratification task (White et al., 1994).

Additionally, studies have also found difference between experimental measures as well. One study found the stop task and go/no-go task were highly related and loaded separately from the other two behavioral methods, the delay-discounting task and the Balloon Analogue Risk Task (Logan et al., 1997). The authors coined these two methods as measuring "impulsive disinhibition" whereas the delay-discounting task and balloon analogue risk task were referred to as "impulsive decision making". This distinction is

often found in literature comparing different assessment instruments of impulsivity (Dougherty, et al, 2003; White, et al., 1994) although terminology is sometimes different.

Behavioral Inhibition. Increasing the confusion surrounding the construct of impulsivity is how it is often used interchangeably with the construct of inhibition, which also takes on different meanings in the literature (Harnishfeger, 1995). For example, interference control is a type of inhibition that refers to suppressing a stimulus that pulls for a competing response so as to carry out a primary response, as measured by the Stroop effect or directed forgetting paradigms (Nigg, 2000). Motor inhibition, also called behavioral inhibition (Nigg, 2000) deficits are typically defined as the ability to inhibit a prepotent response, and are indicated by more errors of commission on tests such as go/no-go paradigm and the continuous performance tasks (Tannock, 2002). Oculomotor inhibition is the effortful suppression of reflexive saccade measured by antisaccade tasks (Nigg, 2000). Each of these tasks involves effortful inhibition of responses. Additionally, a separate subset of inhibition types involves automatic inhibition of attention (Nigg, 2000), but these relate much less to impulsivity that those involving effort.

It is the behavioral inhibition described in the literature that is most strongly associated with behavioral impulsivity. The confusion becomes evident when one considers the tests used to measure each type of construct, behavioral inhibition and behavioral/motor/, are examined. It the commission errors of tasks such as CPT, stop task, and go/no-go task that are reported to measure both impulsivity and inhibition (e.g. Dougherty, et al, 2003; Nig, 2000; Tannock, 2002; White, et al., 1994).

The relationship between impulsivity and inhibition. Enticott and Ogloff (2006) describes inhibitory dyscontrol as one of the proposed causes of impulsivity. However,

Barkley (1997) described the relationship among these constructs in terms of inhibition being subset of impulsivity, and the specific part proposed to be related to ADHD deficits. Among the several dimensions of impulsivity proposed in previous research (Milich and Kramer, 1984; Dougherty et al., 2003), it is the dimension reflected in deferred gratification and resistance to temptation, or what others have called "behavioral inhibition" (White et al., 1994), that is associated with the inhibitory processes described in Barkley's (1997) theory. It is the behavioral dimension of impulse control, rather than the cognitive dimension of impulsiveness that seems to be most stable over development, to correspond more closely to parent or teach ratings of hyperactive-impulsive behavior, and to correlate most highly with later cognitive and social competence (Barkley 1997).

This conceptualization of behavioral inhibition as a subset of impulsivity dimensions is supported in research looking at multiple measures of impulsivity, in which findings consistently indicate a factor specific to inhibitory control (Dougherty et al., 2003; Reynolds et al., 2006; White et al., 1994). Additionally, one study compared self-reported impulsivity according to Esenck's conceptualization to laboratory tests of inhibition. Results indicated that that impulsive people responded slower to signals to inhibit in a laboratory test than non-impulsives (Logan, Schachar, & Tannock, 1997). Therefore, the relationship between impulsivity and behavioral inhibition proposed by Barkley (1997) will remain as an underlying assumption throughout this study. Both impulsivity and behavioral inhibition are constructs related to the two conditions of interest. An additional construct relating to both ADHD and obesity is the concept of sensitivity to reward and punishment.

Sensitivity to reward and punishment. The concept of sensitivity to reward and punishment, as used in this study, is based on Gray's theory of personality (Pickering & Gray, 1999). This model argues two fundamental dimensions of personality; impulsivity and anxiety. Individual differences along these dimensions are argued to reflect variation in the reactivity, or sensitivity, to two basic brain systems. These systems are the behavioral inhibition system (BIS; for anxiety) and the behavioral activation system (BAS; for impulsivity). The BIS is activated by novel stimuli and by conditioned stimuli signaling punishment or frustrative non-reward and is related to trait-anxiety, introversion and neuroticism. The BAS is activated by conditioned stimuli signaling reward or relief from punishment and is related to impulsivity and approach behavior. The neurobiology of each of these systems is strongly influenced by dopaminergic involvement. For example, it is argued that one route to mesolimbic dopamine release is a product of the action of the BAS (Pickering & Gray, 1999). This relates the BIS/BAS functions to a subjects' sensitivity to reinforcers: "subjects with overactive BIS and BAS should have a greater proneness to perceive neutral situations as threatening and rewarding, respectively" (Torrubia et al., 2001). Therefore, BIS is also referred to as sensitivity to punishment while BAS is referred to as sensitivity to reward (Torrubia et al., 2001).

In terms of psychopathology, research suggests that BAS dominance, or increased sensitivity to reward, increases risk for externalizing problems (O'Brien & Frick, 1996; Oosterlaan & Sergeant, 1998), whereas BIS dominance, or increased sensitivity to punishment, increases vulnerability for internalizing problems (Turner, Beidel, & Epstein, 1991). BAS dominance can occur because of a strong BAS or a weak BIS (Colder & O'Connor, 2004).

Two different sets of scales are most commonly used to measure the personality concepts proposed by Gray. The first is the BIS/BAS scales (Carver & White, 1994). These scales consist of one scale measuring the BIS dimension and three measures of BAS dimension called Reward Responsiveness, Drive, and Fun Seeking. The authors did not specifically justify the subdivision of the BAS dimension, but suggested the use of one second-order factor in which all three BAS scales loaded on.

In contrast to the BIS/BAS scales, the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, et al., 2001) consists of only two scales and assessing sensitivity to specific cues, which is more closely related to Gray's theory (Pickering & Gray, 1999). The Sensitivity to Punishment scale was positively related to Eysenck's neuroticism dimension, negatively related to extraversion, not related to psychoticism, and significantly related to the trait anxiety, somatic anxiety, behavioral anxiety, and cognitive anxiety. The Sensitivity to Reward scale was positively related to Eysenck's extraversion and neuroticism, moderately related to psychoticism, positively related to Eysenck's Impulsiveness scale, and positively related to sensation seeking (Torrubia et al., 2001). These two scales, as well as multiple impulsivity measures, have been demonstrated to be related to both of the conditions of interest. The following sections describe the evidence the effect of both impulsivity and sensitivity to reward to the conditions ADHD and obesity.

Potential Mechanisms of Adiposity Variability

Impulsivity and adiposity. Impulsivity is likely to affect eating behavior and therefore result in weight changes and obesity. With respect to eating, impulsive individuals may be less likely to perform a variety of behaviors that contribute to healthy

eating patterns, such as planning meals in advance, eating on a regular basis, and resisting urges to indulge in high-fat foods (Lyke & Spinella, 2003).

Group comparison studies have found that those with eating disorders involving binges, obese women, and restrained eaters score higher on certain measures of impulsivity than non-binging eating disorder, normal weight women and controls, respectively (Nasser, Gluck, & Geliebter, 2004; Nederkoorn, Van Eijs, & Jansen, 2004; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Rosval, Bruce, Israël, Richardson, & Aubut, 2006). Group differences were found on the stop-signal task (Nederkoorn, et al., 2004; Nederkoorn, et al., 2006), the Motor Impulsivity Subscale of the BIS (Nasser et al., 2004; Nederkoorn, et al., 2006; Rosval, et al., 2006), the Nonplanning subscale of the BIS, the BIS total score, and the Go/No-Go task (Rosval, et al., 2006). The Delay Discounting task, Impulsivity Scale of the Eysenck Personality Profiler, Dutch Sensation Seeking Scale, and use of food exposure during laboratory tasks were not sensitive to between group differences (Nederkoorn, et al., 2004, Nederkoorn, et al., 2006). In terms of predicting maladaptive eating behaviors, objective measures of impulsivity such as delinquency and substance abuse were much better predictors than self-report measures of impulsivity (Connolly & Stice, 2004).

When assessing the relationship between impulsivity and eating behaviors specifically, the Motor Impulsivity scale of the BIS-II was significantly correlated with disinhibition scale on the Eating Inventory and the Attentional Impulsivity scale was positively correlated with the Disinhibition scale and the Hunger scale from the Eating Inventory (Lyke & Spinella, 2003). Eating behavior is also related to executive functioning as measured by the Frontal Systems Behavior Scale (FSBS; Grace, Stout, &

Malloy, 1999 as cited in Spinella & Lyke, 2004). All of the FSBS scales were associated with Disinhibition scale and the Hunger scale of the Eating Inventory (Spinella & Lyke, 2004).

Additional studies have found that when participants were categorized as high and low impulsiveness based on the stop-signal task, no differences in food intake during the testing was found (Guerrieri, Nederkoorn, & Jansen, 2007) and that there was no significant difference between obese and non-obese children in the errors made in a reaction timed test (Bonato & Boland, 1983). Additional studies have considered the impact of sensitivity to reward on weight.

Sensitivity to reward/punishment and adiposity. No one has considered the effect of sensitivity to punishment on weight. However, sensitivity to reward is theorized to relate positively to weight. Davis et al. (2004) predicted that food would be more rewarding for those with high sensitivity to reward, fostering the tendency to overeat and thereby contributing to a higher BMI. This mediational relationship was confirmed by using path analysis. The authors found that sensitivity to reward (as measured by a scale assessing physical anhedonia) was positively correlated with emotional overeating, which was in turn positively associated with BMI.

Since this study, both the BIS/BAS scale and the SRSPQ have been used to study sensitivity to reward, all finding a positive link between sensitivity to reward and obesity. Franken and Muris (2005) found that young women who were more sensitive to reward produced more food cravings and had a higher BMI than those less sensitive to reward. Davis et al. (2007b) found support for a structural equation model specifying sensitivity to reward to predict overeating and food preferences for high fat and high sugar food,

which in turn predicted BMI. Both impulsivity and sensitivity to reward and punishment have also been shown to be related to ADHD.

Potential Mechanisms of ADHD

Impulsivity in ADHD. Impulsivity, and more specifically behavioral disinhibition, has been evidenced in the adult ADHD population in a number of studies utilizing a variety of measures including the Matching Familiar Figures Test, CPT, basic Go/No-Go, Stop-Signal, antisaccade, Stroop, and Directed Forgetting tasks (Nigg, 2001). The deficit of behavioral disinhibition in ADHD adults has been supported so often that the research has shifted to the mechanisms behind these inhibitory deficits (Nigg, 2001) and the best approaches or measures to continue studying inhibitory deficits (Hervey, et al., 2001). Several studies have shown that deficits in Stop-Signal reaction time are robust across age (Lijffijt, et al., 2005) and that the reaction time on the stop signal trials of the Stop Signal Task has also had large average effect size when comparing ADHD to non-ADHD adults (Hervey, et al., 2004).

However, one study examining response inhibition by use of both the CPT and Stop-Signal task found response inhibition deficits evidenced in only the CPT (Epstien, et al., 2001). Additionally, a meta-analysis found that the Conners' CPT specifically had a comparable weighted mean effect size to that produced by the stop signal task and that this version of the CPT appears to be better at distinguishing adults with ADHD on the basis of percentage commission errors over the other, traditional versions (Hervey, et al., 2004). It is suggested that this difference is due to the response bias established in each of these test. The Conners' CPT has a higher signal probability and rapid response pace, priming an impulsive response pattern, and therefore producing greater commission

errors (Epstien, et al., 2001). Additionally, the concepts of sensitivity to reward and punishment have been closely related to impulsivity and behavioral inhibition, respectively. As such, numerous evidence of these constructs being over- and under-active in the ADHD population has been documented.

Sensitivity to reward/punishment and ADHD. Quay (1993, 1997) argued that ADHD is linked to a weak BIS and Barkley (1997) also theorizes that the deficits in ADHD are due to an underlying deficit in behavioral inhibition due to an under-active BIS. Therefore, this population would have a decreased sensitivity to punishment. Findings suggest mixed support for these hypotheses (Matthys, van Goozen, de Vries, Cohen-Kettenis, & van Engeland, 1998; Shapiro, Quay, Hogan, & Schwartz, 1988).

In terms of sensitivity to reward, both a reduced sensitivity to reward (Wender, 1971) and an increased sensitivity to reward (Douglas, et al., 1983) have been argued in ADHD children. A reduced sensitivity to reward is hypothesized to result in the inability to delay gratification (Wender, 1971) whereas an increased sensitivity to reward is hypothesized to increase the tendency to seek rewards as well as increase a persons' vulnerability to the arousing effects of reward (Douglas, et al., 1983). Each of these is a problem in ADHD children. Studies have suggested that children with ADHD perform worse under conditions of partial reinforcement than control children (e.g. Barber, Milich, & Welsh, 1996), although these findings have not been consistent (Pelham, Milich, & Walker, 1986).

Tripp and Alsop (1999) found that the performance of the ADHD group compared with control was influenced less by their overall history of reward on the task and more by the last reward they had obtained. The ADHD children's behavior was much

less stable and instances of reward on a particular alternative produced marked shifts in response bias in the direction of that alternative on subsequent trials. These findings support the hypothesis of Douglas (1989) that children with ADHD are more, rather than less, sensitive to the effects of reward. Evidence for both sensitivity to reward and punishment as well as impulsivity playing a role in the conditions of interest, ADHD and obesity, have been described in this review of the literature. Given the overlapping similarities of these two conditions, the following evidence of the comorbidity between ADHD and obesity will be less surprising than it was to the researchers who initially documented these findings.

Comorbidity between ADHD and Obesity

A number of inherently impulsive disorders are consistently found to be highly comorbid with ADHD such as Antisocial Personality Disorder, Conduct Disorder, and Substance Use Disorders (Jackson & Farrugia, 1997). Recently, studies have also found a high comorbidity between childhood ADHD and childhood obesity (Agranat-Meged, Deitcher, Goldzweig, Leibenson, Stein, & Galili-Weisstub, 2005; Holtcamp, Konrad, Heussen, Herpertz-Dahlmann, & Hebebrand, 2004). Holtkamp et al. (2004) compared the weights of a clinical sample of boys with an ADHD diagnosis to a reference population mean and found that proportions of overweight and obese child were significantly higher then expected. Agranat-Meged et al. (2005) assessed school-aged children hospitalized for obesity and found a significantly higher proportion of ADHD in this sample than in the general population, although the ratio of those diagnosed with the specific types of ADHD was similar to those described in population-based studies. However, these results

were not replicated in a sample of non-clinical adolescents in a population study (Rojo, Pharm, Dominquez, Calaf, & Livianos, 2006).

Comorbidity between obesity and ADHD in adult populations is also increasingly evident. One study examined both current and childhood symptoms of ADHD in a clinical sample of obese females and found that significant symptomology was reported in 26.7% of the sample in both childhood and adulthood, primarily including inattentive and impulsive symptoms versus hyperactive symptoms (Fleming & Levy, 2002). A different study examined the records of bariatric patients and found that an overall ADHD prevalence was 27.4%, much higher than the general population, and that the prevalence rate increased to 42.6% when only considering those with a BMI greater than 40 (Altfas, 2002). This same study also found that comorbid obesity and ADHD symptoms rendered treatment less successful compared to non-ADHD counterparts, in that those with comorbid symptoms had more clinic visits, with a trend toward longer treatment duration, and lost lest weight overall while in treatment.

One study, thus far, has focused on the specific behavioral mechanisms which might link ADHD and body size by examining path associations among ADHD symptoms, aspects of overeating, and the body weight in healthy participants from a general population (Davis, Levitan, Smith, Tweed, & Curtis, 2006). ADHD symptoms were measured by the Wender Utah Rating Scale (Ward, Wender, & Reimherr, 1993), assessing childhood ADHD symptoms, and by the total score of the Barratt Impulsiveness Scale-11, assessing impulsivity. Eating behavior was assessed by The Emotional Eating Scale (Arnow, Kenardy, & Agras, 1995), the Emotional Eating subscale and External Eating subscale of the Dutch Eating Behavior Questionnaire

(Strien, Frijters, Bergers, & Defares, 1986) and the Bing Eating Questionnaire (Halmi, Falk, & Schwartz, 1981).

A structural equation model hypothesizing the ADHD symptoms predict aspects of overeating, which in turn is correlated with BMI. A number of indices were utilized; including chi-square analysis, the comparative fit index, the standardized root mean squared residual, and the adjusted goodness of fit index, and each value obtained from the analysis was indicative of a good fitting model. In the discussion, the authors speculate on the mechanisms that could explain the relationship between ADHD and overeating. Several competing hypothesis were proposed including executive function deficits specifically in the domain of deficient inhibitory control, delay aversion based on the motivational hypothesis of the disorder, and the consumption of highly caloric food serving as a self-medicating function because of its ability to activate dopamine in the common reward pathway (Davis et al., 2006). Some of these hypotheses will be explored in the current study, as described below.

Summary and Hypotheses

A small body of evidence has suggested a comorbidity of ADHD and obesity in adulthood although only one known study has examined the specific mechanisms to explain this comorbidity.

In discussing the mechanisms that lead to obesity, both over-eating and under-activity can be examined. Three theories predominate over specific eating behaviors that lead to weight gain and ultimately obesity. These are psychosomatic theory, externality theory, and restraint theory. The mechanisms that lead to these maladaptive eating patterns have also been explored, some of which are closely related to traits also

associated with ADHD. Specifically these include impulsivity, behavioral inhibition, and sensitivity to reward and punishment. These constructs have been previously ill-defined in the literature, although it is theorized that sensitivity to reward is highly positively correlated with impulsivity and sensitivity to punishment is positively correlated with inhibition. Both increased impulsivity and sensitivity to reward as well as decreased behavioral inhibition have been shown to be related to both ADHD and obesity. Exploring each of these constructs and the effect they have on eating behavior will help to unravel the mechanisms that result in the comorbidity between ADHD and obesity.

Additionally, a majority of research on obesity at this point has been based on group comparison methods, comparing obese with non-obese persons. Few studies have examined the process leading to obesity, which is weight gain. This is because weight gain is generally a slow process and therefore a tedious and time-consuming process to study. One way to bypass this obstacle is to measure college students, who have been shown to gain weight at rates much faster than the general public.

The current study intends to further examine the link between adult ADHD and obesity by expanding upon the study of Davis et al. (2006) and examining the mechanisms relating ADHD to eating behaviors and thus to increased adiposity. One goal of this study will be to test the relationship between ADHD symptoms and body fat percentage. A second objective will be to test the relationship of specific constructs relating to ADHD and increased body fat percentage, namely problematic eating behavior styles and impulsivity constructs. Additionally, the study will assess the utility of using college student weight change an alternate method of studying obesity and weight differences.

The overall hypothesis of the study is that the relationship between ADHD symptoms and body fat percentage is mediated by impulsivity and behavioral inhibition as well as problematic eating styles. This overall premise encompasses several sub-hypotheses. The first of these sub-hypotheses is that ADHD symptoms will be a significantly related to body fat percentage. Body fat percentage is also expected to be predicted by 1) problematic eating behavior styles, and 2) impulsivity/behavioral inhibition. It is also hypothesized that eating behavior styles will be predicted by both specific ADHD symptoms as well as impulsivity/behavioral inhibition measures. Each of these hypotheses will tested to include both initial body fat percentage as well as body fat percentage change occurring over the course of students' first semester at college and it is predicted that the results will be similar.

CHAPTER II

METHODS AND ANALYSIS

Participants

Participants in this study were 264 undergraduate students, living on-campus, who were currently enrolled in their first semester at a medium-sized state university in the eastern United States. The students completed the study as per a research requirement for the Introductory Psychology (PSYC 101) course they are currently enrolled in. Students enrolled in 1 of 8 PSYC 101 courses offered in the fall 2007 semester who elected to participate in research to fulfill their research requirement were given a pre-screening measure. A copy of this measure is available in Appendix A. Inclusion criteria are that the student must be enrolled in their first semester at college and living on-campus during this time. Students meeting the inclusion criteria were chosen for the study at random from the psychology department's research subject pool.

Measures

Body fat percentage. Body fat percentage will be measured using the Omron Body Logic Pro Body Fat Analyzer utilizing bioelectric impedance technology. This is a portable, handheld device which sends a low-level electrical current of 50 kHz and 500 uA through the body to determine the amount of fat tissue, which has a lower electric conductivity than muscles, blood vessels, and bones. Body fat percentage refers to the amount of body fat mass as part of the total body weight described as a percentile, such that body fat percentage (BF%) = (Body Fat Mass in Pounds/Body Weight in Pounds) x 100 (Omron Healthcare Inc., 2006).

Bioelectrical impedance technology as been established as a more accurate measurement of body composition than body mass index (Roubenoff, R., Dallal, G., & Wilson, P.W., 1995). Hydrodensitometry, dual-energy x-ray absorptiometry, and air displacement plethysmography are alternative methods of measuring body compositions, but are much less convenient and more costly. Although research still suggests that these methods are optimal, bioelectrical impedance technology is strongly correlated with these measurements (Cox-Reijven, P., van Kreel, B., & Soeters, P.B., 2002), with Pearson product moment correlations for male and female body fat percentages between bioelectrical impedance and hydronsitometry ranging from 0.81 to 0.86 ($p<0.05$) (Williams, C.A. and Bale, P., 1998).

Conners' Adult ADHD Rating Scale – Self Report: Long Version. The Conners' Adult ADHD Rating Scales – Self Report: Long Version (CAARS) is a standardized self-report measure assessing key ADHD symptoms as well as other clinically relevant symptoms (Conners, Erhardt, & Sparrow, 1999). It is designed to take approximately 30 minutes to complete and is written at 4th grade reading equivalency level. Respondents rate each of the 66 items on the Likert-type scale ranging from 0 (not at all, never) to 3 (very much, frequently). Items ask the respondent to report how frequently various behaviors or problems are experienced such as, "I blurt things out" and "Many things set me off easily". Results are reported in the form of T-scores with greater elevations signaling greater symptomology. Raw scores are added up based on the scoring form included with each test form and then converted to T-scores based on the participants' age and gender. The CAARS-S:L includes nine scales; Inattention/Memory Problems (I/MP); Hyperactivity/Restlessness (H/R); Impulsivity/Emotional Lability (I/EL);

Problems with Self-Concept (PSC); DSM-IV Inattentive Symptoms; DSM-IV Hyperactive/impulsive Symptoms, Total ADHD Symptoms, ADHD Index, and Inconsistency Index. Scores for each scaled are computed by adding up individual responses of each item in the scale and then converting the raw score into a T score by using the chart supplied. Five of the 12 items on the ADHD Index are also scored in other scales as well.

Conners et al. (1999) standardized the CAARS on a large sample (n = 1, 026) of non-clinical adults from several locations in the United States and Canada and reported the derived psychometrics. Coefficient alpha ranged from .64 (DSM-Hyperactive/Impulsive Symptoms Scale) to .89 (I/MP) for men between the ages of 18-29 and from .75 (DSM-Hyperactive/Impulsive Symptoms Scale) to .89 (I/MP and H/R) for women between the ages of 10-29. Test-retest reliability over a one month interval ranged from .88 (I/MP) to .91 (PSC).

The confirmatory factor analysis (CFA) of the four-factor structure of the CAARS items that comprise the I/MP, H/R, I/EL, and PSC subscales met the criteria standards for good fit. The correlations among factors from the CFA revealed moderate inter-correlations ranging from .38 between PSC and H/R to .64 between I/EL and I/MP as well as between I/EL and H/R. Erhard et al. (1999) reports on the validity of selected subscales from the CAARS by comparing a group of adults meeting DSM-IV criteria for ADHD (n = 39) and a group of control adults (n = 40). The ADHD group scored significantly higher ($p < .05$) than the control group on the I/MP, H/R, I/EL, and PSC. Additionally, a direct discriminant function analysis was performed using the CAARS subscales as predictors of membership in either the ADHD or control group. The results

of this analysis produced an overall correct classification rate of 85%. The relationship between current levels of ADHD symptoms and childhood symptomotology as measured by the Wender Utah Rating Scale (WURS; Ward, et al., 1993) was examined by Conners et al. (1999). Pearson product-moment correlations between the WURS and the subscales from the CAARS range from .37 (PSCS) to .67 (I/ELS).

Go/no-go task. A computerized, modified version of the Go/No-Go Task was developed using Inquisit Software. Go/No-Go tasks are designed to assess the ability to inhibit inappropriate responses. Participants are presented with different stimuli and are instructed to respond only to certain stimuli (called targets) and then not respond to the other stimuli (referred to as non-targets). The outcome measures are errors of omission (withholding a response when a response should have been made or not responding to a target) and errors of commission (responding to a stimulus, in which the response should have been inhibited or responding to a non-target). Errors of omission are thought to measure inattention, whereas errors of commission are thought to measure impulsivity (Newman et al., 1985). The task was modified in order to best capture commission errors, which is accomplished by having a higher signal probability, or increased targets, and rapid response pace (Epstien, et al., 2001).

This specific task is roughly based on a combination of the Conner's CPT-II (Conner's 2004) and the Newman, Widom and Nathan's Go/No-Go task (1985). Participants are presented with one of ten numerals (0-9) at a variable rate for a total of 12 minutes on average. Participants are instructed to respond to each numeral as quickly as possible, except for the numeral "6", by pressing on the space bar. Numeral "6" is the designated non-target and any responses to this numeral will count as errors of

commission. Each of the numerals are presented in a random order and the non-target will be presented in approximately 10% of the cases, which is the percentage of non-targets used in the CPT-II (Conners, 2004).

The task is broken down into a practice block and five test blocks. Each test block consists of 90 trails, during which the participant is presented with a numeral. Trials have two different durations. During the first test block, numerals are presented after a 250 ms pretrial pause and remain on the screen for as long as 1750 ms or until the participant presses the spacebar. During the second block, numeral are presented after a 1500 ms pretrial pause and remain on the screen for as long as 3000 ms or until the participant presses the spacebar. During the 3rd, 4th, and 5th test block, the two variations of trials are randomly selected. The rate of signal presentations is varied in order to increase the measure's sensitivity. If the interval between stimuli is always the same, any problems in preparing and anticipating will be minimized because subjects can predict when the next stimulus will occur with some certainty (Conners, 2004).

Barratt Impulsiveness Scale- Version 11. The Barratt Impulsiveness Scale - Version 11 (BIS-11) is a paper-and-pencil, self-report measure of the personality trait of impulsiveness (Patton, Standford, & Barratt, 1995). The measure consists of 30 items, answered on a 4-point Likert-type scale (Rarely/Never, Occasionally, Often, Almost Always/Always). Items are scored numerically and higher scores indicate greater impulsiveness. The scale consists of six first-order factors and three second-order factors, which each combined two of the primary factors. The three second-order factor scales are Attentional Impulsiveness (AI), Motor Impulsiveness (MI), and Non-planning Impulsiveness (NPI). Items from these scales ask the respondent to report how frequently

various situations are experienced, such as, “I don’t ‘pay attention’” (AI), “I do things without thinking” (MI), and “I am more interested in the present than the future” (NPI). The current study proposes to use the three second-order factors as separate scales as well the BIS-11 Total Score. Scores are calculated by reverse-scoring all necessary items and then summing the responses to each item on the scale. A copy of the BIS-11 is provided in Appendix B.

Patton et al. (1995) examined the psychometric characteristics of the BIS-11 with four different samples; undergraduates, substance-abuse patients, general psychiatric patients, and prison inmates. Coefficient alpha for the Total BIS-11 scale ranged from .79 in substance abuse patients to .83 in general psychiatric patients. Internal consistency estimates for the individual scales were not reported. Concurrent validity was examined by examining the between-group differences among the four different samples. Substance-abuse patients, general psychiatric patients, and prison inmates all scored significantly higher on the BIS-11 Total Score than the undergraduate population and prison inmates also scored significantly higher ($p < .01$) than both the substance-abuse patients and general psychiatric patients. No significant within-group sex differences were found.

Sensitivity to Punishment/ Sensitivity to Reward Questionnaire. The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) is a self-report measure based on Gray’s model (Pickering & Gray, 1999) of the two motivational systems, the Behavioral Inhibition System (BIS) and the Behavioral Activation System (BAS; Torrubia, Avila, Molto, & Casaras, 2001). The measure consists of 48 yes-no response items, containing two scales: Sensitivity to Punishment (SP; odd items) and Sensitivity to

Reward (SR; even items). Scores for each scale are obtained by adding each of "yes" answers. A copy of the SPSRQ is provided in Appendix C.

Torrubia et al. (2001) examined the reliability of the SPSRQ in an adult community sample. In examining the SP scale, 468 men and 1090 women were given the scale an initial time, after three months, after one year and after three years. Test-rests reliability coefficients were .89, .74, and .57, respectively. In examining the SR scale, 470 men and 1093 women were given the scale an initial time, after three months, after one year, and after three years. Test-retest reliability coefficients were .87, .69, and .61, respectively. Using the same samples, internal consistency estimates ranged between .75 in the SR for females and .83 in SP for males.

Convergent and divergent validity was explored by examining the scales' relationships to Eysenck's personality dimensions (Eysenck & Eysenck, 1978), State/Trait Anxiety Inventory – Trait Scale (STAI-T; Spielberger, Gorsuch, & Lushene, 1970), the Sensation Seeking Scale (SSS; Zuckerman et al., 1978), and the Manifest Anxiety Scale (MAS; Taylor, 1953).

Dutch Eating Behaviour Questionnaire. The Dutch Eating Behaviour Questionnaire (DEBQ) is a self-report measure of eating behaviors based on three leading theories of over-eating; psychosomatic theory, externality theory, and restraint eating theory (Strien, et al., 1986). The measure consists of 33 items. Each item has the response format of never (1), seldom (2), sometimes (3), often (4), and very often (5), although a not relevant response category is included to all items which are cast in a conditional format (i.e. "When you have put on weight, do you eat less than usual."). Items load on only one of three scales: Restrained Eating (10 items), Emotional Eating (13 items), and

External Eating (10 items). The score for each scale is determined by dividing the sum of the items scored by the total number of items on that scale. A copy of the DEBQ is provided in Appendix D.

Strien et al. (1986) examined the psychometric characteristics of the DEBQ in a sample of obese (n = 91) and nonobese (n = 566) adults. Internal consistency estimates for the individual scales, using the entire population, ranged from .80 (External eating) to .95 (Restrained Eating). Allison, Kalinsky, and Gorman (1992) examined two-week test-retest reliability for the Restraint Eating Scale using 34 undergraduate students and results indicate high temporal stability (r = .91).

Criterion-related validity can be determined by examining the differences among the obese and non-obese samples. Using *n,* mean, and standard deviation provided by the authors (Strien, et al., 1986), significant differences were found between the obese and nonobese participants for each of the scales; Restraint Eating (t = 36.36), Emotional Eating (t = 45.83), and External Eating (t = 11.91). Additional evidence of concurrent validity was established by Wardle (1987), finding the DEBQ to be successful in identifying the eating styles that characterize three participant groups; women attending 'weightwatchers' (n = 107), patients diagnosed with anorexia nervosa (n = 33) and patients diagnosed with bulimia nervosa (n = 61).

Survey of College Health Behaviors. The Survey of College Health Behaviors has been created by the researcher based on factors found by Levitsky et al. (2004) to be predictive of college freshman weight gain as well as several general demographic variables (age, gender, etc.). The survey includes seven questions asking about sleep, exercise, and eating habits. Eating behaviors include nighttime snacks, dessert and junk

food consumption, recent dieting, and eating in different environments (i.e. all-you-can-eat facility, off-campus restaurant, dorm room, etc.). Each question is designed to be used independently, as Levitsky et al. (2004) has done, and therefore no specific scoring is necessary. A copy of the Survey of College Health Behaviors is found in Appendix E.

Levitsky et al. (2004) found that consuming evening snacks, high-fat, and other 'junk' foods; all-you-can-eat dining halls; recent dieting; and meal frequency significantly predict weight gain variance when initial body was not controlled for. When initial body was using as a covariate, environmental factors drop out and weight gain is best predicted by junk food and evening snacks, recent dieting, and hours of sleep (Levitsky et al., 2004). These findings support the externality theory of obesity.

The current study assumes that all weight gain, including that which occurs during college, is predominantly attributed to underlying personality traits affecting a specific set of eating patterns. Questions relating to college eating and other health behaviors specifically are included in order to determine if college weight can adequately be used as an alternate method for studying obesity and weight gain in general. If these factors are better predictors of weight gain then general eating behaviors based on eating theories (as measured by the DEBQ) and if they are unrelated to the personality measures assessed (i.e. impulsivity, behavioral inhibition), this would suggest that the process of gaining weight during students' freshman year at college is fundamentally different than general weight gain and would not be an appropriate method for studying weight gain that leads to obesity.

Procedure

The study was conducted through the use of the psychology department subject pool. During the first week of classes, all students consenting to participate in research completed a pre-test form (Appendix A) to assess inclusion criteria for the study. Of those who met inclusion criteria, a randomly selected sample was scheduled for a two separate small group administrations session as early as possible in the semester and then again as late as possible in the fall semester. Given rules and procedures governing the psychology department subject pool, the study officially began in early October and approximately 7 weeks (M = 52.29 days, SD = 2.87) passed between the first and second administration.

During the first session, subjects signed the informed consent form (Appendix F) and then completed the Barratt Impulsiveness Scale-11 (BIS) and the Sensitivity to Punishment/Sensitivity to Reward Questionnaire (SPSRQ). While the participants completed the paper-and-pencil measures, the researcher took each of the participants individually to a private room in order to measure body pat percentage. Using a portable, handheld body fat analyzer utilizing bioelectric impedance technology, participants are instructed to grasp the device with outstretched arms until body fat percentage is displayed on the screen. Participants were not shown the measurements. After the participants completed the paper-and-pencil measures and had their body fat percentage measured, either the researcher or the research assistant set up the computerized Go/No-Go task. Following the completion of the Go/No-Go task, participants set up a follow-up appointment at the end of the semester and received an hour of participation towards their research requirement.

During the follow-up session, participants were administered the Dutch Eating Behavior Questionnaire (DEBQ), College Health Behavior Questionnaire, and the Conners Adult ADHD Rating Scale – Self-report: Long Version (CAARS-S:L). During this time, a second measurement of body fat percentage was taken in a private location as described above. Following the completion of the measures, participants were given the debriefing form (Appendix G) and received a second hour of participation towards their research requirement.

Analyses

Preliminary descriptive analyses were conducted on each of the variables and a series of t-tests were performed to examine gender differences. Partial-order correlations, controlling for the effects of gender, were calculated to get an initial look at the relationship between predictor and criterion variables.

In order to establish impulsivity/behavioral inhibition and eating behavior styles as mediators for the relationship between ADHD symptoms and BF%, several steps of regression analyses will be required. First, ADHD symptoms, as measured by two sub-scores of the CAARS-S: L (DSM-IV Hyperactive-Impulsive Symptoms and DSM-IV Inattentive Symptoms), must be established as significant predictors of BF% and each of the potential mediator variables. Mediator variables include three DEBQ sub-scores (Restraint Eating, Emotional Eating, and External Eating) as measures of problematic eating behavior styles and Go/No-Go Average Commission errors, SPSRQ Sensitivity to Reward and BIS-11 Total Impulsivity Score as measures of Impulsivity/Behavioral Inhibition. Then, these mediator variables must be established as significant predictors of

BF%. This is accomplished in two separate regression analysis, using DEBQ scores and impulsivity measures independently of one another.

If each of these conditions is met, the final step will be a hierarchical regression analysis, where ADHD symptoms will be entered into Model 1 as sole predictor and the mediator variables (either DEBQ scores or Impulsivity measures) will be entered in Model 2. A mediating relationship will be evident if the ability of ADHD symptoms to predict BF% is significantly weakened by adding mediating variables as predictor variables. Although these analyses were planned to be run both initial BF% and BF% change, actual analyses are dependent on the results of preliminary correlations and regression analyses.

CHAPTER III

RESULTS

Demographic Analyses

Data collection began in October, 2008, approximately five weeks after the beginning of the fall semester and the average length of time between Time 1 and Time 2 measurements was 52.29 (SD = 2.87) days. A total of 291 students participated in this study, with 267 participants returning for the second data collection time-point. Of these, three individuals were excluded from analyses due to missing or incomplete data. The remaining sample of 264 was predominantly female (n = 164, 62.1%) and US born (n = 247, 93.6%). The mean age of the sample was approximately 18 years (M = 18.34, *SD* = .691). A total of 6.1% (n = 16) of the sample reported being diagnosed with ADHD and 1.5% (n = 4) reported taking stimulant medication. A total of 16.3% (n = 43) of the sample was considered obese using initial body fat percentage and 31.2% (n = 83) of the sample reported currently engaging in dieting activity or making an effort to lose weight. When asked to estimate biological parents' weight status, 29.9% (n=79) of the sample reported their mother was either overweight or obese and 38.8% (n=84) of the sample reported their father was either overweight or obese. Complete demographic information can be found in Table 1.

Although not directly related to overall goal of the study, several health behaviors were examined in preliminary analyses to get a general sense of the eating, sleeping, and exercise habits of the sample with the idea that this information could be used in the future. Descriptive statistics of these variables as well as correlations to both initial BF% and BF% change are reported in Table 2.

A series of independent samples t-tests were conducted to examine gender differences in each of the independent and dependent variables. Significant results were found for Initial BF% ($t(262) = -7.96$, $p<.01$, d = 1.04) BF% Change ($t(262) = 2.03$, $p<.05$, d = .26), Restrained Eating Scores ($t(262) = -6.17$, $p<.01$, d = .79), and Emotional Eating Scores ($t(262) = -5.90$, $p<.01$, d = .60). Males ($M = 17.45$, $SD = 8.42$) were measured to have less Initial BF% than females ($M = 25.27$, $SD = 6.38$), although males ($M = .795$, $SD = 2.71$) had a greater increase in body fat from Time 1 to Time 2 than females ($M = .117$, $SD = 2.58$). Females reported higher scores on Restrained Eating ($M = 2.52$, $SD = .87$) and Emotional Eating ($M = 2.57$, $SD = .84$) than males ($M = 1.82$, $SD = .81$; $M = 1.90$, $SD = .87$) on each of these scales, respectively. No significant differences were found for the External Eating Score of the DEBQ (t (262) = -.26, *ns*).

When examining gender differences in ADHD symptoms and inattention and impulsivity measures, DSM-IV ADHD Inattentive Symptoms ($t(262) = .12$, *ns*), Sensitivity to Punishment($t(262) = -1.88$, *ns*), GNG Average Omission Errors($t(262) = -1.38$, *ns*), and GNG Average Commission Errors ($t(262) = -.45$, *ns*), were not significantly different. Significant results were found for DSM-IV ADHD Hyperactive-Impulsive Symptoms ($t(262) = -2.34$, $p<.05$), Sensitivity to Reward ($t(262) = 3.73$, $p<.01$), and BIS-11 Total Impulsivity ($t(262) = 2.39$, $p<.05$). While females ($M = 1.10$, $SD = .47$) reported higher scores on DSM-IV Hyperactive-Impulsive Symptoms than males ($M = .87$, $SD = .44$), males reported higher scores on the Sensitivity to Reward ($M = .57$, $SD = .88$) and BIS-11 Total Impulsivity ($M = 6.61$, $SD = 1.23$) measures than females ($M = .48$, $SD = .18$; $M = 6.30$, $SD = .90$) on each of these scales, respectively. Descriptive statistics and the results of these statistical analyses appear in Table 3.

Following the results of these analyses, gender will be used a covariate on all further analyses.

Partial Correlations

Partial correlations were used to explore the relationships among BF% (both Initial and Change), eating behavior, ADHD symptoms, and inattention and impulsivity measures, while controlling for gender. Correlation coefficients can be found on Table 4. Initial BF% was significantly correlated with only Restrained Eating Scores (r = .28, p<.01) and BF% Change (r = -.20, p<.01). BF% Change was not significantly correlated with any of the other variables and was subsequently dropped from further analyses.

Intercorrelations between eating behavior, as reported on the DEBQ, were found between Emotional Eating Scores and Restrictive Eating Scores (r = . 18, p < .01) as well as between Emotional Eating Scores and External Eating Scores (r = .18, p<.10), although not between Restrained and External Eating Scores.

As expected, DSM-IV criteria for ADHD subtypes and total symptoms, as reported on the CAARS-S:L, were also correlated. The strongest correlation was between Inattentive symptoms and total ADHD symptoms (r = .92, p<.01), while the weakest correlation was between Inattentive and Hyperactive symptoms (r = .62, p<.01). The three measures of impulsivity, Sensitivity to Punishment, BIS11 Total Score, and GNG Average Commission Errors were not significantly correlated.

Regression Analyses

In order to examine the additional initial hypotheses, several regression analyses were conducted. First, a series of three hierarchal regression analyses were performed assess the ability of ADHD symptoms, impulsivity measures, and DEBQ scores to

predict initial BF%. The next two sets of hierarchical regression analyses are then performed to assess the ability of ADHD symptoms and impulsivity measures to predict DEBQ subtest scores. Although the intention was to then test the mediating effects of both impulsivity measures and DEBQ scores on the relationship between ADHD symptoms and initial BF%, the following results will display that this step was unnecessary.

Predicting initial body fat percentage. Three hierarchical regressions; using ADHD symptoms, impulsivity measures, and eating behavior styles, respectively; were performed to predict initial BF%. The first hierarchical regression was performed to assess the ability of ADHD symptomology (DSM-IV Hyperactive-Impulsive Symptoms Subscale and DSM-IV Inattentive Symptoms Subscale; CAARS-S:L) to predict initial BF% after controlling for gender. Results of this analysis revealed that the addition of ADHD symptomology variables in step 2 did not significantly increase the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .01, *F* change (2, 260) = - 2.22, *ns).*

A second hierarchical regression analysis was used to assess the ability of impulsivity measures (Sensitivity to Reward, BIS-11 Total Score, and GNG Average Commission Errors) to predict Initial BF% after controlling for gender. Results revealed that the addition of impulsivity scores significantly increased the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .034, *F* change (3, 259) = 3.9, p<.01). Sensitivity to Reward (stand. β = -.12, *t* = -2.05, p<.05) and BIS-11 Total Score (stand. β = -.13, *t* = -2.37, p<.05) both emerged as independent predictors of

initial BF%, such that higher scores of Sensitivity to Reward and BIS-11 Total Impulsivity were associated with lower scores of initial BF%.

The final hierarchical regression analysis computed to predict initial BF% assessed the ability of eating behaviors (Restrained Eating Scores, Emotional Eating Scores, and External Eating Scores; DEBQ) as predictor variables after controlling for gender. Results revealed that the addition of DEBQ scores significantly increased the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .085, *F* change (3, 259) = 10.5, p<.01). Restrained Eating Scores (stand. β = .257, *t* = 4.55, p<.01) and External Eating Scores (stand. β = -.164, *t* = -2.87, p<.01) both emerged as independent predictors of Initial BF%. Restrained Eating was associated with higher initial BF%, whereas External Eating was associated with decreased initial BF%. Results of each of these analyses appear in Table 5.

Predicting eating behaviors. Two sets of hierarchical regression analyses, using ADHD symptoms and impulsivity measures respectively, were performed to predict eating behavior styles. A set of hierarchical regression analyses were used to assess the ability of ADHD symptomology (DSM-IV Hyperactive-Impulsive Symptoms Subscale and DSM-IV Inattentive Symptoms Subscale; CAARS-S:L) to predict the three eating behavior scores from the DEBQ after controlling for gender. For the outcome variable of Restrained Eating Scores, results revealed that the addition of ADHD symptomology scores did not significantly increase the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .009, *F* change (2, 260) = .24, *ns*).

For the outcome variable of Emotional Eating Scores, results indicated that the addition of ADHD symptomology scores significantly increased the predictive ability of

the equation beyond the variable of gender entered in step 1 (R^2 change = .102, *F* change (2, 260) = 16.94, p<.01) and DSM-IV Inattentive Symptoms scores (stand. β = .295, *t* = 4.22, p<.01) emerged as an independent predictor. Increased DSM-IV Inattentive Symptoms were associated with increased Emotional Eating Scores. Similarly, for the outcome variable of External Eating Scores, results indicated that the addition of ADHD symptomology scores significantly increased the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .118, *F* change (2, 260) = 17.41, p<.01) and DSM-IV Inattentive Symptoms scores (stand. β = .336, *t* = 4.53, p<.01) emerged as an independent predictor. Increased DSM-IV Inattentive Symptoms were associated with increased External Eating Scores. Results of these analyses appear in Table 6.

A second set of hierarchical regression analyses were used to assess the ability of impulsivity measures (Sensitivity to Reward, BIS-11 Total Score, and GNG Average Commission Errors) to predict the three eating behavior scores from the DEBQ after controlling for gender. For the outcome variable of Restrained Eating Scores, results revealed that the addition of impulsivity measures did not significantly increase the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .024, *F* change (3, 259) = 2.46, *ns*).

For the outcome variable of Emotional Eating Scores, results indicated that the addition of impulsivity measures significantly increased the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .049, *F* change (3, 259) = 5.12, p<.01). Sensitivity to Reward scores (stand. β = .132, *t* = 2.24, p<.05) and BIS-11 Total Scores (stand. β = .162, *t* = 2.78, p<.01) were indicated as independent

predictors, such that higher scores on these measures were associated with higher Emotional Eating Scores. Similarly, for the outcome variable of External Eating Scores, results indicated that the addition of impulsivity measures significantly increased the predictive ability of the equation beyond the variable of gender entered in step 1 (R^2 change = .087, *F* change (3, 259) = 8.23, p<.01). Sensitivity to Reward scores (stand. β = .175, *t* = 2.83, p<.01) and BIS-11 Total Scores (stand. β = .215, *t* = 3.53, p<.01) were also indicated as independent predictors for this outcome variable, such that higher scores on these measures were associated with higher External Eating Scores. Results of these analyses appear in Table 7.

CHAPTER IV

DISCUSSION

The current study had several objectives related to further examining the relationship between ADHD and adiposity and the mechanisms related to this relationship. The first two goals of the study were to test the relationship between ADHD and obesity found in recent literature and assess the utility of using college student BF% change as an alternate method of studying obesity. Additionally, the present study aimed to test the relationship of specific constructs relating to ADHD and increased adiposity, specifically maladaptive eating behavior styles and impulsivity constructs.

In regards to the first goal of examining the comorbidity between ADHD and obesity, it was hypothesized that ADHD symptoms and initial BF% would be positively correlated and that increased ADHD symptoms would be associated with increased BF%. This hypothesis was not supported[1]. Neither DSM-IV hyperactivity-impulsivity symptoms nor DSM-IV inattention symptoms were significantly related to initial BF%. Although this result is contradictory to several recent findings (e.g Altfas; 2002; Eremis et al., 2004; Fleming & Levy, 2002), it is not altogether surprising given the limited amount of previous evidence suggesting a relationship between ADHD and obesity. Furthermore, the current study was based on a non-clinical sample with a range of body compositions whereas all of the above studies were conducted in clinical settings by focusing on obese individuals. In research examining the comorbidity between ADHD and obesity in childhood, a similar trend is found. Several studies (e.g. Agranat-Meged et al., 2005; Holtcamp et al., 2004; Waring & Lapane, 2008) examining clinical populations found a relationship whereas the only two studies utilizing non-clinical

samples (Mustillo et al., 2003; Rojo et al., 2006) were unable to replicate these findings. The current study as well as previous research all support the theory proposed by Cortese et al., (2008) that the relationship between ADHD and obesity may only hold true in clinical samples only.

The second objective of the present study was to assess the potential utilization of college student BF% change as an alternate method of studying obesity and change in body composition. It was hypothesized that the relationships found above for initial BF% would be duplicated when using BF% Change as the criterion variable. This hypothesis was not supported and BF% Change was not significantly related to any of the other variables examined in this study. Although the majority of recent research agrees that most college freshman gain weight at a rate much faster than the general population, the jury is still out as to why this is. Most research has focused on changes in environment or daily routines, with any attention given to individual differences being unfounded (Hodge et al., 1993). This study also failed to confirm that college freshman adiposity change was related to individual or personality differences and, despite suggestions (Levitsky et al., 2004) that this population could be generalized to study universal adiposity change, this study does not support this claim. Although results may suggest that the change in BF% occurring over the first semester of college students' freshman year may be qualitatively different than examining BF% at one time point, an alternative explanation may be to consider the flawed methodology. As described in more detail in the limitations section, measuring initial BF% was substantially delayed from the beginning of the semester and, therefore, a relatively short time frame existed between the first and second measurements of BF%. Therefore, the present study may not be an accurate

representation of college freshman weight gain and these results should be interpreted with caution.

The final aim of this study was to test the relationship of specific constructs relating to both ADHD and obesity, including problematic eating behavior styles and impulsivity measures. This objective contains several hypotheses: (1) increased impulsivity will significantly predict increased initial BF%; (2) higher reported maladaptive eating behavior styles will significantly predict initial BF%; (3) increased impulsivity will significantly predict increased maladaptive eating patterns; and (4) greater reported ADHD symptoms would significantly predict increased maladaptive eating patterns. Although initial hypotheses also predicted that both maladaptive eating behaviors and impulsivity would mediate the relationship between ADHD and initial BF%, this was not specifically tested given that no relationship was found between ADHD and initial BF%.

The first of these hypotheses was not supported. Although scores on the BIS-11 Total Impulsivity and Sensitivity to Reward significantly predicted initial BF%, it was in the opposite direction hypothesized. Results found that impulsivity, as measured by these two scales, was negatively associated with BF%. In terms of impulsivity, previous research has been mixed in this construct's relationship to body composition. Most of this variability tends to be associated with the construct and methods used to study impulsivity, which is consistent with the current study where relationships were found using self-report measures but not using the Go/No-Task. However, previous research using the BIS-11 and a Go/No-Go task specifically found that obese women were more likely to score higher on these measures than normal weight women (Nederkoorn, et al.,

2006). These results are also in stark contrast to results of Davis et al. (2006) who used the BIS-11 and ADHD symptoms to predict BMI in healthy participants of a general population and Davis et al. (2004) who also found that Sensitivity Reward to be predictive of BMI. The present study is similar to each of these former studies in its use of measures and to the studies of Davis et al. (2006) and Davis et al. (2004) in its use of a non-clinical sample, making these results all the more surprising. However, unlike these previous studies, the present study utilized body fat percentage as opposed to BMI as a body composition measure and a college student sample versus an adult community sample. Although gender was accounted for in the analysis, the present study's sample also included males whereas previous studies have only included females in their samples.

The second hypothesis, predicting maladaptive eating behavior styles will be significantly related to initial BF%, was partially supported. Of the three eating behavior styles used as predictor variables, Restrained Eating and External Eating emerged as independent predictors. Restrained Eating scores were positively related to initial BF%. However, in direct contrast to the hypothesis, External Eating Scores were negatively related to initial BF%. Although previous research has supported each of these three eating behavior styles as significant factors in weight gain and obesity, there is still a significant amount of literature and controversy devoted to this topic and the importance of each of these eating styles in weight. In the past decade, restraint theory has dominated as the most important factor in over-eating and obesity. However, recent research has found that external eating (Wansink, Payne, & Chandon, 2007; Burton, Smit, & Lightowler, 2007) and emotional eating (Gelievbter, A. & Aversa, 2003) are still both

associated with BMI. Additionally, Ouwens, van Strien, and van der Staak (2003) found that restraint eating was not associated with food consumption or body composition. Generally, researchers have yet to agree that any or all of these factors is important in predicting weight or discriminating among obese versus normal-weight individuals and conclude that obesity is still a multi-determined and extremely complex process.

An alternate explanation for the current findings could be to consider self-reporting bias in that overweight/obese individuals may be more likely to deny emotional and external eating as sign of weakness while being more likely to endorse a restrained eating style as this could be taken as a sign of strength or looked highly upon. This theory has some empirical support in studies (Rennie, Siervo, & Jebb, 2006; Vansant & Hulen, 2006) finding that restrained eating was associated with under-reporting actual dietary intake.

The third hypothesis, predicting impulsivity will be positively related to maladaptive eating patterns, was partially supported. Although Restrained Eating scores were not significantly associated with impulsivity measures, both External and Emotional Eating Scores were significantly related to BIS-11 Total Impulsivity and Sensitivity to Reward. BIS-11 Total Impulsivity scores and Sensitivity to Reward scores were positively associated with scores on Emotional Eating and External Eating. The results of Sensitivity to Reward as a predictor variable are consistent with previous finding relationships between Sensitivity to Reward and Emotional Eating (Davis et al., 2004) as well as to the total DEBQ score (Davis et al., 2007b). Previous research examining the relationship of impulsivity and eating behavior styles have been confused by a) lack of agreement on the construct of impulsivity, and b) lack of clear conceptualization of eating

styles, making comparisons difficult. For example, Lyke and Spinella (2003) found that sub-scores on the BIS-11 are significantly correlated with Disinhibition and Hunger Scales on the Eating Inventory, but it is unclear how these scales relate to different theories of eating behavior styles or DEBQ subscales. Additionally, when the DEBQ is used, a combined score of all three subscales is often used as a single variable, making it difficult to compare the present results to these results. A prime example of this is the inspiration for the present study, conducted by Davis et al. (2006), finding that "maladaptive eating behaviors," as measured by the total DEBQ score, mediated the relationship between ADHD symptoms and BMI.

While there has been a great deal of effort to support one theory of maladaptive eating pattern in contrast to another, a different school of thought has combined these eating styles in view that they are not mutually exclusive and that there could be a cumulative maladaptive effect. By examining the relationships between DEBQ sub-scores in the present study, the independence of these constructs is supported over their interdependence. There was only a small relationship between Restrained Eating and Emotional Eating, a moderate relationship between Emotional Eating and External Eating, and no significant relationship between Restrained Eating and External Eating.

As highlighted in the review of literature, the construct of impulsivity is also extremely inconsistent. Data of the present study supports the current theory that Sensitivity to Reward is related to Impulsivity, although only mildly so. The present study also concurs with previous literature finding differences between self-reported impulsivity and impulsivity as measured experimentally, although these results should be interpreted with caution. While the lack of significant correlation between omission or

commission errors on the Go/No-Go task and self-reported measures may be due to the fact that we are comparing experimental results with those attained via self-report method, it should also be noted that this measure was created by the experimenter, based very closely on Conner's CPT, and has not been specifically validated.

The final hypothesis, predicting ADHD symptoms would be positively related to maladaptive eating patterns, was partially supported. Although ADHD symptoms were not significantly predictive of Restrained Eating scores, increased DSM-IV Inattentive symptoms significantly predicted increased External Eating scores and Emotional Eating scores. Two topics related to this hypothesis are worth noting. First, as mentioned previously, prior research has lumped these eating behavior styles together when examining their relationship with ADHD. Davis et al. (2006) grouped the Emotional and External DEBQ scores with two other measures of "disordered" eating behavior and found that increased ADHD symptoms were able to positively predict higher overall disordered eating. Secondly, it is important to point out that DSM-IV Hyperactive-Impulsive Symptoms was not a significant predictor of any of the types of eating behavior styles. This may be due to the fact that hyperactive and impulsive symptoms are grouped together, both for purposes of the study as well as in the DSM-IV. A study where these symptoms were separated found that obese women reported significantly higher inattentive and impulsive symptoms than normal-weight counterparts versus hyperactive symptoms (Fleming & Levy, 2002).

Theoretical and Clinical Implications

A primary objective of the study was to assess the possibility of utilizing college freshman adiposity change as an alternative method for studying the theory of weight

gain. This study did not support this possibility, which may suggest that the mechanisms related to adiposity change in the first semester of college may be both quantitatively and qualitatively different than adiposity change throughout a persons' lifetime. Although specific methodological issues must be considered when interpreting these results, at this time, prospective, longitudinal studies remain the gold standard for examining the mechanisms that lead to weight gain over time.

In an effort to examine and combine theories of both ADHD and obesity, the present study is able to add a small piece to a very large and complicated set of literature. The first goal of the study was to examine the comorbidity of ADHD and obesity in a non-clinical sample. Similar to the results of previous studies summarized by Cortese et al. (2008), this study was unable to support a relationship between ADHD symptoms and body composition in a non-clinical sample. Although several studies have supported this phenomenon in clinical populations, ADHD symptoms and body composition do not appear to be related when examined as a continuum of normal behavior. This suggests it may be necessary to view and study these symptoms in terms of pathology alone.

Likewise, the present study also examined all of the mechanisms hypothesized to link ADHD to obesity as continuous trait variables as well. An opposing, and potentially enhanced, view would be to examine only pathological amounts of the traits. For example, one theory of impulsivity (Carver, 2005; Dickman, 1990) is that a certain amounts of this trait are useful and it only becomes problematic when a person is *too* impulsive. Perhaps this "some is good but a lot is not" view would lead to a better understanding of the relationships among these variables. Statistically, the relationships between impulsivity, eating behavior styles, ADHD symptoms, and body composition

may not be linear and may only be present when examining the extremes scores of each of these variables[2].

Although a great deal of information was gained from examining the relationships between ADHD, BF%, eating behavior styles, and impulsivity as continuous, linear constructs, this information does not fit easily into preexisting literature. This is partially because much of the preexisting literature regarding eating behavior styles and impulsivity is theoretically complex and contradictory in terms of empirical support. Additionally, both of these constructs tend to vary based on how they are measured and utilized, as demonstrated in this study's impulsivity measures. Factor in the possibility of self-reporting bias and the sheer number of variables that go into measuring mechanisms of weight gain make it difficult to develop concrete conclusions.

From a clinical standpoint, it would be presumptuous to conclude that people tending toward overweight would be more impulsive than average or those who are more likely to act on impulsive are at increased risk for gaining weight. However, it may still be appropriate to 1) screen for ADHD in patients with obesity and 2) to look for abnormal eating behaviors in patients with ADHD, given previous findings this comorbidity in clinical populations.

Limitations

The current study had several limitations. Despite deliberating choosing to study the college student sample, results of study indicate that this decision may have ultimately been problematic. The primary problem with using a non-clinical sample is that results may be less salient than if the sample was derived from a clinical population. Previous studies finding a comorbidity between ADHD and obesity, for example, used

samples of clinically obese patients. The two other studies found looking at this relationship in an adult community sample did not replicate these results. Of the current sample, only 6.1% were currently diagnosed with ADHD and 16.3% were categorized as obese.

Despite the potential limitation of using a college freshman population, this population was necessary and deliberately chosen given the goal of examining college freshman weight gain. A primary limitation in regards to this goal was the limited amount of time between the first BF% measurement and the second BF% measurement. The average length of time between Time 1 and Time 2 measurements was only 52.29 (SD = 2.87) days which resulted from rules and procedures to recruit participants. Furthermore, the first measurements were not taken until the beginning of October, after students had been living on campus for over a month, allowing them time to gain weight before the study began. It is likely that results of the study would be affected by attaining BF% measurements closer to the beginning of the school year and creating more time between the first and second measurements.

The final primary limitation worth noting deals with methodology. Due to financial resources, a custom-made version of the Go/No-Go task was used rather than purchasing the well known and equally well validated Conner's CPT. Although the custom version was designed to be very similar to the Conner's version without violating copyright protection, it was not independently validated prior to the current study. Given that results from this measure were not related to any of the other ADHD or impulsivity measures used in the study, it is certainly possible that the design of the measure itself

was not psychometrically sound. Results may vary if an alternate method of measuring behavioral impulsivity is used.

Directions for Future Research

Given the implication and limitations of the present study, there are many possibilities and suggestions for future research. The first of these is to conduct a similar study after making minor adjustments to improve on the current study's limitations. Future studies focusing on the potential mechanisms underlying the association between ADHD and obesity should focus on clinical populations, as this is still the only population where this association is known to exist. In addition to testing pathological populations, pathological eating patterns should also be assessed. Specifically examining binge eating behaviors would be useful as this has been shown to be prevalent in ADHD populations. In order to minimize self-reporting bias, it would be beneficial to utilize more experimental methods of data collection such as examining actual food consumption and a validated version of the CPT or a Go/No-Go task.

Additionally, alternate hypotheses of the mechanisms relating ADHD and obesity should be examined. The present study found that DSM-IV Inattentive symptoms, versus hyperactive-impulsive symptoms, were positively related to maladaptive eating styles and Cortese et al. (2008) posits that being inattentive to internal signs of hunger and satiety may also lead to overeating. Additionally, recent attention has been given to the potential that obesity and ADHD are different expressions of common underlying biological mechanisms. Specific theories currently being considered include the reward deficiency syndrome, which relates to insufficient dopamine receptors, or alterations in Brain Derived Neurotropic Factor (BDNF). Optimally, the theories of impulsivity, inattention,

and potential biological mechanisms could all be tested during the same study to determine which of these is most important in understanding the relationship between these two conditions.

Ultimately, before examining the constructs linking ADHD and obesity, it is important to have these constructs clearly conceptualized and defined. Future studies looking at the relationships between the variety of measures to test for impulsivity and the relationships between self-reported impulsivity, behavioral impulsivity, and sensitivity to reward and punishment would be beneficial. Similar studies are needed to further evaluate the concepts of emotional, external, and restraint eating styles as well as their specific relationships to the consumption of food and weight gain.

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Footnotes

[1]In order to further investigate the comorbidity between ADHD and obesity, a Chi-square test of independence was conducted between participants categorized as obese using initial BF% and those who self-reported being previously diagnosed with ADHD. The difference in proportions between these two groups was not significant, $x^2(1, N = 264) = 2.03$, *ns.* Additionally, an independent samples t-test was performed utilizing the categories of obese versus non-obese as the independent variable. Non-significant differences were found when examining DSM-IV Inattentive Symptoms ($t(262) = .642$, *ns*) and DSM-IV Hyperactive-Impulsive Symptoms ($t(262) = 1.45$, *ns*) as dependent variables.

[2]However, when only looking at the current subsection of this study's sample that were categorized as obese, partial correlations found that initial BF% was not significantly related to DSM-IV Inattentive Symptoms, DSM-IV Hyperactive-Impulsive Symptoms, BIS11 Total Impulsivity, Sensitivity to Reward, or GNG Average Commission Errors after controlling for gender.

Appendix A

Pre-Screening Form

Student Identification (PIN) Number: @_____

1. Is this your first semester attending college? Yes No

2. Do you live in a university dormitory? Yes No

3. Do you currently use medical electronic implants (i.e. pacemaker) or electronic life support system (i.e. artificial heart/lung)? Yes No

Appendix B

BIS – 11

Circle the response that best describes your behavior.

1. I plan tasks carefully.	Rarely/Never	Occasionally	Often	Almost Always/Always
2. I do things without thinking.	Rarely/Never	Occasionally	Often	Almost Always/Always
3. I make up my mind quickly.	Rarely/Never	Occasionally	Often	Almost Always/Always
4. I am happy-go-lucky.	Rarely/Never	Occasionally	Often	Almost Always/Always
5. I don't "pay attention".	Rarely/Never	Occasionally	Often	Almost Always/Always
6. I have "racing" thoughts.	Rarely/Never	Occasionally	Often	Almost Always/Always
7. I plan trips well ahead of time.	Rarely/Never	Occasionally	Often	Almost Always/Always
8. I am self-controlled.	Rarely/Never	Occasionally	Often	Almost Always/Always
9. I concentrate easily.	Rarely/Never	Occasionally	Often	Almost Always/Always
10. I save regularly.	Rarely/Never	Occasionally	Often	Almost Always/Always
11. I "squirm" at plays or theatres.	Rarely/Never	Occasionally	Often	Almost Always/Always
12. I am a careful thinker.	Rarely/Never	Occasionally	Often	Almost Always/Always
13. I plan for job security.	Rarely/Never	Occasionally	Often	Almost Always/Always
14. I say things for job security.	Rarely/Never	Occasionally	Often	Almost Always/Always
15. I like to think about complex problems.	Rarely/Never	Occasionally	Often	Almost Always/Always
16. I change jobs.	Rarely/Never	Occasionally	Often	Almost Always/Always
17. I act "on impulse".	Rarely/Never	Occasionally	Often	Almost Always/Always
18. I get easily bored when solving thought problems.	Rarely/Never	Occasionally	Often	Almost Always/Always
19. I act on the spur of the moment.	Rarely/Never	Occasionally	Often	Almost Always/Always
20. I am a steady thinker.	Rarely/Never	Occasionally	Often	Almost Always/Always
21. I change residences.	Rarely/Never	Occasionally	Often	Almost Always/Always
22. I buy things on impulse.	Rarely/Never	Occasionally	Often	Almost Always/Always
23. I can only think about one problem as a time.	Rarely/Never	Occasionally	Often	Almost Always/Always
24. I change hobbies.	Rarely/Never	Occasionally	Often	Almost Always/Always

25. I spend or charge more than I earn.	Rarely/Never	Occasionally	Often	Almost Always/Always
26. I often have extraneous thoughts when I am thinking.	Rarely/Never	Occasionally	Often	Almost Always/Always
27. I am more interested in the present than the future.	Rarely/Never	Occasionally	Often	Almost Always/Always
28. I am restless at the theaters or lectures.	Rarely/Never	Occasionally	Often	Almost Always/Always
29. I like puzzles.	Rarely/Never	Occasionally	Often	Almost Always/Always
30. I am future oriented.	Rarely/Never	Occasionally	Often	Almost Always/Always

Appendix C

SPSRQ

1. Do you often refrain from doing something because you are afraid of it being illegal?	Yes	No
2. Does the good prospect of obtaining money motivate you strongly to do some things?	Yes	No
3. Do you prefer not to ask for something when you are not sure you will obtain it?	Yes	No
4. Are you frequently encouraged to act by the possibility of being valued in your work, in your studies, with your friends or with your family?	Yes	No
5. Are you often afraid of new or expected situations?	Yes	No
6. Do you often meet people that you find physically attractive?	Yes	No
7. Is it difficult for you to telephone someone you do not know?	Yes	No
8. Do you like to take some drugs because of the pleasure you get from them?	Yes	No
9. Do you often renounce your rights when you know you can avoid a quarrel with a person or an organization?	Yes	No
10. Do you often do things to be praised?	Yes	No
11. As a child, were you troubled by punishments at home or in school?	Yes	No
12. Do you like being the center of attention at a party or a social meeting?	Yes	No
13. In tasks that you are not prepared for, do you attach great importance to the possibility of failure?	Yes	No
14. Do you spend a lot of your time on obtaining a good image?	Yes	No
15. Are you easily discouraged in difficult situations?	Yes	No
16. Do you need people to show their affection for you all the time?	Yes	No
17. Are you a shy person?	Yes	No
18. When you are in a group, do you try to make your opinions the most intelligent or the funniest?	Yes	No
19. Whenever possible, do you avoid demonstrating your skills for fear of being embarrassed?	Yes	No
20. Do you often take the opportunity to pick up people you find attractive?	Yes	No
21. When you are with a group, do you have difficulties selecting a good topic to talk about?	Yes	No
22. As a child, did you do a lot of things to get people's approval?	Yes	No
23. Is it often difficult for you to fall asleep when you think about things you have done or must do?	Yes	No
24. Doe the possibility of social advancement, move you to action, even if this involves not playing fair?	Yes	No

25. Do you think a lot before complaining in a restaurant if your meal is not well prepared?	Yes	No
26. Do you generally give preference to those activities that imply an immediate gain?	Yes	No
27. Would you be bothered if you had to return to a store when you noticed you were given the wrong change?	Yes	No
28. Do you often have trouble resisting the temptation of doing forbidden things?	Yes	No
29. Whenever you can, do you avoid going to unknown places?	Yes	No
30. Do you like to compete and do everything you can to win?	Yes	No
31. Are you often worried by things that you said or did?	Yes	No
32. Is it easy for you to associate tastes and smells to very pleasant events?	Yes	No
33. Would it be difficult for you to ask your boss for a raise (salary increase)?	Yes	No
34. Are there a large number of objects or sensations that remind you of pleasant events?	Yes	No
35. Do you generally try to avoid speaking in public?	Yes	No
36. When you start to play with a slot machine, is it often difficult for you to stop?	Yes	No
37. Do you, on a regular basis, think that you could do more thinks if it was not for your insecurity or fear?	Yes	No
38. Do you sometimes do things for quick gains?	Yes	No
39. Comparing yourself to people you know, are you afraid of many things?	Yes	No
40. Does you attention easily stray from your work in the presence of an attractive stranger?	Yes	No
41. Do you often find yourself worrying about things to the extent that performance in intellectual abilities is impaired?	Yes	No
42. Are you interested in money to the point of being able to do risky jobs?	Yes	No
43. Do you often refrain from doing something you like in order not to be rejected or disapproved of by others?	Yes	No
44. Do you like to put competitive ingredients in all of your activities?	Yes	No
45. Generally, do you pay more attention to threats than to pleasant events?	Yes	No
46. Would you like to be a socially powerful person?	Yes	No
47. Do you often refrain from doing something because of your fear of being embarrassed?	Yes	No
48. Do you like displaying your physical abilities even though this may involve danger?	Yes	No

Appendix D

DEBQ

Please use the following scoring key to answering the following questions:
Never (1) Seldom (2) Sometimes (3) Often (4) Very Often (5) Not Relevant (NA)

1. If you have put on weight, do you eat less than you usually do?	1 2 3 4 5 NA
2. Doe you try to eat less at mealtimes than you would like to eat?	1 2 3 4 5
3. How often do you refuse food or drink offered because you are concerned about your weight?	1 2 3 4 5
4. Do you watch exactly what you eat?	1 2 3 4 5
5. Do you deliberately eat foods that are slimming?	1 2 3 4 5
6. When you have eaten too much, do you eat less than usual the following days?	1 2 3 4 5 NA
7. Do you deliberately eat less in order not to become heavier?	1 2 3 4 5
8. How often do you try not to eat between meals because you are watching your weight?	1 2 3 4 5
9. How often in the evening to you try not to eat because you are watching your weight?	1 2 3 4 5
10. Do you take into account your weight with what you eat?	1 2 3 4 5
11. Do you have desire to eat when you are irritated?	1 2 3 4 5 NA
12. Do you have a desire to eat when you have nothing to do?	1 2 3 4 5 NA
13. Do you have a desire to eat when you are depressed or discouraged?	1 2 3 4 5 NA
14. Do you have a d desire to eat when you are feeling lonely?	1 2 3 4 5 NA
15. Do you have desire to eat when somebody lets you down?	1 2 3 4 5 NA
16. Do you have desire to eat when you are cross?	1 2 3 4 5 NA
17. Do you have a desire to eat when you are anticipating something unpleasant to happen?	1 2 3 4 5
18. Do you get the desire to eat when you are anxious, worried, or tense?	1 2 3 4 5
19. Do you have desire to eat when things are going against you or when things have gone wrong?	1 2 3 4 5
20. Do you have a desire to eat when you are frightened?	1 2 3 4 5
21. Do you have desire to eat when you are disappointed?	1 2 3 4 5 NA
22. Do you have desire to eat when you are emotionally upset?	1 2 3 4 5 NA
23. Do you have a desire to eat when you bored or restless?	1 2 3 4 5 NA

24. If food tastes good to you, do you eat more than usual?	1 2 3 4 5
25. If food smells and looks good, do you eat more than usual?	1 2 3 4 5
26. If you see or smell something delicious, do you have a desire to eat it?	1 2 3 4 5
27. If you have something delicious to eat, do you it straight away?	1 2 3 4 5
28. If you walk past the baker do you have the desire to buy something delicious?	1 2 3 4 5
29. If you walk past a snack-bar or a café, do you have the desire to buy something delicious?	1 2 3 4 5
30. If you see others eating, do you also have the desire to eat?	1 2 3 4 5
31. Can you resist eating delicious foods?	1 2 3 4 5
32. Do you eat more than usual when you see others eating?	1 2 3 4 5
33. When preparing a meal, are you inclined to eat something?	1 2 3 4 5

Appendix E

Survey of College Health Behaviors

1. Gender (circle one): Male Female Other

2. Age: _____

3. For your ***biological mother***, provide your best estimate on which weight class she is in:

a. under-weight
b. normal weight
c. over weight
d. very over weight/obese
e. unknown/not applicable

3. For your ***biological father,*** provide your best estimate on which weight class he is in:

a. under-weight
b. normal weight
c. over-weight
d. very over weight/obese
e. unknown/not applicable

4. Have you ever been diagnosed with Attention Deficit Disorder or Attention Deficit Hyperactivity Disorder (ADD or ADHD)? yes no

5. Are you currently taking medication for ADD or ADHD?

Yes No

If yes, what is the name of your medication _____.

Please answer the following question based on your regular activities and lifestyle from the current semester at IUP.

1. How many hours of *consecutive* sleep do you get at night during the weekdays? _____
2. How many times do you exercise in a given week? _____
3. How many snacks do you consume <u>after</u> dinner in a day? _____
4. How many meals per week are followed by 'dessert'? _____

5. How many times per week do you eat ‘junk food’? _____

6. Do you purposefully engage in dieting activity or making an effort to lose weight?

   Yes / No

7. Please fill in the following chart based on how many times per week you ate at the following locations on average:

Location	**Number of times eaten here per week**
In your dormitory (room, kitchen, etc.)	
At an all-you-can-eat dining hall	
At a pay-per-item dining hall/food court	
At an off-campus restaurant	
Other: (please specify)	

Appendix F

Informed Consent Form

You are invited to participate in this research study. The following information is provided in order to help you to make an informed decision whether or not to participate. If you have any questions please do not hesitate to ask. You are eligible to participate because you are a first semester, freshman student in PSYC 101 General Psychology at Indiana University of Pennsylvania (IUP) who is currently living on-campus.

The purpose of this study is to examine the relationship between several personality characteristics, eating behaviors, and weight during students' first semester at college. You will be asked to complete several questionnaires and a computerized task today. Additionally, your body mass index and body fat percentage will be measured using a common electronic device utilizing bioelectric impedance technology. The study will conducted at two separate time points during the semester. In addition to participating in this study for approximately one hour today, you will be asked to sign up for and return to participate in the second half of the study in a later point in the semester for approximately one additional hour. There are no known risks associated with participation in the study. However, some of the questions do ask about information of a personal nature such as previous diagnosis and symptoms of Attention Deficit/Hyperactivity disorder and your current health practices such as eating and exercise behaviors.

Participation in this study will require approximately two hours of your time total, during two separate time points, and is not considered a part of PSYC 101. Participation or non-participation will not effect the evaluation of your performance in this class. However, by participating in both parts of this study, you will earn 2 credits towards your subject pool requirement for this course.

Your participation in this study is voluntary. You are free to decide not to participate in this study or to withdraw at any time without adversely affecting your relationship with the investigators or IUP. Your decision will not result in any loss of benefits to which you are otherwise entitled. If you choose to participate, you may withdraw at any time by notifying the Project Director or informing the person administering the questionnaires. Upon your request to withdraw, all information pertaining to you will be destroyed.

If you choose to participate, all information will be held in strict confidence and will have no bearing on your academic standing or services you receive from the University. Your responses will be considered only in combination with those from other participants. Collected data will be retained for a minimum of three years in compliance with federal regulations. The data collected during this study may be published in psychological research journals or presented at conferences. As a participant, if you are interested, you are entitled to a meeting with the Principal Investigator to discuss the results of the study once all of the data have been collected. Contact information is provided below.

If you are willing to participate in this study, please sign the statement below and return it to the experimenter. Take the extra unsigned copy with you. If you choose not to participate, deposit the unsigned copies to the experimenter.

For further information about this study or to request a meeting with the Principal Investigator to learn the results of the study, please contact:

Principal Investigator: Katherine Ratcliff
Graduate Student
Psychology Department
220 Uhler Hall
Indiana, PA 15705
k.l.ratcliff@iup.edu
(724) 357-6227

Faculty Sponsor: Donald U. Roberston, Ph.D.
Professor of Psychology
Psychology Department
222 Uhler Hall
Indiana, PA 15705
Donald.Robertson@iup.edu
(724) 357-4522

This project has been approved by the Indiana University of Pennsylvania Institutional Review Board for the Protection of Human Subjects (Phone: 724/357-2223).

Informed Consent Form (continued)

VOLUNTARY CONSENT FORM

I have read or have had read to me the information contained on the informed consent form. Any questions that I have regarding the study have been answered by the principal investigator or one of his assistants. I have been told of the risks or discomforts and possible benefits of the study. I understand my participation is voluntary and that participation includes completing the questionnaires and tasks presented today as well as being weighed at the beginning and the end of the semester. I understand that my refusal to participate will involve no penalty or loss of rights to which I am entitled. I may withdraw from participation at any time without penalty. I also understand that the results of this study may be published, but my individual scores and responses are used only in combination with those from other participants. I have received an unsigned copy of the informed consent form to keep in my possession.

I understand my rights as a research participant and I voluntarily consent to participate in this study. I understand what the study is about and how and why it is being done.

_____ _____

Participant's Signature Date

_____

Participant's Name (Print)

I certify that I have explained to the above individual the nature and purpose, the potential benefits, and possible risks associated with participating in this research study, have answered any questions that have been raised, and have witnessed the above signature.

_____ _____

Investigator's Signature Date

Appendix G

Debriefing Form

1. Rationale for the current study. The current study is an examination of the relationship between Attention Deficit/Hyperactivity Disorder (ADHD) and obesity. It is designed to investigate the ways that symptoms of ADHD, such as impulsivity, can affect maladaptive eating behaviors and weight gain. Previous research had found that ADHD and obesity are linked, but researchers have not studies why this might be the case. In order to study this, the present study will measure eating behaviors and ADHD symptoms via survey method as well as a computerized task. Body Mass Index and Body Fat Percentage will also be collected at the beginning an the end of the semester and statistical analysis will examine the relationships among these variables. The results of this study may result in a better understanding of the relationship between these two conditions and later research may examine how this relationship will affect treatment and risk factors for both conditions. It is expected that certain ADHD symptoms, such as impulsivity, will result in increased poor eating choices, which will result in increased body mass and body fat.

2. Obtaining results of this study. As a participant in this study, you are entitled to a meeting with the Principal Investigator once all of the data have been collected. You may also contact the Principal Investigator to obtain results of the study, even if you do not desire a meeting. To schedule a meeting or to obtain a copy of the results you can contact Katherine Ratcliff at (724) 357-6227 anytime after March 1, 2008.

Thank you for your participation in this study.

Sincerely,

Katherine Ratcliff, M.A.
Doctoral Candidate
Department of Psychology
Indiana University of Pennsylvania
Indiana, PA 15705

Appendix H

Table H1

Descriptive Statistics for Demographic Variables

Variable	Response	Frequency	Valid Percent
Gender	Male	100	37.9%
	Female	164	62.1%
Age	17-years	1	.4%
	18-years	185	70.1%
	19-years	73	27.7
	20+ years	5	2%
ADHD Diagnosis	Yes	16	6.1%
	No	248	93.9%
ADHD Medication	Yes	4	1.5%
	No	260	98.6%
Obese*	Yes	43	16.3%
	No	221	83.7%
Dieting	Yes	81	30.7%
	No	177	67.0%
Biological Mother	Under-weight	17	6.4%
	Normal Weight	163	61.7%
	Over-weight	76	28.8%
	Very over-weight/obese	3	1.1%
	Unknown/not applicable	5	1.9%
Biological Father	Under-weight	8	3.0%
	Normal Weight	157	59.5%
	Over-weight	74	28.0%
	Very over-weight/obese	10	3.8%
	Unknown/not applicable	15	5.7%

Note: Valid percentages may not add up to 100% due to invalid/missing responses.
*Obese is defined as an initial BF% >32% in females and >25% in males.

Table H2

Descriptive Statistics and Correlations of Reported Health Behaviors to BF%

Measure	Mean (SD)	*r(Initial BF%)*	*r (BF% Change)*
Consecutive Hours Slept per Night 7 hours (median)	6.64 (1.10)	.04	-.123*
No. of Times Exercised Per Week	1.55 (1.35)	-.15*	.01
No. of After-Dinner-Snacks Consumed Per Week	1.54 (1.01)	-.29**	-.08
No. of Desserts Consumed Per Week	2.41 (2.31)	.04	.002
No. of Junk Foods Consumed Per Week	4.9 (3.02)	-.10	-.003
Locations Eaten:			
No. of times eaten in Dorm Room	4.64 (3.31)	.001	-.001
No. of times Eaten in All-you-can-eat dining facility	6.64 (4.17)	-.07	.059
No. of Times Eaten in pay-per-item dining facility	2.87 (2.83)	-.23**	.001
No. of times eaten off campus	.91 (1.26)	-.25**	.06
No. of times Eaten "other" locations	.25 (1.03)	-.01	.10

Note: Participants were asked to estimate an average amount of the above health behaviors.
*p<.05; **$p < .01$

Table H3

Descriptive Statistics and Analyses for Variables of Interest

Measure	Sex	Mean (SD)	*t* (262)
Initial BF%	Male	17.48 (8.42)	
	Female	25.27 (6.38)	-7.96**
BF% Change	Male	.80 (2.71)	
	Female	.12 (2.58)	2.03**
Restrained Eating Score	Male	1.84(.81)	
	Female	2.51(.89)	-6.30**
Emotional Eating Score	Male	1.90(.87)	
	Female	2.55(.84)	-5.85**
External Eating Score	Male	2.94(.60)	
	Female	2.97(.62)	-.27
DSM-IV Inattentive Symptoms	Male	1.01(.54)	
	Female	1.00(.56)	.120
DSM-IV Hyperactive-Impulsive Symptoms	Male	.87(.45)	
	Female	1.01(.47)	-2.37*
Sensitivity to Reward	Male	.57(.18)	
	Female	.48(.18)	3.73**
Sensitivity to Punishment	Male	.43(.21)	
	Female	.49(.22)	-1.88
BIS-11 Total Impulsivity	Male	6.61(1.23)	
	Female	6.30(.90)	2.40*
GNG Avg. Omission	Male	.97(.11)	
	Female	.98(.07)	.01
GNG Avg. Commission	Male	.72(.17)	
	Female	.73(.15)	.13

*p<.05 **p < .01

Table H4

Partial Correlations

	2	3	4	5	6	7	8	9	10	11	12	13
1. Initial BF%	-.20**	.28**	.08	-.13	-.04	-.06	-.06	-.15	-.02	-.17	-.03	.01
2. BF% Change		.05	.03	.03	.02	.08	.06	.10	.01	.04	.00	.00
3. Restrained Eating Score			.18**	.05	-.04	.05	.00	.14	.12	-.06	.12	.00
4. Emotional Eating Score				.42**	.34**	.23**	.32**	.16	.25**	.19**	.16	-.03
5. External Eating Score					.34**	.22**	.32**	.20**	.17	.24**	.15	.04
6. DSM-IV Inattentive Symptoms						.62**	.92**	.24**	.20**	.53**	.09	.03
7. DSM-IV Hyperactive-Impulsive Symptoms							.88**	.31**	-.06	.42**	.08	.04
8. DSM-IV ADHD Total Symptoms								.30**	.09	.54**	.09	.04
9. Sensitivity to Reward									-.07	.16**	.05	.00
10. Sensitivity to Punishment										.07	.08	.00
11. BIS11 Total Impulsivity											.02	-.07
12. GNG Avg. Omission												-.01
13. GNGAvg.Commission												

Note: The Restrained Eating Score, Emotional Eating Score, and External Eating Score are components of the Dutch Eating Behaviour Questionnaire (DEBQ). DSM-IV Inattentive Symptoms, DSM-IV Hyperactive Symptoms, and DSM-IV ADHD Total Symptoms are subscales from the Conner's Adult ADHD Rating Scale – Self Report: Long Version (CAARS-S:L). Sensitivity to Reward and Sensitivity to Punishment are the two subscales in the Sensitivity to Reward/Sensitivity to Punishment Questionnaire (SPSRQ). BIS-11 Total Impulsivity is the total scale of the Barratt Impulsivity Scale – Version 11. GNG Avg. Omission is the average omission errors made on the Go/No-Go task and GNG Avg. Commission is the average commission errors made on the Go/No-Go task.

** $p < .01$

Table H5

Hierarchical Regression Analyses to Predict Initial Body Fat Percentage

Step and Predictor Variables	β	t	R	R^2	$F(df)$	R^2 change	F change
Regression Analysis 1							
Step 1: Gender	.47	8.50*	.47	.21	72.2 (1, 262)**		
Step 2: ADHD Symptomology			.47	.22	24.3 (3, 260)**	.003	.55
DSM-IV Hyperactive-Impulsive	-.002	-.80					
DSM-IV Inattentive	-.057	-.03					
Regression Analysis 2							
Step 1: Gender	.47	8.50**	.47	.21	72.2 (1, 262)**		
Step 2: Impulsivity Scores	.		.50	.25	21.58 (4, 259)**	.034	3.9**
BIS-11 Total Score	-.13	-2.37*					
Sensitivity to Reward	-.12	-2.05*					
GNG Avg. Commission Errors	.000	-.001					
Regression Analysis 3							
Step 1: Gender	.47	8.50**	.47	.21	72.2 (1, 262)**		
Step 2: DEBQ Scores .			.55	.30	21.58 (4, 259)**	.085	10.5**
Restrained Eating Score	.257	4.55**					
Emotional Eating Score	.097	1.57					
External Eating Score	-.164	-2.87**					

Note: Beta coefficients are reported in standardized values.
*p<.05; **$p < .01$.

Table H6

Hierarchical Regression Analyses Using Gender and ADHD Symptoms to Predict DEBQ Scores

Step and Predictor Variables	β	t	R	R^2	$F(df)$	R^2 change	F change
Regression Analysis 1: Predicting Restrained Eating Scores							
Step 1: Gender	.356	6.17**	.35	.13	38.01 (1, 262)**		
Step 2: ADHD Symptomology .			.37	.14	13.67 (3, 260)**	.009	1.42
DSM-IV Inattentive	.122	-1.46					
DSM-IV Hyperactive-Impulsive	.146	1.56					
Regression Analysis 2: Predicting Emotional Eating Scores							
Step 1: Gender	.343	5.90**	.34	.12	34.84 (1, 262)**		
Step 2: ADHD Symptomology .			.47	.22	13.67 (3, 260)**	.102	16.9**
DSM-IV Inattentive	.295	4.22**					
DSM-IV Hyperactive-Impulsive	.038	.532					
Regression Analysis 3: Predicting External Eating Scores							
Step 1: Gender	.016	.264	.01	.00	.07 (1, 262)		
Step 2: ADHD Symptomology .			.34	.12	11.63 (3, 260)**	.118	17.4**
DSM-IV Inattentive	.336	4.52**					
DSM-IV Hyperactive-Impulsive	.164	.870					

Note: Beta coefficients are reported in standardized values.
*p<.05; **p < .01.

Table H7

Hierarchical Regression Analysis Using Gender and Impulsivity Measures to Predict DEBQ Scores

Step and Predictor Variables	β	t	R	R^2	$F(df)$	R^2 change	F change
Regression Analysis 1: Predicting Restrained Eating Scores							
Step 1: Gender	.356	6.17**	.35	.13	38.01 (1, 262)**		
Step 2: Impulsivity Scores .			.39	.15	11.52 (3, 260)**	.024	2.46
Sensitivity to Reward	.291	2 Table 5: *Hierarchical Regression Analysis Using Gender and Impulsivity*					
Measures to Predict DEBQ Scores.51							
BIS-11 Total Score	-.083	-1.42					
GNG Commission Errors	-.010	-.171					
Regression Analysis 2: Predicting Emotional Eating Scores							
Step 1: Gender	.343	5.90**	.34	.12	34.84 (1, 262)**		
Step 2: Impulsivity Scores .			.41	.16	12.96 (3, 260)**	.049	5.12**
Sensitivity to Reward	.132	2.24*					
BIS-11 Total Score	.162	2.78**					
GNG Commission Errors	-.017	-.300					
Regression Analysis 3: Predicting External Eating Scores							
Step 1: Gender	.016	.264	.01	.00	.07 (1, 262)		
Step 2: Impulsivity Scores .			.30	.09	6.19 (3, 260)**	.087	8.29**
Sensitivity to Reward	.175	2.83**					
BIS-11 Total Score	.215	2.53**					
GNG Commission Errors	.050	.402					

Note: Beta coefficients are reported in standardized values. *p<.05; **$p < .01$.