ADHD SYMPTOMOLOGY AND OVERWEIGHT AMONG COLLEGE MEN

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Thesis Prepared for the Degree of

MASTER OF SCIENCE

UNIVERSITY OF NORTH TEXAS

December 2011

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Johnson, Leslee M. ADHD Symptomology and Overweight among College Men. Master of Science (Psychology), December 2011, 106 pp., 8 tables, 9 figures, references, 139 titles.

Attention deficit/hyperactivity disorder (ADHD) is a childhood disorder that often persists into adulthood. Among adults, ADHD is highly comorbid with addictive behaviors (e.g., substance abuse and dependence), and depressive disorders. Recently, an association between ADHD and obesity has been reported in the literature; emotional and binge eating may be "addictive behaviors" that contribute to weight gain in this population. The purpose of this study was to test competing models of the hypothesized link between ADHD symptomology and overweight. Specifically, in Model 1, symptoms of depression are expected to mediate the relationship between symptoms of impulsivity and inattention and emotional and binge eating which, in turn, leads to weight gain (i.e., increased BMI). In Model 2, however, the impulsive symptoms have direct relationships with emotional and binge eating in addition to being mediated by depressive symptoms. Structural equation modeling (SEM) was employed to test how the models fit the data of 790 college men. Both models fit the data well, with Model 2 being preferred because of its greater connection to theory. All paths were significant indicating that increased impulsive and inattentive symptoms predicted increased symptoms of depression that, in turn, predicted increased emotional/binge eating, which has a direct and positive relationship with increased BMI. Moreover, impulsive symptoms were also directly related to emotional/binge eating, suggesting different paths to overweight across ADHD subtypes. The findings of the current study elucidate the links between ADHD symptoms and overweight (i.e., increased BMI).

ACKNOWLEDGEMENTS

I would like to express my appreciation for the patience, guidance, and ongoing support provided by my major professor throughout this endeavor. I would also like to thank my family and friends for their ongoing encouragement.

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CHAPTER 1

INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) has been studied in children for decades, with much less emphasis on its persistence into adulthood. ADHD is one of the most common behavioral disorders diagnosed in children and adolescents with a prevalence rate of 8.6%, with males being over-represented among youth who are diagnosed (National Institutes of Health, 2009). Most people with ADHD report continued difficulties into adulthood (Adler, 2008; Kessler et al., 2006). The presence of ADHD among college students has also been reported as 4% (Heiligenstein, Conyers, Berns, & Miller, 1998) and recent studies suggest that between 4% and 5% of adults meet the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition – Text Revision* (*DSM-IV-TR*) criteria for ADHD (Davidson, 2008; Kessler et al., 2006). See Appendix A for a review of ADHD *DSM-IV-TR* criteria, differences between the symptoms in children and adults, possible diagnostic problems with an ADHD diagnosis in adulthood, and common comorbidities with the diagnosis of ADHD.

While numerous psychiatric disorders and conditions have been identified as comorbid, only in recent years have clinicians reported an apparent overrepresentation of overweight and obese people among adults with ADHD (Altfas, 2002; Fleming & Levy, 2002). A few investigators tested this observation empirically and identified emotional/binge eating as a mediating construct of ADHD and overweight. To date, only one mechanism has been put forward to explore the connections between ADHD, disordered eating, and obesity (Davis, Levitan, Smith, Tweed, & Curtis, 2006). Thus, the purpose of the current study is to expand on what little is known to explain the connection between ADHD and obesity in adulthood.

ADHD and Addictive Behavior

There is a fairly extensive body of literature that addresses the links between ADHD, ADHD-like symptoms, and addictive behaviors (Lambert, 2005; Milberger, Biederman, Faraone, Chen & Jones, 1997; Ohlmeier et al., 2007; Pomerleau, Downey, Stelson & Pomerleau, 1995) and a variety of hypotheses have been proposed to explain the high rates of addiction among people with ADHD or ADHD symptoms. The three most prominent explanations are reviewed. These are that addictive behaviors are more likely to develop among people who have trouble with inhibition, addictive behaviors begin as attempts to cope with the stressors associated with having ADHD, and addictive behaviors may alter neurochemistry in ways that offset some of the neurotransmitter anomalies of ADHD.

To become addicted to anything, an individual must first initiate the behavior to which they are addicted. Individuals who struggle with impulse control are more likely to engage in a wide range of risky behaviors, some of which (e.g., illicit drug use) increase the risk for addiction (Kalbag & Levin, 2005; Wilson, 2007) A possible explanation for the high comorbidity between substance abuse and symptoms of ADHD might be the disinhibition symptom that is at the core of the disorder (Barkley, 1999; Dinn et al., 2004; Mason et al., 2007). Disinhibition can be related to both psychoemotional factors and neurochemical factors. Individuals oftentimes know and understand the negative effects of the risky behaviors they are engaging in, but continue the behaviors anyway due to the transient alleviation of distress they are currently experiencing (Dinn et al., 2004). Additionally, individuals with ADHD or ADHD-like symptoms may not contemplate or anticipate the risks or long-term effects (i.e., poor impulse control) of smoking, drinking, or drug use. These individuals might discount future

consequences, only focusing their attention on immediate rewards (i.e., alleviation of the negative symptoms of ADHD).

The symptoms of ADHD may cause daily struggles in the lives of the individuals they affect and, even when treated, people may use substances in an effort to help them cope. In addition, individuals who suffer with ADHD symptoms experience a myriad of secondary stressors (Able, Johnston, Adler, & Swindle, 2007; Wadsworth & Harper, 2007; Wilens, Faraone, & Biederman, 2004). For example, impulsive behaviors increase the likelihood of interpersonal conflict (Ratey, Greenberg, Bemporad, & Lindem, 1992), and problems with organization contribute to daily hassles such as chronically running late, forgetting needed items, and getting traffic tickets (Riccio, Wolfe, Davis, Romine, George, & Lee, 2005). Adults with ADHD are also more likely than adults without the disorder to experience certain negative life events such as divorce (Mccann & Roy-Byrne, 2004; Murphy & Barkley, 1996; Weiss & Hechtman, 1993; Weiss & Murray, 2003), job loss (Able et al., 2007; Barkley, Fischer, Edelbrock, & Smallish, 1990), and driving-related accidents (Murphy & Barkley, 1996). Ohlmeier et al. (2007) suggest that using and abusing substances may be a form of self-soothing, attempting to cope with the aversive symptoms they are feeling. These substances may also act as a temporary escape (i.e., a form of coping, albeit, not the most adaptive) from the adversities associated with ADHD symptomlogy (Dinn et al., 2004; Spencer, Biederman, & Mick, 2007). See Appendix A for a more detailed discussion of empirical studies related to ADHD symptomology and addiction.

In addition to the use of substances in an attempt to cope with stress, some authors have suggested that adolescents and adults with ADHD use substances as a means of self-medication (Dinn et al., 2004; Levin et al., 1996; Mason et al., 2007). Nicotine and some other psychoactive

substances with stimulant properties can reduce ADHD symptoms such as inattention and poor concentration for some people with ADHD (Levin, Conners, Silva, Canu, & March, 2001). Additionally, clinicians have suggested that children may ingest high amounts of caffeine as a form of self-medication prior to being diagnosed with ADHD or taking appropriate medication (e.g., psychostimulants) to treat ADHD symptomology (Leon, 2000).

Many drugs of abuse are stimulants (i.e., nicotine, cocaine, and amphetamines) that elevate mood and increase attention and concentration via neurotransmitters: norepinephrine, serotonin, and dopamine. A common reward deficiency syndrome (RDS) has been hypothesized to be associated with the neurotransmitter anomalies associated with ADHD, drug and alcohol use, and disordered eating (i.e., binge eating and emotional eating). The dopamine D2 receptor (DRD2) is the main neurotransmitter that has been supported by research to be associated with addictive behaviors and compulsive behaviors, such as smoking, alcoholism, drug dependency, pathological gambling, ADHD, and disordered eating that may lead to becoming overweight (Blum, Sheridan, Wood, Braverman, Chen, and Comings, 1995). Insufficient dopamine D2 receptors and tendencies to engage in compulsive rewarding behaviors, such as drug use and overeating characterize the reward deficiency syndrome (Barry, Clarke, & Petry; 2009; Blum et al., 2008).

These studies (see Appendix A) and possible explanations suggest that individuals with ADHD or ADHD symptomology may be at a higher risk to engage in addictive behaviors. Recently, researchers have identified a possible link between ADHD and obesity and suggested binge eating as a type of addictive behavior that could explain the link (Barry, Clarke, & Petry, 2009; Cortese, Bernardo, & Mouren, 2007). Food consumption can no longer be thought of as only a means of providing appropriate nutrients to our bodies. Rather, like substance abuse,

eating can involve disinhibition, be used as a form of coping, and as a form of self-medication (Barry, Clarke, & Petry, 2009). One type of eating that appears to involve disinhibition and may serve as a short-term form of coping and/or self-medication is binge eating. See Appendix A for *DSM-IV-TR* criterion for binge-eating disorder.

There is a case report (see Appendix A for detailed information) in the literature where onset of binge eating occurs when ADHD is no longer managed with medication. According to these authors, the binge eating might have been used as a form of coping (i.e., a compensatory mechanism) with the frustration this girl felt concerning the attention and concentration difficulties she was experiencing. Another hypothesis might be that individuals binge-eat as a form of self-medication. Numerous researchers (Gold, Frost-Pineda, & Jacobs, 2003; Hernandez & Hoebel, 1990) have suggested that food intake, especially food that is high in fat and/or sugar, increases the amount of dopamine released from the presynaptic terminal in the brain. When individuals ingest large amounts of food (e.g. binge eaters), higher concentrations of dopamine are found in the synaptic gap in the brain (Gold, Frost-Pineda & Jacobs, 2003).

ADHD and Obesity

Obesity is an epidemic in the United States, increasingly beginning in childhood and adolescence. Compared to 1980, the percentage of children (ages 6-11 years) classified as obese has doubled, while the percentage of adolescents (ages 12-19 years) classified as obese has tripled (Centers for Disease Control and Prevention, 2010). In 2010, the percentage of adults (i.e. ages 20 and above) who are classified as overweight totaled 68.0% (i.e., males 72.3% and females 64.1%) and those classified as obese totaled 33.8% (i.e., males 32.2% and females 35.5%; Flegal, Carroll, Ogden, & Curtin, 2010). Obese and overweight are both used to classify individuals whose body weight is higher than what is considered healthy for their height. The

body mass index (BMI) has been viewed as the best way to define and classify individuals in specific weight ranges (e.g., overweight versus obese; National Heart Lung and Blood Institute, 2010). It is determined by dividing an individual's weight in kilograms by the square of their height in meters. Overweight and obesity are not mutually exclusive; an individual with a BMI between 25-29.9 is classified as overweight, whereas a BMI of 30 or greater would classify an individual as obese therefore a person who is obese is also overweight (National Heart Lung and Blood Institute, 2010). See Appendix A for a review of health concerns related to overweight and obesity.

One population of children and adolescents who appears to be at an increased risk for obesity is those who have high symptoms of ADHD. For example, in a study involving: 1) adolescents in treatment for obesity; 2) adolescents who were obese but not in treatment, and 3) a normal weight control group from a high school, teens in Group 1 were much more likely to have ADHD (i.e., 13.3%) than adolescents in either of the other groups (ADHD prevalence in Groups 2 and 3 was 3.3% in each group) (Eremis et al., 2004). The Eremis et al. (2004) study suggests a clear link between ADHD and obesity for some adolescents and other researchers report findings that support this association. For example, Cortese et al. (2008) comprehensively reviewed the literature on the connection between ADHD and obesity across all age groups (i.e., 13 of the 15 studies reviewed included participants under age of 18). They excluded "case reports, non-empirical studies," and studies where "ADHD diagnostic criteria" were not used (p. 524). All of the studies reviewed reported that, on average, those diagnosed with ADHD were higher in weight than expected given their height (i.e., according to their calculated BMI) (Agranat-Meged et al., 2005; Biederman et al., 2003; Erermis et al., 2004; Faraone et al., 2005; Holtkamp et al., 2004; Mustillo et al., 2003; Spencer et al., 1996; Swanson et al., 2006).

Scientists who have studied the risk factors of and etiological mechanisms that lead to obesity have identified negative affect, poor impulse control, dopamine deficiencies, and unrestrained eating as important phenomena and mechanisms (Davis, Levitan, Smith, Tweed, Curtis, 2006; Davis et al., 2009; Delahanty, Hayden, Williamson, & Nathan, 2002; Lluch, Herbeth, Mejean, & Siest, 2000; Shepard, 2009; Wansink, Painter, & North, 2005). It is notable that each of these phenomena is also associated with ADHD (American Psychiatric Association, 2000; Davis et al., 2006; Davis et al., 2009; Malloy-Dinz, Fuentes, Leite, Correa & Bechara, 2007).

Indeed, Davis et al. (2006) utilized structural equation modeling (SEM) to test their hypotheses that ADHD symptoms predict abnormal eating patterns and overeating behaviors, which in turn predict overweight and obesity. Davis et al. (2006) utilized a nonclinical sample of women (N = 110) recruited from universities, hospitals, and community centers (see Appendix A for measures used to assess ADHD symptomology and eating behaviors). The researchers hypothesized that ADHD symptoms were positively related to disordered eating (i.e., eating in response to environmental cues, negative mood, and binge eating), which led to increased BMI. Results suggested that all the paths the researchers predicted were statistically significant, and their model fit their data well.

Davis et al. (2006) explain how symptoms of ADHD that might be connected with disordered eating. By definition, most individuals with ADHD struggle with controlling their impulses. If their ADHD manifests in disinhibited eating or impulsive food choices, it is not difficult to see how this symptom of ADHD could indirectly increase overweight and obesity. For example, those with lower impulse control may be more prone to eating when they want to versus when they need to because they are quick to respond to the enticing smells, sights and

tastes of foods. Similarly, disinhibition could explain overeating and even binge eating. Over time, impulsive and/or disinhibited eating could contribute to overweight and obesity.

ADHD coexists with other psychiatric disorders such as major depression and anxiety disorders for approximately 50 – 75% of adults with ADHD (Adler, 2008) and may also contribute to higher risk for overweight and obesity in this population (Davis et al., 2009; Lluch, Herbeth, Mejean, & Siest, 2000). In addition to these obvious sources of negative affect, adults with ADHD are more likely than their non-ADHD peers to experience negative life events such as job loss, marital discord, financial problems, and legal trouble (Biederman, Faraone, & Spencer, 1993, 1994; Murphy & Barkley, 1996). Inordinate stress such as that which accompanies aversive life events and negative affect increase the likelihood for maladaptive coping that may take the form of addictive behaviors such as substance abuse, and of particular interest here, emotional eating and binge eating (Burton, Stice, Bearman, & Rohde, 2007).

Emotional eating can include consuming foods with high glucose and fat content for self-soothing, as well as using food as a coping strategy to control and minimize negative emotions (Goossens, Braet, Van Vlierberghe & Mels, 2009). This type of eating, along with binge eating, might be an effective temporary solution because food overconsumption can temporarily decrease negative affect by altering dopamine concentration in the brain. Just as the reward deficiency syndrome was hypothesized to play a role in the relationship between ADHD and addiction, it is hypothesized to play a similar role between ADHD and binge eating. For example, Blum et al. (2008) studied reward deficiency syndrome (RDS). RDS is a dysfunction of the reward centers in the brain, which involve dopamine transmission. The resulting effect is a lack of receptor sites for the neurotransmitter, dopamine, which results in insufficient amounts of dopamine in the brain. Because of this, individuals who experience this deficit are compelled to

partake in activities that will increase transmission of dopamine, including gambling, risk taking, smoking, disordered eating (i.e., binge eating and emotional eating), and drug and alcohol use. Davis et al. (2006; 2009) provide evidence that individuals with ADHD experience low levels of dopamine, linking ADHD and RDS. Similarly, in a study conducted by Wang et al., (2001), obese individuals had reduced dopamine D2 receptors when compared to the normal weight control group, indicating that these individuals would have to eat larger amounts in order to experience the rewarding effects of dopamine. It could be hypothesized that ADHD individuals might use food as a means to "self-medicate" the low dopaminergic state associated with the disorder. Hoebel, Avena, and Rada (2007) further acknowledge this connection due to the findings that eating food, especially sweet foods, triggers the release of dopamine in the brain, which tends to elevate mood.

ADHD, Obesity and Depression

The literature suggests a connection between ADHD and depressive symptoms (Able et al., 2007; Goodman, 2009; Kessler et al., 2006), while the literature on obesity and depressive symptoms is inconsistent (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Roberts, Deleger, Strawbridge, & Kaplan, 2003; Sammel et al., 2003; Tanofsky-Kraff et al., 2006). Depressive symptoms and disorders are highly comorbid with ADHD symptoms (Able et al., 2007; Goodman, 2009; Kessler et al., 2006), such that a high percentage (18.6%) of individuals diagnosed with ADHD will also be diagnosed with MDD. Further, it is important to note those individuals who experience ADHD symptoms, but have no previous diagnosis, screened positive for current depression more often than those participants who already had a formal ADHD diagnosis (Able

et al., 2007). This may indicate that individuals who experience ADHD symptoms, but no formal diagnosis, might be struggling with current depressive symptoms as well.

Limited research has been conducted on the connections between ADHD and obesity. Davis et al. (2006) suggest the symptoms of ADHD predict overeating, which further and indirectly predicts overweight and obesity. Specifically, the disinhibition, poor planning, impulsivity, inattentiveness, and hypo-dopaminergic levels associated with ADHD could play a role in the overconsumption of food. Notably, the researchers also suggest that many *other* factors; for example, depression, could mediate the relationship between ADHD and obesity (Davis et al., 2006).

The literature on the relationship between obesity and depression is inconsistent. Some studies suggest a relationship between obesity and depression (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Sammel et al., 2003) and others report nonsignificant results (Roberts, Deleger, Strawbridge, & Kaplan, 2003; Tanofsky-Kraff et al., 2006). The inconsistencies may be related to the variety of assessment approaches for measuring BMI. Specifically, some of the studies used self-reported height and weight to calculate BMI. Kvaavik, Tell, and Klepp (2003) document how a downward bias exists when individuals are self-reporting their height and weight (i.e., most individuals overestimate their height and underestimate their weight). Most of the studies assessing the relationship between depression and measured obesity have suggested depression as a risk factor for weight gain and obesity. Specifically, when high levels of depression exist, motivation, planning, impulse control, and activity levels tend to be low, making overconsumption of food and/or poor food choices more likely (Rooke & Thorsteinsson, 2008). Markowitz, Friedman, and Arent (2008) propose a theoretical model of depression and weight

gain that highlights two possible mediators of the link between depression and obesity: physiological means as well as low motivational levels. The "de-motivational means" is of particular importance because it includes symptoms in common with ADHD -- low impulse control, poor organization and planning -- as associated with features of depression that could lead to the overconsumption of food and/or more fattening food choices.

To date, however, researchers have not investigated how ADHD, obesity, and depression interrelate. Although ADHD symptoms and obesity are each associated with depression, depression might also be a condition that helps explain the connection between one or both of the core symptoms of ADHD and obesity. Furthermore, only a few studies have explored the specific relationship between impulsive symptoms and emotional/binge eating and their effects on weight gain. This connection is notable because of the literature findings suggesting strong connections between ADHD and addiction (Lambert, 2005; Ohlmeier et al., 2007) and proposing disordered eating as an addictive behavior (Barry, Clarke, & Petry, 2009; Cortese, Bernardo, & Mouren, 2007). Thus, two separate models were proposed: one with paths from ADHD symptoms predicting symptoms of depression, which, in turn, predicts emotional/binge eating and another that includes an additional path from impulsive symptoms directly to emotional/binge eating. Specifically, the hypotheses were that (a) impulsive and inattentive symptoms would be directly and positively related to symptoms of depression, (b) symptoms of depression would be directly and positively related to emotional and binge eating, and (c) emotional and binge eating would be directly and positively related to increased BMI. An additional path, which was added to the alternative model, hypothesized that impulsive symptoms would also be directly and positively related to emotional and binge eating.

CHAPTER 2

METHOD

Participants

The current study made use of an archival dataset that was recently collected in 2009 and 2010. Participants were recruited from a large university in the southwestern United States in psychology classes that offered extra credit for research participation and through flyers posted on campus. The overall sample contains 665 participants who ranged in age from 18 to 24 years-old. Men 24 years of age and older (n = 14) were excluded from all further analyses because of missing data from the Conners' Adult ADHD Rating Scale (CAARS). As shown in Table 1, almost half of the participants were 18 or 19 years old (n = 181, 27.2% and n = 162, 24.4%, respectively), followed by 20 years old (n = 126, 18.9%), 21 years old (n = 80, 12.0%), 22 years old (n = 47, 7.1%), 23 years old (n = 44, 6.6%), and 24 years old (n = 25, 3.8%). Also shown in Table 1, the majority of participants had never been diagnosed with Attention deficit/hyperactivity disorder (ADHD; N = 543, 81.7%), but 15.0% of the men (n = 100) had been. The remaining 3.3% of the participants did not answer the item about diagnostic history. Due to small group sizes, participants who identified as Native American (n = 2, 0.3%) and Other (n = 22, 3.3%) were excluded from analyses comparing ethnic groups. The majority of participants were European American (n = 400, 60.2%), followed by African American (n = 93, 14.0%), Latin American (n = 95, 14.3%), and Asian American (n = 52, 7.8%). Only one participant did not report his ethnicity. Regarding sexual orientation, 89.5% of the sample identified as heterosexual/straight, 4.5% identified as gay, and 2.7% identified as bisexual, while 3.3% of the participants did not identify their sexual orientation. See Table 1 for frequencies and percentages from Sample 1 and Sample 2.

Means and standard deviations of continuous demographic variables for the overall sample are reported in Table 2. Participants' measured weight ranged from 112 pounds to 385 pounds (M = 177.86, SD = 43.95), whereas their self-reported weight ranged from 110 pounds to 360 pounds (M = 176.31, SD = 40.00). Measured height ranged from 60 inches to 81.6 inches (M = 70.75, SD = 2.90), whereas their self-reported height ranged from 55 inches to 80 inches (M = 70.85, SD = 3.03). When both height and weight as measured by study personnel were used to calculate BMI, it ranged from 16.14 to 59.26 (M = 24.56; SD = 5.27). All BMI variables were calculated using measured height. Past highest BMI ranged from 16.81 to 65.07 (M = 26.20, SD = 6.24), and past lowest BMI ranged from 13.10 to 67.92 (M = 22.74, SD = 4.97). Participants' ideal BMI ranged from 13.95 to 48.80 (M = 23.98, SD = 3.58). See Table 3 for detailed information of the means and standard deviations of the continuous variables in each sample. Independent Samples t tests were conducted to test for difference between these measures and the two random samples. No significant differences were found, all *p*s, *ns*.

Measures

Demographic questionnaire. The Demographic questionnaire (see Appendix D) was prepared specifically for this study in order to have the participants report characteristics such as age, ethnicity, and year in school. The questionnaire also addresses weight, height, sexual orientation, and current level of exercise.

Conners' Adult ADHD Rating Scale (CAARS). The CAARS' subscales were used to represent the impulsive symptoms and inattentive symptoms constructs in the model. Specifically, the impulsivity/emotional lability, hyperactivity/restlessness, and the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition – Text Revision* (*DSM-IV-TR*) hyperactive impulsive symptoms subscales were used to represent the impulsive symptoms

construct in the model. Further, the inattention/memory problems and *DSM-IV-TR* inattentive symptoms subscales were parceled by conducting a factor analysis to create four separate indicators that were equally strong measures of the inattentive symptoms construct. The Conners' Adult ADHD Rating Scale was designed to aid in the diagnosis of ADHD in adults age 18 and above (Conners, Erhardt, & Sparrow, 1999). The 66-item long form of the CAARS was used. It includes four subscales that were derived by factor analysis (Inattention/Memory Problems, Hyperactivity/Restlessness, Impulsivity/Emotional Lability, 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. The purpose of the ADHD Index is to identify individuals who are likely to meet the criteria of ADHD as adults, while the function of the Inconsistency Index is to detect possible response deviations that might be due to random responding or uncooperative behavior.

The CAARS was normed on a large sample (1,026 adults for the self-report form), which consisted of adult community members (i.e., a nonclinical sample) in the United States and Canada (Conners, Erhardt, & Sparrow, 1999). Four age ranges are represented in the normative sample: 18 to 29 years, 30 to 39 years, 40 to 49 years, as well as 50 years of age and older. The CAARS manual does not include ethnicity or specific locations of the normative sample. Internal consistency reliability for the Self-Report forms ranged from $\alpha = .86$ to $\alpha = .92$ (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999), $\alpha = .66$ to $\alpha = .90$ (Conners et al., 1999), and $\alpha = .74$ to $\alpha = .95$ (Alder et al., 2007) in previous studies. Internal consistency reliabilities for the current subsamples were acceptable to excellent ($\alpha = .78$ - $.90$)(see Table 4).

The test-retest coefficients were measured on the long form over a one-month period and proved to be sufficient ranging from $r = .88$ to $r = .91$ (Conners, Erhardt, & Sparrow, 1999;

Erhardt et al, 1999). Additionally, Erhardt et al. (1999) compared the CAARS Self-Report long form to the Wender Utah Rating Scale (Ward, Wender, & Reimharr, 1993), an ADHD rating scale for children. Acceptable moderate correlations (r = .37 to r = .67) between the two measures were found, which provided satisfactory construct validity for the CAARS. Moreover, it is important to note the CAARS is similar to other measures that can be used with multiple reporters in that the strength of the construct validity can very across raters. Nevertheless, the CAARS has been shown to have good internal consistency when used both with self-rated and investigator-rated forms (Alder et al., 2007).

Criterion validity of the CAARS was assessed by comparing CAARS scores of individuals who have been diagnosed with ADHD (met DSM-IV criteria) and non-ADHD individuals (Erhardt et al., 1999). The CAARS has high sensitivity and specificity, correctly identifying 82% of the participants who truly were diagnosed with ADHD and correctly identifying 87% of the participants who did not meet the criteria for ADHD. At the same time, however, the CAARS falsely identified participants with ADHD 13% of the time and failed to notice the disorder 18% of the time.

Symptom Checklist-90-R (SCL-90-R). The SCL-90-R depression subscale was one variable that was used to represent the symptoms of depression construct in the model. This subscale was parceled by conducting a factor analysis to create two indicators that were equally strong measures of the symptoms of depression construct. The SCL-90-R (Derogatis, 1994; See Appendix D) is a self-report 90-item inventory that is used to assess a variety of symptoms within the past 7 days. The DEP subscale will be used in the current study to assess dysphoric mood. Respondents are asked to indicate how much they were distressed by the problems listed in the 90 items based on a 5-point scale (0-4), specifying *not at all*, *a little bit*, *moderately*, *quite*

a bit, and *extremely*. The SCL-90-R has been reviewed and deemed a valuable measure to use in research studies (Pauker, 2004; Payne, 2004).

The SCL-90-R has shown strong reliability and validity throughout, with the depression subscale, in particular, providing impressive results (Derogatis, 1994). Convergent validity between the Beck Depression Inventory and the SCL-90-R has proved to be strong (r = .45 to r = .73), with the highest being on the depression dimension (Gotlib, 1984). Internal consistency is satisfactory with alpha coefficients ranging from α = .77 to α = .90, again with depression being the highest. Internal consistency reliabilities with the current subsamples were considered very good to excellent (α = .80 - .90), (see Table 4). Further, test-retest reliability also proves to be satisfactory with correlation coefficients ranging from r = .68 to r = .90, with 10 weeks between the administration of the SCL-90-R and r = .78 to r = .90, with one week between administrations (Derogatis, Rickels, & Rock, 1976). Derogatis (1994) provides many studies in the SCL-90-R manual to showcase the satisfactory content, convergent, and discriminant validity.

In studies that have used college-aged samples, results have shown that higher symptomology is found when the college sample is compared to adult norms as opposed to the adolescent norms, suggesting that college-aged samples are more closely related to the adolescent norm group than they are to adult norm group (Johnson, Ellison, & Heikkinen, 1989; Todd, Deane, & McKenna, 1997). Further, Johnson et al. (1989) and Todd et al. (1997) encourage researchers to be aware of this increase in symptomology when analyzing the results due to the unique characteristics of college-aged samples. Additionally, the sensitivity of the SCL-90-R with nonpatient undergraduates deemed sufficient, correctly classifying the nonpatient

samples 70% of the time (Todd, Deane & McKenna, 1997). However, it is important to be aware that the SCL-90-R is a screening measure and, therefore, designed to minimize false negatives.

Beck Depression Inventory – 2nd edition (BDI-II). The BDI-II was the other variable that was used to represent the symptoms of depression construct in the model. This subscale was parceled by conducting a factor analysis to create two indicators that were equally strong measures of the symptoms of depression construct. The BDI-II (Beck, Steer, & Brown, 1996; see Appendix C) is a 21-item self-report inventory that is used to measure depression symptomology and severity in association to the *DSM-IV* criteria for depressive disorders and can be used with adolescents (age 13 years and older) and adults. Assessing the severity of symptoms among people already diagnosed with major depression disorder and detecting depression symptoms in the normal population are two ways the BDI-II can be utilized. Each of the 21 items Beck et al. (1996) included on the BDI-II consists of a list of four statements, each representing an increasing level of severity of depressive symptoms. For example, Item 1 is "Sadness: 0: I do not feel sad, 1: I feel sad much of the time, 2: I am sad all of the time, and 3: I am so sad or unhappy I can't stand it," with the level of severity increasing from response 0 to response 3 (Beck, Steer, & Brown, 1996). Respondents choose the statement (by circling the corresponding number beside the statement) that best represents how they have felt during the past two weeks.

Easy and quick administration (5-10 minutes) and scoring are among the strengths associated with the BDI-II. Further, 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). Internal consistency reliability for the BDI-II is excellent ranging from $\alpha = .92$ (outpatient sample) to $\alpha = .93$ (college student sample). Internal consistency reliabilities in the current subsamples ranged

from acceptable to excellent (α = .77 - .90), (see Table 4). Moreover, the test-retest yielded a correlation of r = .93 over a 1-week period, which provides evidence that the respondent's answers will be consistent when repeating the BDI-II. One of the goals of the BDI-II was to assess the *DSM-IV* diagnostic criteria, which provides adequate content validity. Convergent validity 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). The BDI-II showed a stronger positive correlation with the Hamilton Psychiatric Rating Scale for Depression than with the Hamilton Anxiety Rating scale, which provides evidence for its discriminant validity (i.e., anxiety vs. depression). Moreover, concurrent validity between the BDI-II total score proved satisfactory between the State-Trait Depression Inventory (r = .76) and State-Trait Anxiety Inventory (r = .69) factor scores, indicating that the BDI-II correlates well with other depression and anxiety measures (Storch, Roberti, & Roth, 2004).

Additionally, Steer and Clark (1997) administered the BDI-II to a college sample to assess whether the psychometrics were as reliable for the college sample as they were for the psychiatric populations. Although the BDI-II appears to have very good internal consistency reliability with a college student population (α = .93), Steer and Clark warn clinicians to be careful about overinterpreting the changes in appetite and sleep items because inconsistent sleeping and eating habits are common among college students who are not depressed.

The Bulimia Test – Revised (BULIT-R). The BULIT-R was one variable that was used to represent the emotional and binge eating construct in the model. This measure was parceled by conducting a factor analysis to create two indicators that were equally strong measures of the

emotional and binge eating construct. The BULIT-R (Thelen, Mintz, & VanderWal, 1996; See Appendix F) is a 36-item self-report measure used to assess disordered eating habits in clinical and nonclinical populations, specifically bulimia nervosa in relation to the *DSM-IV* criteria. In the current study, the BULIT-R will be used to assess disordered eating, such as binge eating. Internal consistency reliabilities with the current subsamples ranged from acceptable to excellent (α = .78 - .90), (see Table 4). Further, test-retest also proved to be reliable (r = .95) with the nonclinical college female population over a 2-month interval period (Thelen, Farmer, Wonderlich, & Smith, 1991). High reliability coefficients were shown for construct validity between the BULIT-R and the Binge Scale (r = .85) (Hawkins & Clement, 1980), as well as the original BULIT (r = .99) (Smith & Thelen, 1984). Thelen, Mintz, and VanderWal (1996) also showed sufficient construct validity between the BULIT-R and *DSM-IV* criteria with an overall high correlation, r = .73. The BULIT-R has shown to be a cost-effective and valid measure to use in nonclinical populations to assess for bulimia symptomology (Thelen, Mintz, & VanderWal, 1996).

Male Eating Behavior and Body Image Evaluation (MEBBIE). The MEBBIE Emotional and Binge Eating (EBE) subscale was the other variable that was used to represent the emotional and binge eating construct in the model. This subscale was parceled by conducting a factor analysis to create two indicators that were equally strong measures of the emotional and binge eating construct. The MEBBIE (Kaminski & Caster, 1994; Kaminski et al., 2002; See Appendix G) 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 EBE subscale (9 items) assesses an individual's tendency to overeat in response to mood and feelings of losing control.

All MEBBIE subscales have yielded satisfactory internal consistency reliability ranging from $\alpha = .71$ to $\alpha = .92$ (Chapman, Kaminski, Haynes, & Own, 2004; McFarland & Kaminski, 2008). Internal consistency reliabilities in the current subsamples ranged from acceptable to excellent ($\alpha = .74$ - .82), (see Table 4). Moreover, the test-retest reliability demonstrated with a sample of undergraduate men was sufficient with a coefficient of $r = .72$ (Kaminski et al., 2002). Convergent validity was shown between the MEBBIE, EAT, and EDI-2 with the Body Dissatisfaction Scale being significantly correlated ($r = .74$, $p < .01$ and $r = -.65$, $p < .01$) with the equivalent subscales on the EDI-2 and EAT, respectively (see Chapman et al., 2004 & Kaminski et al., 2002 for additional information).

Procedure

Upon receiving IRB approval (see Appendix G), psychology student participants were recruited through the use of flyers (see Appendix H) posted around campus. Recruitment also took place through a website (SONA) utilized by students to receive extra credit in their classes. Participants were given a brief explanation of the study, as well as a detailed consent form. Research assistants (RAs) obtained informed consent before any data was collected. Participants completed one of three counter-balanced questionnaire packets. Each packet contained a demographic questionnaire, the CAARS (Conners, Erhardt, & Sparrow, 2004), BDI-II (Beck, Steer, & Brown, 1996), RSES (Rosenberg, 1965), BULIT-R (Thelen, Mintz, & VanderWal, 1996), SCL-90-R (Derogatis, 1994), and the MEBBIE (Kaminski & Caster, 1994; Kaminski, Slaton, Caster, Own, Baker, & Chapman, 2002). The participants completed four additional measures as part of a larger research project.

After spending 90 – 120 minutes completing the questionnaire packet, each participant was taken by a RA to a private room to have his actual weight and height measured. RAs

repeated measurements and entered their average in the database. The Body Mass Index (see Appendix E) is a measure utilized to classify individuals 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). Participants who are "very muscular" are likely to have high BMIs that would normally place them in an overweight or obese category. To account for this problem, the participants who were classified as "very muscular" were taken out of the current study. Researchers identified the different body types by indicating those body types of famous people through images found in the media. RAs were trained to distinguish between the different body types by practicing differentiating the body types in the pictures of the famous people. Therefore, the RAs recorded the participant's observed body type (OBT; i.e. underweight, low-average, high-average, overweight, very overweight, and very muscular) based on their appearance and training.

Debriefing forms (see Appendix J) were given to each participant and extra credit was assigned as well. In particular, students were given extra credit on SONA, which is a website utilized by universities to provide extra credit to students.

Data Preparation

Before the initial structural model was tested, the data was examined to check the accuracy of data entry, attend to missing values, identify outliers, and test statistical assumptions. Three different RAs checked each participant's responses as compared to the values entered in the dataset to assess for data entry accuracy. If more than 5% of the items on a subscale were missing for a participant, then that participant was deleted from any analysis that required that

subscale (Field, 2009). To replace specific items that were randomly missing, researchers used a procedure called ipsative imputation (Schafer & Graham, 2002) and maximum likelihood estimation (Schlomer, Bauman, & Card, 2010).

Preliminary analyses assessed univariate normality, multivariate normality, and multicollinearity to check for violations of the assumptions required for structural equation modeling. Specifically, skewness and kurtosis values of observed variables were investigated. Overall, absolute kurtosis and skewness values greater than 3 can affect the fit of the SEM model (Kline, 1998; 2005). All of the skewness values were within acceptable range, suggesting no severe deviations from normality (see Table 5). Kurtosis values for the parceled variables were also acceptable with the exception of the BDI-II Parcels, and the BULIT-R Parcel II (Kurtosis = 3.05 – 4.99; see Table 5). Kurtosis values greater than 3 only indicate problems if there are multicollinearity issues, which did not exist among these parcels (Bentler & Chou, 1987). Additionally, means, standard deviations, skewness, and kurtosis for all of the measured variables are reported in Table 5.

For the participants diagnosed with ADHD, the two random samples were compared with ADHD medication status (yes vs. no). A nonparametric χ^2 test of association revealed no significant relationship between the two samples of ADHD diagnosed participants on taking ADHD medication, $\chi^2(1) = .01$, $p = .910$, (see Table 6).

To check for multicollinearity problems, the correlation coefficients, tolerance, and variance inflation factor (VIF) were inspected. Correlation estimates of .85 or higher indicate bivariate relationships that may indicate a multicollinearity problem. 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). Absolute

correlation coefficients among observed variables separately for the two random samples ranged from .008 to .881 (see Table 7), indicating no problem with bivariate multicollinearity, with the exception of the correlations between Hyperactivity/Restlessness subscale and Hyperactive/Impulsive subscale (r = .881), Inattention/Memory Parcel 1 and Inattention/Memory Parcel 2 (r = .864), DSM-IV Inattentive Parcel 1 and DSM-IV Inattentive Parcel 2 (r = .854), Beck Depression Inventory – II Parcel 1and Beck Depression Inventory – II Parcel 2 (r = .852), BULIT-R: The Bulimia Test – Revised Parcel 1 and BULIT-R: The Bulimia Test – Revised Parcel 2 (r = .855) for sample 1, and Beck Depression Inventory – II Parcel 1and Beck Depression Inventory – II Parcel 2 (r = . 878) for random sample 2, indicating potential bivariate collinearity. However, the tolerance values ranged from .221 to .814 and the VIF ranged from 1.76 to 4.25, indicating no multicollinearity problem at the multivariate level.

Analysis

Structural equation modeling (SEM) is a complex, two-step statistical technique that allows researchers to test the relationships between multiple variables simultaneously (Schumacker & Lomax, 2010). The measurement model was tested through confirmatory factor analysis (CFAs) to investigate validation of the proposed constructs of interest using the maximum likelihood (ML) estimation method, assuming multivariate normality. The CFA signifies whether the indicators are adequately measuring the specific latent variables. The second step tested the structural models – both the initial structural model and the alternative structural model.

In the current study, the linear structural relations software (LISREL; Joreskog & Sorbom, 2006) was used to conduct the SEM analysis. LISREL creates a mathematical and statistical model to analyze the structural equation 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. 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. The chi-square statistic is sensitive to sample size, so that, in a large sample, small discrepancies between the initial structural model and the observed data could cause the chi-square to be significant and falsely indicate a poor fit (Martens, 2005; Schumacker & Lomax, 2010). Thus, researchers suggest using additional fit indices to measure a model's fit (Bentler, 2006; Martens, 2005; Worthington & Whittaker, 2006).

In the current study, in order to thoroughly evaluate the goodness of model fit, 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), standardized root mean square residual (SRMR), the comparative fit index (CFI), 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 .90 and lower AIC values indicate good fit (Martens, 2005; Weston & Gore; 2006; Worthington & Whittaker, 2006).

CHAPTER 3

RESULTS

Measurement Model – Sample 1

A confirmatory factor analysis was conducted to test the measurement model on Sample 1 (see Figure 3). The three variables measuring impulsive symptoms (i.e., impulsivity/emotional lability, hyperactivity/restlessness, and *Diagnostic and Statistical Manual of Mental Disorders – 4th Edition – Text Revision* (*DSM-IV-TR*) hyperactive impulsive symptoms) had strong, positive path coefficients to the latent construct and ranged from .77 to .90. The amount of variance accounted for in predicting impulsive symptoms from these three measures was 70%. The two parcels from inattention/memory problems and the two parcels from *DSM-IV-TR* inattentive symptoms positively loaded on the Inattentive Symptoms construct with path coefficients ranging from .88 to .90 and accounting for 78% of the variance. Additionally, the four parcels measuring symptoms of depression (Symptom Checklist-90-R (SCL-90-R) Depression Parcel 1 and 2 and Beck Depression Inventory – 2nd edition (BDI-II) Parcel 1 and 2) had strong, positive path coefficients to the latent construct and ranged from .77 to .84. The amount of variance accounted for in predicting symptoms of depression from these four parcels are 65%. The two parcels from The Bulimia Test – Revised (BULIT-R) and the two parcels from Male Eating Behavior and Body Image Evaluation – Emotional and Binge Eating (MEBBIE EBE) positively loaded on the Emotional and Binge Eating construct with path coefficients ranging from .72 to .83 and accounting for 59% of the variance. As expected, in the measurement model of Sample 1, all indicators loaded positively on the latent constructs. Overall, the measurement model for Sample 1 had good fit (see Table 8).

Alternative Structural Model – Sample 1

The structural model of both the initial and the alternative models (see Figures 4 and 5) exhibited excellent fit. Significant differences were found between the two models, $\Delta\chi^2 = 5.56$, $df = 1$, $p < .05$, and $\Delta AIC = 1.07$, (see Table 8), indicating that the alternative structural model was a better fit than the initial structural model. Although the initial structural model is more parsimonious, the alternative structural model is equally as strong and accounts for more of the theoretical constructs. Past research suggests a link between ADHD symptoms and depressive symptoms (see Figure 4; Able et al., 2006; Goodman, 2009; Kessler et al., 2006). This comorbidity, however, occurs at higher rates among people with predominantly inattentive subtype than either of the other two subtypes where hyperactive and impulsive symptoms are more central (*DSM-IV-TR*, 2000). Moreover, theorists have argued that Attention deficit/hyperactivity disorder (ADHD) sufferers who do not meet hyperactive/impulsive diagnostic criteria are likely coping with a disorder that is sufficiently distinct from the type of ADHD experienced by those who have difficulties with inhibition (Barkley, 1999; Murphy & Barkley, 1996; Able et al., 2007).

The alternative structural model (see Figure 5) includes a direct path from impulsive symptoms (as related to ADHD diagnosis) and emotional and binge eating. This path was hypothesized because poor impulse control may contribute to the overconsumption of food (Rooke & Thorsteinsson, 2008). The fit indices between the initial structural model and alternative structural model were not significantly different, indicating that either model provides a good fit with the sample data. Because the alternative structural model includes another path that is supported by research, it is the model described.

In the alternative structural model (see Figure 5), all pathways are significant at the $p <$ .001 level. Specifically, there are direct and positive relationships between increased impulsive and inattentive symptoms and increased symptoms of depression, which accounted for 65% of the variance in symptoms of depression. Additionally, increased impulsive symptoms had a direct and positive relationship with increased emotional and binge eating, indicating that the relationship between impulsive symptoms and emotional and binge eating is only partially mediated by symptoms of depression. On the other hand, the relationship between inattentive symptoms and emotional and binge eating is fully mediated by symptoms of depression. 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).

Measurement Model – Sample 2

The measurement model from Sample 1 was tested on Sample 2 (see Figure 7). The path coefficients for the impulsive symptoms construct remained strong, ranging from .71 to .83 and the three indicators accounted for 65% of the variance. The two parcels from inattention/memory problems and the two parcels from DSM-IV inattentive symptoms positively loaded on the Inattentive Symptoms construct with path coefficients ranging from .87 to .89 and accounting for 77% of the variance. Additionally, the four parcels measuring symptoms of depression (SCL-90-R Depression parcel 1 and 2 and BDI-II parcel 1 and 2) continued to have strong, positive path coefficients to the latent construct and ranged from .78 to .85. The amount of variance accounted for in predicting symptoms of depression from these four parcels is 66%. The two parcels from BULIT-R and the two parcels from MEBBIE EBE positively loaded on the Emotional and Binge Eating construct with path coefficients ranging from .71 to .80 and accounting for 56% of the

variance. See Figure 7 for more detailed information on the path coefficients of the measurement model on Sample 2. The measurement model for Sample 2 also indicated good fit (see Table 8).

Alternative Structural Model – Sample 2

The alternative structural model was cross-validated on Sample 2 (see Figure 8) and all pathways are significant at the $p < .001$ level. Specifically, there are direct and positive relationships between increased impulsive and inattentive symptoms and increased symptoms of depression, which accounted for 66% of the variance in symptoms of depression. Additionally, increased impulsive symptoms had a direct and positive relationship with increased emotional and binge eating, indicating that the relationship between impulsive symptoms and emotional and binge eating are only partially mediated by symptoms of depression. 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 8). The alternative structural model for Sample 2 indicated good fit (see Table 8).

CHAPTER 4

DISCUSSION

The purpose of this study was to test models of hypothesized pathways between Attention deficit/hyperactivity disorder (ADHD) symptoms and overweight. Past research suggested a link between these two constructs (Davis et al., 2006; Eremis et al., 2004) and the current study was conducted to explore possible pathways that might further explain the relationship between ADHD and overweight. The initial structural model suggested that the relationships of both core symptoms of adult ADHD (i.e., impulsivity and inattention) to emotional/binge eating would be mediated by depression and disordered eating would predict overweight (i.e., increased BMI). The alternative structural model added a direct pathway from impulsive symptoms to emotional/binge eating to test a direct relationship between those latent constructs. Structural equation modeling (SEM) was employed to test the models and a second sample was used to cross-validate the findings. The alternative structural model demonstrated a good fit in both samples and all hypothesized paths were significant.

Based on the results of the current study, ADHD symptomology, specifically impulsivity and inattentiveness, predicts increased symptoms of depression, which, in turn, predicts increased emotional/binge eating, which has a direct and positive relationship with increased BMI. Additionally, an alternative pathway showed a significant relationship between impulsive symptoms and emotional/binge eating, suggesting that impulsive symptoms, regardless of the presence of depressive symptoms, have a direct and positive relationship with emotional/binge eating. All of these findings help elucidate the relationships between ADHD symptoms and overweight (i.e., increased body mass index (BMI). That is, there are at least three pathways and two variables that help explain the relationship between ADHD symptoms and overweight.

Along with the entire model, each pathway provides information about the possible connections and implications between ADHD symptomology and overweight.

ADHD symptoms were directly and positively related to symptoms of depression, indicating that increased symptoms of impulsivity and, separately, inattentiveness, predicted increased symptoms of depression. This finding is consistent with past research that suggests a high comorbidity between ADHD symptomology with depressive symptoms and disorders (Able et al., 2006; Goodman, 2009; Kessler et al., 2006). Further, it is not uncommon for individuals who experience ADHD symptoms (i.e., impulsivity and inattentiveness) without a formal diagnosis, to screen positive for a current depression diagnosis (Able et al., 2006). One explanation for increased current depressive symptoms among people who experience ADHD symptoms is the additional stress experienced due to a chronic condition that can negatively affect interpersonal relationships, job performance, time management, and day-to-day planning and organization (Able et al., 2007; Wadsworth & Harper, 2007; Wilens et al., 2004). This interpretation is consistent with reports of adults with ADHD having lower self-esteem and a poorer quality of life than their non-ADHD peers (Able et al., 2007). These adverse correlates of ADHD symptomology can be further explained by exploring the symptoms of impulsivity and inattentiveness separately.

In adulthood, hyperactive/impulsive behaviors may present themselves differently than in childhood (Faraone, 2000). Hyperactivity in adulthood may appear more like restlessness (Adler, 2008), where adults struggle to stay at their desks or in work meetings, which could potentially lead to job loss. Moreover, adults who experience impulsivity are more likely to experience interpersonal and marital conflict than less impulsive adults (Ratey et al., 1992; Weiss & Murray, 2003) and at a higher frequency than those who do not struggle with ADHD

symptoms (Riccio et al., 2005). Thus, a possible explanation for the relationship between impulsive symptoms and symptoms of depression is that the secondary stressors (e.g., poor performance at work and interpersonal conflict) associated with ADHD symptoms may increase feelings of and/or risk for depression.

Individuals cope with stressors in many different ways and those who experience poor impulse control are more likely to engage in risky behaviors, such as drinking, smoking, and drug use (Kalbag & Levin, 2005; Wilson, 2007). These potentially addictive behaviors may also help explain the link between impulsivity and depression given that there is high comorbidity between addiction and depressive symptoms and disorders (Dinn et al., 2004; Spencer et al., 2007).

In the current study, impulsive symptoms were also directly and positively related to emotional/binge eating. Thus, a potential explanation is that the negative stressors (e.g., job loss, interpersonal conflict) associated with impulsivity might influence individuals to emotionally eat in an effort to feel better. In this way, adults who experience impulsivity might 'self-medicate' with food in order to cope. Emotional eating may be viewed as a form of self-soothing, but binge eating tends to be more complex. Specifically, individuals who engage in binge eating episodes feel a loss of control and disinhibition (Barry et al., 2009), meaning that knowing the negative consequence does not dissuade the compulsive eating behavior. There is also an impulsive component to binge eating, where individuals consume large amounts of food without thinking about it (American Psychiatric Association, 2000). By definition, individuals who struggle with impulsivity tend to act before they think. Eating what looks or smells good without considering hunger or nutrient value would be expected. 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). Furthermore, Davis et al. (2006), suggest that disinhibited eating and impulsive food selection might result in overconsumption of food which, in turn, relates to weight gain (i.e., increased BMI).

A direct and positive relationship was found between inattentive symptoms and symptoms of depression. Although the paths between both ADHD symptoms (i.e., impulsive and inattentive) were significant, the path between inattentive symptoms and symptoms of depression was relatively stronger. Inattentiveness becomes the more dominant symptom of ADHD as individuals enter adulthood (Davidson, 2008; Erk, 2000; Faraone, 2000). Therefore, the adults who reported ADHD symptoms in the current study might have been more likely to struggle with inattentiveness versus impulsivity. Furthermore, adults with inattentive symptoms tend to struggle with poor time management and have difficulty completing tasks (Adler, 2008). Additionally, adults with inattentive symptoms tend to have low levels of motivation and avoid activities (Adler, 2008). An explanation for the strong, positive relationship between inattentive symptoms and symptoms of depression may be that individuals who struggle with their day-to-day organization and time management, as well as interpersonal isolation, might be more likely to experience depressive symptoms related to those stressors.

Based on the findings pertaining to the two main symptoms of ADHD in adulthood (i.e., impulsivity and inattentiveness), it is important to consider how ADHD subtypes may differentially relate to emotional/binge eating. Specifically, the distinguishing factor of ADHD, Combined Type (ADHD-C) from ADHD, Predominantly Inattentive Type (ADHD-I) is that the

latter does not require impulsive symptoms to be present (American Psychiatric Association, 2000). Given the role of impulsive symptoms in the alternative model, adults who meet criteria for ADHD-C might engage in emotional/binge eating in both the presence and absence of depressive symptoms. By contrast, the data suggests that the risk for emotional/binge eating among adults with ADHD-I is only notable when depressive symptoms are present.

As hypothesized, symptoms of depression were directly and positively related to emotional/binge eating, indicating that increased symptoms of depression predicted increased emotional/binge eating. Although the literature is inconsistent on the relationship between depression and emotional/binge eating (Franko et al., 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Sammel et al., 2003), possibly due to depression being oftentimes marked by poor appetite, the results of the current study suggest, at least for some people, there is a relationship between symptoms of depression and emotional/binge eating.

Depression and emotional/binge eating share common criterion. Specifically, hyperphagia is a diagnostic criterion of both disorders and conditions (American Psychiatric Association, 2000), which would further explain the strong relationship between the constructs. Individuals who experience higher levels of depressive symptoms also tend to struggle with low motivation, poor planning, and lower activity levels, which increases the likelihood of overeating (Konttinen, Männistö, Sarlio-Lähteenkorva, Silventoinen, & Haukkala, 2010; Rooke & Thorsteinsson, 2008). Some of those symptoms (i.e., poor planning and impulse control) are shared symptoms with a diagnosis of ADHD, suggesting that those individuals who suffer from ADHD symptomology and depressive symptoms might be more likely to engage in emotional/binge eating behaviors than those who do not experience ADHD and depressive symptoms. Moreover, emotional eating may be used as a means of coping with negative

emotions (i.e., using food to temporarily decrease depressive symptoms). Specifically, some research (e.g., Blum et al., 2008) has focused on the relationship between emotional/binge eating and the reward deficiency syndrome (RDS). RDS involves a dysfunction in the reward system in the brain where relative deficits in dopamine (D2) receptor sites are thought to contribute to dysphoria. Addictive behaviors such as smoking, alcoholism, drug use, gambling, ADHD, and disordered eating (i.e., emotional/binge eating; Barry et al., 2009; Blum et al., 1995; Blum et al., 2008) are thought to explain the increase in D2, which can help decrease symptoms like dysphoria. In particular, this disordered eating increases the likelihood of consuming more food than is nutritionally necessary. Eating food that is high in fat and sugar releases dopamine in the brain, which, in turn, elevates mood (Hoebel et al., 2007).

Past research findings (Millstein, Wilens, Biederman, & Spencer, 1997) suggesting differences between the ADHD subtypes provides another explanation for the direct link between impulsive symptoms and emotional/binge eating. Specifically, adults diagnosed with ADHD-C were also more likely to be diagnosed with a substance use disorder than adults diagnosed with ADHD-I (Millstein et al., 1997). The hyperactive/impulsive symptoms related to the ADHD-C diagnosis might have been more related to addiction and addictive behaviors than the inattentive symptoms related to the ADHD-I diagnosis. Thus, those experiencing impulsive symptoms might be more likely to engage in the addictive act of emotional /binge eating.

Consistent with hypotheses, a direct and positive relationship was found between emotional/binge eating and BMI, indicating that increased emotional/binge eating predicted increased BMI. Individuals who engage in emotional/binge eating behaviors would be more likely to have increased BMIs due to overeating (e.g., a result of disinhibited eating or emotional disequilibrium; Goossens et al., 2008).

Clinical Implications

These findings are of importance to clinicians to increase their awareness of the links between ADHD symptoms and overweight. It will further help clinicians create treatment plans that include skills such as meal planning, impulse control related to food, and better manage overeating behaviors that contribute to weight gain. Further, it would be advantageous for clinicians to understand how the presence of depressive symptoms affects the relationship between ADHD symptomology and overweight. If clinicians are more aware of the connections, aforementioned prevention practices may help clients stay mentally and physically healthier.

Due to ADHD being previously thought of as a childhood disorder, it is also important for clinicians to assess ADHD symptomology and ADHD diagnosis in adulthood. The results of the current study can aid clinicians in being more knowledgeable and aware of the many symptoms that ADHD shares with other disorders. Knowing these connections between multiple disorders, especially between ADHD, depression, and binge eating, may prevent clinicians from overlooking possible dual diagnoses. If clinicians better understand the effects multiple symptoms and disorders have on clients, then they will be more likely to understand how to help alleviate the stressors the clients are experiencing. In turn, the clinicians will be able to explain the connections between the symptoms and diagnoses to the client in hopes that the client will better understand their situation. Oftentimes, clients do not understand the interactions of symptoms and, in turn, feel a loss of hope in making progress in treatment. If clinicians are able to address and explain all the symptoms, there's a possibility treatment outcome might improve.

Limitations and Directions for Future Research

Although the current results were strong and supported both models with significance on all hypothesized paths, it is important to address some limitations of the study. Because the data

was collected for the use with multiple studies, the sample was comprised solely of male college students. While this is not something that creates problems with the analysis, it does limit generalizability. Specifically, researchers were unable to explore gender differences among variables. Future research might explore and compare gender differences by including females in the sample. Furthermore, the sample in the current study was large, but included mostly individuals who identified as European-American (White).

Another limitation of the current study was that researchers explored the possible relationships among symptoms of ADHD, depression, and binge-eating disorder (BED) instead of formal diagnoses. The results of the current study are unable to be generalized to those who are formally diagnosed with ADHD, BED, or depressive disorders. Instead, the results of the current study provide information about the connections and possible relationships between the symptoms of these disorders (i.e., impulsivity, inattentiveness, depressive symptoms, and emotional/binge eating). Future research could replicate the current study with adults who have a formal diagnosis of ADHD and explore the possible relationships and pathways that lead to overweight (i.e., increased BMI).

Another limitation of the current study was the method used to obtain the participant's body mass index. While the current study did not rely on self-report of height and weight (i.e., it was measured with weight and height by RAs), a better and more accurate alternative would have been to assess body fat percentage with calipers using a skin-fold test ("Bye-bye BMI," 2007, Lukasi, 1987). Skin-fold tests using calipers actually pinch the fat away from the bone as a means of providing the most accurate measure of body fat percentage in individuals.

Conclusions

The findings of the current study provide information about the relationships between impulsive symptoms and inattentive symptoms, symptoms of depression, emotional/binge eating and overweight. Symptoms of depression fully mediated the relationship between inattentive symptoms and emotional/binge eating, while it only partially mediated the relationship between impulsive symptoms and emotional/binge eating. Thus, the findings suggest that a diagnosis of ADHD-C and ADHD-I might differ in their connections to overweight. Specifically, ADHD-C is more related to addiction (Millstein et al., 1997) when compared with ADHD-I and those diagnosed with ADHD-C might engage in emotional/binge eating regardless of the absence or presence of depressive symptoms. Furthermore, the findings suggest that adults diagnosed with ADHD-I are at risk for emotional/binge eating only when depressive symptoms are present. Thus, adults diagnosed with ADHD, Combined Type (ADHD-C) might not experience depressive symptoms, but still struggle with emotional/binge eating. The findings of the current study indicate that the link between inattentive symptoms and emotional/binge eating are mediated through a third construct, depressive symptoms. Thus, those who struggle with inattentive symptoms (e.g., adults diagnosed with ADHD-I) are more likely to engage in emotional/binge eating when depressive symptoms are also present. The findings of the current study highlight the connections between ADHD symptoms (i.e., impulsivity and inattentiveness) and overweight, which is a relatively new area of research. Symptoms that are linked to an ADHD diagnosis are correlated with increased weight, which affects the health of individuals. Obtaining more knowledge about the connections between these two constructs is of importance to health providers, clinicians, and clients in an effort for these adults to maintain optimal mental and physical health.

Table 1

Frequencies and Percentages for Categorical Variables

	Total Sample		Random Sample 1		Random Sample 2	
	n	%	*n*	%	*n*	%
Ethnicity[a]						
Asian-American (Asian)	52	7.8	28	8.8	24	7.4
African-American (Black)	93	14.0	51	16.1	42	13.0
European-American (White)	400	60.2	190	59.9	209	64.7
Latin-American (Hispanic)	95	14.3	48	15.1	48	14.9
Native-American (Indian)	2	0.3	1	.3	1	.3
Other	22	3.3	13	3.9	9	2.7
Age[b]						
18 years old	181	27.2	88	26.5	93	27.9
19 years old	162	24.4	83	25.0	78	23.4
20 years old	126	18.9	58	17.5	68	20.4
21 years old	80	12.0	40	12.0	41	12.3
22 years old	47	7.1	30	9.0	17	5.1
23 years old	44	6.6	20	6.0	24	7.2
24 years old	25	3.8	13	3.9	12	3.6
ADHD Diagnosis[c]						
ADHD Diagnosis	100	15.0	54	16.9	46	14.2
No Diagnosis	543	81.7	266	83.1	277	85.8
Sexual Orientation[d]						
Straight/Heterosexual	595	89.5	295	92.2	300	92.9
Gay/Homosexual	30	4.5	15	4.7	15	4.6
Bisexual	18	2.7	10	3.1	8	2.5
BMI Category[e]						
Underweight	29	5.0	13	4.4	16	5.6
Normal	344	58.8	176	59.3	168	58.3
Overweight	133	22.7	57	19.2	76	26.4
Obese	79	13.5	51	17.2	28	9.7

Note. Frequencies not summing to 665 and percentages not summing to 100% reflect missing data. Nonparametric χ^2 for random sample by: [a] ethnicity (does not include Native American or other, $\chi^2(3) = 2.03$, $p = .567$; [b] age $\chi^2(6) = 5.10$, $p = .567$; [c] ADHD diagnosis $\chi^2(1) = .85$, $p = .357$; sexual orientation $\chi^2(2) = .25$, $p = .882$; BMI category $\chi^2(3) = 5.77$, $p = .221$.

Table 2

Means and Standard Deviations for Continuous Demographic Variables (*N*= 665)

	n	Mean	*SD*	Min	Max
Age	665	19.83	1.70	18	24
Self-Reported Weight	642	176.31	40.00	110	360
Self-Reported Height	643	70.75	2.90	60	81.6
Measured Weight	642	177.86	43.95	112	385
Measured Height	641	70.85	3.03	55	80
Past Highest BMI	615	26.25	6.21	16.81	67.92
Past Lowest BMI	610	22.67	4.78	13.10	53.45
Ideal BMI	612	23.98	3.58	13.95	48.80
Measured BMI	589	24.56	5.27	16.14	59.26
% Difference of Ideal BMI & BMI	584	-.63	2.76	-20.80	6.26

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

Table 3

Means and Standard Deviations of Age, Measured Height and Measured BMI by Random Samples

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
Age				.47	.636				
RS1	332	19.86	1.72			18	24	.77	-.35
RS2	333	19.80	1.69			18	24	.85	-.12
Self-Reported Weight				1.40	.161				
RS1	320	178.41	43.47			110	360	1.34	1.81
RS2	322	173.97	36.47			110	347	1.35	3.05
Self-Reported Height				.28	.780				
RS1	320	70.78	2.30			61	81.6	-.02	.45
RS2	323	70.71	2.88			60	81	-.07	.67
Measured Weight				1.57	.116				
RS1	322	180.44	47.71			112	364	1.48	2.25
RS2	320	175.01	39.34			113	385	1.55	4.06
Measured Height				.49	.628				
RS1	322	70.90	3.04			55	79.5	-.47	2.25
RS2	319	70.78	3.15			55	80	-.27	2.09
Past Highest BMI				.52	.601				
RS1	308	26.38	6.04			16.81	52.37	1.14	1.30
RS2	307	26.12	6.38			16.95	65.07	1.88	7.47
Past Lowest BMI				1.43	.153				
RS1	304	22.95	4.95			13.75	43.04	1.38	2.13
RS2	306	22.40	4.59			13.1	67.92	2.28	9.76
Ideal BMI				.73	.467				
RS1	308	24.07	3.70			13.95	36.26	.69	.63
RS2	304	23.86	3.59			16.05	48.8	2.11	9.99

Table 3 (*continued*).

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
Measured BMI				1.56	.120				
RS1	299	24.88	5.51			16.14	51.65	1.41	2.39
RS2	290	24.21	4.83			16.74	59.26	2.13	9.23
% Difference of Ideal BMI & BMI*				-1.59	.113				
RS1	297	-.81	3.01			-20.8	5	-2.24	8.60
RS2	287	-.44	2.48			-12.55	6.26	-1.36	4.33

Note. RS1 = Random Sample 1; RS2 = Random Sample 2. *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 and Parcels for Sample 1 (N = 332) and Sample 2 (N = 333)

Subscale	Random Sample 1 α	Random Sample 2 α
CAARS *Imp/Emo Lab* – (12 items)	.90	.85
CAARS *Hyp/Rest* – (12 items)	.89	.87
CAARS *DSM-IV Hyp/Imp* – (9 items)	.87	.81
CAARS *Inattn/Mem* - All Items (12 items)	.90	.88
CAARS *Inattn/Mem* - Parcel 1 (6 items)	.80	.79
CAARS *Inattn/Mem* - Parcel 2 (6 items)	.82	.78
CAARS *DSM-IV Inattn* - All Items (9 items)	.90	.89
CAARS *DSM-IV Inattn* - Parcel 1 (4 items)	.79	.79
CAARS *DSM-IV Inattn* - Parcel 2 (5 items)	.82	.81
SCL-90-R *Dep* - All Items (13 items)	.89	.90
SCL-90-R *Dep* - Parcel 1 (7 items)	.80	.82
SCL-90-R *Dep* - Parcel 2 (6 items)	.81	.83
BDI-II - All Items (21 items)	.88	.90
BDI-II - Parcel 1 (11 items)	.78	.81
BDI-II - Parcel 2 (10 items)	.77	.83
BUILT-R - All Items (28 items)	.89	.90
BUILT-R - Parcel 1 (14 items)	.82	.81
BUILT-R - Parcel 2 (14 items)	.78	.82
MEBBIE EBE - All Items (9 items)	.82	.80
MEBBIE EBE - Parcel 1 (4 items)	.79	.78
MEBBIE EBE - Parcel 2 (5 items)	.79	.74

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

Table 5

Means and Standard Deviations of Continuous Measures by Random Samples

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
Impulsive/ Emotional Lability				-.32	.750				
RS1	332	.71	.54			0	2.83	1.00	.98
RS2	330	.72	.49			0	2.58	.90	.86
Hyperactivity/ Restlessness				-.28	.779				
RS1	332	1.10	.62			0	3	.71	.31
RS2	330	1.11	.59			0	2.75	.48	-.38
DSM-IV Hyperactive/ Impulsivity				-.21	.833				
RS1	332	.78	.60			0	2.89	1.04	1.03
RS2	330	.79	.54			0	2.67	.63	-.18
Inattention/ Memory Problems P1				-.02	.988				
RS1	332	.95	.61			0	3	.80	.59
RS2	330	.95	.61			0	2.83	.59	-.10
Inattention/ Memory Problems P 2				.70	.487				
RS1	332	1.01	.67			0	3	.51	-.37
RS2	330	.97	.62			0	2.83	.44	-.55
DSM-IV Inattentive Symptoms P1				-.82	.413				
RS1	326	.92	.71			0	3	.83	.33
RS2	324	.96	.71			0	3	.52	-.41
DSM-IV Inattentive Symptoms P2				-.28	.780				
RS1	326	.91	.67			0	3	.86	.42
RS2	324	.93	.64			0	3	.66	.09
SCL-90-R Depression P1				.40	.693				
RS1	331	.96	.74			0	3.5	1.02	.78
RS2	332	.94	.75			0	3.3	.78	.03

(*table continues)*

Table 5 (*continued*).

	n	Mean	*SD*	*t*	*p*	Min	Max	Skewness	Kurtosis
SCL-90-R									
Depression P2				-.68	.494				
RS1	331	.68	.67			0	3.67	1.36	1.94
RS 2	332	.72	.75			0	3.83	1.37	1.72
BDI-II P1				1.15	.251				
RS1	332	.48	.38			0	2	1.13	1.38
RS2	332	.44	.37			0	2.27	1.44	3.05
BDI-II P2				.75	.456				
RS1	332	.43	.37			0	2.33	1.46	3.16
RS2	332	.41	.40			0	2.6	1.78	4.99
BULIT-R P1				1.20	.229				
RS1	331	1.83	.56			1	4.21	1.26	1.56
RS2	333	1.78	.53			1	4	1.29	1.74
BULIT-R P2				1.60	.111				
RS1	331	1.83	.48			1	3.93	1.50	3.11
RS2	333	1.78	.50			1	4.14	1.80	4.81
MEBBIE EBE P1				1.25	.212				
RS1	332	2.22	.91			1	5.5	.70	.07
RS2	332	2.13	.86			1	5.25	.74	.20
MEBBIE EBE P2				.10	.917				
RS1	332	1.92	.79			1	5.6	1.25	2.18
RS2	332	1.91	.70			1	4.4	.71	.13

Note: RS = Random Sample; P = Parcel 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 Random Sample for Participants with ADHD Diagnosis

	Random Sample 1		Random Sample 2			
	n	%	*n*	%	χ^2	*p*
ADHD Medication					.01	.910
ADHD Medication	17	31.5	14	30.4		
No ADHD Medication	37	68.5	32	69.6		

Note. ADHD subsample only

Table 7

Correlation Matrix of Measured Variables in Sample 1 (N = 332) and Sample 2 (N = 333)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	--	.486**	.756**	.547**	.617**	.570**	.498**	.491**	.523**	.553**	.564**	.204	.223	.337**	.287*	-.026
2	.724**	--	.751**	.571**	.482**	.467**	.613**	.234*	.208	.273*	.255*	.155	.037	.135	.278*	.132
3	.811**	.881**	--	.708**	.706**	.699**	.723**	.357**	.397**	.459**	.394**	.163	.087	.141	.256*	.091
4	.680**	.634**	.634**	--	.778**	.718**	.797**	.507**	.477**	.544**	.454**	.212	.122	.170	.255*	.051
5	.748**	.650**	.664**	.864**	--	.820**	.791**	.469**	.492**	.562**	.468**	.229	.185	.219	.358**	.054
6	.706**	.684**	.653**	.831**	.846**	--	.769**	.361**	.493**	.495**	.437**	.199	.269*	.195	.236*	-.007
7	.767**	.702**	.691**	.826**	.841**	.854**	--	.451**	.454**	.479**	.435**	.254*	.175	.149	.294*	.045
8	.536**	.381**	.399**	.518**	.573**	.561**	.520**	--	.750**	.649**	.671**	.344**	.252*	.333**	.355**	.065
9	.540**	.365**	.404**	.547**	.547**	.479**	.499**	.786**	--	.727**	.661**	.276*	.232	.277*	.294*	-.036
10	.593**	.488**	.484**	.564**	.586**	.578**	.560**	.647**	.643**	--	.878**	.313**	.285*	.289*	.194	-.011
11	.538**	.381**	.395**	.506**	.504**	.484**	.485**	.611**	.664**	.852**	--	.429**	.407**	.404**	.287*	.080

(*table continues*)

Table 7 (*continued*)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
12	.485**	.427**	.472**	.400**	.440**	.385**	.403**	.340**	.375**	.461**	.339**	--	.786**	.517**	.545**	.328**
13	.502**	.473**	.534**	.354**	.400**	.387**	.386**	.212*	.259**	.406**	.294**	.855**	--	.501**	.352**	.219
14	.306**	.333**	.321**	.173	.245**	.271**	.233**	.293**	.278**	.284**	.156	.458**	.468**	--	.598**	.276*
15	.312**	.301**	.309**	.264**	.291**	.222*	.282**	.243**	.314**	.251**	.177*	.550**	.537**	.573**	--	.217
16	.046	.073	.105	.008	.024	-.056	-.046	-.024	.052	.060	-.009	.197*	.197*	.173	.069	--

Note. Sample 1 correlations are shown below the diagonal and Sample 2 above the diagonal. 1 = Conners' Adult ADHD Rating Scales, Impulsivity/ Emotional Lability subscale; 2 = Conners' Adult ADHD Rating Scales, Hyperactivity/Restlessness subscale; 3 = Conners' Adult ADHD Rating Scales, Hyperactive/Impulsive subscale; 4 = Conners' Adult ADHD Rating Scales, Inattention/Memory Parcel 1; 5 = Conners' Adult ADHD Rating Scales, Inattention/Memory Parcel 2; 6 = Conners' Adult ADHD Rating Scales, DSM-IV Inattentive Parcel 1; 7 = Conners' Adult ADHD Rating Scales, DSM-IV Inattentive Parcel 2; 8 = Symptom Checklist 90 – Revised, Depression Parcel 1; 9 = Symptom Checklist 90 – Revised, Depression Parcel 2; 10 = Beck Depression Inventory – II Parcel 1; 11 = Beck Depression Inventory – II Parcel 2; 12 = BULIT-R: The Bulimia Test – Revised Parcel 1; 13 = BULIT-R: The Bulimia Test – Revised Parcel 2; 14 = MEBBIE EBE: Male Eating Behavior and Body Image Evaluation, Emotional and Binge Eating Parcel 1; 15 = MEBBIE EBE: Male Eating Behavior and Body Image Evaluation, Emotional and Binge Eating Parcel 2; 16 = Measured BMI; * $p < .05$; ** $p < .01$.

Table 8

Fit Indices

Model	χ^2	*df*	*p*	Adj. χ^2 (< 3)	RMSEA	R^2	CFI	SRMR	AIC
Random Sample 1									
Measurement Model	163.22	76	< .001	2.14	.042	.682	.943	.033	75.75
Proposed Model	187.54	91	< .001	2.06	.040	.751	.980	.032	83.68
Alternative Model	181.98	90	< .001	2.02	.039	.751	.986	.031	82.61
Random Sample2									
Measurement Model	127.40	76	< .001	1.67	.045	.667	.893	.031	64.76
Alternative Model	138.23	90	< .001	1.53	.040	.725	.913	.026	69.22

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), R^2 = amount of variance explained by the model, CFI = Comparative Fit Index (> .90 indicates good fit), SRMR = Standardized Root Mean Square Residual (< .08 indicates good fit), AIC = Akaike Information Criterion (lower values indicate good fit.

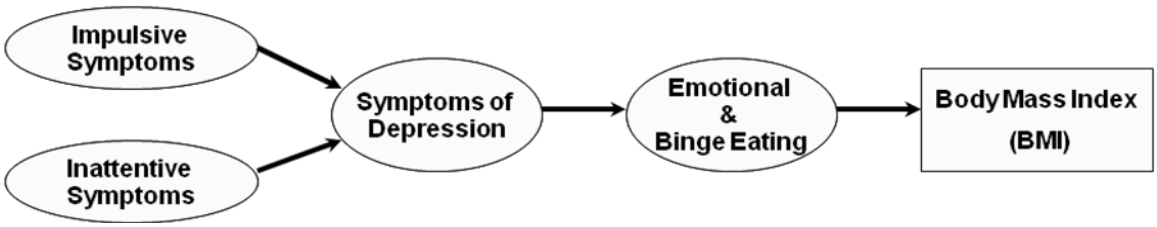

Figure 1. Initial structural model.

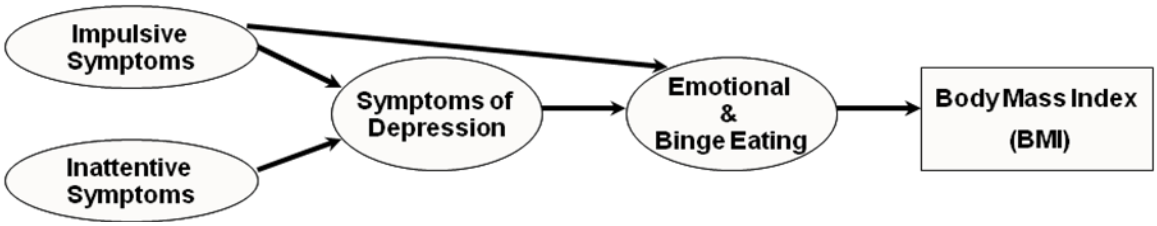

Figure 2. Alternative structural model.

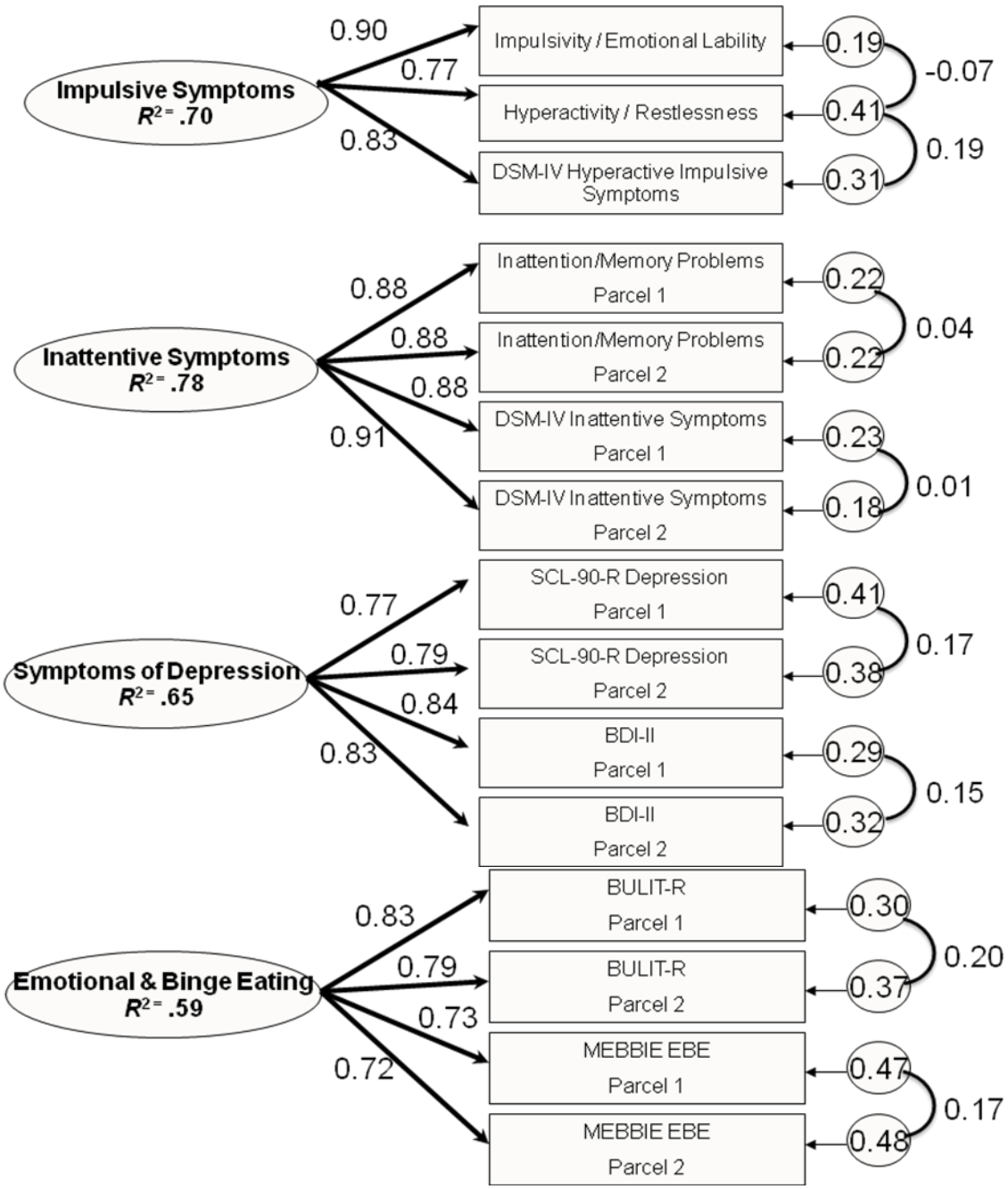

Figure 3. Measurement model fit indices for Random Sample 1 (N = 322)

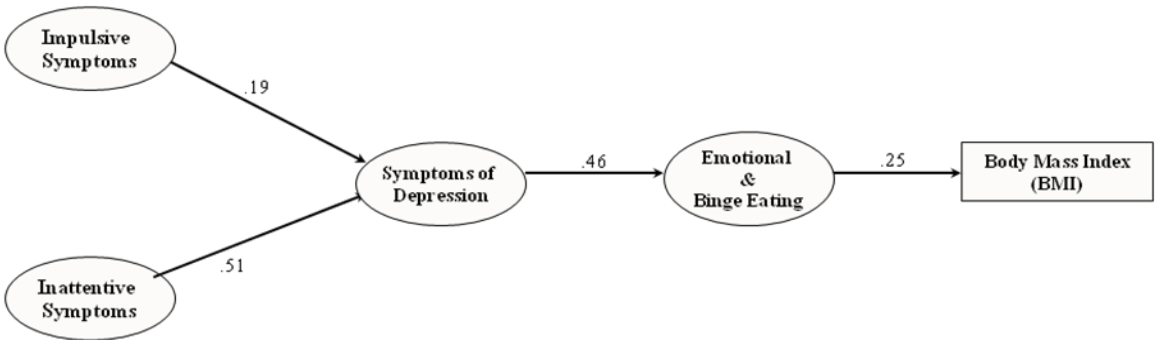

Figure 4. Initial structural model for Random Sample 1 (N = 322). All coefficients and paths are significant at $p < .001$.

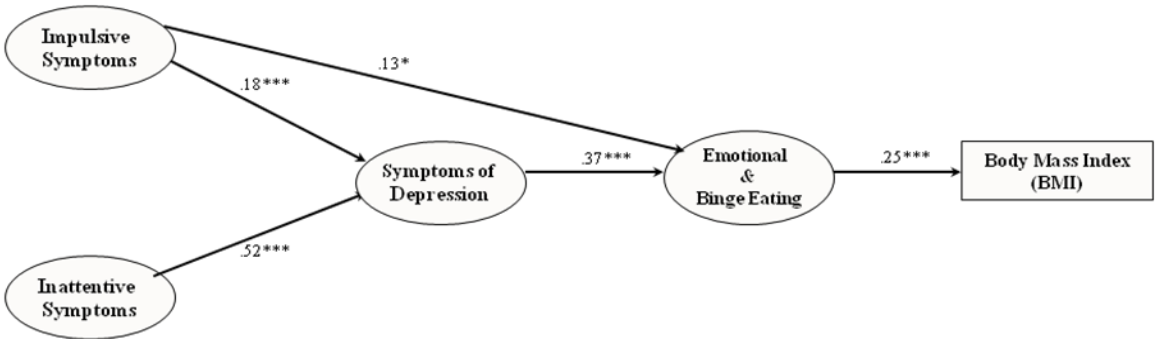

Figure 5. Alternative structural model for Random Sample 1 (N = 322). * = significance at $p < .05$. *** = significance at $p < .001$.

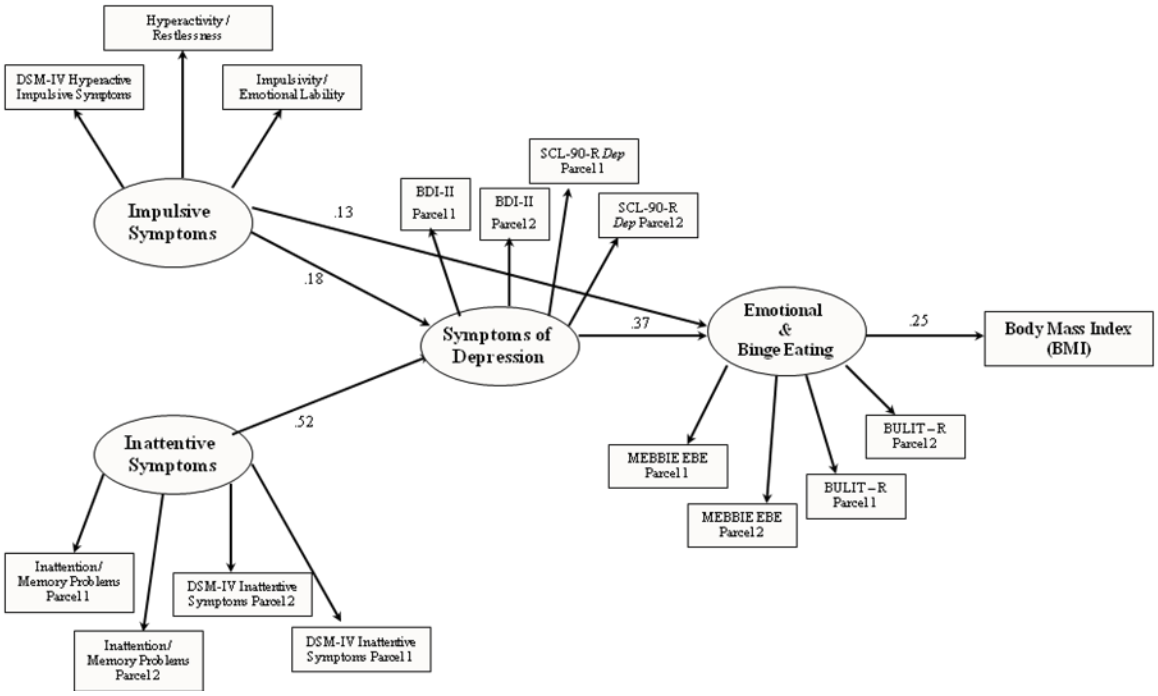

Figure 6. Alternative structural model fit indices for Sample 1 ($N = 322$). All coefficients and paths are significant at $p < .001$.

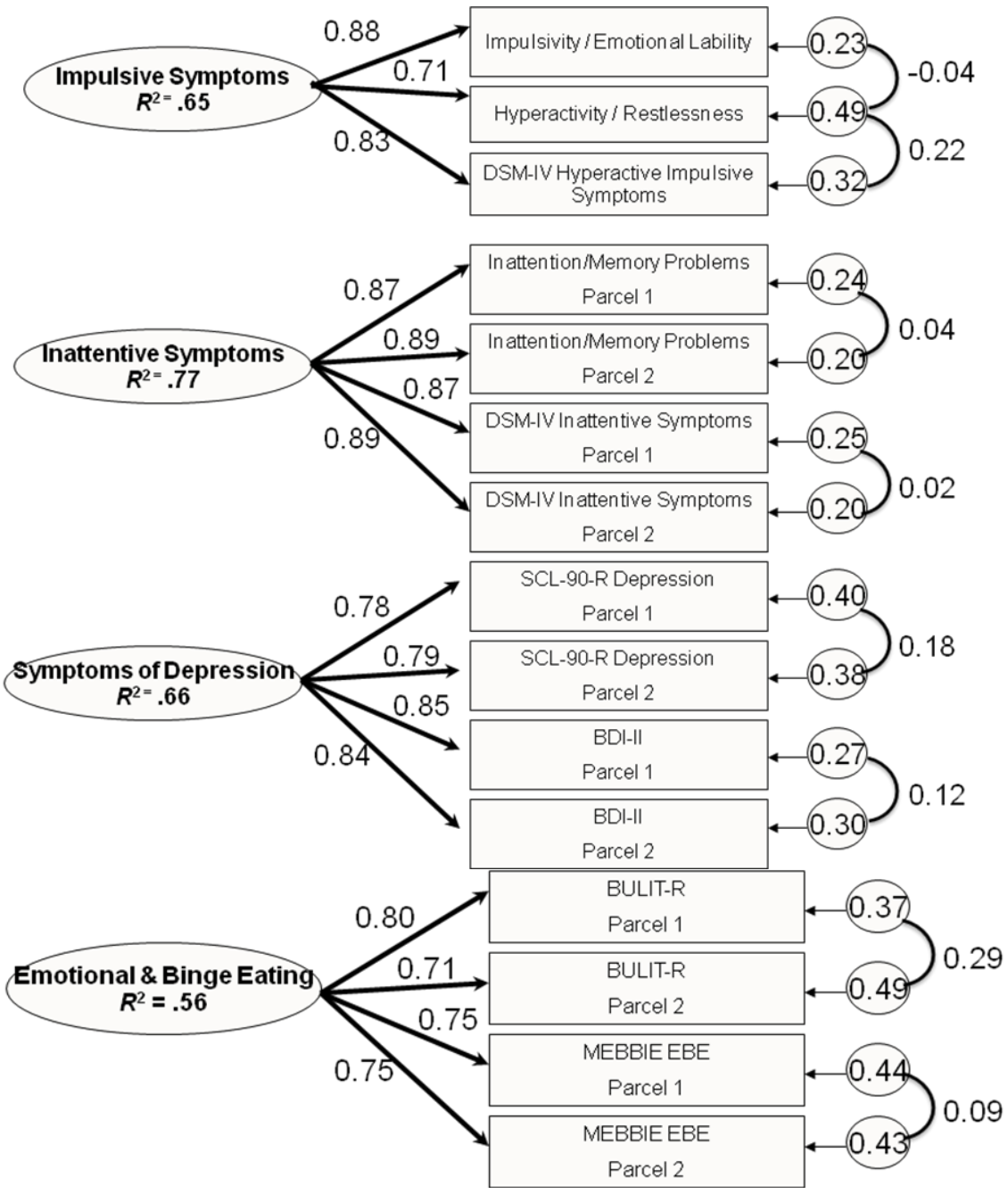

Figure 7. Measurement model fit indices for Random Sample 2 (N = 333)

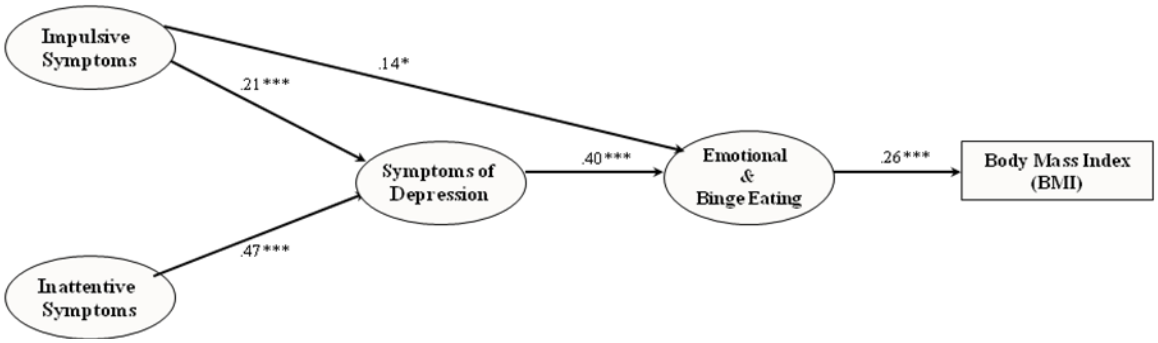

Figure 8. Alternative structural model for Random Sample 2 ($N = 333$). * = significance at $p < .05$. *** = significance at $p < .001$.

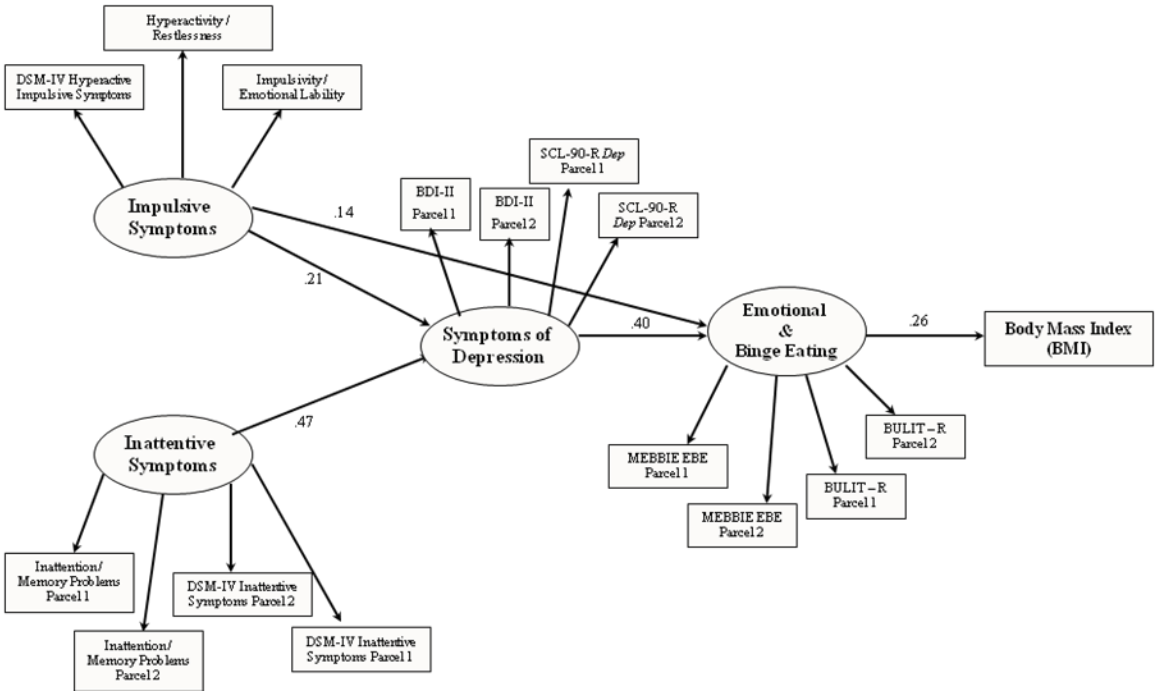

Figure 9. Alternative structural model fit indices for Sample 2 ($N = 333$). All coefficients and paths are significant at $p < .001$.

APPENDIX A

INITIAL LITERATURE REVIEW

Attention-deficit/hyperactivity disorder (ADHD) has been studied in children for decades, with much less emphasis on its persistence into adulthood. ADHD is among one of the most common behavioral disorders diagnosed in children and adolescents with a prevalence rate of 8.6%, with males being over-represented among youth who are diagnosed (National Institutes of Health, 2009). Most people with ADHD report continued difficulties into adulthood (Adler, 2008; Kessler et al., 2006) and recent studies suggest that between 4% and 5% of adults meet *Diagnostic and Statistical Manual of Mental Disorders – 4th Edition – Text Revision* (*DSM-IV-TR*) criteria for ADHD (Davidson, 2008; Kessler et al., 2006). The presence of ADHD among college students has also been reported as 4% (Heiligenstein, Conyers, Berns, & Miller, 1998).

According to the American Psychiatric Association *DSM-IV-TR* criteria for ADHD, symptoms of inattentiveness and/or hyperactivity and impulsivity must have been present and causing impairment prior to age seven, must cause clinically significant impairment in two or more settings (work, home, or school), and cannot occur due to another disorder, general medical condition, or substance (2000). ADHD includes three subtypes: ADHD combined type (ADHD-C; both inattentive and hyperactive-impulsive symptoms), ADHD predominantly inattentive type (ADHD-I), and ADHD predominantly hyperactive-impulsive type (ADHD-H), with ADHD-I being the most predominant in adults (Davidson, 2008; Erk, 2000; Spencer, Biederman, & Mick, 2007).

The symptom picture of ADHD seen in children may look different from adults because of developmental changes that occur throughout a lifetime and how task demands vary from childhood to adulthood. Inattentive and hyperactive symptoms may present themselves differently in adulthood than in childhood. For example, what appears as distractibility and inattentiveness in children may be seen in adults as poor time management, difficulty completing

tasks and avoidance of activities (Adler, 2008). With age, hyperactive symptoms tend to decrease and inattentiveness becomes dominant, but this does not imply that the impulsive behavior ceases to exist (Faraone, 2000). Many individuals with ADHD still struggle with both maintaining attention and controlling impulsive behavior into their adult lives (Wadsworth & Harper, 2007; Wilens, Faraone, & Biederman, 2004). In order to compensate, adults might purposefully create situations in which they can cope with restlessness (hyperactive/impulsive symptoms) in adaptive ways, such as over-working or having multiple jobs (Adler, 2008).

Adults with histories of ADHD in childhood recall significant impairments in many areas of their lives (Biederman et al., 1993; Murphy & Barkley, 1996). When adults with ADHD were compared with non-ADHD adults, higher rates of tutoring, repeated grades, special placement in classes, and reading disabilities were associated with the ADHD group. Adults with ADHD also reported a history of lower grades and higher instances of disciplinary action (Biederman et al., 1993; Murphy & Barkley, 1996).

Difficulty in school in childhood and adolescence, which are symptoms of ADHD in childhood, present themselves differently once individuals with ADHD become adults. For example, when adults with ADHD were compared with non-ADHD controls in other studies, similar findings surfaced suggesting adults with ADHD engage in more risky behaviors (McCann & Roy-Byrne, 2004; Murphy & Barkley, 1996) and experience higher rates of marital and interpersonal difficulties (Mccann & Roy-Byrne, 2004; Murphy & Barkley, 1996; Weiss & Murray, 2003). Moreover, they exhibited lower levels of motivation and job performance than the non-ADHD control group (Goldman, Genel, Bezman, & Slanetz, 1998; McCann & Roy-Byrne, 2004; Wender, 1998).

In another study, similar results illustrated that individuals with ADHD typically continue to struggle with symptoms and associated features of the disorder as they age, especially if their condition had not been formerly diagnosed or treated. A cross-sectional study included 752 adults who either: 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. Numerous differences between the groups emerged, with the undiagnosed ADHD group always faring worse. For example, they were less likely to hold full-time jobs and more likely to be unemployed than members of the comparison groups (Able, Johnston, Adler, & Swindle, 2007). Moreover, adults with undiagnosed ADHD experienced more anxiety and mood lability and reported lower self-esteem and quality of life than the diagnosed ADHD comparison group and the cohort-matched comparison group. They also were significantly more likely than the comparison groups to be accident-prone at home and on the job (Able et al., 2007; Murphy & Barkely, 1996). As this study suggests, symptoms of ADHD can negatively affect an individual's work and home life.

A common description of ADHD in adulthood is that those with the disorder primarily struggle with executive functions, such as planning, organizing, and initiating/inhibiting appropriate actions. In an effort to provide evidence concerning the stability of executive function deficits into adulthood, a seven-year longitudinal study of 85 males between 9 and 22 years of age was conducted (Biederman et al., 2007). Executive function deficits appeared stable over a seven-year period into young adulthood. The executive functions included in this study were: sustained attention, planning and organization, response inhibition (impulsivity), selective

attention, visual scanning, verbal and visual learning, and memory (Biederman et al., 2007). Individuals with ADHD continue to struggle with executive functions into their adult lives, which may cause problems in various areas. McGough and Barkley (2004) propose that ADHD be re-conceptualized as a "life-span disorder" (p. 1948). The symptoms of ADHD do not cease to exist once children become adults. Rather, viewing ADHD as a "life-span disorder" recognizes the idea that these potentially debilitating symptoms often persist into adulthood.

Recognizing these potentially debilitating symptoms and diagnosing adults with ADHD can be complicated. Shaffer (1994) discusses three diagnostic problems when attempting to diagnose adults with ADHD: requirement of a childhood history of symptoms, similar clinical features with other disorders, as well as the likelihood of comorbidity with other psychiatric disorders. Once children become adults, it can be difficult to obtain possible ADHD symptoms the individual experienced as a child. Self-report measures are available for the adult individual to complete, but that does not always provide a clear picture of the adult's childhood. It is usually better to obtain information from the parents as well, although this is not always possible. However, important information about an adult's current symptoms can be obtained through self-report measures (Brown, 1996; Conners, Erhardt, & Sparrow, 2004).

The second diagnostic problem discussed by Shaffer (1994) is that some of the symptoms included in the ADHD diagnostic criteria can be caused by a different disorder and this symptom overlap between different diagnoses can make diagnosing ADHD more complicated and difficult. For example, impaired concentration, mood swings, irritability, restlessness, and memory deficits are also symptoms of major depressive disorder (Adler, 2004; Kessler et al., 2006). Moreover, children in chaotic families with inconsistent rules and discipline can have cognitive and behavioral problems that mimic ADHD (Granero, Ezpeleta, Domenech, & de la

Osa, 2008). Thus, even a reliable report of ADHD-like symptoms in childhood does not guarantee that the symptoms were due to ADHD.

Additionally, ADHD is a disorder that individuals frequently experience with one or more other psychiatric disorders simultaneously (i.e., comorbidity). In adulthood, these include antisocial personality disorder, substance use disorders, and major depressive disorder (Shaffer, 1994; Triolo, 1999). Kessler et al. (2006) provide statistics on the comorbidity of major depressive disorder (MDD) and ADHD. There is an 18.6% chance an individual diagnosed with ADHD will also have MDD, and a 9.4% chance an individual diagnosed with MDD will also have ADHD. Similarly, Able et al. (2007) found that individuals who were in the "undiagnosed ADHD" group (screened positive for ADHD symptoms, but no formal diagnosis) showed more indicators of current depression than the "non-ADHD controls" (46.4% vs. 21.0%). Additionally, high rates of anxiety disorders (43% to 52%), antisocial personality disorder (12%), major depressive disorder (31%), conduct disorder (20%), and alcohol and drug dependencies (27% and 18%, respectively) accompanied ADHD diagnoses in a clinic adult sample (i.e., formally diagnosed individuals referred to a psychiatric clinic) (Biederman, Faraone, Spencer, Wilens, Norman, & Lapey, 1993). These complications make it imperative for clinicians to be aware of the shared symptoms of multiple disorders, in order to effectively and accurately rule out other psychiatric disorders and differentiate between symptom overlap and actual comorbidity.

ADHD and Addictive Behavior

There is a fairly extensive body of literature that addresses the links between ADHD, ADHD-like symptoms, and addictive behaviors (Lambert, 2005; Milberger, Biederman, Faraone, Chen & Jones, 1997; Ohlmeier et al., 2007; Pomerleau, Downey, Stelson & Pomerleau, 1995) and a variety of hypotheses have been proposed to explain the high rates of addiction among

people with ADHD or ADHD symptoms. The three most prominent explanations will be reviewed. These are that addictive behaviors are more likely to develop among people who have trouble with inhibition, that addictive behaviors begin as attempts to cope with the stressors associated with having ADHD, and that addictive behaviors may alter neurochemistry in ways that offset some of the neurotransmitter anomalies of ADHD.

To become addicted to anything, an individual must first initiate the behavior to which they are addicted. Individuals who struggle with impulse control are more likely to engage in a wide range of risky behaviors, some of which (e.g., illicit drug use) increase the risk for addiction (Kalbag & Levin, 2005; Wilson, 2007) A possible explanation for the high comorbidity between substance abuse and symptoms of ADHD might be the disinhibition symptom that is at the core of the disorder (Barkley, 1999; Dinn et al., 2004; Mason et al., 2007). People with ADHD or ADHD symptoms and substance abuse share traits such as impulsivity and disinhibition. Having increased disinhibition, like those with ADHD, is likely to increase the risk for substance abuse. Disinhibition can be related more to emotional factors rather than cognitive factors. Individuals oftentimes know and understand the negative effects of the risky behaviors they are engaging in, but continue the behaviors anyway due to the transient alleviation of distress they are currently experiencing (Dinn et al., 2004). Additionally, individuals with ADHD or ADHD-like symptoms may not contemplate or anticipate the risks or long-term effects (i.e., poor impulse control) of smoking, drinking, or drug use. These individuals might discount future consequences, only focusing their attention on immediate rewards (i.e., alleviation of the negative symptoms of ADHD).

The symptoms of ADHD may cause daily struggles in the lives of the individuals they affect and, even when treated, people may use substances in an effort to help them cope. In

addition, individuals who suffer with ADHD symptoms experience a myriad of *secondary* stressors (Able, Johnston, Adler, & Swindle, 2007; Wadsworth & Harper, 2007; Wilens, Faraone, & Biederman, 2004). For example, impulsive behaviors increase the likelihood of interpersonal conflict (Ratey, Greenberg, Bemporad, & Lindem, 1992), and problems with organization contribute to daily hassles such as chronically running late, forgetting needed items, and getting traffic tickets (Riccio, Wolfe, Davis, Romine, George, & Lee, 2005). Adults with ADHD are also more likely than adults without the disorder to experience certain negative life events such as divorce (Mccann & Roy-Byrne, 2004; Murphy & Barkley, 1996; Weiss & Hechtman, 1993; Weiss & Murray, 2003), job loss (Able et al., 2007; Barkley, Fischer, Edelbrock, & Smallish, 1990), and driving-related accidents (Murphy & Barkley, 1996). Ohlmeier et al. (2007) suggest that using and abusing substances may be a form of self-soothing, attempting to cope with the aversive symptoms they are feeling. These substances may also act as a temporary escape (i.e., a form of coping, albeit, not the most adaptive) from the adversities associated with ADHD symptomlogy (Dinn et al., 2004; Spencer, Biederman, & Mick, 2007).

A longitudinal study of 91 male and female adults was conducted over a 6-month period to examine the relationship between ADHD symptomology and alcohol dependence (Ohlmeier et al., 2007). More than 20% of the alcohol-dependent participants met the DSM-IV criteria for the presence of ADHD, which is four times the rate of ADHD seen in the general population (Davidson, 2008; Kessler et al., 2006; Ohlemeier et al., 2007). Not only did the researchers find that a high rate of the alcohol-dependent participants met the DSM-IV criteria for ADHD, but they also found that nicotine use was higher in those participants with comorbid alcohol dependence and ADHD when compared to alcohol-dependent participants without ADHD.

In addition to the use of substances in an attempt to cope with stress, some authors have suggested that adolescents and adults with ADHD use substances as a means of self-medication (Dinn et al., 2004; Levin et al., 1996; Mason et al., 2007). Nicotine and other psychoactive substances with stimulant properties can reduce ADHD symptoms such as inattention and poor concentration for some people with ADHD (Levin, Conners, Silva, Canu, & March, 2001). Additionally, clinicians have suggested that children may ingest high amounts of caffeine as a form of self-medication prior to being diagnosed with ADHD or taking appropriate medication (e.g., psychostimulants) to treat ADHD symptomology (Leon, 2000).

Many drugs of abuse are stimulants (i.e., nicotine, cocaine, and amphetamines) that elevate mood and increase attention and concentration via neurotransmitters: norepinephrine, serotonin, and dopamine. When individuals use stimulants, increased amounts of norepinephrine, serotonin, and dopamine are released from the presynaptic terminals. The stimulants bind to those neurotransmitters, therefore, preventing reuptake into storage vesticles and increasing the concentration of those neurotransmitters in the synaptic gap (Sofuoflu & Sewell, 2008). Chronic use and high doses of these drugs can cause the brain chemistry to be altered over time, requiring increased amounts of the drug to elicit an effect (i.e., elevated mood and increased concentration and attention; Chabner, 2007).

A common reward deficiency syndrome (RDS) has been hypothesized to be associated with the neurotransmitter anomalies associated with ADHD, drug and alcohol use, and disordered eating (i.e., binge eating and emotional eating). The dopamine D2 receptor (DRD2) is the main neurotransmitter that has been supported by research to be associated with addictive behaviors and compulsive behaviors, such as smoking, alcoholism, drug dependency, pathological gambling, ADHD, and disordered eating that may lead to becoming overweight

(Blum, Sheridan, Wood, Braverman, Chen, Comings, 1995). Insufficient dopamine D2 receptors and tendencies to engage in compulsive rewarding behaviors, such as drug use and overeating characterize the reward deficiency syndrome (Barry, Clarke, & Petry; 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. With extended drug or alcohol use, the number of dopamine D2 receptors decreases, causing individuals to intake higher doses of drugs and alcohol in order to experience the rewarding release of dopamine in the brain (Barry, Clarke, & Petry, 2009; Volkow & Fowler; 2000). Individuals that struggle with addictive and/or compulsive behaviors might have genetic defects associated with dopamine D2 receptors. Specifically, those individuals may be over-engaging in certain behaviors (e.g., nicotine, alcohol, and drug use) in order to increase the dopamine levels in their brains (Wang et al., 2001).

In a four-year longitudinal study, cigarette use among 6- to 17- year old boys diagnosed with ADHD was compared to use among a non-ADHD control group (Milberger, Biederman, Faraone, Chen, & Jones, 1997). Children who were diagnosed with ADHD showed higher rates of cigarette smoking in adolescence (i.e., 19% of the children diagnosed with ADHD were smokers) when compared with their non-ADHD controls (i.e., 10% of the non-ADHD controls were smokers). Additionally, the children diagnosed with ADHD began smoking at an earlier age than their peers without ADHD. In other studies (Levin & Razvani, 2002; Newcorn, 2008), when comparing the general population to the adult ADHD population, rates of nicotine use are doubled. Specifically, nicotine may also help decrease attention deficits among people with ADHD by signaling the brain to release serotonin, dopamine, and norepinephrine from the presynaptic terminals, causing an overabundance of these neurotransmitters in the brain. Moreover, some researchers have speculated that nicotine acts as a form of self-medication to

alleviate and manage the symptoms of ADHD, as this stimulant may improve attention and concentration (Milberger et al., 1997). Similarly, Lambert (2005) proposed the idea that individuals with ADHD symptoms "self-medicate," that is, they may use or abuse stimulants such as tobacco, cocaine, and amphetamines as a means to manage their symptoms of inattention and impulsivity. Early onset of cigarette smoking in children and adolescents with ADHD, used as a means to decrease negative symptomology associated with the diagnosis, may increase comfort with drug use and/or set them on a path to addictive behavior and further, more serious use of psychostimulants in adulthood (Lambert, 2005). Other clinicians have proposed a similar phenomenon that begins with caffeine abuse for the initial, and not necessarily conscious, purpose of self-medication that may pose a risk for later interest in and willingness to try illegal stimulants (P. L. Kaminski, personal communication, July 28, 2010).

These studies and possible explanations suggest that individuals with ADHD or ADHD symptomology may be at a higher risk to engage in addictive behaviors. Recently, researchers have identified a possible link between ADHD and obesity and suggested binge eating as a type of addictive behavior that could explain the link (Barry, Clarke, & Petry, 2009; Cortese, Bernardo, & Mouren, 2007). Food consumption can no longer be thought of as only a means of providing appropriate nutrients to our bodies. Rather, like substance abuse, eating may involve disinhibition, be used as a form of coping, as well as a form of self-medication (Barry, Clarke, & Petry, 2009). One type of eating that appears to involve disinhibition and may serve as a short-term form of coping and/or self-medication is binge eating. A binge eating episode is defined in the DSM-IV-TR (American Psychiatric Association, 2000) as "eating within any 2-hour period an amount of food that is definitely larger than most people would eat in a similar period of time

under similar circumstances and a sense of lack of control over eating during the episode" (p. 787).

There are case reports in the literature where onset of binge eating occurs when ADHD is no longer managed with medication. For example, Schweickert, Strober, and Moskowitz (1997) described a woman who was diagnosed with ADHD as a child, was taken off medication at age 9 and saw a return of ADHD symptoms, began binge eating and purging at age 13, and eventually began experimenting with drugs and alcohol at age 14. According to these authors, the binge eating might have been used as a form of coping (i.e., a compensatory mechanism) with the frustration this girl felt concerning the attention and concentration difficulties she was experiencing. Another hypothesis might be that individuals binge-eat as a form of self-medication. Numerous researchers (Gold, Frost-Pineda, & Jacobs, 2003; Hernandez & Hoebel, 1990) have suggested that food intake, especially food that is high in fat and/or sugar, increases the amount of dopamine released from the presynaptic terminal in the brain. When individuals ingest large amounts of food (e.g. binge eaters), higher concentrations of dopamine are found in the synaptic gap in the brain (Gold, Frost-Pineda & Jacobs, 2003).

Moreover, individuals who experience ADHD symptoms and struggle with compulsive eating behaviors (e.g., binge eating, overeating certain types of foods) might have reduced amounts of the dopamine D2 receptors in their brains. Wang et al. (2001) results suggested that this reduction of dopamine D2 receptors may represent a downregulation to compensate for the increased amount of dopamine release caused by continual overstimulation from eating (Wang et al., 2001). Therefore, it is possible that those individuals who partake in binge eating episodes eat foods that are high in fat and/or sugar, in an effort to increase the dopamine levels in their brains. This type of abnormal eating can be used as a form of self-medication, controlling the

negative symptoms that are related to ADHD while also increasing mood. Additionally, binge eating also involves impulsivity and disinhibition (Cortese, Bernardo, & Mouren, 2007; Davis et al., 2006), seeking the immediate reward of feeling better while binge eating, but discounting the long-term consequences and health concerns it may cause in the future, as well as knowing the future adverse consequences but being unable to inhibit the behaviors.

ADHD and Obesity

Obesity is an epidemic in the United States, increasingly beginning in childhood and adolescence. Compared to 1980, the percentage of children (ages 6-11 years) classified as obese has doubled, while the percentage of adolescents (ages 12-19 years) classified as obese has tripled (Centers for Disease Control and Prevention, 2010). In 2010, the percentage of adults (i.e. ages 20 and above) who are classified as overweight totaled 68.0% (i.e., males 72.3% and females 64.1%) and those classified as obese totaled 33.8% (i.e., males 32.2% and females 35.5%) (Flegal, Carroll, Ogden, & Curtin, 2010). Obese and overweight are both used to classify those individuals whose body weight is higher than what is considered healthy for their height. Overweight and obesity are not mutually exclusive, meaning that a person who is obese is also overweight (National Heart Lung and Blood Institute, 1998). The body mass index (BMI) has been viewed as the best way to define, determine, and classify individuals in specific weight ranges (i.e., overweight versus obese) (National Heart Lung and Blood Institute, 1998). The BMI is determined by dividing an individual's weight in kilograms by the square of their height in meters. Specifically, an individual is classified as overweight if they have a body mass index (BMI) between 25 – 29.9, whereas a BMI of 30 or greater would classify an individual in the obese range (National Heart Lung and Blood Institute, 2010).

Overweight and obese individuals have increased physical risks that those individuals within their weight range might not experience. Individuals with BMIs of 30 or greater are at increased risk for diabetes, coronary heart disease, high cholesterol, and high blood pressure, which all increase the risk of mortality as well (Akbartabartoori, Lean, & Hankey, 2008).

In recent years, researchers have continued to identify new risk factors for the development of obesity, especially among children and adolescents. Erermis et al. (2004) emphasize the importance of identifying and treating obesity by adolescence because, without intervention, most obese adolescents will remain obese into and throughout adulthood. It is important to recognize weight concerns as early as possible in an effort to prevent the myriad of associated health problems that become more and more likely and serious as people age.

One population of children and adolescents who appears to be at an increased risk for obesity is those who have high symptoms of ADHD. For example, in a study involving: 1) adolescents in treatment for obesity; 2) adolescents who were obese but not in treatment, and 3) a normal weight control group from a high school, teens in group 1 were much more likely to have ADHD (i.e., 13.3%) than adolescents in either of the other groups (ADHD prevalence in groups 2 and 3 was 3.3% in each group) (Eremis et al., 2004). The Eremis et al., 2004 study suggests a clear link between ADHD and obesity for some adolescents and other researchers report findings that support this association. For example, Cortese et al. (2008) comprehensively reviewed the literature on the connection between ADHD and obesity across all age groups. They included 15 studies in their review, excluding "case reports, non-empirical studies," and studies where "ADHD diagnostic criteria" were not used (p. 524). All of the studies reviewed reported that, on average, those diagnosed with ADHD were higher in weight than expected given their height (i.e., according to their calculated BMI) (Agranat-Meged et al, 2005; Biederman et al., 2003;

Erermis et al., 2004; Faraone et al., 2005; Holtkamp et al., 2004; Mustillo et al., 2003; Spencer et al., 1996; Swanson et al., 2006). Most of these studies (i.e., 13 of the 15), however, were conducted on children and adolescents (18 years of age and under), indicating that additional research that specifically focuses on the possible relationships and connections between ADHD symptoms, ADHD diagnosis, and obesity in adults (i.e., older than 18 years of age) might be helpful for researchers who are looking into the possible relationships that exist among those three variables.

Scientists who have studied the risk factors of and etiological mechanisms that lead to obesity have identified negative affect, poor impulse control, dopamine deficiencies, and distracted eating as important phenomena (Davis, Levitan, Smith, Tweed, Curtis, 2006; Davis et al., 2009; Delahanty, Hayden, Williamson, & Nathan, 2002; Lluch, Herbeth, Mejean, & Siest, 2000; Shepard, 2009; Wansink, Painter, & North, 2005). It is notable that each of these phenomena are also associated with ADHD (American Psychiatric Association, 2000; Davis et al., 2006; Davis et al., 2009; Malloy-Dinz, Fuentes, Leite, Correa & Bechara, 2007).

Indeed, Davis et al. (2006) utilized structural equation modeling (SEM) to test their hypotheses that ADHD symptoms predict abnormal eating patterns and overeating behaviors, which in turn predict overweight and obesity. Davis et al. utilized a nonclinical sample of women (n = 110) recruited from universities, hospitals, and community centers. ADHD symptomology was measured using the Wender Utah Rating Scale (WURS) and the Barratt Impulsivity Scale. Eating behaviors were measured using the Emotional Eating Scale (EES), the Dutch Eating Behavior Questionnaire, and the Binge Eating Questionnaire (BEQ). The researchers hypothesized that ADHD symptoms were positively related to disordered eating (i.e., eating in response to environmental cues, negative mood, and binge eating). Results suggested that all the

paths the researchers predicted were statistically significant, indicating a possible relationship between ADHD symptoms, overeating, and higher BMIs.

Davis et al. (2006) explain symptoms of ADHD that might be connected with disordered eating. By definition, most individuals with ADHD struggle with controlling their impulses. If their ADHD manifests in disinhibited eating or impulsive food choices, it is not difficult to see how this symptom of ADHD could indirectly increase overweight and obesity. For example, those with lower impulse control may be more prone to eating when they want to versus when they need to because they are quick to respond to the enticing smells, sights and tastes of foods. Similarly, disinhibition could explain overeating and even binge eating. Over time, impulsive and/or disinhibited eating could contribute to overweight and obesity.

Individuals diagnosed with ADHD and others who have trouble with organization and planning may see those difficulties affect their eating patterns and food choices (Davis et al., 2009; Shepherd, 2009). Specifically, poor organization and planning can influence an individual's ability to adequately plan for meals, which might result in eating what is convenient and easily obtainable (e.g., snack foods and fast food as opposed to healthier options).

Factors related to the high rates of psychiatric comorbidity among individuals with ADHD might also contribute to higher risk for overweight and obesity in this population (Davis et al., 2009; Lluch, Herbeth, Mejean, & Siest, 2000). For example, ADHD coexists with other psychiatric disorders such as major depression and anxiety disorders for approximately 50 – 75% of adults with ADHD (Adler, 2008). In addition to these obvious sources of negative affect, adults with ADHD are more likely than their non-ADHD peers to experience negative life events such as job loss, marital discord, financial problems, and legal trouble (Biederman, Faraone, & Spencer, 1993, 1994; Murphy & Barkley, 1996). Inordinate stress such as that which

accompanies negative life events and negative affect are risk factors for maladaptive coping that may take the form of addictive behaviors such as substance abuse, and of particular interest here, emotional eating and binge eating (Burton, Stice, Bearman, & Rohde, 2007).

Emotional eating can include consuming foods with high glucose and fat content for self-soothing. Gossens, Braet, Van Vlierberghe and Mels (2008) suggest that emotional eating may be used as a coping strategy to control and minimize negative emotions. This type of eating, along with binge eating, might be an effective temporary solution because binge eating can temporarily decrease negative affect.

Just as the reward deficiency syndrome was hypothesized to play a role in the relationship between ADHD and addiction, it is hypothesized to play a similar role between ADHD and binge eating. For example, Blum et al. (2008) studied reward deficiency syndrome (RDS). RDS is a dysfunction of the reward centers in the brain, which involve dopamine transmission. The resulting effect is a lack of receptor sites for the neurotransmitter, dopamine, which results in insufficient amounts of dopamine in the brain. Because of this, individuals who experience this deficit are compelled to partake in activities that will increase transmission of dopamine, including gambling, risk taking, smoking, disordered eating (i.e., binge eating and emotional eating), and drug and alcohol use. Davis et al. (2008; 2009) provide evidence that individuals with ADHD experience low levels of dopamine, linking ADHD and RDS. Similarly, in a study conducted by Wang et al., (2001), obese individuals had reduced dopamine D2 receptors when compared to the normal weight control group, indicating that these individuals would have to eat larger amounts in order to experience the rewarding effects of dopamine. It could be hypothesized that ADHD individuals might use food as a means to "self-medicate" the low dopaminergic state associated with the disorder. Hoebel, Avena, and Rada (2007) further

acknowledge this connection due to the findings that eating food, especially sweet foods, triggers the release of dopamine in the brain, which tends to elevate mood.

ADHD, Obesity and Depression

The literature concerning ADHD and depressive symptoms suggests a connection (Able et al., 2006; Goodman, 2009; Kessler et al., 2006), while the literature on obesity and depressive symptoms in inconsistent (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Roberts, Deleger, Strawbridge, & Kaplan, 2003; Sammel et al., 2003; Tanofsky-Kraff et al., 2006). Depressive symptoms and disorders are highly comorbid with ADHD symptoms (Able et al., 2006; Goodman, 2009; Kessler et al., 2006), such that a high percentage (18.6%) of individuals diagnosed with ADHD will also be diagnosed with MDD. Further, it is important to note that the results of Able et al. (2006) suggest that those individuals who experience ADHD symptoms, but have no previous diagnosis, screened positive for current depression more often than those participants who already had a formal ADHD diagnosis), indicating that individuals who experience ADHD symptoms, but no formal diagnosis, might be struggling with current depressive symptoms as well (i.e., due to not having learned healthy coping skills or received treatment for their distressing ADHD symptoms).

Limited research has been conducted on the connections between ADHD and obesity. Davis et al. (2006) suggest the symptoms of ADHD predict overeating, which further and indirectly predicts overweight and obesity. Specifically, the disinhibition, poor planning, impulsivity, inattentiveness, and hypo-dopaminergic levels associated with ADHD could play a role in the overconsumption of food. Notably, the researchers also suggest that many *other*

factors could mediate the relationship between ADHD and obesity (Davis et al., 2006). For example, it is possible that depressive symptoms and disorders might mediate this relationship.

The literature on the relationship between obesity and depression is inconsistent. Some studies suggest a relationship between obesity and depression (Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Goodman & Whitaker, 2002; Noppa & Hallstrom, 1981; Sammel et al., 2003) and others report nonsignificant results (Roberts, Deleger, Strawbridge, & Kaplan, 2003; Tanofsky-Kraff et al., 2006). The inconsistencies may be related to the variety of assessment approaches for measuring BMI. Specifically, some of the studies used self-reported height and weight to calculate BMI. Kvaavik, Tell and Klepp (2003) document how a downward bias exists when individuals are self-reporting their height and weight (i.e., most individuals overestimate their height and underestimate their weight). Most of the studies assessing the relationship between depression and measured obesity have suggested depression as a risk factor for weight gain and obesity. Specifically, when high levels of depression exist, motivation, planning, impulse control, and activity levels tend to be low, making overconsumption of food and/or poor food choices more likely (Rooke & Thorsteinsson, 2008). Markowitz, Friedman, and Arent (2008) propose a theoretical model of depression and weight gain that highlights two possible mediators of the link between depression and obesity: physiological means as well as low motivational levels. The "de-motivational means" is of particular importance because it includes symptoms in common with ADHD -- low impulse control, poor organization and planning -- as associated with features of depression that could lead to the overconsumption of food and/or more fattening food choices.

To date, however, researchers have not investigated how ADHD, obesity, and depression interrelate. Although ADHD symptoms and obesity are each associated with depression,

depression might also be a condition that helps explain the connection between ADHD and obesity.

APPENDIX B

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: (1) 18 (2) 19 (3) 20 (4) 21 (5) 22 (6) 23
(7) 24 (8) Over 24

3. Class Rank: (1) Freshman (3) Junior
(2) Sophomore (4) Senior

4. Are you a serious athlete? (i.e., play(ed) for a high school or college team, or train(ed) on your own) (1) Yes (2) No

If yes, what sports or activities are you <u>currently</u> involved in?

College	(1)Yes	(2)No	**Intramural**	(1)Yes	(2)No
a. football			j. flag football		
b. basketball			k. golf		
c. golf			l. water polo		
d. track			m. tennis		
e. hockey			n. volleyball		
f. lacrosse			o. soccer		
g. cross country			p. basketball		
h. swimming			q. weightlifting		
i. diving			r. softball		
			s. martial arts		

t. other (Please describe) (1) _____
_____
_____

5. In an average week, about how many hours do you spend working out? (Please describe)
_____
_____
_____
_____
_____

6. What is your current weight? _____

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

8. How certain are you that the number in question 6 is your accurate weight?
(1) Very uncertain (2) Somewhat uncertain (3) Somewhat certain (4) Very certain

9. How certain are you that the number in question 7 is your accurate height?
(1) Very uncertain (2) Somewhat uncertain (3) Somewhat certain (4) Very certain

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

11. What was the least you have weighed as an adult? _____

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

13. How many hours of television do you watch per day?

Less than 2 2 to 4 4 to 6 7 or more

14. How would you classify your sexual orientation?

(1) Straight/heterosexual (2) Gay/homosexual (3) Bisexual

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

(1) Yes (2) No

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

(1) Yes (2) No

17. If yes, please list the name of the medication and dosage:

APPENDIX C

BODY MASS INDEX (BMI): FOR ADULT MEN

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

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

APPENDIX D

HEIGHT/WEIGHT FORM

Code #: _____
Height: _____
Weight: _____
Shoe Size: _____ Frame Size: _____

Body Type: (1) ☐ Underweight (2) ☐ Low-Average (3) ☐ High-Average

(4) ☐ Overweight (5) ☐ Very Overweight

(6) ☐ Very Muscular (higher weight due to high muscle mass)

APPENDIX E

IRB APPROVAL LETTER

UNT
UNIVERSITY OF
NORTH·TEXAS
Discover the power of ideas.

OFFICE OF THE VICE PRESIDENT FOR RESEARCH AND ECONOMIC DEVELOPMENT
Research Services

February 1, 2010

Elissa Woodruff
Department of Psychology
University of North Texas

RE: Human Subjects Application No. 09001

Dear Ms. Woodruff:

The UNT Institutional Review Board has reviewed and approved the extension you requested to your project titled "The Development of Symptoms of Muscle Dysmorphia: An Examination of Mediating and Moderating Variables." Your extension period is for one year, **January 30, 2010 through January 29, 2011. Federal policy 45 CFR 46.109(e) stipulates that IRB approval is for one year only.**

Enclosed is your consent document with stamped IRB approval. Please copy and **use this form only** for your study subjects.

The UNT IRB must re-review this project prior to any modifications you make in the approved project. It is your responsibility according to U.S. Department of Health and Human Services regulations to submit annual and terminal progress reports to the IRB for this project. Please mark your calendar accordingly.

Please contact Shelia Bourns, Research Compliance Administrator, ext. 3940 or Boyd Herndon, Director of Research Compliance, ext. 3941 if you need additional information.

Sincerely,

Debbie Rohwer, Ph.D.
Associate Professor
Faculty Member
Institutional Review Board

DR/sb

CC: Dr. Patricia Kaminski

1155 Union Circle #305250 | Denton, Texas 76203-5017 | TEL 940.565.3940 | FAX 940.565.4277
TTY 940.369.8652 | research.unt.edu

APPENDIX F

RECRUITMENT FLYER

Male Research Participants Needed

- WHAT: Make **$10** in an hour for filling out questionnaires about body image in adult men
- WHO: Male students @ UNT (you must be 18 years old or older)
- WHERE: Terrill Hall Room 281

To schedule, email untbodyimage@yahoo.com or call 972-345-6705

To schedule, email untbodyimage@yahoo.com or call 972-345-6705

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APPENDIX G

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 disturbed eating and body image in adult males. The majority of research about eating and body image disturbances addresses females only, and the research from this study will facilitate a better understanding of these problems among men.

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 eating and/or body image disturbance, 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 men with problems they might have dealing with eating and/or body image disturbances.

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

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

Elissa Woodruff
Graduate student

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