PATHWAYS FROM ADHD SYMPTOMS TO OBESITY IN A COLLEGE POPULATION

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Attention deficit hyperactivity disorder (ADHD) is more recently being recognized as a lifetime disorder that continues to affect individuals into their adult lives. Recent research studies have found connections between ADHD and overweight/obesity. The current study was designed to further explore these relationships and better understand the connections between these two constructs among 340 college students. It was hypothesized that the ADHD symptoms (i.e., inattention and impulsivity) would positively predict depressive symptoms, which in turn would predict emotional/binge eating and lead to overweight/obesity. Additionally, it was hypothesized that impulsivity would predict substance use, which would predict emotional/binge eating and also predict overweight/obesity. The model was tested and exhibited excellent fit. ADHD positively predicted depressive symptoms, which in turn positively predicted emotional/binge eating and led to overweight/obesity. Further, ADHD symptoms also positively predicted substance use, which in turn predicted emotional/binge eating and led to overweight/obesity. All paths were statistically significant and findings suggest there are at least two paths that connect ADHD symptoms and overweight/obesity in adults. The current results are of importance to practicing clinicians because they provide increased clarity and depth regarding the connections and relationship between symptoms of ADHD and overweight/obesity.

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CHAPTER 1

INTRODUCTION

Recent advances in the neuroscience of human behavior have given researchers the opportunity to explore strong links and common factors between attention deficit hyperactivity disorder (ADHD) and obesity in adults (Davis, Levitan, Smith, Tweed, & Curtis, 2006; Davis et al., 2009; Pagoto, 2008). To date, there are a few hypotheses for this association. Possible explanations include: decision-making difficulties, reward-punishment deficits, and decreased levels of or decreased receptivity for certain neurotransmitters (Barry, Clarke, & Petry, 2009; Blum, Sheridan, Wood, Braverman, Chen, & Comings, 1995; Blum et al., 2008; Davis et al., 2006). Recent studies using structural equation modeling (SEM) have found links between ADHD, aspects of overeating, and increased BMI (Davis et al., 2006; Johnson, 2011; see Figures 1 and 2, respectively). The current study aims to advance this line of research by including additional theoretically supported potential mediators and moderators such as symptoms of depression and substance use (see Figure 3). The structural model in the current study was compared to Johnson's (2011) initial structural model to determine which model is a better explanation of the pathways between ADHD and obesity.

There are strong associations between ADHD symptoms, depressive symptoms, substance abuse/dependency, and obesity in adults (Davis, Levitan, Smith, Tweed, & Curtis, 2006; Davis et al., 2009; Ohlmeier et al., 2007; Pagoto, 2008). ADHD individuals, individuals with substance dependency, individuals with depression, and overweight/obese individuals share many common factors, including: decision-making difficulties, reward deficiencies and neurotransmitter deficits. Specifically, Davis and colleagues (2006) suggest that when making decisions to guide behavior, individuals with ADHD are more likely to respond to immediate

sensory stimuli than to engage in complex processing, a quality also present in overweight and obese women (Davis et al., 2006) and substance dependent individuals (Bechara et al., 2002). Similarities in the reward cascade center in the brain have been noted between ADHD individuals, substance dependence individuals, individuals with depression, and overweight/obese individuals (Blum et al., 2008; Blum & Noble, 2001). Results suggest that these individuals have decreased amounts of dopamine receptor sites, resulting in decreased amounts of dopamine in the brain. Due to this deficiency, individuals with ADHD symptoms, substance abuse/dependence, and overweight are at a high risk to engage in compulsive, impulsive, and addictive behaviors in an effort to increase the dopamine concentration in their brains (Blum et al., 2008; Barry et al., 2009).

When comparing adults diagnosed with ADHD to non-ADHD controls in other studies, findings suggested those diagnosed with ADHD engage in more risky behaviors, including substance abuse, and experience higher rates of relational difficulties (Mccann & Roy-Byrne, 2004; Murphy & Barkley, 1996). Moreover, they exhibit lower levels of job performance, as well as low levels of motivation (which might contribute to depressive symptoms) than adults without ADHD (Goldman, Genel, Bezman, & Slanetz, 1998; McCann & Roy-Byrne, 2004).

ADHD is conceptualized as a "lifespan disorder" (McGough & Barkley, 2004; p. 1948) in which those with ADHD continue to struggle with symptoms, as well as associated features of the disorder into their adult years, especially if they have never been formerly diagnosed or treated for their condition. A cross-sectional study included 752 adults that were separated into three categories: 1) reported a history of and current symptoms that met criteria for ADHD but had never been diagnosed or treated for that disorder ("undiagnosed ADHD"), 2) adults with ADHD who had been previously diagnosed with or treated for ADHD ("diagnosed ADHD

comparison group"), and 3) a cohort-matched comparison group that never met diagnostic criteria for ADHD. A variety of differences emerged between the groups. The undiagnosed ADHD group experience worse outcomes when compared to the other two groups in the study. For example, the undiagnosed ADHD group were more likely to be unemployed and struggle to hold full-time jobs than adults in the comparison groups (Able, Johnston, Adler, & Swindle, 2007). Moreover, those individuals in the undiagnosed ADHD group reported experiencing higher levels of anxiety and mood lability, as well as lower levels of quality of life and self-esteem than members of the comparison groups. Further, when compared to the diagnosed ADHD group and the cohort-matched comparison group, adults with undiagnosed ADHD reported higher levels of current depression, as well as being more likely to consume alcohol as a means of coping (Able et al., 2007). When adults with ADHD were compared to a control group, those diagnosed with ADHD had a greater prevalence of substance use disorders, as well as reported greater use of illegal substances (Murphy & Barkley, 1996)

Even when adults have been diagnosed with ADHD and treated with medication, they may continue to struggle with distressing symptoms associated with the disorder. For example, in another cross-sectional study that included 105 adults who were diagnosed with ADHD and treated with medication, researchers attempted to assess which domains of life impairments were most affected by ADHD (Safren et al., 2010). Researchers described life impairments as including four different domains: work, interpersonal relationships, recreation, and global satisfaction. Findings suggested that work and interpersonal relationship impairments were particularly associated with ADHD severity (Safren et al., 2010). As these studies suggest, symptoms of ADHD can negatively affect an individual's work and home life, which might lead

them to self-medicate with substances or food (i.e., emotional/binge eating) in an effort to decrease negative affect and improve mood.

ADHD Diagnostic Difficulties

It can be complicated for clinicians to recognize these potentially debilitating symptoms and diagnose adults with ADHD. One diagnostic complication is that some of the symptoms included in the ADHD diagnostic criteria can also occur during the course of other disorders (Shaffer, 1994), and can be mistaken for ADHD. For example, mood swings, restlessness, impaired concentration, memory deficits, and irritability are all symptoms that also are indicative of major depressive disorder (Adler, 2004; Kessler et al., 2006). Additionally, even when properly diagnosed, ADHD is a disorder that is oftentimes simultaneously experienced with other psychiatric disorders (i.e., comorbidity), such as major depressive disorder, substance use disorders, and antisocial personality disorder (Shaffer, 1994; Triolo, 1999). Due to these complications, it is imperative and necessary for clinicians to be cognizant of the many shared symptoms of various disorders, in order to effectively understand that the presence of a particular symptom does not clearly denote a specific disorder or diagnosis.

ADHD and Addictive Behavior

An extensive body of literature explores the connections between ADHD-like symptoms, ADHD diagnosis, and addictive behaviors (see Ohlmeier et al., 2007 for review). Many different hypotheses attempt to explain and explore high rates of addiction among individuals with ADHD. The three most prominent explanations are: disinhibition, attempts to cope with the stressors related to an ADHD diagnosis, and that addictive behaviors, such as self-medicating with substances, may modify neurochemistry in ways that offset some of the neurotransmitter anomalies of ADHD.

ADHD and substance dependency are highly co-morbid. In a six-month longitudinal study of 91 male and female adults, 23.1% of the alcohol-dependent participants met the *Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition - Text Revision (DSM-IV –TR)* criteria for the presence of ADHD, which is about four times the prevalence rate of ADHD when compared to the general population (Ohlmeier et al., 2007). Researchers found that a high amount of the alcohol-dependent participants met the *DSM-IV-TR* criteria for ADHD. Moreover, those participants with comorbid alcohol-dependence and ADHD had higher nicotine use (76.2%) when compared to alcohol-dependent participants without ADHD (45.7%). These differences suggest that nicotine might be serving as a form of self-medication for those also diagnosed with ADHD.

Those with ADHD are at a particularly high risk for nicotine dependence, as findings suggest that individuals with ADHD start smoking at an earlier age when compared to non-ADHD control groups (Lambert & Hartsough, 1998; Milberger et al., 1997). When comparing the general population to the adult ADHD population, findings indicate that rates of nicotine use in the adult ADHD population is double that of the general population (Levin & Razvani, 2002; Newcorn, 2008). Additionally, findings suggest that when compared to non-ADHD smokers, individuals with ADHD experience greater irritability, more difficulty concentrating, and decreased response inhibition upon withdrawal from nicotine, indicating that when individuals with ADHD stop smoking, some of their ADHD symptoms are exacerbated (McClernon et al., 2008; Pomerleau et al., 2003).

Individuals with ADHD and those who abuse substances share traits such as disinhibition and impulsivity that increase risk for engaging in potentially dangerous behaviors that offer short-term rewards. Moreover, there is likely a higher risk for substance abuse when individuals

are experiencing low inhibition and impulse control, both of which as core symptoms of ADHD. Thus, it is not difficult to understand why individuals with ADHD are more prone to addiction and addictive behaviors (Dinn et al., 2004; Kalbag & Levin, 2005; Mason et al., 2007; Wilson, 2007).

Adults diagnosed with ADHD or those with ADHD-like symptoms experience a variety of secondary stressors (Able, Johnston, Adler, & Swindle, 2007; Wadsworth & Harper, 2007) from which many individuals seek to escape. For example, impulsivity increases the risk of interpersonal conflict (Ratey, Greenberg, Bemporad, & Lindem, 1992) along with organizational problems, forgetting needed items, and getting traffic tickets (Riccio et al., 2005). When compared to adults that are not diagnosed with ADHD, adults with ADHD are more likely than to experience job loss (Able et al., 2007), divorce (Mccann & Roy-Byrne, 2004; Murphy & Barkley, 1996), and driving-related accidents (Murphy & Barkley, 1996). Ohlmeier et al. (2007) suggest that substance use and abuse may be used as a method of self-soothing. Individuals may use and abuse substances in an attempt to better cope or escape the negative effects related to ADHD symptomology (Dinn et al., 2004; Spencer, Biederman, & Mick, 2007).

In addition to using substances to cope with stress, some researchers suggest that adolescents and adults with ADHD use substances, including nicotine, to self-medicate, that is, using substances to alleviate negative symptoms and associated distress of ADHD (Dinn et al., 2004; Mason et al., 2007). Researchers propose that the serotonergic and dopaminergic neurotransmitter systems interact to contribute to the dysfunction experienced by those diagnosed with ADHD (Quist & Kennedy, 2001). These two neurotransmitter systems need to sustain balance in order to maintain more adaptive behaviors in people with ADHD. If there is a higher concentration of serotonin versus dopamine in the reward center of the brain (or vice

versa), one possible result is maladaptive levels of inattention, hyperactivity, and impulsivity (Quist & Kennedy, 2001). Ingestion of nicotine, alcohol, and some other drugs, as well as binge eating on sugary/high-fat foods can temporarily alter concentrations of these neurotransmitters (Davis et al., 2004; McClernon et al., 2008; Pomerleau et al., 2003), leading to a temporary reduction of the aversive symptoms of ADHD (i.e., self-medication; Gehricke, Whalen, Jamner, Wigal, & Steinhoff, 2006; Lambert, 2005; Levin, Conners, Silva, Canu, & March, 2001).

ADHD and Neurobiological Effects

Neurotransmitters that primarily make individuals "feel" better such as serotonin, dopamine, and norepinephrine are related to ADHD, substance dependence, and obesity (Blum et al., 1995). ADHD is characterized as a dopamine-dysfunction disorder and is associated with the reward deficiency syndrome (RDS) (Blum & Noble, 2001). RDS is characterized by an insufficient number of receptors in the mesolimbic pathway of the brain, which is known to be involved in modulating behavioral responses to stimuli that activate feelings of reward through the neurotransmitters dopamine and serotonin. Individuals who have ADHD, a history of substance abuse, and more recently, those who overeat (e.g., binge eating, emotional eating) have a reduced ability to produce these rewarding neurochemicals, contributing to RDS. This under-activation of the reward center in the brain means that self-medicating behaviors that increase dopamine and serotonin also increase mood, motivation, attention, and even produce a sense of calmness.

RDS may be associated with the neurotransmitter anomalies commonly tied to ADHD, drug and alcohol use, and some types of disordered eating (e.g., emotional/binge eating) (Blum et al., 1995). Many abused substances are stimulants (i.e., nicotine, cocaine, amphetamines) that elevate mood and, potentially, increase attention and concentration by altering neurotransmitters

such as norepinephrine, serotonin, and dopamine. The dopamine D2 receptor gene (DRD2) is the main neurotransmitter associated with addictive and compulsive behaviors, such as substance abuse and dependency, pathological gambling, ADHD symptoms, and disordered eating (e.g., compulsive or binge eating) that may lead to becoming overweight (Blum et al., 1995). RDS is characterized by insufficient DRD2 receptors and engaging in compulsive rewarding behaviors (i.e., drug use and overeating; Barry et al., 2009; Blum et al., 2008). Studies that focused on individuals with chronic drug and alcohol use suggested a reduced availability of dopamine D2 receptors in their brains, specifically in the mesolimbic pathway (a reward pathway in the brain). With extended drug or alcohol abuse, the number of dopamine D2 receptors decreases, further causing individuals to take in higher doses of drugs and alcohol in order to experience the rewarding release of dopamine in the brain (Barry et al., 2009; Volkow & Fowler, 2000). Individuals that struggle with addictive and/or compulsive behaviors (e.g., substance abuse and disordered eating) might have genetic defects associated with dopamine D2 receptors (Blum et al., 1995; Wang et al., 2001). Specifically, those individuals may be over-engaging in certain behaviors (e.g., nicotine, alcohol, drug use, and compulsive/binge eating) in order to increase the dopamine levels in their brains (Wang et al., 2001).

These studies (Barry et al., 2009; Cortese, Bernardo, & Mouren, 2007) suggest that individuals with ADHD may be at a higher risk to engage in addictive behaviors and become addicted due to the genetic differences in their D2 receptors compared to non-ADHD individuals. Researchers have recently identified and explored possible connections between ADHD and obesity. In particular, binge eating has been proposed as a type of addictive behavior that might explain the link between those two constructs (i.e., ADHD and obesity; Barry et al., 2009; Cortese, Bernardo, & Mouren, 2007). The three explanations associated with high rates of

addiction among people with ADHD (i.e., disinhibition, attempts to cope with the stressors associated with having ADHD, and that addictive behaviors may alter neurochemistry in ways that offset neurotransmitter anomalies of ADHD) can also be applied to eating behaviors.

Eating, particularly binge eating, may involve disinhibition or may be used as a form of coping (Barry et al., 2009). Emotional eating, eating in an attempt to cope with aversive emotions, can include the intake of high glucose and fat content for self-soothing (Goossens, Braet, Van Vlierberghe & Mels, 2009). Emotional eating, along with binge eating, may be an effective temporary solution, or form of self-medication, as both can temporarily decrease negative affect by increasing the concentration of serotonin and dopamine in the brain. Similar to the hypothesis that RDS plays a role in the relationship between ADHD and addiction, RDS is also hypothesized to play a role between ADHD and binge eating.

Several researchers (Gold, Frost-Pineda, & Jacobs, 2003; Hernandez & Hoebel, 1990) have postulated that consuming large amounts of food, notably food high in fat and/or sugar, results in an increase in the amounts of dopamine released from the presynaptic terminals in the brain, which in turn, leads to higher concentrations of dopamine in the synaptic gaps in the brain (Gold, Frost-Pineda & Jacobs, 2003). Individuals who experience this dopamine deficiency (e.g., individuals diagnosed with ADHD) are compelled to partake in activities that will increase transmission of dopamine, one example being disordered eating (i.e., binge eating and emotional eating). Similar to substance use/abuse, it could be hypothesized that ADHD individuals may use food as a means to "self-medicate" the low dopaminergic state associated with the disorder.

ADHD and Obesity

Over the past 30 years, the amount of individuals who were classified as overweight and obese has significantly increased. The amount of children (ages 6-11 years) classified as obese

has doubled. Similarly, the percentage of adolescents (ages 12-19 years) classified as obese has tripled (Centers for Disease Control and Prevention, 2010). In 2010, 68.0% (i.e., males 72.3% and females 64.1%) of adults (ages 20 and above) classified as overweight while 33.8% (i.e., males 32.2% and females 35.5%) classified as obese (Flegal, Carroll, Ogden, & Curtin, 2010).

Children and adolescents with high symptoms of ADHD appear to be at an increased risk for obesity. For example, in a study of adolescents (ages 11-17), the prevalence of an ADHD diagnosis was statistically significantly higher for overweight and obese adolescents (i.e., 7%) when compared to adolescents of normal weight (3.5%) and adolescents who were underweight (4.9%) (Erhart et al., 2012). Additionally, Cortese et al. (2008) conducted a meta-analysis on the links between ADHD and overweight/obesity among each age group. They included 15 studies in their review, 13 of the 15 studies included participants under age 18. Each study reported, based on individual's current height, that those individuals diagnosed with ADHD weighed higher than what would be expected (see Cortese et al., 2008 for review). Erhart and colleagues' (2012) and Cortese and colleagues' (2008) findings support a clear link between ADHD and obesity in adolescents.

Poor impulse control, negative affect, dopamine deficiencies, and distracted eating are important risk factors and mechanisms that may lead to obesity (Davis et al., 2006; Davis et al., 2009; Shepard, 2009). Notably, each of these constructs is also associated with ADHD (American Psychiatric Association, 2000; Davis et al., 2006; Davis et al., 2009; Malloy-Dinz, Fuentes, Leite, Correa & Bechara, 2007). In a recent study, structural equation modeling was used with a nonclinical sample of 110 women to explore the connections between ADHD symptoms and obesity (Davis et al., 2006). Researchers hypothesized that symptoms of ADHD predict disordered eating patterns and overeating, which also, in turn, predict obesity (see Figure

1). ADHD symptoms were positively related to disordered eating (e.g., eating in response to negative mood, environmental cues, and binge eating), indicating a possible relationship between ADHD symptoms, overeating, and higher BMIs.

Because many individuals with ADHD struggle with impulsivity, disinhibition, or other psychiatric disorders (e.g., mood disorders, substance use disorders, or eating disorders), they may be at a higher risk of becoming overweight/obese (Able et al., 2007; Cortese et al., 2008). If ADHD manifests in disinhibited or impulsive eating, poor impulse control could indirectly increase overweight and obesity. Moreover, ADHD is highly comorbid with other disorders. For example, approximately 37 – 47% of adults diagnosed with ADHD also meet criteria for anxiety disorders and major depressive disorder (Cumyn, French, & Hechtman, 2009), which might increase the risk for overweight/obesity in this population (Davis et al., 2009; Lluch, Herbeth, Mejean, & Siest, 2000). In addition to these sources of negative affect, when compared to their non-ADHD peers, ADHD individuals are more likely to experience marital difficulties, job loss, legal trouble, financial problems, and other negative life events (Biederman, Faraone, & Spencer, 1993, 1994; Murphy & Barkley, 1996). Negative affect and undesirable life events are likely to cause significant amounts of stress, which increases the risk for individuals to utilize maladaptive coping mechanisms such as substance use and disordered eating behaviors (i.e., emotional eating/binge eating; Burton, Stice, Bearman, & Rhode, 2007).

Limited research has been conducted on the connections between ADHD and obesity. Davis and colleagues (2006) suggest the symptoms of ADHD predict types of overeating, which further and indirectly predicts overweight and obesity. Specifically, ADHD symptoms such as disinhibition, impulsivity, inattentiveness, poor planning, and hypo-dopaminergic levels may play a role in food overconsumption. Notably, researchers also suggest that many other factors;

for example, depression, could mediate the relationship between ADHD and obesity (Davis et al., 2006; Johnson, 2011).

ADHD, Obesity and Depression

Past literature suggests a link between ADHD and depressive symptoms (Able et al., 2007; Goodman, 2009; Kessler et al., 2006), such that a high percentage (18.6%) of ADHD individuals will also have an MDD diagnosis. Notably, individuals who have never been diagnosed with ADHD, but experience ADHD symptoms screened positive for depressive disorders more often than those with a formal ADHD diagnosis (Able et al., 2007). This may indicate that individuals who experience symptoms of ADHD, but no formal diagnosis, might be struggling with current symptoms of depression due to the aversive symptoms and secondary stressors that are associated with ADHD.

Past literature has produced contradictory results on the relationship between overweight/obesity and depression. While some studies support a relationship between the two constructs, (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Sammel et al., 2003; Tanofsky-Kraff et al., 2006) others report results that are nonsignificant (Roberts, Deleger, Strawbridge, & Kaplan, 2003; Tanofsky-Kraff et al., 2006). Many of the studies exploring the relationship between obesity (as measured by BMI) and depression concluded that the presence of depression increases the risk for weight gain and obesity. When individuals feel more depressed, they are likely to experience lower levels of motivation, impulse control, planning, and activity, which in turn, make it more likely that they will overeat and make poorer food choices (Rooke & Thorsteinsson, 2008).

In a theoretical model of depression, Markowitz, Friedman, and Arent (2008) propose two possible mediators of the relationship between depression and obesity: low motivational

levels and physiological means. Specifically, Markowitz et al. (2008) propose that depressed individuals have a hypothalamic-pituitary-adrenal (HPA) dysregulation, which contributes to an elevated amount of cortisol. Higher levels of cortisol have been directly linked to weight gain, as well as an indirect pathway connecting depressive symptoms to obesity. Markowitz et al., propose binge eating as an indirect pathway (i.e., a mediator) between depressive symptoms and obesity. Previous studies have suggested negative mood as a precursor to binge eating episodes and that eating in response to aversive emotions is associated with more frequent binge episodes and depression among obese individuals (Arnow, Kenardy, & Agras, 1995; Masheb & Grilo, 2006). The "low motivational level" mediator includes symptoms such as poor organization, low impulse control, and poor planning, which are all shared symptoms of ADHD, which when paired with depression could lead to food choices higher in fat and/or overeating (Markowitz et al., 2008).

The connections between ADHD, obesity, and depression have not been widely studied and only a few studies have explored how these three constructs interrelate. Although depression is associated with ADHD symptoms and obesity separately, it might be a condition that elucidates the relationship between ADHD and obesity. Johnson (2011) conducted a study to explore possible pathways that might further explain the relationship between ADHD and overweight. Specifically, Johnson (2011) examined the possible relationships between ADHD symptoms, symptoms of depression, emotional and binge eating, and overweight/obesity (see Figure 2). Based on the results of the study, ADHD symptoms, particularly inattentiveness, predict increased symptoms of depression, which, in turn, predict increased emotional/binge eating, which has a direct and positive relationship with BMI. Additionally, an alternative pathway showed a statistically significant relationship between hyperactive and impulsive (H/I)

symptoms and emotional/binge eating, suggesting that H/I symptoms, regardless of the presence of depressive symptoms, have a positive relationship with emotional/binge eating.

Consistent findings in past research suggest that symptoms of ADHD and ADHD diagnoses are highly comorbid with addictive behaviors and diagnoses (Lambert, 2005; Milberger et al., 1997; Ohlmeier et al., 2007; Pomerleau et al., 1995). More recent studies hypothesized binge eating as a type of addictive behavior that might connect ADHD symptomology and diagnoses to overweight and obesity (Barry et al., 2009; Cortese et al., 2007). Johnson's (2011) findings help elucidate the relationships between ADHD symptoms and increased BMI, but there were limitations of that study. In an effort to improve upon Johnson's (2011) findings, the structural model of the current study will be compared to Johnson's (2011) model to determine which better explains the connections and pathways between ADHD and obesity.

CHAPTER 2

METHOD

Participants

Participants were recruited from a four-year university in the southern part of the United States. All participants were recruited from psychology classes that offered incentive in the form of extra credit points for each student's research participation. The overall sample included 340 participants, 105 males (30.9%) and 235 females (69.1%). See Table 1 for detailed information regarding the participants' ethnicity, age ranges, previous and current mental health diagnoses, sexual orientation, and body mass index category (BMI). Refer to Table 3 for information on the means and standard deviations of continuous variables by gender (i.e., age, self-reported height/weight, measured height/weight, past highest/past lowest/measured BMI/ideal BMI).

Measures

Demographic Questionnaire. Created specifically for the current study and based on participants self-report, The Demographic Questionnaire (see Appendix A) was utilized to gather participant's individual characteristics such as age, sex, ethnicity, and year in school. The questionnaire was also used to collect data related to weight, height, exercise habits, nicotine use, and ADHD and other psychiatric diagnoses.

Measures of ADHD Symptoms

Conners' Adult ADHD Rating Scale (CAARS). The Conners' Adult ADHD Rating Scale was designed to assist in the diagnosis of ADHD in adults age 18 and above (Conners, Erhardt, & Sparrow, 1999). In the current study, the long form (66-items) of the CAARS was used. Included on the long form are: four subscales (inattention/memory problems, impulsivity/emotional lability, hyperactivity/restlessness, and problems with self-concept), three

DSM-IV ADHD symptom subscales (inattentive symptoms, hyperactive-impulsive symptoms, and total ADHD symptoms), an ADHD index, and an inconsistency index. In the current study, every subscale is utilized except for the total ADHD symptoms, the ADHD index, and the inconsistency index.

For the self-report form, the CAARS was normed on a sample of 1,026 adults, including age ranges of 18 to 29 years, 30 to 39 years, 40 to 49 years, and 50 years and older. In previous studies, the self-report forms' internal consistency reliability observed scores ranged from α = .74 to α = .95 (Alder et al., 2007), α = .66 to α = .90 (Conners et al., 1999), and α = .86 to α = .92 (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999). In the current study, reliability proved to range from adequate to excellent with α = .85 to α = .91.

Over a one-month period, the test-retest coefficients on the long form of the CAARS were adequate ranging from r = .88 to r = .91 (Conners, Erhardt, & Sparrow, 1999; Erhardt et al., 1999). When the CAARS self-report long form was compared to the Wender Utah Rating Scale (Erhardt et al., 1999; Ward, Wender, & Reimharr, 1993), acceptable moderate correlations (r = .37 to r = .67) were found between the two measures. These findings indicated the scores from the CAARS long form have satisfactory construct validity.

To assess criterion validity of the CAARS, Erhardt et al. (1999) compared scores of individuals who had been formally diagnosed with ADHD against those individuals who did not meet criteria for an ADHD diagnosis (based on *DSM-IV-TR* criteria). Findings suggest the CAARS has high specificity and sensitivity. Based on the obtained score from the CAARS, findings suggest the CAARS correctly identified 82% of the individuals who had a formal ADHD diagnosis, as well as correctly identified 87% of the individuals who did not meet criteria for an ADHD diagnosis. However, the obtained CAARS scores from this study also suggested

that the CAARS falsely identified individuals as meeting criteria for a formal ADHD diagnosis 13% of the time, as well as 18% of the time overlooked an ADHD diagnosis (when individuals actually met criteria for an ADHD diagnosis).

Brown Attention-Deficit Disorder Scales (BADDS). The BADDS (Brown, 1996) is a self-report 40-item inventory that is used to assess symptoms and associated features of ADHD. Five dimensions are included to measure these symptoms: organizing and activating to work, sustaining attention and concentration, sustaining energy and effort, managing affective interference, and utilizing "working memory" and accessing recall. The sustaining attention and concentration subscale was used in the current study as an indicator of inattention as related to an ADHD diagnosis. Respondents are asked to indicate how much they believe that a feeling or behavior has been a problem over the past 6 months based on a 4-point scale (0-3), specifying *never*, *once a week or less*, *twice a week*, and *almost daily*.

The BADDS (adult self-report form) was normed on 143 adult community members (i.e., a nonclinical sample), as well as 142 adults who were seeking consultation services for attentional difficulties (i.e., a clinical sample) in the United States (Brown, 1996). Three age ranges are represented in the normative sample: 18 to 28 years, 29 to 39 years, and those who are 40 years of age and older. Three ethnicity groups are represented in the normative sample: African American, Hispanic, and White. Internal consistency reliability for the self-report form ranged from $\alpha = .86$ to $\alpha = .93$ (for the clinical and nonclinical samples respectively), with an overall internal consistency of $\alpha = .96$ (i.e., for the combined sample of clinical and nonclinical) (Brown, 1996). In previous studies, internal consistency reliability ranged from $\alpha = .71$ to $\alpha = .79$ for the adult self-report form (Kooij et al., 2008). In the current study, reliability proved to be excellent with $\alpha = .93$.

The test-retest coefficients were measured over a 2-week period and proved to be satisfactory (r = .87) (Brown, 1996). Additionally, Brown (1996) compared the clinical and nonclinical samples, which provided strong discriminant validity. Specifically, the clinical sample raw scores averaged 78, while the nonclinical sample raw scores averaged 31 (SD = 16), for a discrepancy of almost two standard deviations (Brown, 1996; Kaufman & Kaufman, 2001).

Criterion validity of the BADDS was assessed by comparing BADDS scores of individuals who have been diagnosed with ADHD and non-ADHD individuals, as well as setting the cut-off score of 50 to indicate the presence of ADHD symptoms (Brown, 1996). In that study, the BADDS obtained scores falsely identified participants with ADHD 4% of the time and failed to notice the disorder 6% of the time.

Attention-Deficit Scales for Adults (ADSA). The ADSA (Triolo & Murphy, 1996) is a self-report 54-item inventory that is used to assess symptoms associated with a diagnosis of ADHD. For each item, the respondent is asked to indicate the frequency of occurrence on a 5-point Likert scale: *never, seldom, sometimes, often, and always.* Nine content subscales are included in the ADSA: attention-focus/concentration, interpersonal, behavior-disorganized activity, coordination, academic theme, emotive, consistency/long-term, childhood, and negative-social. In the current study, the attention-focus/concentration subscale was used to assess inattentive symptoms as related to an ADHD diagnosis.

The ADSA was normed on 306 adult community members (M = 33.95; SD = 11.61) in Georgia, Massachusetts, Florida, Alabama, New York, and New Hampshire (Triolo & Murphy, 1996). Six ethnic groups are represented in the normative sample: White, Black, Asian, Hispanic, Native American, and other. Overall, the internal consistency reliability for the self-report form was adequate (α = .89). Previous studies also showed high internal consistency

reliability (α = .93) (West, Mulsow, Arredondo, 2003). Internal consistency reliability coefficients for each individual subscale varied with some subscales having low coefficients. The internal consistency reliability for the obtained scores of the subscale (attention-focus/concentration) that was used in the current study was acceptable (α = .76) (Triolo & Murphy, 1996). In the current study, reliability proved to be adequate with α = .87.

Convergent validity of the obtained scores between the ADSA and the ADHD Rating Scale – IV has proved to be strong, with the highest being on the attention-focus/concentration dimension (r = .50) (West, Muslow, & Arredondo, 2003). Triolo and Murphy's (1996) findings suggested that the ADSA is useful to help identify adults with ADHD and that the obtained scores of the ADSA correctly identified individuals with an ADHD diagnosis 88% of the time.

Measures of Depression

Symptom Checklist-90-R (SCL-90-R). The SCL-90-R (Derogatis, 1994) is a self-report 90-item inventory that is used to assess a variety of symptoms within the past 7 days. To measure these symptoms, nine dimensions are included: somatization (SOM), obsessive-compulsive (O-C), interpersonal sensitivity (I-S), depression (DEP), anxiety (ANX), hostility (HOS), phobic anxiety (PHOB), paranoid ideation (PAR), and psychoticism (PSY). In the current study, the DEP subscale was used to assess dysphoric mood. Respondents are asked to specify how distressed they are by the problems included in the 90 items.

The obtained scores of the SCL-90-R have shown strong reliability and validity (Derogatis, 1994). The SCL-90-R and the Beck Depression Inventory proved to have strong convergent validity (r = .45 to r = .73), with the highest being on the depression dimension (Gotlib, 1984). The SCL-90-R also proves to have satisfactory internal consistency of the scores with alpha coefficients ranging from α = .77 to α = .90, again with depression being the highest.

Moreover, based on two different time frames between administration, test-retest reliability of the scores has proved to be satisfactory with correlation coefficients ranging from $r = .68$ to $r = .90$ (10 weeks between administration) and $r = .78$ to $r = .90$ (one week between administration; Derogatis, Rickels, & Rock, 1976). In the current study, reliability proved to be excellent with $\alpha = .90$.

Based on the SCL-90-R obtained scores, the sensitivity of the measure with nonclinical undergraduate students was adequate. The nonclinicial participants were correctly classified 70% of the time (Todd, Deane, & McKenna, 1997).

Beck Depression Inventory – 2nd Edition (BDI-II). The Beck Depression Inventory – 2nd Edition (Beck, Steer, & Brown, 1996) is a 21-item self-report inventory and is used to measure symptoms of depression and depression severity based on the *DSM-IV* criteria. The BDI-II is appropriate for use with adolescents over 13 years of age and adults. The BDI-II can be used to identify symptoms of depression in the normal population (i.e., nonclinical population), as well as used to assess symptom severity among those individuals with a formal diagnosis of major depression. Respondents are instructed to choose one out of four statements for each item that best represents their feelings over the past two weeks.

The BDI-II was normed on 500 individuals (age range 13-86 years; *SD* = 15.91) from 4 different psychiatric outpatient clinics and 120 college students (mean age = 19.58 years; *SD* = 1.84) from a university in Canada (Beck et al., 1996). The obtained scores of the BDI-II have proven to have excellent internal consistency reliability ranging from $\alpha = .92$ (outpatient sample) to $\alpha = .93$ (college student sample). In the current study, reliability proved to be excellent with $\alpha = .90$.

Over a one-week period, test-retest scores yielded a high correlation of r = .93. Convergent validity of the obtained scores is shown through the moderate and strong positive correlations between the BDI-II and several other psychological scales, including the Hamilton Psychiatric Rating Scale for Depression (r = .71), Hamilton Anxiety Rating Scale (r = .47), Beck Hopelessness Scale (r = .68), and Beck Anxiety Inventory (r = .60). Notably, there was a stronger positive correlation between the BDI-II obtained scores and the Hamilton Anxiety Rating Scale than between the BDI-II obtained scores and the Hamilton Psychiatric Rating Scale for Depression, indicating discriminant validity.

Past studies have been interested in how reliable the BDI-II is when used with college students. Specifically, Steer and Clark (1997) administered the measure to a group of college undergraduate students and findings suggest that the BDI-II is acceptable to be used with college students as well as inpatient populations. Internal consistency reliability of the obtained scores of the BDI-II proved to be excellent (α = .93; Steer & Clark, 1997).

The Hamilton Depression Scale – Short Form (HDI-SF). The HDI-SF (Reynolds & Kobak, 1995) is a 9-item self-report screening measure used to assess and screen for symptoms of depression. Respondents choose the statement (by circling the corresponding number beside the statement) that best represents how they have felt during the past 2 weeks. The HDI-SF was normed on 510 adults (age range 18-89 years; mean age = 41.06; *SD* = 15.97) from the community in the Midwestern and Western parts of the country. Internal consistency reliability of the scores for the HDI-SF is excellent ranging from α = .92 (female sample) to α = .93 (male sample) (Reynolds & Kobak, 1995). In the current study, reliability proved to be adequate with α = .87. Moreover, test-retest scores yielded a correlation of r = .93 over a 1-week period, which

provides evidence of consistency among respondent's answered when repeating the HDI-SF (Reynolds & Kobak, 1995).

Bech (1992) indicated six different domains that make up the depression domain: depressed mood, anxiety, motor symptoms, cognitive symptoms, social impairment, and vegetative symptoms. Based on Bech's (1992) criterion, the HDI-SF exhibits adequate content validity as it contains items that measure mood, anxiety, motor, cognitive, social, and vegetative content. Moreover, the obtained scores of the HDI-SF shows excellent convergent validity (Reynolds & Kobak, 1995), as evidenced by the moderate to strong, statistically significant correlations with the BDI (r = .92), Beck Hopelessness Scale (r = .81), and the Beck Anxiety Inventory (BAI) (r = .72). These high correlation coefficients signify that the BDI-II and the HDI-SF are measuring the same construct of depression and depressive symptoms. The HDI-SF scores showed a stronger positive correlation with the BDI than with the BAI, which provides evidence for its discriminant validity (i.e., anxiety vs. depression).

Measures of Emotional and Binge Eating

The Bulimia Test – Revised (BULIT-R). The BULIT-R (Thelen, Mintz, & VanderWal, 1996) is a self-report measure that consists of 36 items measuring disordered eating habits. In particular, the BULIT-R measures bulimia nervosa symptomology in both clinical and nonclinical populations. The BULIT-R was used to assess disordered eating, such as binge eating. With a nonclinical female college population, the test-retest obtained scores were strong and reliable (r = .95) over a 2 -month interval period (Thelen, Farmer, Wonderlich, & Smith, 1991). Construct validity of the obtained scores of the Binge Scale and the BULIT-R was sufficient (r = .85) (Hawkins & Clement, 1980). Additionally, Thelen, Mintz, and VanderWal (1996) also showed adequate construct validity of the scores between the BULIT-R and *DSM-IV*

criteria (r = .73). In the current study, the BULIT-R was parceled to create two indicators so that the emotional and binge eating construct would have three indicators, which is considered ideal (Schmaker & Lomax, 2010). The parcels were created using factor analysis, where the items with the highest loadings were separated to create the two parcels: BULIT-R Parcel 1 and BULIT-R Parcel 2. Internal consistency reliability of the BULIT-R Parcel 1 and BULIT-R Parcel 2 obtained scores were adequate, α = .86 and α = .84 respectively.

Male Eating Behavior and Body Image Evaluation (MEBBIE). The MEBBIE (Kaminski & Caster, 1994; Kaminski et al., 2002) is a 57-item self-report measure used to assess men's attitudes and behaviors with regard to eating habits, exercise, and body image. The MEBBIE is composed of seven subscales: body dissatisfaction (BD), drive for muscularity (DM), emotional and binge eating (EBE), over-exercise (OE), drive for thinness (DT), fear of fatness (FF), and distorted cognitions (DC) (Kaminski & Caster, 1994; Kaminski et al., 2002). In the current study, the EBE subscale was utilized. The EBE scale consists of 9 items and measures participant's overeating tendencies, as well as their experiences of loss of control while eating.

Internal consistency reliability of the seven MEBBIE subscales obtained scores were satisfactory, ranging from α = .71 to α = .92 (Chapman, Kaminski, Haynes, & Own, 2004; McFarland & Kaminski, 2008). In the current study, reliability proved to be acceptable with α = .77. Further, test-retest scores proved to be reliable (r = .72) with a sample of undergraduate men (Kaminski et al., 2002).

The Questionnaire for Eating Disorder Diagnoses (Q-EDD). The Q-EDD (Mintz, O'Halloran, Mulholland, & Schneider, 1997) is a brief (i.e., 5-10 minutes to complete) 50-tem self-report questionnaire that assesses symptoms of eating disorders based on criteria from the *DSM-IV-TR* (APA, 2000). Respondents are asked to answer the questions regarding their

behaviors and beliefs over the past 3 months, including the frequency and duration of binge eating, purging behaviors, and body concerns/satisfactions. Based on the respondent's total responses, participants were classified as eating disordered (i.e., anorexia, bulimia, or EDNOS), symptomatic (the presence of some eating disorder symptoms, but no *DSM-IV-TR* diagnosis), or asymptomatic (no eating disorder symptoms). Six specific diagnostic categories comprise the eating disorder classification: anorexia, bulimia (two *DSM-IV-TR* diagnoses), subthreshold bulimia, menstruating anorexia, nonbinging bulimia, and binge eating disorder (four *DSM-IV-TR* EDNOS diagnoses). Mintz et al. (1997) provided information that individuals who are classified as asymptomatic, but are grossly obese may not be reporting honestly and may suffer from binge eating. In the current study, the Q-EDD was used to assess disordered eating, such as binge eating.

Mintz et al. (1997) provided high test-retest reliability scores over a 1-3 month period with kappa values ranging from .54 - .64. Further, the Q-EDD obtained scores show excellent convergent validity, as evidenced by statistically significant t-tests and analyses of variance conducted on individuals completing the Eating Attitudes Test, BULIT-R, and the Q-EDD, where scores were significantly different between the measures (Mintz et al., 1997). Criterion validity scores were assessed by comparing the Q-EDD diagnosis with individuals who have been diagnosed with a structured clinical interview. The obtained scores from the Q-EDD correctly identified participants with an eating disorder 97% of the time, as well as correctly identified participants without an eating disorder 98% of the time (Mintz et al., 1997).

In the current study, three questions were utilized from the QEDD to create a new variable called Binge Number. Specifically, the questions that were utilized were (1) Do you engage in recurrent episodes of binge eating, meaning eating in a discrete period of time (e.g.,

within any 2-hour period) an amount of food that is definitely larger than most people would eat during a similar time period?, (2) If yes to question 1, Do you have a sense of lack of control during the binge eating episodes (i.e., the feeling that you cannot stop eating or control what or how much you are eating?), and (3) Circle the number in each set of brackets that best fits for you: On average, I have had [1, 2, 3, 4, 5, 6 or more] binge eating episodes per week for at least [1 month, 2 months, 3 months, 4 months, 5 months, 6-12 months, more than one year]. The participant's answers to these questions were then used to create the variable binge number, which assess the amount of binges each participant engages in that would meet criteria for the DSM-IV-TR diagnosis of binge eating disorder. Each participant that indicated that they engaged in binge eating episodes for at least 3 months or longer had a binge number created for them. If they have not engaged in binge eating episodes for more than 3 months, then their binge number was zero, indicating they did not struggle with binge eating enough to meet criteria for binge eating disorder.

The Eating Disorder Inventory – 3rd Edition (EDI-3). The EDI-3 (Garner, 2004) is a self-report measure that is used to assess psychological traits that are associated with people who have been diagnosed with eating disorders. The EDI-3 is a 91-item questionnaire, in which respondents are asked to indicate the most accurate response based on a 6-point Likert scale: *always, usually, often, sometimes, rarely, and never.* In the current study, the bulimia subscale was used.

The EDI-3 obtained scores have shown strong reliability and validity (Clausen, Rosenvinge, Friborg, & Rokkedal, 2011). Specifically, internal consistency of the scores is excellent, ranging from $\alpha = .90$ to $\alpha = .97$. In a previous study (Clausen et al., 2011), internal consistency of the scores proved to be excellent for the bulimia subscale in a clinical ($\alpha = .92$)

and nonclinical sample (α = .87). In the current study, reliability proved to be adequate with α = .82. Over a 7-day period, test-retest reliability scores also proved to be excellent (r = .98) (Garner, 2004; Wildes, Ringham, & Marcus, 2010). Moreover, Cumella (2006) suggests that the EDI-3 obtained scores yields adequate convergent and divergent validity based on high correlations with other eating disorder measures.

Since the MEBBIE was only completed by the males and the EDI-3 was only completed by the females, z-scores were created in order to combine and standardize these 2 subscales (MEBBIE EBE and EDI-Bulimia), creating a new indicator "zmebedi."

Measures of Substance Use

Michigan Alcohol Screening Test (MAST). The MAST (Selzer, 1975) is a 22-item questionnaire that is used as a screening measure to assess drinking behavior, drinking consequences, and attempts to obtain help for drinking problems. Parsons, Wallbrown, and Meyers (1994) provide evidence that the MAST is suitable to use with the college population. All of the items on the MAST are in "yes/no" format and intended to be face valid.

In previous studies, internal consistency reliability scores ranged from α = .84 to α = .88 (Conley, 2001; Laux, Mewman, & Brown, 2004; Selzer, 1975). In the current study, reliability proved to be acceptable with α = .76. An item was removed in order to increase the internal consistency reliability (measure item #s 12). The test-retest coefficients scores were measured over a 1- and 3-day period and proved to be excellent (r = .97 and r = .94 respectively) (Teitelbaum & Carey, 2000; Zung, 1982). The obtained scores from the MAST show adequate convergent validity (Conley, 2001), as evidenced by the statistically significant correlations with the Alcohol Dependence Scale (r = .79) and the presence of *DSM-IV-TR*- diagnosed alcohol disorder (r = .65). The obtained scores from the MAST correctly identified participants with

alcohol-related problems 74% of the time and correctly identified participants with the absence of alcohol-related problems 98% of the time (Okay, Sengul, Acikgoz, Ozan, & Dilbaz, 2010).

The Drug Abuse Screening Instrument (DAST). The DAST (Skinner, 1982) is a 28-item self-report measure that assesses drug use problems. The DAST parallels the questions on the MAST, but for drug use and respondents are asked to indicate "yes" or "no" to the 28 questions.

In previous studies, internal consistency reliability scores ranged from $\alpha = .92$ to $\alpha = .95$ (El-Bassel et al., 1997; McCann, Simpson, Ries, & Roy-Byrne, 2000; Skinner, 1982; Staley & El-Guebaly, 1990). In the current study, the DAST subscale was parceled by conducting a factor analysis to create two indicators that were equally strong measures of the substance use construct. Reliability scores proved to be adequate for DAST Parcel 1 and Parcel 2, with $\alpha = .76$ and $\alpha = .85$ respectively. Some items were removed in order to increase the internal consistency reliability (measure items #s 7, 21, and 23). The test-retest reliability scores also proved to be sufficient over a 2-week period ($r = .85$) (El-Bassel et al., 1997). Using the cutoff score of 6, the sensitivity of the DAST ranges from 81% to 96% and the specificity of the DAST ranges from 71% to 94%, indicating that the obtained scores from the DAST correctly identified those with and with out drug use problems (Yudko, Lozhkinda, & Fouts, 2012).

Nicotine Per Day. A variable was created to assess the amount of nicotine each participant used per day. Undergraduate RAs called each participant and asked them four questions related to nicotine use. Specifically, each participant was asked (1) Do you currently smoke cigarettes?, (2) If answered "yes" to question 1, then how many cigarettes do you smoke per day and what brand?, (3) Do you currently chew tobacco (sometimes called "dipping" or "chewing")?, and (4) If you answered "yes" to question 3, then how many times a day do you "chew" and how long (30 minutes, 1 hour)? In the current study, three variables were created:

Cigarette use per day (amount of nicotine in each cigarette), Chewing tobacco use per day (amount of nicotine in each "dip"), and total amount of nicotine use per day (which was calculated by adding cigarette use per day with chewing tobacco use per day). The amounts of nicotine in different brands of cigarettes and chewing tobacco were found in the "Report of tar, nicotine, and carbon monoxide of the smoke of 1206 varieties of domestic cigarettes for the year 2005" (Federal Trade Commission, 2005).

Procedure

After receiving IRB approval, psychology student participants were recruited through a website (SONA) utilized by students to receive extra credit in their classes. Participants viewed and signed a detailed consent form before completing the measures. Participants completed one of three counter-balanced questionnaire packets. Each packet contained a demographic questionnaire, the CAARS (Conners, Erhardt, & Sparrow, 2004), BADDS (Brown, 1996), ADSA (Triolo & Murphy, 1996), BDI-II (Beck, Steer, & Brown, 1996), SCL-90-R (Derogatis, 1994), HDI (Reynolds & Kobak, 1995), BULIT-R (Thelen, Mintz, & VanderWal, 1996), MEBBIE (Kaminski & Caster, 1994; Kaminski, Slaton, Caster, Own, Baker, & Chapman, 2002), Q-EDD (Mintz, O'Halloran, Mulholland, & Schneider, 1997), EDI-2 (Garner, 2004), MAST (Selzer, 1975), and the DAST (Skinner, 1982).

After completing the questionnaire packet, each participant was taken by a research assistant (RA) to a private room to have their actual weight and height measured, in order to accurately calculate the participant's BMIs. The body mass index (see Appendix B) is a method of measuring if an individual should be classified as underweight, normal, overweight, or obese. Specifically, BMI is calculated by dividing a person's weight in kilograms by the square of their height in meters. A BMI between 25 and 29.9 would represent an overweight adult, while a BMI

of 30 or higher would represent an obese adult (Centers for Disease Control and Prevention, 2010; National Institutes of Health, 2010).

Upon completion of the measures, each participant was given a debriefing form (see Appendix C). Each participant who fully completed all measures was assigned extra credit on SONA, which is a website through which universities assign extra credit to students.

Data Preparation, Design, and Analysis

Initially, the data was checked for the accuracy of data entry, outliers were identified, statistical assumptions were tested and the sample was screened for missing values. To ensure the data was accurately entered, one research assistant (RA) entered the data and two different RAs compared the data entered with each participant's responses. In the sample, less than 2% of all the possible data was missing on each subscale. Through the missing values analysis in SPSS, the data showed to be missing values completely at random (MCAR). Multiple imputation was used to effectively deal with the missing data (Peugh & Enders, 2004). Specifically, multiple imputation was conducted through the multiple imputation module of the LISREL software program, which implements the expected maximization algorithm and the Markov Chain Monte Carlo method for imputing participant's missing values in the dataset (Jöreskog & Sörbom 2005).

Total scores, means, standard deviations, and distributional properties (i.e., skewness and kurtosis) were obtained for each measure. Multivariate normality tests were calculated in LISREL and assessed as Mardia's (1970) multivariate kurtosis coefficient and tests for multivariate normality. While a z score greater than 1.86 is considered statistically signifcant, SEM recommendations suggest that less than 3.0 or even < 10.0 is within acceptable range (Bollen, 1989; Kline, 2004). Mardia's coefficient was 1.078. Multivariate skew was 6.927

and multivariate kurtosis was 3.664. Total scores from the binge number, MAST, DAST, and nicotine per day scales were positively skewed and leptokurtic (see Table 5). Square root and logarithmic procedures (Tabachnick & Fidell, 2007) were used to transform these measures; however, the transformed scores still remained skewed and kurtotic. Therefore, the decision was made to keep the nontransformed values in the data and rely on the fit statistics from LISREL. Specifically, the approach for dealing with nonnormality was with scaled chi-square and robust standard errors using the method developed by Satorra and Bentler (1988; 1994). Conceptually, one would expect these variables to be skewed because most participants reported that they do not binge, smoke, or drink/use drugs excessively. Lastly, the internal consistency reliabilities and correlations among the total scales were computed (see Table 4).

Correlation coefficients, tolerance, and variance inflation factor (VIF) were inspected to assess possible multicollinearity problems. To assess multicollinearity, tolerance values of less than 1.0, and/or VIFs of greater than 10 at the multivariate level might result in a multicollinearity problem in the SEM analysis (Kline, 1998; 2005). Correlation estimates were explored for bivariate relationships (i.e. correlation estimates of .85 or higher may indicate a bivariate relationship). Absolute correlation coefficients among observed variables ranged from -.098 to .908 (see Table 7), indicating no problem with bivariate multicollinearity, with the exception of the correlations between Hyperactivity/Restlessness subscale, DSM-IV Hyperactive/Impulsive subscale (r = .852), Inattention/Memory subscale and DSM-IV Inattentive subscale (r = .903), and BULIT-R Parcel 1 subscale and BULIT-R Parcel 2 subscale (r = .908). The tolerance values ranged from .214 to .828 and the VIF ranged from 1.21 to 6.89, indicating a possible multicollinearity problem at the multivariate level (Kline, 1998; 2005). When variables are highly correlated, there is potential to affect the model, but the research on

when multicollinearity causes problems, as well as what to do about multicollinearity is ambiguous (Grewal, Cote, & Baumgartner, 2004). Researchers suggest increasing sample size, deleting observed variables from the study, and exploring a higher order factor as options for dealing with multicollinearity problems (Grewal et al., 2004).

The proposed and alternative models were tested through structural equation modeling (SEM), which is a two-step statistical method that allows the relationships between multiple variables to be tested at the same time (Schumacker & Lomax, 2010). First, we tested the measurement model through Confirmatory Factor Analysis (CFAs) to investigate whether the indicators adequately measured the specific latent variables. Maximum Likelihood (ML) estimation was utilized, which assumes multivariate normality (Schumaker & Lomax, 2010). Second, both the initial and alternative models were tested.

Linear Structural Relations software (LISREL; Joreskog & Sorbom, 2006) was used to conduct the SEM analysis. To analyze the structural equation model, LISREL creates a mathematical and statistical model. The mathematical model uses covariance and mean structure models to determine and analyze the relationships between the independent and dependent variables in the model. The statistical model generates estimates of the free (i.e., unknown) parameters. Because latent variables do not possess a scale, common practice is to set one path from an indicator to the latent variable to 1.0 and let the other paths be estimated freely (Brown, 2006; Keith, 2006). Once LISREL estimates the parameters, a chi-square statistic assesses how large of a discrepancy exists between the covariances and variances of the initial structural model and the sample data. Researchers suggest using multiple fit indices to evaluate overall model fit (Weston & Gore, 2006; Martens, 2005; Hoyle & Panter, 1995).

In order to fully evaluate model fit in the current study, normed chi-square (χ^2), the ratio of chi-square to degrees of freedom (χ^2/df), the root mean square error of approximation (RMSEA), the comparative fit index (CFI), standardized root mean square residual (SRMR), and the Akaike Information Criterion (AIC) were calculated and provided for each construct and combination of constructs. Specifically, larger chi-square values suggest that the model does not fit the data and the ratio of chi-square to degrees of freedom need to be less than 3 to show adequate fit (Martens, 2005; Weston & Gore; 2006; Worthington & Whittaker, 2006). Additionally, a RMSEA less than .06 indicates good fit, while a SRMR less than .08 indicates good fit. Lastly, a CFI greater than .95 and lower AIC values indicate good fit (Hu & Bentler, 1999; Martens, 2005; Weston & Gore; 2006; Worthington & Whittaker, 2006).

CHAPTER 3

RESULTS

Measurement Model and Proposed Initial Model

A confirmatory factor analysis (CFA) was conducted to the test the measurement model of the proposed initial model. This model had the two core symptoms of attention deficit hyperactivity disorder (ADHD) separated into their own latent constructs, as was done in Johnson (2011) study. The measurement model exhibited adequate fit, although the impulsive and inattentive symptoms (latent constructs) were highly correlated (r = .92), as were their respective indicators (see Table 7). Based on the CFAs, the nicotine per day indicator measuring the substance use construct and the binge number indicator measuring the emotional/binge eating construct were not loading strongly. Due to the weak factor loadings to the latent variables they were supposed to be measuring, it was decided to delete these two indicators from the model in order to strengthen the measurement of the model. These two indicators had low construct validity and were not measuring the theoretical construct they were supposed to be measuring. It is common practice to remove an indicator that does not meet criteria (e.g., construct validity; Paswan, 2009). The drug abuse screening test (DAST) and the bulimia test-revised (BULIT-R) were then parceled using factor analysis (i.e., separating items to two parcels based on highest loading to the latent construct) in order to include at least three indicators per latent construct.

The proposed initial model was originally tested with the data (see Figure 3). The model produced some paths that were inconsistent with past research. Specifically, the path between inattentive symptoms of ADHD and symptoms of depression was negative, indicating an inverse relationship between these two constructs. The model suggested that individuals who experience higher levels of inattentive symptoms would experience lower levels of depression. Inattentive

symptoms are a core symptom of ADHD and past research findings consistently report positive relationships between ADHD symptoms and depressive symptoms and disorders (Goodman, 2009; Johnson, 2011; Kessler et al., 2006; Wilens et al., 2005). Modification indices were examined and suggested

One of the signs of multicollinearity is when a path coefficient is negative when; theoretically, there should be a positive relationship (Grewal et al., 2004). Some ways to remedy multicollinearity are: (1) increasing the sample size, (2) removing the most intercorrelated variable from the analysis (which should not be done if theoretically it should be there), and (3) to combine the variables into a higher order factor (only if theoretically justifiable; Gordon, 1968; Grewal et al., 2004; Kaplan, 1994; Marsh, Dowson, Pietsch, & Walker, 2004). Because of this inconsistency in the results between the latent constructs, the model was further explored to better understand the relationships between the constructs included in the model and how they might be impacting and affecting the results. Theoretically, inattentive symptoms (of an ADHD diagnosis) and depressive symptoms should have a strong, positive relationship.

The model was tested in LISREL with impulsive symptoms deleted and interesting findings surfaced. When impulsive symptoms were taken out of the model, the path coefficient between inattentive symptoms and symptoms of depression became strong, positive, and statistically significant. Similarly, when inattentive symptoms were removed from the model, the path coefficient between impulsive symptoms and symptoms of depression remained strong and positive. This signified that impulsive symptoms and inattentive symptoms were so highly correlated that they both conveyed essentially the same information and the model was treating them as the same variable (Gordon, 1968; Grewal et al., 2004; Kaplan, 1994; Marsh, Dowson, Pietsch, & Walker, 2004). Impulsive and inattentive symptoms were then combined in a higher

order factor: ADHD symptoms, and the model was tested. The initial and alternative models stayed the same as originally proposed with the exception of combining the two core ADHD symptoms, impulsive and inattentive symptoms, into an ADHD symptoms construct (see Figures 5 and 6). Those are the models discussed.

Measurement Model of the Revised Initial Model

A confirmatory factor analysis was conducted to test the measurement model (see Figure 4). All latent constructs were allowed to correlate. The seven variables measuring ADHD symptoms included: three Conners' adult ADHD rating scale (CAARS) impulsivity subscales (i.e., impulsivity/emotional lability, hyperactivity/restlessness, and DSM-IV hyperactive impulsive symptoms), two CAARS inattention subscales (i.e., inattention/memory problems and DSM-IV inattentive symptoms), Barkley attention deficit disorder scale (BADDS) subscale (i.e., sustaining attention and concentration), and the attention deficit scale for adults (ADSA) subscale (i.e., attention-focus/concentration). All subscales measuring the ADHD symptoms construct had strong, positive path coefficients and ranged from .72 to .96. The amount of variance accounted for in predicting ADHD symptoms from these seven measures was 68%.

Two measures of impulsive symptoms of ADHD (CAARS: impulsivity/ emotional lability and CAARS: DSM-IV hyperactive/impulsive symptoms) have measurement error that is correlated, which theoretically makes sense because the measurement errors that are correlated are measuring the same global construct. Allowing measurement error to correlate is an accepted practice as long as it is warranted theoretically and does not significantly alter the path coefficients (Bagozzi, 1981; Fornell, 1983). Theoretically, all the indicators measuring impulsivity and inattention are globally measuring symptoms of ADHD, therefore; it theoretically makes sense that their measurement errors would correlate. Additionally, the three

variables measuring symptoms of depression (Symptom checklist – 90 – revised (SCL-90-R) Depression, Beck depression inventory – second edition (BDI-II), and the Hamilton depression inventory – short form (HDI-SF) all positively loaded on the symptoms of depression construct with path coefficients ranging from .84 to .91 and accounting for 78% of the variance. The BDI-II and the HDI-SF had correlated measurement error, indicating that these two indicators share something in common (e.g., construct inherent variance). The three variables measuring emotional and binge eating (i.e., BULIT-R Parcel 1, BULIT-R Parcel 2, and ZMEB/EDI) had positive path coefficients to the latent construct and ranged from .73 to .96. The amount of variance accounted for in predicting emotional and binge eating from these three measures was 79%. The Michigan alcohol screening test (MAST), DAST Parcel 1, and DAST Parcel 2 positively loaded on the substance use construct with path coefficients ranging from .44 to .63 and accounting for 28% of the variance. The DAST parcels' measurement error was correlated (see Figure 4). This is likely due to both of the parcels containing items that comprise the same measure, as well as both measuring the same construct (i.e., substance use). As expected, in the measurement model, all other indicators loaded positively on the latent constructs. Overall, the measurement model had good fit (see Table 7).

Revised Initial Model

The fit indices of the revised initial and alternative model (see Figures 5 and 6) are shown in Table 8. Based on the adjusted χ^2, RMSEA, and SRMR, the evidence suggests that the revised initial model fits the data better than the alternative model (Revised initial model adjusted $\chi^2 = 2.66$, RMSEA = .070, and SRMR = .065; Alternative model adjusted $\chi^2 = 3.34$, RMSEA = .083, and SRMR = .070). However, as the alternative model is more parsimonious

(fewer parameters) it is not surprising that the AIC value for the alternative model is lower (AIC = 305.09) than the proposed model (AIC = 379.20; Tanaka, 1993).

In the revised initial model (see Figure 5), all of the pathways are statistically significant at the $p < .001$ and $p < .01$ levels. Specifically, there are direct and positive relationships between increased ADHD symptoms (i.e., impulsive and inattention symptoms) and increased symptoms of depression. Direct and positive relationships were also found between ADHD symptoms and substance use and substance use and emotional and binge eating. Finally, a direct and positive relationship was found between symptoms of depression and emotional and binge eating, as well as between emotional and binge eating and BMI (see Figure 5).

CHAPTER 4

DISCUSSION

The purpose of the current study was to further explore and determine the relationships between the core symptoms of attention deficit hyperactivity disorder (ADHD; i.e., impulsive and inattentive symptoms), symptoms of depression, substance use, emotional and binge eating, and overweight (i.e., increased BMI). In order to examine and explore the relationships, a structural and alternative model were hypothesized and tested through structural equation modeling (SEM). Initially, the proposed initial model was tested (see Figure 3) and yielded some results that were inconsistent with past research findings. Specifically, the path between inattentive symptoms and symptoms of depression suggested a negative relationship between the two constructs, indicating that when inattentive symptoms were high, depressive symptoms were low.

Due to this unlikely result, further exploration of the model was conducted and it was revealed that the two core symptoms of ADHD (i.e., impulsive and inattentive symptoms) were highly correlated and significantly affecting the relationships from each separate construct to other constructs in the model. When impulsive symptoms were removed from the model, the path between inattentive symptoms and depressive symptoms became positive and statistically significant. After further exploration of this model, it was indicated that a higher order factor was needed between impulsive and inattentive symptoms and, thus, one latent construct comprised of impulsive and inattentive symptoms together (i.e., ADHD symptoms) was created (see Figure 5). The revised initial and alternative models differ from the originally proposed models only in that the ADHD symptoms of impulsive and inattention were combined to create ADHD symptoms. Otherwise, the models stayed the same as what was proposed. The resulting

models are now closely related to Davis' (2006) model (see Figure 1), in that the ADHD symptoms are one higher order factor versus being separated into the two core symptoms, as was done in Johnson's (2011) study. Both the resulting revised initial and alternative models exhibited a good fit, although the revised initial model provided a better fit then the alternative model. All pathways of the revised initial model were statistically significant.

Findings from the current study suggest that ADHD core symptoms (i.e., impulsive and inattentive symptoms) predict increased use of substances (i.e., alcohol and illegal drugs), which in turn predicts increased emotional/binge eating. Additionally, ADHD symptoms predicts increased depressive symptoms, which in turn also predicts increased emotional/binge eating. Lastly, emotional/binge eating has a direct, positive relationship with overweight/obesity (i.e., increased BMI). Based on the current study, there are two separate paths and three constructs that help explain the connections between ADHD symptoms and overweight/obesity. Exploring each path separately will help to better understand the possible complex relationships between these constructs.

Due to the inclusion of a substance use construct in the current model, new findings have emerged in the connections between ADHD symptoms and overweight/obesity. A direct and positive relationship was found between ADHD symptoms and substance use, as well as between substance use and emotional/binge eating constructs in the model, which can lead to overweight/obesity. These findings suggest that some individuals who experience ADHD symptoms may be at an increased risk to use/abuse substances, as well as abnormal eating patterns. Extensive research has been conducted on the high rates of comorbid diagnoses with an ADHD diagnosis. Substance use disorder is one of those disorders that is highly comorbid with ADHD, such that between 15 – 25% of those with ADHD have a lifetime history of substance

use disorder (SUD; Wilens, 2004). Additionally, those individuals with ADHD-Combined Type (i.e., experience both impulsive and inattentive symptoms) were more likely to have a substance use disorder when compared to those who were diagnosed with ADHD-Inattentive Type and ADHD-Impulsive Type (Cumyn et al., 2009). Wilens (2004) and Ohlmeier et al. (2007) suggest that it is imperative for clinicians to be aware of the high risk factor for substance use/abuse among the ADHD population. There are three prominent hypotheses for high substance use/abuse among ADHD individuals that are discussed in the literature: (1) disinhibition, (2) coping, and (3) self-medicating. These same three explanations can be applied to emotional/binge eating as well.

ADHD, substance use/abuse, and emotional/binge eating have shared symptoms of disinhibition, which involves a lack of restraint, poor risk assessment, and difficulty thinking about the consequences of behaviors. Individuals with ADHD, substance abuse, and those who engage in emotional/binge eating might use drugs or overeat knowing the potential negative side effects (Barry et al., 2009; Ohlmeier et al., 2007). Additionally, those individuals with ADHD are more likely to eat in response to environmental cues (e.g., the smell of food) versus eating in response to hunger (Davis et al., 2006). Furthermore, those with ADHD experience a variety of secondary stressors such as interpersonal troubles, difficulty keeping a job, and legal troubles, all of which have the potential to increase negative affect (Able et al., 2007; Murphy & Barkley, 1996). Individuals might turn to drugs, alcohol, and emotional/binge eating as a means to more effectively cope with these stressors (Able et al., 2007; Barry et al., 2009). Similar to coping with drugs, alcohol, and emotional/binge eating, some individuals use these substances and food as a means to self-medicate. In recent studies, researchers have explored the neurochemistry of individuals with ADHD and substance abuse/dependence, as well as overweight/obese

individuals (Blum et al., 1995). Findings showed that these individuals have lower concentrations of dopamine receptor sites in their brains. Drugs/alcohol and eating foods that are highly palatable (i.e., food that are high in fat and/or sugar) increases the amount of dopamine in the brain, and in turn, reduces the distressing symptoms of ADHD. These new results advance the past findings of Davis et al. (2006; 2009) and Johnson (2011) suggesting there is another pathway that connects ADHD symptoms and overweight/obesity, through the use and abuse of substances.

Consistent with past research findings, ADHD symptoms (i.e., impulsive symptoms and inattentive symptoms) were positively related to symptoms of depression (Johnson, 2011). The relationship between ADHD symptoms and symptoms of depression has been well documented for children and adolescents (Humphreys et al., 2013; Drabick, Gadow, & Sprafkin, 2006), as well as adults (de Zwaan et al., 2012; Alpert et al., 1996). The core symptoms of ADHD are associated with numerous detrimental outcomes and lower qualities of life, including job loss, poor interpersonal relationships, substance abuse/dependence, academic failure, legal problems, and car accidents (Barkley et al., 1996; Barkley, 2006; Klassen et al., 2010), which all have the potential to contribute to depressive symptomology. Additionally, Chao et al. (2008) examined the relationship between adult ADHD and depression and found that individuals in the ADHD group had more severe depression when compared to the non-ADHD control group.

More related to this specific sample, college students diagnosed with ADHD exhibited lower levels of academic functioning and adjustment, lower self esteem, underdeveloped social skills, and lower levels of academic achievement when compared with a non-ADHD control group (Frazier, Youngstrom, Glutting, & Watkins, 2007; Shaw-Zirt, Popali-Lehane, Chaplin, & Bergman, 2005). Individuals who struggle with impulse control in particular are more likely to

encounter the aforementioned secondary stressors (Ratey et al., 1992). Thus, a possible explanation for the direct and positive relationship between ADHD symptoms and depressive symptoms is that the presence of secondary stressors, as well as ADHD symptoms, may increase risk for and/or feelings of depression. Furthermore, individuals who struggle with impulse control are more likely to engage in risky behaviors, such as alcohol, drug, and nicotine use (Wilson, 2007; Kalbag & Levin, 2005). Because of the high comorbidity between these addiction and depressive disorders (Compton, Thomas, Stinson, & Grant, 2007; Hasin et al., 2002), this might better help to explain the relationship between ADHD symptoms and depressive symptoms in the current model.

As hypothesized, depressive symptoms and emotional/binge eating were directly and positively related. There was a statistically significant relationship between these two constructs, indicating that increased depressive symptoms predicted increased emotional/binge eating. Although the literature is inconsistent in regards to the relationships between depressive symptoms and emotional/binge eating (Goldschmidt, Wall, Loth, Bucchianeri, & Neumark-Sztainer, 2013; Franko et al., 2005; Noppa & Hallstrom, 1981; Hawkins & Clement, 1984; Polivy & Herman, 1993; Haedy-Matt & Keel, 2011; Johnson, 2011), the current results suggest that a relationship between the two constructs exists, at least for some people.

The affect regulation model has been used to explain the relationships between depressive symptoms (i.e., negative affect) and emotional/binge eating (Polivy & Herman, 1993; Hawkins & Clement, 1984). Two main hypotheses have been studied in relation to the affect regulation model: (1) that depressive symptoms precede and increase the act of emotional/binge eating and (2) that emotional/binge eating decreases negative affect (Haedt-Matt & Keel, 2011). Several studies have yielded results in support of the first hypothesis (Bruce & Agras, 1992;

Lynch, Everingham, Dubitzky, Hartman, & Kasser, 2000; Davis & Jamieson, 2005; Vanderlinden et al., 2004). Results have shown that between 69 - 100% of individuals diagnosed with binge eating disorder (BED) reported that depressive symptoms preceded emotional/binge-eating episodes. The results of the current study further support this hypothesis.

Further supporting the affect regulation model, individuals may use emotional/binge eating as a means of coping with their depressive symptoms. That is, using food to temporarily decrease the presence of negative emotions. Recent research has focused on the connection between the reward deficiency syndrome (RDS) and emotional/binge eating (Blum et al., 2008; Blum et al., 1995). RDS is the dysfunction of the reward cascade process in the brain where a reduced amount of dopamine (D2) receptor sites are thought to contribute to feelings of depression. In contrast, in individuals who do not have this dysfunction, the reward cascade involves the release of serotonin, which allows encephalin to be released at the hypothalamus, which inhibits the release of GABA at the substania nigra, which in turn regulates the amount of dopamine released at the nucleus accumbens (i.e., the reward site; Blum et al., 2000). In those individuals who experience dysfunction of the reward cascade process, the amount of dopamine released is reduced. Addictive behaviors (e.g., drug use, alcohol use, gambling, and smoking), ADHD, and emotional/binge eating are understood to regulate and increase the release of dopamine in the reward site of the brain, which, in turn, reduces the experience of negative affect (Blum et al., 2000; Blum et al., 2008).

Of importance to this study, the higher concentration of dopamine in the brain as a result of emotional/binge eating increases the likelihood that individuals will cope with their depressive symptoms by eating more than is needed for survival. Individuals may begin to engage in eating highly palatable foods (i.e., foods high in fat and sugar) in an effort to release dopamine in the

brain and, in turn, elevate their moods (Hoebel et al., 2007; Blum et al., 2000). In this way, emotional/binge eating can be viewed as an addictive behavior that temporarily eases depressive symptoms.

Depression and ADHD are highly comorbid (Murphy & Barkley, 1996; Goodman, 2009; Able et al., 2007), with major depressive disorder (MDD) being the most common co-occurring diagnosis with ADHD. In fact, 24.4 - 31% of individuals diagnosed with ADHD will also be diagnosed with MDD in their lifetime (Fischer et al., 2007; Biederman et al., 1993), which is higher than the lifetime prevalence rate of being diagnosed with MDD in the general population (16.2%; Kessler et al., 2003). Some individuals who experience ADHD and depression have shared symptoms of poor planning, low motivation, and problems with impulse control, which could contribute and increase the likelihood of overeating (Rooke & Thorsteinsson, 2008). This also suggests that those individual who struggle with both ADHD and depression might be at an increased risk to engage in emotional/binge eating than those who do not experience comorbid ADHD and depression.

A direct and positive relationship was found between emotional/binge eating and increased BMI, indicating that increased emotional/binge eating predicted increased BMI. More specifically, individuals who engage in emotional eating and binge eating are at a higher risk than those who do not engage in these behaviors of being overweight (i.e., having increased/higher BMIs) due overeating (e.g., eating in response to emotions or disinhibited eating; Goossens et al., 2008; Presnell, Bearman, & Stice, 2004).

Clinical Implications

The current results are of importance to practicing clinicians because they provide increased clarity and depth regarding the connections and relationship between symptoms of

ADHD and increased BMI (i.e., overweight/obese). Similar to the clinical implications Johnson (2011) discussed, the results of the current study may be used to help clinicians develop more appropriate treatment plans for their clients. Based on these findings, clinicians will know to incorporate behavioral interventions such as meal & exercise planning, as well as skills related to better management of distracted eating and impulse control as related to food choice. Additionally, self-management skills training and environmental restructuring will help these individuals who struggle with ADHD symptoms increase the amount of structure, organization, and routines into their daily lives. Knowing that substance use is highly comorbid with ADHD, and positively related to emotional/binge eating, clinicians can be cognizant of the interplay of these constructs on their clients current levels of distress. It will also be helpful to notice these connections to know when clinicians might need to refer a client to a drug/alcohol specialist. Further, it would be advantageous for clinicians to be aware of all of these connections so that they may talk candidly with their clients about eating behaviors to better understand how, if at all, it relates to their symptom picture and level of distress. Moreover, based on the current findings, as well as being knowledgeable about the potential connections between these constructs, clinicians would benefit from screening for substance use and emotional/binge eating after a client discloses or they suspect an ADHD diagnosis.

Additionally, clinicians should remain aware that ADHD is no longer considered to be only a childhood disorder. Instead, it is more helpful to think of the disorder as a life-span disorder that continues to have the potential to negatively impact the lives of adults as well. Because of the many shared symptoms of ADHD with other psychiatric disorders, it might be difficult to differentiate symptoms of specific disorders. This is important for clinicians to note because a diagnosis of adult ADHD has the potential to be overlooked and left untreated,

especially for those cases when it was never diagnosed in childhood. Therefore, the hope is that the findings of the current study help to increase clinician's awareness of ADHD symptoms in adulthood and how they can be interconnected with substance use and symptoms of depression, as well as related to emotional/binge eating and increased BMI. In turn, if clinicians are knowledgeable about the connections between these constructs, they will be more capable of explaining these relationships to their clients. If clients are able to better understand how their symptoms are related, it might instill hope and positive feelings about meeting their treatment goals.

Limitations and Directions for Future Research

Although our models both provided excellent fit and yielded positive results, it is important to discuss the limitations of this study. Future research might attempt to include a more diverse sample. About half of the current sample consisted of individuals who identified as European-American (White), which is double that of any other ethnicity represented in the sample. Furthermore, approximately 75% of the current sample was between the ages of 18-20.

Future research might include more measures of substance use in order for it to be a stronger latent construct. Although the current study yielded statistically significant paths between the substance use construct, the subscales used to measure substance use could have been stronger. Further, the substance use construct could have been better represented among our sample. It is possible that the participants were engaging in impression management and underreporting their alcohol and drug use. Further, the participants in the current study were mainly under the legal drinking age (~75%), thus participants might have been unwilling to disclose the amount of alcohol and drugs they consume, due to fear of engaging in illegal activities. Future studies might want to oversample individuals who are of legal drinking age to

get a more accurate representation of the presence of substance use and how it relates to ADHD symptoms, depressive symptoms, emotional/binge eating, and overweight.

Another limitation of our study was that the nicotine question was inadvertently left off the demographic form. Researchers contacted all the participants that disclosed their contact information to ask a follow-up question about their nicotine use. It is possible that participants also engaged in impression management when answering about their nicotine use. Most of the sample denied any nicotine use (92.6% reported they did not smoke and 99.1% reported they did not chew tobacco). Future research should take care to include this question on the demographic form in order to get more accurate results. That way, it could possibly be used in future studies as an indicator for an addiction construct.

In the current study, the possible relationships between the symptoms of ADHD, depression, and emotional/binge eating were explored versus exploring the connections between the formal diagnoses of these disorders (i.e., ADHD, MDD, BED). To further contribute to the literature, future studies could explore the relationships between the formal diagnoses of these disorders in order to generalize the results of the current study to those individuals formally diagnosed with ADHD, MDD, and BED. Further exploration will help to clarify the possible connections between formal diagnoses of ADHD, MDD, and BED and how those diagnoses are related to increased BMI (overweight).

Table 1

Frequencies and Percentages for Categorical Variables

	Total Sample	
	n	%
Sex[a]		
Male	105	30.9
Female	235	69.1
Ethnicity[b]		
Asian-American (Asian)	22	6.5
African-American (Black)	66	19.4
European-American (White)	141	41.5
Latin-American (Hispanic)	84	24.7
Bi-Ethnic	21	6.2
Other	6	1.8
Age[c]		
18 years old	170	50
19 years old	51	15
20 years old	36	10.6
21 years old	31	9.1
22 years old	17	5
23 years old	10	2.9
24 years old	3	.9
25 years old	1	.3
26 years old	5	1.5
27 years old	1	.3
28 years old	1	.3
29 years old	4	1.2
30 years old	1	.3
31 years old	2	.6
33 years old	1	.3
34 years old	1	.3
36 years old	1	.3
37 years old	1	.3
48 years old	1	.3
53 years old	1	.3
ADHD Diagnosis[d]		
ADHD Diagnosis	48	14.1
No Diagnosis	237	69.7
Never diagnosed, but suspect I have the disorder	51	15

(table continues)

Table 1 *(continued)*

	Total Sample	
	n	%
Eating Disorder Diagnosis[e]		
Eating Disorder Diagnosis	3	.9
No Diagnosis	328	96.5
Never diagnosed, but suspect I have the disorder	7	2.1
Major Depressive Disorder (MDD) Diagnosis[f]		
MDD Diagnosis	30	8.8
No Diagnosis	299	87.9
Never diagnosed, but suspect I have the disorder	9	2.6
Alcohol Abuse or Dependence[g]		
Alcohol Abuse/Dependence Diagnosis	4	1.2
No Diagnosis	334	98.2
Never diagnosed, but suspect I have the disorder	1	.3
Drug Abuse or Dependence[h]		
Drug Abuse/Dependence Diagnosis	4	1.2
No Diagnosis	332	97.6
Never diagnosed, but suspect I have the disorder	2	.6
Sexual Orientation[i]		
Straight/Heterosexual	303	89.1
Gay/Homosexual	19	5.6
Bisexual	17	5
BMI Category[j]		
Underweight	7	2.1
Normal	193	56.8
Overweight	82	24.1
Obese	58	17.1

Note. Frequencies not summing to 340 and percentages not summing to 100% reflect missing data.

Table 2

Means and Standard Deviations for Continuous Demographic Variables (*N*= 340)

	n	Mean	*SD*	Min	Max
Age	339	19.87	3.74	18	53
Self-Reported Weight	338	151.29	39.63	95	350
Self-Reported Height	339	65.80	3.71	58.5	76.0
Measured Weight	340	154.7	40.66	96.6	357.8
Measured Height	340	65.49	3.87	40.4	75.9
Past Highest BMI	334	26.19	5.97	16.66	50.29
Past Lowest BMI	334	23.02	5.02	13.56	46.11
Ideal BMI	332	22.85	3.85	16.31	65.18
Measured BMI	340	25.15	5.54	16.86	50.79

Note. Height is recorded in inches and weight is recorded in pounds.

Table 3

Means and Standard Deviations of Age, Self-Reported Weight/Height, Measured Weight/Height, Past Highest/Lowest BMI, Ideal BMI, and Measured BMI by Sex

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
Age				-.47	.639				
Male	104	19.92	3.31			18	36	2.75	8.28
Female	235	19.84	3.92			18	53	5.11	34.3
Self-Reported Weight				-7.81	<.001				
Male	104	172.51	44.28			95	350	1.44	2.44
Female	234	141.87	33.39			95	270	1.61	3.01
Self-Reported Height				-19.35	<.001				
Male	104	69.53	2.70			59	76	-.89	1.86
Female	235	64.15	2.79			58.5	72	.23	-.28
Measured Weight				-7.08	<.001				
Male	105	174.31	44.72			97.6	357.8	1.50	2.65
Female	235	145.95	35.45			96.6	287.3	1.53	2.56
Measured Height				-5.58	<.001				
Male	105	69.08	2.85			57.6	75.9	-.85	2.15
Female	235	64.03	2.76			56.8	73	.21	.11
Past Highest BMI				-1.21	.228				
Male	103	27.03	6.68			18.4	49.68	1.28	1.36
Female	231	25.81	5.60			16.7	50.29	1.41	2.61
Past Lowest BMI				-.59	.555				
Male	104	23.45	5.21			15.5	45.65	1.66	3.39
Female	230	22.83	4.93			13.6	46.11	1.50	3.22
Ideal BMI				-3.98	<.001				
Male	104	24.27	3.12			17.8	33.33	.54	.38
Female	228	22.21	3.99			16.3	65.18	5.83	58.9

(table continues)

Table 3 *(continued)*

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
Measured BMI				-.73	.468				
Male	105	25.60	6.01			17.39	50.79	1.67	3.41
Female	235	24.94	5.31			16.86	46.88	1.36	2.21
% Difference of Ideal BMI & BMI*				-5.27	<.001				
Male	104	-3.19	12.12			-49.69	28.11	-.60	1.65
Female	228	-10.28	9.47			-47.79	11.63	-.89	1.38

Note. *Computed by dividing Ideal BMI by Actual BMI and subtracting 1, then multiplying by 100 to get the percentage as a whole number. Positive number indicates the participant prefers to be heavier, 0 indicates the participant is content with weight, and negative number indicates the participant prefers to be lighter.

Table 4

Reliability Analysis of Subscales (N = 340)

Subscale	α
CAARS *Imp/Emo Lab* – (12 items)	.90
CAARS *Hyp/Rest* – (12 items)	.90
CAARS *DSM-IV Hyp/Imp* – (9 items)	.85
CAARS *Inattn/Mem* - (12 items)	.89
CAARS *DSM-IV Inattn* - (9 items)	.91
BADDS *Attn-Con* - (9 items)	.93
ADSA - (13 items)	.87
SCL-90-R *Dep* - (13 items)	.90
BDI-II - (21 items)	.90
HDI-SF - (9 items)	.87
MAST - (24 items)	.76
DAST Parcel 1 - (13 items)	.76
DAST Parcel 1 - (13 items)	.85
BUILT-R Parcel 1- (14 items)	.86
BUILT-R Parcel 2- (14 items)	.84
MEBBIE EBE - (9 items)	.77
EDI-*Bul* - (8 Items)	.82

Note. SCL-90-R *Dep* = Symptom Checklist 90 – Revised, Depression subscale; BDI-II = Beck Depression Inventory – II; BULIT-R = The Bulimia Test – Revised; MEBBIE EBE = Male Eating Behavior and Body Image Evaluation, Emotional and Binge Eating subscale; CAARS *Inattn/Mem* = Conners' Adult ADHD Rating Scales, Inattention/Memory subscale; CAARS *DSM-IV Inattn* = Conners' Adult ADHD Rating Scales, DSM-IV Inattentive subscale; BADDS *Attn-Con* = Brown Attention Deficit Disorder Scale, Attention-Concentration Subscale; ADSA = Attention Deficit Scales for Adults; HDI = Hamilton Depression Inventory-Short Form; MAST = Michigan Alcohol Screening Test; DAST = Drug Abuse Screening Test; EDI-*Bul* = Eating Disorder Inventory, Bulimia Subscale

Table 5

Means and Standard Deviations of Continuous Measures

	n	Mean	*SD*	Min	Max	Skewness	Kurtosis
Impulsive/ Emotional Lability	336	.76	.58	0	2.75	.84	.31
Hyperactivity/ Restlessness	336	1.20	.69	0	3	.44	-.56
DSM-IV Hyp/ Impulsivity	336	.92	.63	0	2.78	.63	-.26
Inattention/ Memory Problems	336	.92	.62	0	2.67	.54	-.38
DSM-IV Inattentive Symptoms	336	.99	.73	0	3	.60	-.43
ADSA	339	1.90	.75	0	4	-.01	-.52
BADDS Attn – Concentration	335	1.42	.85	0	3	.21	-1.08
SCL-90-R Depression	340	.85	.73	0	4	1.18	1.22
BDI-II	339	.47	.39	0	4	1.13	1.38
HDI-SF	340	.61	.47	0	2	1.12	1.31
BULIT-R	340	1.78	.58	1.04	3.93	1.32	1.42
MEBBIE EBE	105	1.89	.76	1	5	1.21	1.21
EDI-Bulimia	234	.41	.57	0	2.88	1.99	4.09
Binge Number	334	3.33	15.86	0	157.5	6.54	48.96
MAST	335	.97	1.64	0	13	3.26	14.80
DAST	334	1.59	2.60	0	24	4.12	24.19
NicPD	340	.96	8.56	0	150	15.91	273.54

Note: CAARS *Imp/Emo Lab* = Conners' Adult ADHD Rating Scales, Impulsivity/ Emotional Lability subscale; CAARS *Hyp/Rest* = Conners' Adult ADHD Rating Scales, Hyperactivity/Restlessness subscale; CAARS *DSM-IV Hyp/Imp* = Conners' Adult ADHD Rating Scales, Hyperactive/Impulsive subscale; CAARS *Inattn/Mem* = Conners' Adult ADHD Rating Scales, Inattention/Memory subscale; CAARS *DSM-IV Inattn* = Conners' Adult ADHD Rating Scales, DSM-IV Inattentive subscale; SCL-90-R *Dep* = Symptom Checklist 90 – Revised, Depression subscale; BDI-II = Beck Depression Inventory – II; BULIT-R = The Bulimia Test – Revised; MEBBIE EBE = Male Eating Behavior and Body Image Evaluation, Emotional and Binge Eating subscale.

Table 6

Frequencies and Percentages ADHD Medication by Sex for Participants with ADHD Diagnosis

	Male		Female			
	n	%	*n*	%	χ^2	*p*
ADHD Medication					.09	.765
ADHD Medication	22	91.7	30	93.8		
No ADHD Medication	2	8.3	2	6.3		

Note. ADHD subsample only

Table 7

Correlation Matrix of Measured Variables (N = 340)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	--	.852**	.733**	.590**	.640**	.698**	.763**	.473**	.417**	.410**	.348**	.409**	.390**	.143**	.190**	.125**	.012
2	.852**	--	.605**	.515**	.612**	.633**	.716**	.418**	.371**	.367**	.266**	.343**	.305**	.162**	.170**	.092	-.042
3	.733**	.605**	--	.604**	.565**	.685**	.681**	.627**	.471**	.524**	.311**	.347**	.354**	.142**	.200**	.110**	-.009
4	.590**	.515**	.604**	--	.786**	.761**	.765**	.535**	.472**	.500**	.383**	.368**	.365**	.135	.142	.050	-.066
5	.640**	.612**	.565**	.786**	--	.742**	.792**	.449**	.440**	.411**	.315**	.333**	.335**	.171**	.190**	.086	-.063
6	.698**	.633**	.685**	.761**	.742**	--	.903**	.521**	.480**	.535**	.358**	.370**	.355**	.131	.188**	.041	-.020
7	.763**	.716**	.681**	.765**	.792**	.903**	--	.494**	.457**	.484**	.344**	.373*	.363**	.132	.192**	.075	-.018
8	.473**	.418**	.627**	.535**	.449**	.521**	.494**	--	.756**	.830**	.414**	.454**	.474**	.220**	.225**	.095	.017
9	.417**	.371**	.471**	.472**	.440**	.480**	.457**	.756**	--	.837**	.403**	.482**	.509**	.177**	.265**	.070	.042
10	.410**	.367**	.524**	.500**	.411**	.535**	.484**	.830**	.837**	--	.404**	.452**	.491**	.195**	.267**	.102	.014
11	.348**	.266**	.311**	.383**	.315**	.358**	.344**	.414**	.403**	.404**	--	.691**	.693**	.078	.107	.123	.219**
12	.409**	.343**	.347**	.368**	.333**	.370**	.373**	.454**	.482**	.452**	.691**	--	.908**	.154**	.219**	.186**	.251**

(table continues)

Table 7 *(continued)*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
13	.390**	.305**	.354**	.365**	.335**	.355**	.363**	.474**	.509**	.491**	.693**	.908**	--	.158**	.235**	.215**	.264**
14	.143**	.162**	.142**	.135	.171**	.131	.132	.220**	.177**	.195**	.078	.154**	.158**	--	.365**	.272**	-.098
15	.190**	.170**	.200**	.142**	.190**	.188**	.192**	.225**	.265**	.267**	.107	.219**	.235**	.365**	--	.539**	-.042
16	.125	.092	.110	.050	.086	.041	.075	.095	.070	.102	.123	.186**	.215**	.272**	.539**	--	-.029
17	.012	-.042	-.009	-.066	-.063	-.020	-.018	.017	.042	.014	.219**	.251**	.264**	-.098	-.042	-.029	--

Note. 1 = Conners' Adult ADHD Rating Scales, DSM-IV Hyperactive/Impulsive subscale; 2 = Conners' Adult ADHD Rating Scales, Hyperactivity/Restlessness subscale; 3 = Conners' Adult ADHD Rating Scales, Impulsivity/ Emotional Lability subscale; 4 = ADSA = Attention Deficit Scales for Adults; 5 = BADDS *Attn-Con* = Brown Attention Deficit Disorder Scale, Attention-Concentration Subscale; 6 = Conners' Adult ADHD Rating Scales, Inattention/Memory; 7 = Conners' Adult ADHD Rating Scales, DSM-IV Inattentive; 8 = Symptom Checklist 90 – Revised, Depression; 9 = Beck Depression Inventory – II; 10 = HDI = Hamilton Depression Inventory-Short Form; 11 = ZMEB/EDI: Standardized Scores for the MEBBIE EBE: Male Eating Behavior and Body Image Evaluation, Emotional and Binge Eating and the EDI: Eating Disorder Inventory; 12 = BULIT-R: The Bulimia Test – Revised Parcel 1; 13 = BULIT-R: The Bulimia Test – Revised Parcel 2; 14 = MAST = Michigan Alcohol Screening Test; 15 = DAST = Drug Abuse Screening Test Parcel 1; 16 = DAST = Drug Abuse Screening Test Parcel 2; 17 = Measured BMI; ** $p < .01$.

Table 8

Fit Indices

Model	χ^2	*df*	*p*	Adj. χ^2 (< 3)	RMSEA	C.I. RMSEA	Adj. R^2	CFI	SRMR	AIC
Measurement Model	250.79	93	< .001	2.69	.071	.060,.081	.644	.981	.046	336.79
Revised Initial Model	293.20	110	< .001	2.66	.070	.060, .080	.614	.978	.065	379.20
Alternative Model	237.09	71	< .001	3.34	.083	.072, .095	.682	.979	.070	305.09

Note: χ^2 = Model Chi-square (larger values suggest that the model does not fit the data), (*df*) = degrees of freedom, *p* = probability, χ^2/df = ratio of chi-square to degrees of freedom (normed chi-square), RMSEA = Residual Mean Squared Error of Approximation (<.06 indicates good fit), C.I. RMSEA = Confidence Intervals of the Residual mean Squared Error of Approximation, Adj. R^2 = amount of variance explained by the model when adjusting for the overall number of parameters, CFI = Comparative Fit Index (> .95 indicates good fit), SRMR = Standardized Root Mean Square Residual (< .08 indicates good fit), AIC = Akaike Information Criterion (lower values indicate good fit).

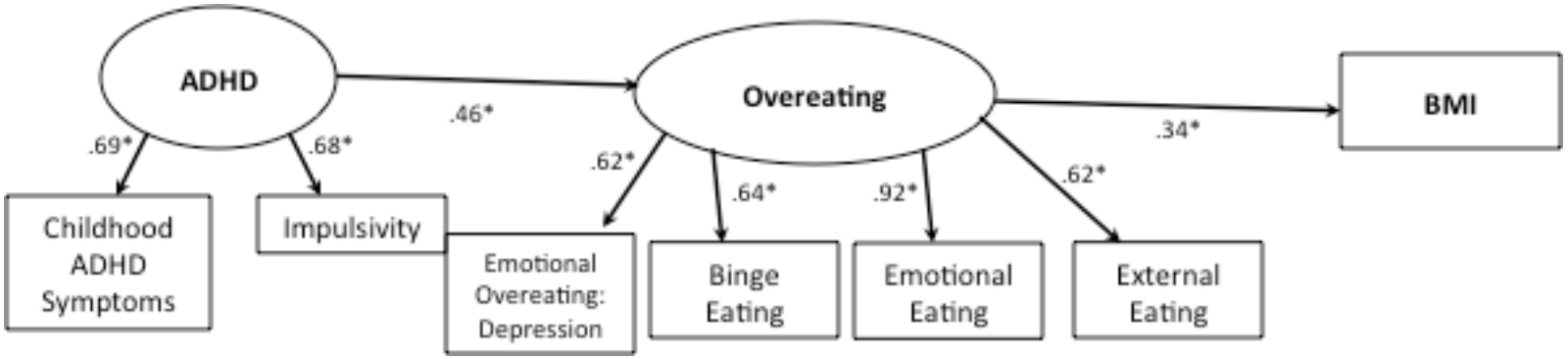

Figure 1. Davis et al. (2006) model.

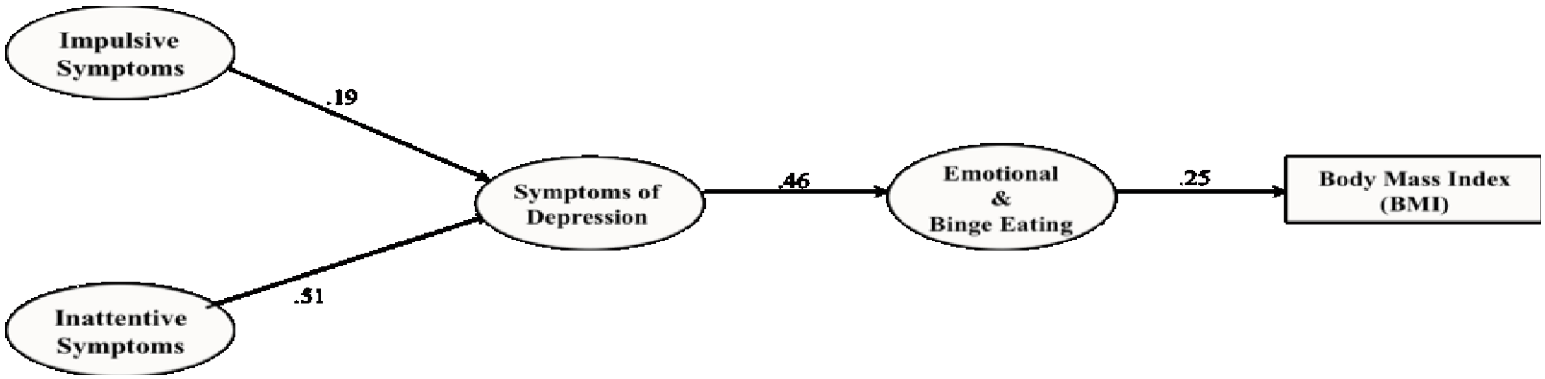

Figure 2. Johnson (2011) initial model; all paths statistically significant at $p < .001$.

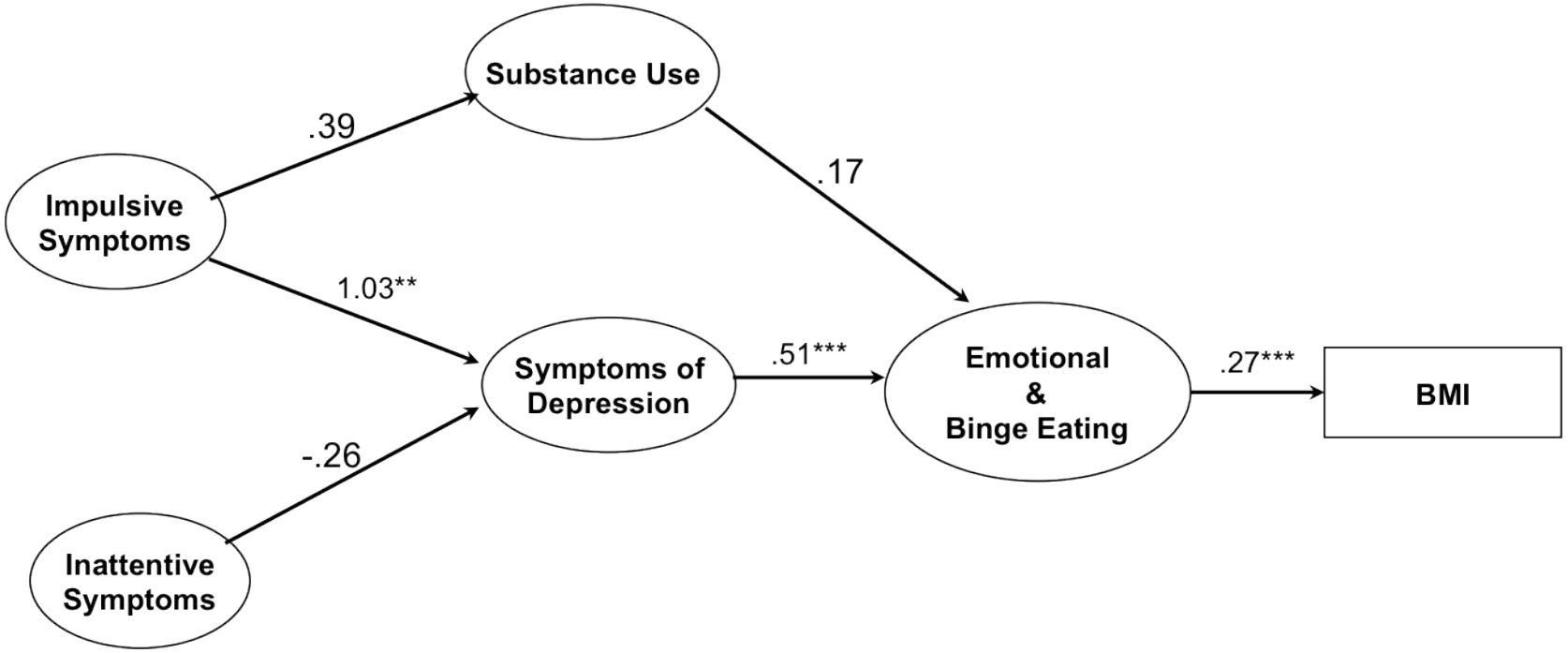

Figure 3. Proposed initial model with ADHD symptoms separated (N = 340). Standardized paths, ** = statistically significant at p < .01. *** = statistically significant at p < .001.

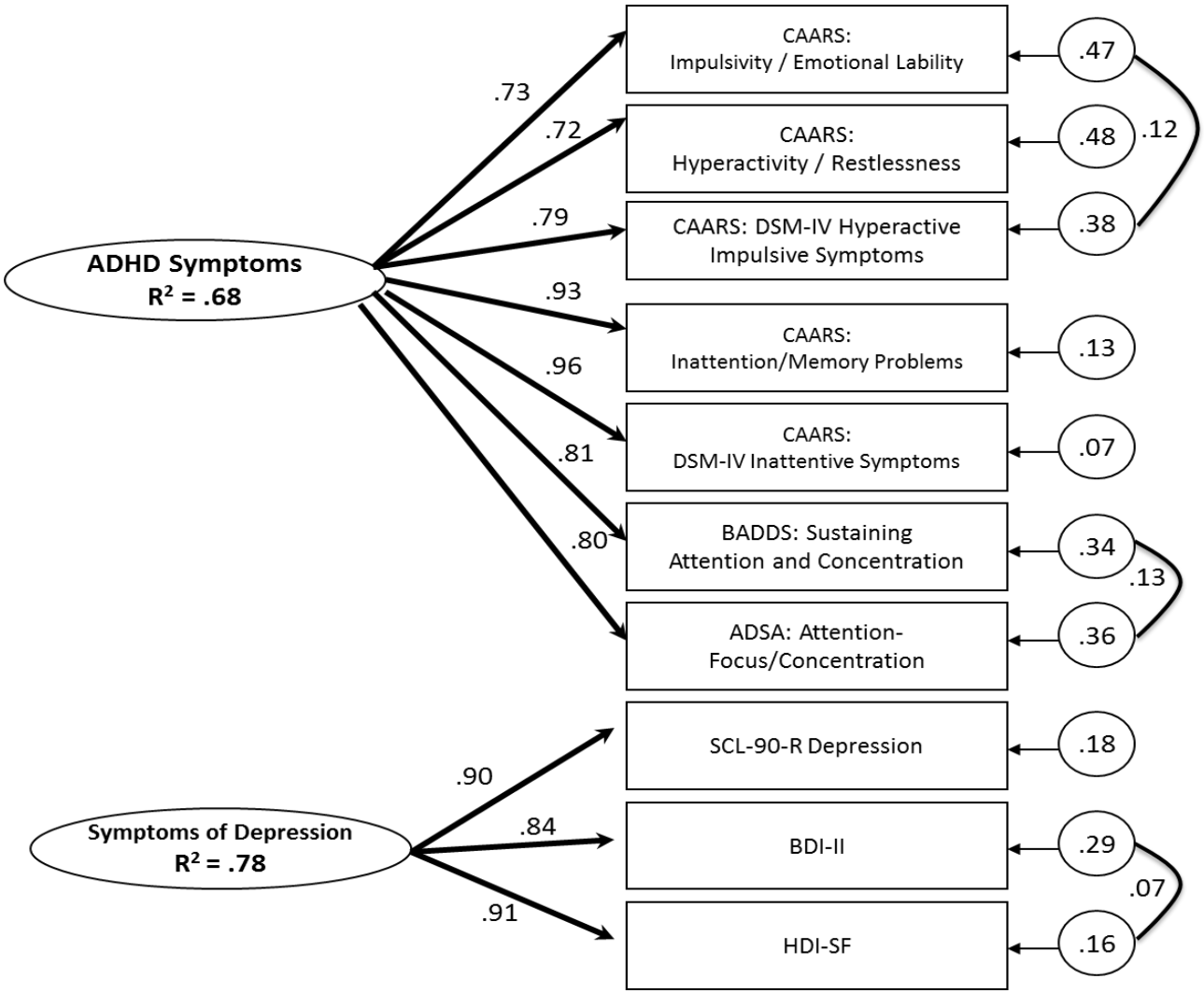

Figure 4. Measurement model fit indices (*N* = 340).

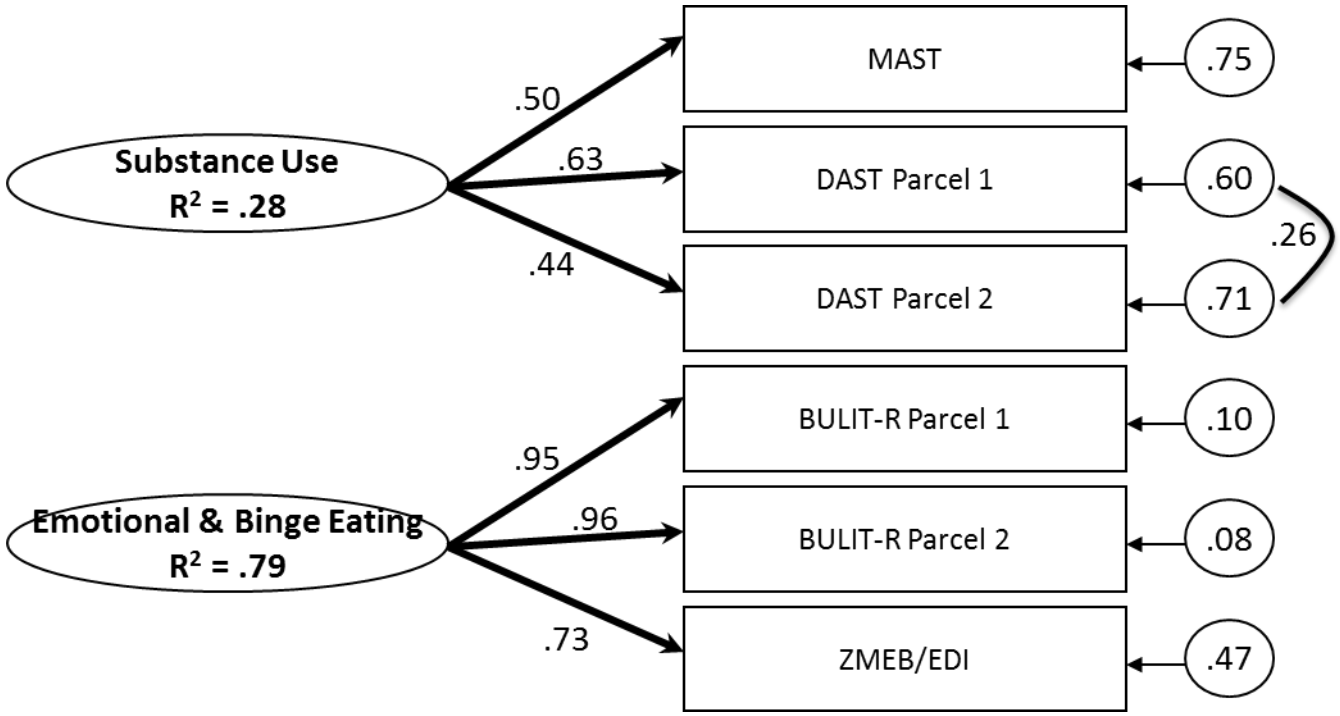

Figure 4 cont. Measurement model fit indices (N = 340).

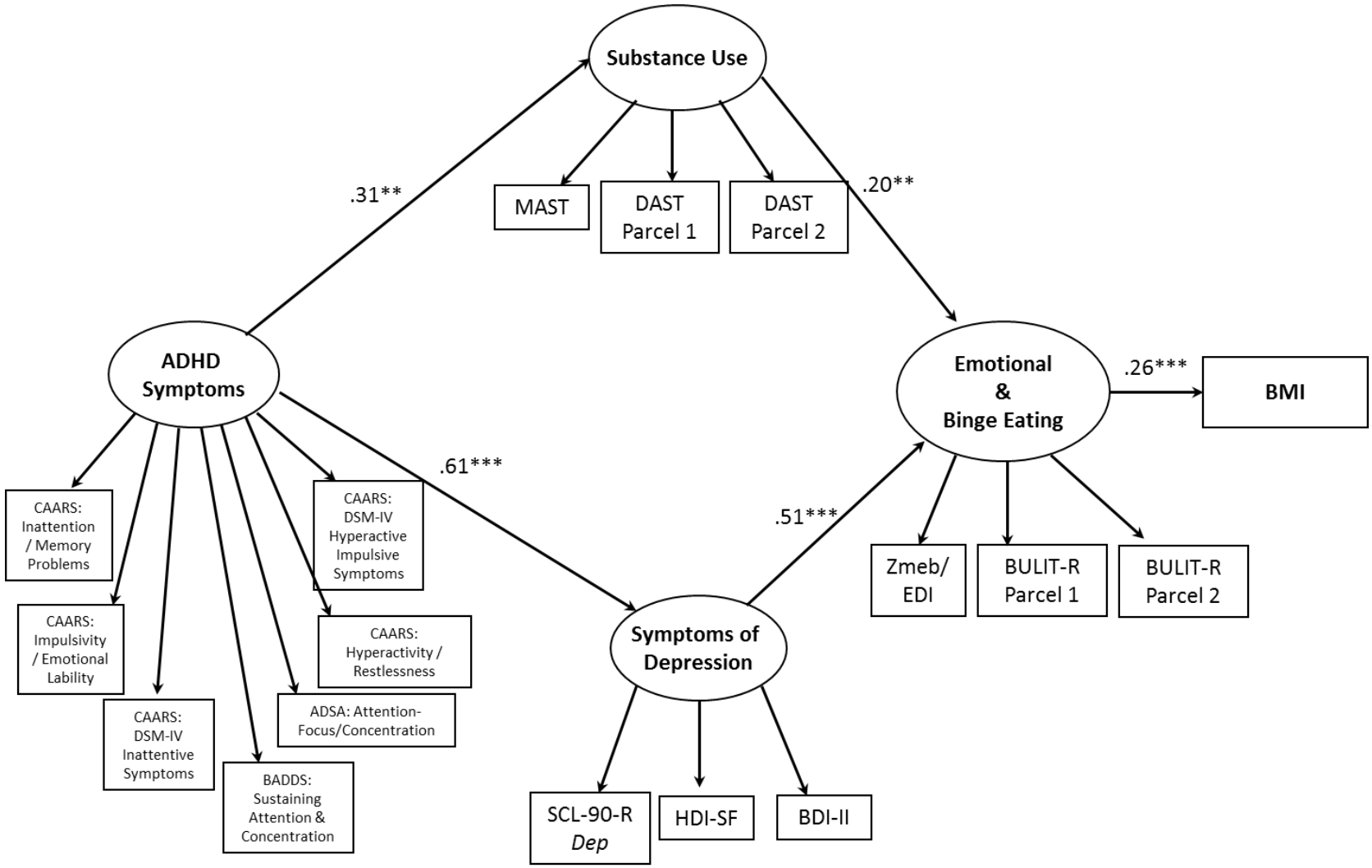

Figure 5. Revised initial model (N = 340). Standardized paths, ** = statistically significant at $p < .01$. *** = statistically significant at $p < .001$.

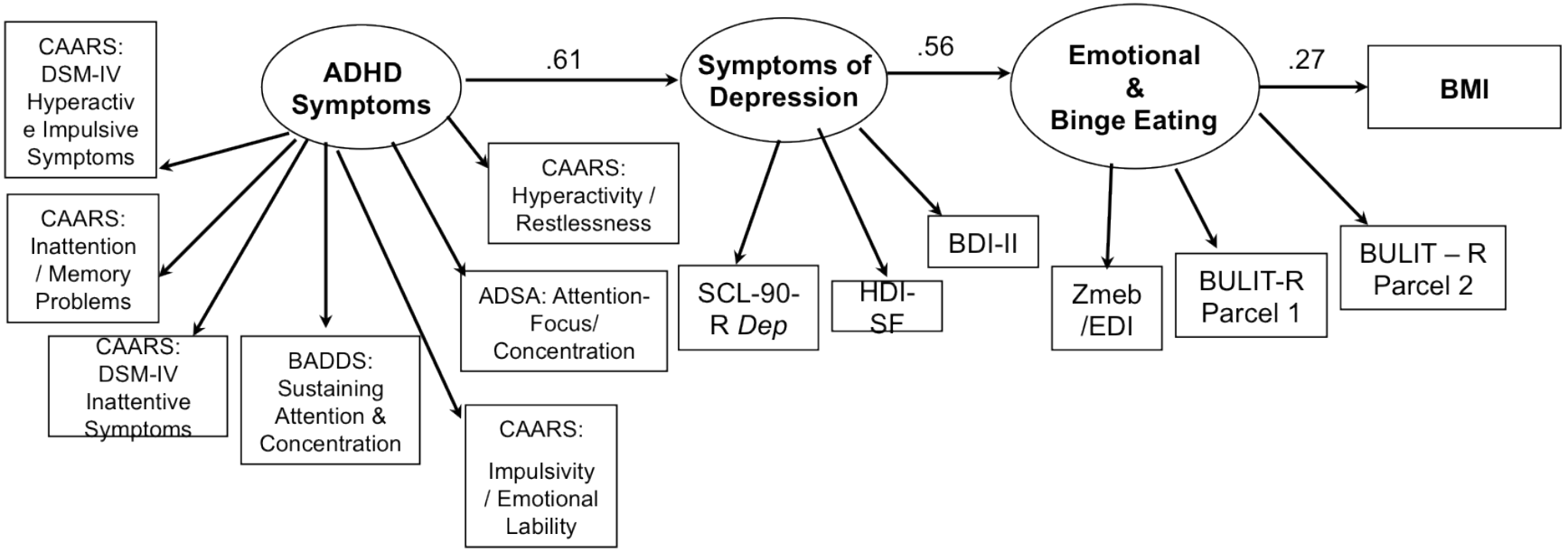

Figure 6. Alternative model (N = 340). All standardized paths are statistically significant at $p < .001$.

APPENDIX A

DEMOGRAPHIC SURVEY

1. Ethnicity: (1) ☐ Asian American (4) ☐ Latin American (Hispanic)
(2) ☐ African American (Black) (5) ☐ Native American (Indian)
(3) ☐ European American (Caucasian) (6) ☐ Other

2. Age: _____ years old

3. Class Rank: (1) ☐ Freshman (3) ☐ Junior

(2) ☐ Sophomore ☐ (4) ☐ Senior ☐

4. In an average week, how much time do you spend working out? (example: lifting weights, cardio, walking) (Please describe)

_____
_____
_____
_____
_____

5. What is your current weight? _____

6. How certain are you about your weight?
(1) ☐ Very uncertain (2) ☐ Somewhat uncertain (3) ☐ Somewhat certain (4) ☐ Very certain

7. How tall are you? Ft._____In._____

8. How certain are you about your height?
(1) ☐ Very uncertain (2) ☐ Somewhat uncertain (3) ☐ Somewhat certain (4) ☐ Very certain

9. What was the most you have ever weighed? _____

10. What was the least you have weighed since your 18th birthday? _____

11. What do you think is your ideal weight? _____

12. How many hours of television do you watch and hours spent on the computer per day?

(1) ☐ Less than 2 (2) ☐ 2 to 4 (3) ☐ 4 to 6 (4) ☐ 7 or more

13. How would you classify your sexual orientation?

(1) ☐ Straight/heterosexual (2) ☐ Gay/homosexual (3) ☐ Bisexual (4) ☐ Questioning/Unsure

14. Have you ever been diagnosed with Attention-Deficit/Hyperactivity Disorder, sometimes called ADHD, ADD, or Hyperactivity?

(1) ☐ Yes (2) ☐ No

15. If answered yes to above question, are you currently taking ADHD medication?

(1) ☐ Yes (2) ☐ No

16. If yes, please list the medication(s) and dosage(s):

---

17. If you answered Yes to question 14, to the best of your recollection, at what age were you diagnosed with ADHD? _____

17a. Who diagnosed you with ADHD? (e.g., psychologist, pediatrician, doctor, etc.)_____

18. Do you currently smoke cigarettes?

(1) ☐ Yes (2) ☐ No

19. If Yes, please specify the brand of cigarettes and how many cigarettes you smoke per day?

---

20. Do you currently chew tobacco, sometimes called ‘dipping’ or ‘chewing?’

---

21. If you answered Yes to question #21, please specify how many times per day that you chew tobacco and, on average, how long (e.g., 30 minutes, 1 hour, etc.)?

APPENDIX B

BODY MASS INDEX (BMI): FOR ADULT MEN & WOMEN

BMI	Weight Status
Below 18.5	Underweight
18.5 – 24.9	Normal
25.0 – 29.9	Overweight
30.0 and Above	Obese

For Adult Men:

To use the BMI chart, find the appropriate height (in inches) in the left-hand column labeled Height. Move across to a given weight (in pounds). If you do not find your weight on the first chart, please consult the following table. The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off.

BMI	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**	**31**	**32**	**33**	**34**
Height (inches)	**Body Weight (pounds)**															
58	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162
59	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168
60	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174
61	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180
62	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186
63	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191
64	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197
65	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204
66	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210
67	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217
68	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223
69	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230
70	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236
71	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243
72	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250
73	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257
74	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264
75	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272
76	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279

To use the BMI chart, find the appropriate height (in inches) in the left-hand column labeled Height. Move across to a given weight (in pounds). If you do not find your weight on the first chart, please consult the following table. The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off.

BMI	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**	**45**	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**
Height (inches)	**Body Weight (pounds)**																			
58	167	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258
59	173	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267
60	179	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276
61	185	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285
62	191	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295
63	197	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304
64	204	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314
65	210	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324
66	216	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334
67	223	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344
68	230	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354
69	236	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365
70	243	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376
71	250	257	265	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386
72	258	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397
73	265	272	280	288	295	302	310	318	325	333	340	348	355	363	371	378	386	393	401	408
74	272	280	287	295	303	311	319	326	334	342	350	358	365	373	381	389	396	404	412	420
75	279	287	295	303	311	319	327	335	343	351	359	367	375	383	391	399	407	415	423	431
76	287	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443

For Adult Women:

	NORMAL						OVERWEIGHT					OBESE										EXTREME OBESITY		
BMI	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
Height (Feet-Inches)	**Weight** (Pounds)																							
4'10"	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167	172	177	181	186	191	196	201
4'11"	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208
5'00"	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179	184	189	194	199	204	209	215
5'01"	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185	190	195	201	206	211	217	222
5'02"	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191	196	202	207	213	218	224	229
5'03"	107	112	118	124	130	135	141	146	152	158	163	169	174	180	186	191	197	203	208	214	220	225	231	237
5'04"	110	116	122	128	134	140	145	151	157	163	169	175	180	186	191	197	204	209	215	221	227	232	238	244
5'05"	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210	216	222	228	234	240	246	252
5'06"	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216	223	229	235	241	247	253	260
5'07"	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223	230	236	242	249	255	261	268
5'08"	125	131	138	144	151	158	164	171	177	184	190	197	204	210	216	223	230	236	243	249	256	262	269	276
5'09"	128	135	142	149	155	162	169	176	182	189	196	203	210	216	223	230	236	243	250	257	263	270	277	284
5'10"	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243	250	257	264	271	278	285	292
5'11"	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250	257	265	272	279	286	293	301
6'00"	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258	265	272	279	287	294	302	309
6'01"	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265	275	280	288	295	302	310	318
6'02"	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272	280	287	295	303	311	319	326
6'03"	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279	287	295	303	311	319	327	335
6'04"	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287	295	304	312	320	328	336	344

Adapted from: George Bray, Pennington Biomedical Research Center, *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report*, National Institutes of Health, National Heart, Lung, and Blood Institute, September 1998.

APPENDIX C

DEBRIEFING STATEMENT

Dear Research Participant:

Thank you for participating in our study. Our goal was to collect data to understand the psychological and social factors that contribute to the link between adult ADHD and overweight/obesity. The majority of research about ADHD focuses on children and adolescents and the research from this study will facilitate a better understanding of disturbances associated with ADHD in an adult population. Further, the research from this study will contribute to the existing literature concerning other variables that may or may not be associated with ADHD in adulthood.

We hope that taking this questionnaire was not stressful for you. Nevertheless taking a questionnaire can cause stress and tension about life problems. If you have any questions about ADHD symptoms, symptoms of depression, or eating disturbances, please let the researcher know right now. We can help you get an appointment with a mental health professional. If you have any questions after you leave today or would like help at a later date, call Dr. Trish Kaminski at (940-565-2671).

The following is a list of names and phone numbers of help lines and agencies that offer counseling and other services to help individuals with problems they might have that are associated with an ADHD diagnosis, as well as eating disturbances.

Counseling and Testing Services (UNT, Denton) – offers personal counseling services on campus to all students at no charge [940-565-2741].

Children and Adults with Attention Deficit/Hyperactivity Disorder (CHADD) – National Resource Center on ADHD – offers one on one conversation with someone who can help to answer your questions related to an ADHD diagnosis [1-800-233-4050].

United Way Crisis Help line – offers one on one conversation with someone who can help to answer your questions, and is toll free [1-800-233-HELP].

National Alliance for The Mentally Ill – offers one on one conversation with someone who can help answer your questions, and is toll free [1-800-950-NAMI].

Massachusetts Eating Disorder Association, Inc. (www.medainc.org) Help line – staffed by trained/supervised individuals that can help you with your questions [617-558-1881].

National Eating Disorders Association (www.nationaleatingdisorders.org) – provides resources, education, and support to individuals affected by eating and body image disturbances [1-800-931-2237]

Psychology Clinic (UNT, Denton) – individual assessment and therapy with fees set according to income level [940-565-2631].

Denton County Friends of the Family, Denton, TX (www.dcfof.org) – offers support and counseling services to those affected by relationship violence and assault [toll free crisis hotline: 1-800-572-4031]

The results of our study will be available to you in the future. If you would like a copy of our results, please give us your address now or contact us at a later date. You may keep this sheet for your records.

Sincerely,

Trish Kaminski, PhD
Associate Professor of Psychology

Leslee Marcom, M.S.
Graduate student

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