INVESTIGATION OF THE RELATIONSHIP BETWEEN ADHD AND
BODYWEIGHT IN A CANADIAN SAMPLE OF CHILDREN

by

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for the degree of Doctor of Philosophy
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Abstract

Objectives: To investigate whether Attention Deficit Hyperactivity Disorder (ADHD) is associated with abnormal bodyweight (underweight or obesity) in a national sample of Canadian children.

Methods: Data were derived from children aged 4-11 years in the National Longitudinal Survey of Children and Youth (N =10,200). Data were analysed using linear modelling and logistic regression, controlling for variables known to be associated with ADHD and bodyweight, including age, sex, SES, medication, and other psychological difficulties. We examined ADHD using an ADHD symptom scale (dimensional approach), symptom severity rating (no, mild, moderate or severe symptoms), a proxy ADHD diagnosis (to approximate a clinical categorical condition), and hyperactive/impulsive and inattentive ADHD symptom severity.

Results: Higher scores on the ADHD scale were associated with increased odds of obesity (adjusted odds ratio [AOR] = 1.04, 95% confidence interval [CI95] = 1.02-1.06, p = .000), but not underweight. Children with mild (AOR = 1.39, CI95 = 1.16-1.67, p=.001) and moderate (AOR = 1.63, CI95 = 1.31-2.01, p=.000) ADHD symptoms had higher odds of obesity relative to children with no symptoms. Among children not taking ADHD medication, those with severe ADHD symptoms also had higher odds of obesity.
(AOR = 1.43, CI95 = 1.02-2.01, p = .040). In terms of specific ADHD symptom dimensions, low-moderate (AOR = 1.40, CI95 = 1.19-1.64, p = .000) and severe (AOR = 1.39, CI95 = 1.05-1.85) hyperactive/impulsive and low-moderate (AOR = 1.30, CI95 = 1.15-1.46, p = .000) inattentive symptoms of ADHD were significantly associated with increased odds of obesity. On the other hand, proxy ADHD diagnosis was associated with higher odds of underweight (AOR = 1.34, CI95 = 1.01-1.78, p = .046), but not obesity. Finally, when bodyweight classification was entered as the predictor variable and ADHD variables served as the outcome variables, the pattern of results was essentially the same.

**Conclusions:** Results demonstrated a relationship between ADHD and abnormal bodyweight, although the associations were different for underweight and obesity. The association between ADHD and obesity also varied with symptom severity, suggesting a non-linear relationship. Further research is needed to clarify the mechanisms involved in this complex relationship.
Acknowledgements

This project is the culmination of several years of interest in the area of ADHD and its association with abnormal bodyweight. This is my third thesis on this topic, and it was my hope that this project would help me to resolve some of the issues that had been highlighted by my own previous work, as well as that of other researchers in the field. I encountered many challenges along the way, but learned more than I could have anticipated in the process. I learned many skills related to the process of analysing data from a large, national database and writing a thesis on a complex topic in clear, concise and logical way. However, perhaps most importantly I learned much about adapting to and overcoming challenges, perseverance, and the critical role of receiving support and feedback from others along the way.

I was extremely lucky to have the support of a number of incredible people throughout the years I worked on this thesis, and I would very much like to take a moment to acknowledge just a few of these people. First, my supervisor, Dr. Rosemary Tannock, who helped me throughout the entire process from the initial planning stages through the final to the final edits. Dr. Tannock’s ongoing support and expertise was invaluable, and I am extremely grateful to have had the opportunity to work with her. Second, I would like to thank my incredible thesis committee, who in addition to Dr. Tannock included doctors Michele Peterson-Badali and Katreena Scott. Each of my committee members provided me with invaluable feedback and support throughout this process, and their unique perspectives enabled me to write in a way that will be meaningful to people from a variety of fields, with different areas of expertise. I would also like to thank those who volunteered to be on my final oral exam committee, which
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Chapter 1: Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that has a significant impact on the health, safety, personal relationships, academic and occupational achievement, and overall well-being of those who have the disorder (see review by Biederman, 2005). Abnormal bodyweight has also been found to have a significant impact on the physical, social, and emotional health and well-being of children (Pulgarón, 2013), and is associated with increased health care utilization and increased psychiatric referrals. Furthermore, both obesity and underweight are associated with increased mortality in adulthood (Flegal, Graubard, Williamson, & Gail, 2007).

It is only recently that researchers have begun to identify a relationship between ADHD and abnormal bodyweight, with a number of studies reporting associations across diverse populations, including both clinical and non-clinical samples (for instance, see review by Cortese & Vincenzi, 2012). Given the impact of ADHD and abnormal bodyweight on affected children, their families, and society, it is critical to develop a better understanding of the association between these conditions. Clarifying the extent and nature of this association also has important theoretical and clinical implications.

The goal of this study was to investigate the potential association between ADHD and abnormal bodyweight in a large, national sample of Canadian children. Furthermore, this study sought to address some of the presently unresolved issues raised by previous research studies by approaching this investigation from multiple angles. For instance, the present study investigated the influence of ADHD on bodyweight outcomes, as well as the influence of abnormal bodyweight on ADHD outcomes. Furthermore, ADHD was
examined in three ways: ADHD symptoms as a continuous variable, ADHD symptom severity as a non-dichotomous, categorical variable (ranging from no symptoms to severe symptoms), and ADHD as a discrete, dichotomous variable, to approximate a clinical diagnosis of ADHD. This chapter will provide a foundation for the current research study, including the specific objectives and methods used. First, the following key issues will be discussed for ADHD and abnormal bodyweight individually: definition and prevalence, and relationship to development in childhood, social economic status (SES), sex, and comorbid psychopathology. Each of these variables has a significant influence on both conditions, and therefore may influence the relationship between them. Second, research regarding the association between ADHD and bodyweight will be reviewed, including the history of this area of research, current empirical support, and unresolved issues.

1.1 Overview of ADHD

1.1.1 Definition and Prevalence of ADHD. ADHD is a heterogeneous neurodevelopmental disorder arising from of a complex interaction between genetic and environmental factors (Archer, Oscar-Berman, & Blum, 2011). It is characterized by a persistent pattern of hyperactivity-impulsivity and/or inattention that causes functional or developmental impairment (American Psychiatric Association, 2013). The world-wide estimated prevalence in children is 5.2% (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007), and 4.8% in Canadian children, specifically (Waddell, Offord, Shepherd, Hua, & McEwan, 2002).

According to the DSM 5 (American Psychiatric Association, 2013), a diagnosis of ADHD requires that a child have six or more symptoms of inattention and/or six or more
symptoms of hyperactivity and impulsivity. Symptoms of inattention include problems sustaining attention during tasks, organizing materials, completing tasks and following instructions. Symptoms of hyperactivity and impulsivity include fidgeting, excessive or inappropriate physical activity, restlessness, difficulty waiting, and frequently interrupting or intruding on others. Three types of presentations can be specified: predominantly inattentive, predominantly hyperactive/impulsive, and combined type, depending on the number of symptoms that are met within each symptom category in the preceding six months. Severity should also be specified as mild, moderate or severe (American Psychiatric Association, 2013).

DSM 5 criteria further state that ADHD symptoms must have been present prior to the age of 12 years, be present in two or more settings, such as home and school, and cause impairment in social, academic or occupational functioning. Finally, to diagnose ADHD, symptoms must not occur only in the course of a psychotic disorder or be better explained by any other mental disorder (American Psychiatric Association, 2013).

Although there have been changes to the DSM criteria for ADHD since the publication of the DSM-III-R, diagnostic continuity has been demonstrated between the DSM-III-R and DSM-IV (Biederman et al., 1997), and the symptom criteria for ADHD in the DSM 5 are the same as those in the DSM-IV-R, with changes made to examples provided for each criterion (American Psychiatric Association, 2013). Although DSM 5 did include changes related to the age of onset and number of symptoms required for diagnosis in late adolescence and adulthood, these changes were made to improve diagnostic accuracy for older adolescents and adults, and should not significantly impact
ADHD diagnosis in children. Overall, the core features of ADHD, including inattention, hyperactivity and impulsivity, have been consistent since DSM-III-R.

The symptom domains of inattention and hyperactivity/impulsivity described by the DSM have received considerable support (for instance, see McLoughlin, Ronald, Kuntsi, Asherson, & Plomin, 2007; Toplak et al., 2009). For instance, some studies have found empirical support for a model of ADHD that includes these distinct symptom domains using factor analysis by investigating how well various models of ADHD are able to account for actual data (Coghill & Sonuga-Barke, 2012). In general, studies that have approached these types of questions using confirmatory factor analysis have found support for a general ADHD factor and specific inattention and hyperactive/impulsive factors. Similarly, other studies using latent class analysis have also generally found that there is support for three subgroups within ADHD: inattentive, hyperactive/impulsive and combined groups.

However, although widely used in North America the DSM approach to defining ADHD has also been widely criticized as not being an effective or valid approach for identifying or defining ADHD. For instance, in one study (Power, Costigan, Leff, Eiraldi, & Landau, 2001), researchers found that a DSM approach to defining diagnostic criteria was the least valid approach of those investigated, and that other approaches that considered the predictive value of individual criteria were more helpful. On the other hand, the alternate approaches investigated in that study sacrificed sensitivity in the process of improving specificity, suggesting that all categorical approaches tried had limitations.
There is, in fact, considerable evidence to suggest that the categorical conceptualization of ADHD used in the DSM may not be appropriate, and that a dimensional approach is required (Haslam et al., 2006; Levy, Hay, McStephen, Wood, & Waldman, 1997; Marcus & Barry, 2011). In a recent review (Coghill & Sonuga-Barke, 2012), researchers considered the issue of categorical versus dimensional approaches in conceptualizing psychopathology in children and adolescents in great depth. In that paper, authors reviewed several approaches for investigating the relative merit of categorical and dimensional approaches to defining ADHD, including factor analysis and taxometrics, each of which provides different information regarding the validity and clinical and research utility of both approaches to defining ADHD.

In that paper (Coghill & Sonuga-Barke, 2012), authors point out that while approaches such as factor analysis provide support for the factor structure of ADHD based on models such as that used by the DSM-5 (see above), they cannot answer the question as to whether or not ADHD is best described as a dimensional construct or a discrete diagnostic category, or “taxon.” Instead, this question is better answered by taxometric analysis, which employs a group of mathematical algorithms that can distinguish discrete and continuous constructs. So far, three such studies have been conducted regarding ADHD, specifically (Haslam et al., 2006; Levy et al., 1997; Marcus & Barry, 2011), and none were able to find support for ADHD as a taxonomic category. Instead, these studies found that ADHD was best conceptualized as a dimensional construct. Furthermore, Coghill and Sonuga-Barke (2012) suggest that while there are still some caveats that need to be investigated further, the results of these three studies are consistent with findings from genetic studies. For example, in one genetics study (Levy et
al., 1997), researchers found that the heritability of ADHD based on a categorical classification system (as in the DSM approach) was not higher than the heritability of ADHD as a trait (i.e., a dimensional approach), suggesting that children with ADHD represented the tail end of a distribution, as opposed to a discrete group.

Given that these issues have yet to be resolved in a satisfactory way, the current study, consistent with the recommendations of Coghill and Sonuga-Barke (2012), sought to use a variety of approaches for defining ADHD – including both categorical and continuous approaches, and measures of specific symptom domains (inattentive and hyperactive/impulsive).

1.1.2 ADHD and Development. Given the high degree of variability associated with typical development, ADHD is not easily distinguished from normal development before the age of 4 years, although hyperactivity is often first noted when children are toddlers (American Psychiatric Association, 2013). In typically developing children, the ability to focus attention and inhibit behavior begin to advance around the age of four years, and those children who have problems with hyperactivity and impulsivity that continue into early grade school are the ones who are most frequently referred for ADHD assessment (see review by Nussbaum, 2012). In later elementary school and middle school, however, problems with inattention become more prominent with increasing demands for self-organization and independent academic work, and referrals for inattention become increasingly likely.

Given that symptoms associated with ADHD are associated with changes in normative development, the current study included age as a covariate in order to control for changes in ADHD symptoms that occur due to ongoing maturation processes.
1.1.3 ADHD and Socioeconomic Status (SES). ADHD has been found to be associated with lower SES in several studies. For instance, in a study by Scahill et al. (1999), ADHD was associated with low income in a community sample of children between the ages of 6 and 12 years. In another study (Sauver et al., 2004), researchers found that ADHD was negatively related to parents’ highest level of education. The authors suggested that lower parental education may be associated with less effective parenting practices and less structure in the home, which could worsen symptoms of ADHD. However, the association between ADHD and SES may be better explained by complex interactions between genetics and environment (Archer, 2011), and low SES is not considered a causative mechanism for the development of ADHD. Given the statistical association between SES and ADHD, this variable was also considered a potential confound in the present study.

1.1.4 ADHD and Sex. According the DSM 5, ADHD is more prevalent in males than females, with a male to female ratio of approximately 2:1 in children (American Psychiatric Association, 2013). However, there is considerable variability in prevalence estimates by sex. For instance, in a recent review (Nussbaum, 2012) male-to-female ratios for ADHD ranged from 2:1 to 9:1. Given the variability in sex ratios for ADHD found in studies to date, some researchers have questioned the validity of estimated prevalence-by-sex ratios. For instance, Nussbaum (2012) points out that the criteria for ADHD were developed on samples that consisted primarily of male children, and females with ADHD exhibit less disruptive characteristics of ADHD, leading to a referral bias that favors male children with more overt hyperactive/impulsive symptoms.
Several studies have investigated sex differences in the presentation of ADHD in children. Several studies suggest that males exhibit higher levels of hyperactivity and/or impulsivity (Gaub & Carlson, 1997; Gershon, 2002; Hartung et al., 2002; Newcorn et al., 2001), although a study by Rucklidge and Tannock (2001) found that females had higher symptoms of hyperactivity than males. There are also studies that report higher levels of inattention in female children with ADHD than males (Rucklidge & Tannock, 2001) or that girls are more likely to have the predominantly inattentive presentation of ADHD than boys (Biederman et al., 2002). Conversely, other studies have reported higher levels of inattention in males (Gaub & Carlson, 1997; Gershon, 2002), and still others reported no significant sex differences (Hartung et al., 2002; Newcorn et al., 2001).

Outcomes in research on sex differences in ADHD seem to depend on several factors. For instance, of the studies that found sex differences in ADHD symptom presentation, two (Gershon, 2002; Hartung et al., 2002) only found significant differences in the above-mentioned symptoms when ratings were based on teacher report, and a third (Gaub & Carlson, 1997) found significant associations in non-referred samples (for example, population and community studies), but not clinical samples. Furthermore, the study by Rucklidge and Tannock (2001) was unusual in that females were found to have greater impairment on all ADHD symptoms based on teacher and parent ratings. On the other hand, that study was also unusual in that it consisted primarily of adolescents with the predominantly inattentive presentation of ADHD (although the authors point out that many of those children had previously met criteria for the combined presentation). Therefore, it would appear that sex differences in ADHD are strongly influenced by factors that may not be specific to the clinical features of ADHD, including sample
source, rater, referral bias, and predominant ADHD symptom presentation of the sample. However, given the potential influence of sex on ADHD presentation, it was considered to be a potential confound in the current study.

**1.1.5 ADHD and Comorbid Psychopathology.** Comorbidity is common in children with ADHD; commonly reported co-occurring problems include conduct disorder, mood and anxiety disorders, and learning disorders (Biederman, 2005). As children with ADHD progress into adolescence, problems with substance use and eating disorders also become more common (see review by Nussbaum, 2012; see also Hinshaw et al., 2012). Recent research also reports increased risk of suicide attempts, non-suicidal self-injury, and overall global impairment in females with ADHD (Hinshaw et al., 2012). Children with ADHD are also reported to use health and mental health services at a higher rate than non-ADHD children, and to engage in behaviors that are deleterious to physical health – such as smoking and substance abuse – more often than their non-ADHD peers (Rowland, Lesesne, & Abramowitz, 2002).

In a recent study (Newcorn et al., 2001), researchers found that comorbid conditions may not just co-exist with ADHD, but may also be associated with the clinical presentation of children with ADHD. For instance, in that study children with ADHD and oppositional defiant or conduct disorders were, overall, more impulsive than any of the groups of children who did not have either oppositional or conduct disorders. Also, children with ADHD and anxiety disorders were found to be less impulsive than children with ADHD and oppositional or conduct disorders. Given the potential impact of comorbid psychopathology on ADHD symptoms, both mood/anxiety and conduct/physical aggression were controlled for in the present study.
1.2 Overview of Abnormal Bodyweight

1.2.1 Definition and Prevalence of Abnormal Bodyweight. Bodyweight can be measured, and therefore “abnormal bodyweight,” including underweight and obesity, can be defined in a number of ways. Body mass index (BMI), is currently the most commonly used method of assessing bodyweight for the purpose of classifying weight status in research (Militão Abrantes, Alves Lamounier, & Antônio Colosimo, 2002). BMI is defined as weight (kg) divided by squared height (m^2) based on the observation that weight changes in proportion to squared height (for more information on this formula see Heymsfield, Gallagher, Mayer, Beetsch, & Pietrobelli, 2007), and is relatively easy to measure and calculate. BMI is recommended above other approaches to assessing bodyweight if only one measure can be used, and provides a valid marker of weight status in children (Ball & McCargar, 2003).

BMI values vary widely depending on the age and sex of the child, necessitating the use of age and sex-adjusted comparison values in order to classify BMI into normal and abnormal categories in children. Presently, there are three sets of percentiles that are most commonly used to generate BMI cut-points for the classification of bodyweight in children and adolescents for research purposes. One set of percentiles was produced by the Centers for Disease Control and Prevention (CDC), based on national data from the United States (Kuczmarski et al., 2002). Another set was produced by the International Obesity Task Force (IOTF), based on international data from six countries (Cole, Flegal, Nicholls, & Jackson, 2007). The most recent approach was developed by the World Health Organization (WHO), and is based on international data for children up to 5 years of age (De Onis & World Health Organization, 2006), and on data from the United States.
for children ages 5 to 19 years of age (Onis et al., 2007). Each method has a similar goal, which is to provide a measure of nutritional and health status based on bodyweight. Each approach provides specific BMI cut-points that help identify children with increased nutrition and overall health risks as a result of abnormally high (obese) or low (underweight) bodyweight (Kuczmarski et al., 2002).

Each set of reference values provides specific cut-off BMI values for boys and girls, separately, at specific age intervals. The cut-points used in the CDC method are based on the 5th and 95th percentiles of BMI for age and gender (Kuczmarski et al., 2002), while the WHO method uses cut-points at 2 standard deviations (SD; approximately equivalent to the 3rd and 97th percentiles) below and above the mean to identify children as underweight and obese, respectively. The IOTF cut-offs, on the other hand, are based on values from centile curves that were produced to pass through the cut points for BMI established for adults, at the age of 18 years. These curves were averaged to generate BMI cut points for abnormal bodyweight for boys and girls (separately) between the ages of 2 and 18 years (Cole, Bellizzi, Flegal, & Dietz, 2000; Cole et al., 2007).

Each of these three methods results in different prevalence estimates of obesity and underweight due to variations in cut-points. For Canadian children, specifically, the differences in estimates are smallest, overall, between the CDC and WHO cut-points, and largest between the IOTF and WHO cut-points (Shields & Tremblay, 2010). Given that both the CDC and WHO methods for classifying children’s bodyweight rely on data from the US, it is worth noting that estimates of obesity for American children (10%) are only slightly higher than those for Canadian children (8%; Shields, 2006). Given that the WHO and CDC cut-points demonstrated smaller differences in prevalence of obesity for
Canadian children, and of these, the CDC approach has been used more often in previous studies on the association among ADHD and abnormal bodyweight, the current study used CDC percentiles to classify children into underweight, normal weight or obese categories.

Furthermore, although one might expect that the WHO estimates of obesity, based on two standard deviations above the mean (or roughly the 97th percentile) would result in more conservative estimates of obesity than those of the CDC, which use the 95th percentile as the cut-off, this is not expected to have a significant influence in the current study, given a recent study that compared prevalence estimates using each of the above methods of classifying children as obese (Shields & Tremblay, 2000). In that study, researchers found that as of 2004, 8-13% of Canadian children were obese, with both the CDC and WHO approaches producing equivalent estimates (13%), while the IOTF-based estimate was lower (8%), with additional variation arising from different approaches to data collection method (Shields & Tremblay, 2010). Furthermore, both the CDC and WHO estimates are consistent with those of a study of Canadian children in 1996 (Tremblay & Willms, 2000), which estimated the prevalence of obesity to be 11.8 and 13.5% for girls and boys, respectively, providing further support for the use of the CDC method in the current study.

Irrespective of the specific classification approach used, the estimates described above (Shields & Tremblay, 2010; Tremblay & Willms, 2000) represent a dramatic increase in obesity over the last few decades. According to Shields (2006), obesity rates have increased by more than 2.5 times between 1978/79 and 2004. Although specific estimates of prevalence of underweight in Canadian children are not yet available, a
recent study (Lazzeri et al., 2013) found that the prevalence of underweight has decreased in many European countries and the US since 1997/98. Underweight is seldom investigated in developed countries, and warrants further investigation (Lazzeri et al.).

1.2.2 Abnormal Bodyweight and Development. Generally, obesity has been found to increase in prevalence as children get older, with lowest prevalence in young children, and highest prevalence in adolescents (Mustillo et al., 2003; Shields, 2006; Wake et al., 2013), although some studies have found decreasing odds of obesity with age (Waring & Lapane, 2008) and higher obesity prevalence among younger children (Turer, Lin, & Flores, 2013). Conversely, underweight has more often been found to be more prevalent in younger children, less prevalent older children and adolescents (Lazzeri et al., 2013; Meyers et al., 2013; Wake et al., 2013), and odds of underweight may even decrease with age (Kim et al., 2014; Waring & Lapane, 2008). That prevalence of underweight decreases and prevalence of obesity rises as children get older is consistent with changes in BMI associated with development. Specifically, children’s BMI tends to decrease from the ages of one to six years, and then generally increases with age throughout the remainder of childhood and adolescence (Cole et al., 2000).

Health risks associated with underweight and obesity vary with age. In one study (Wake et al., 2013), researchers found that poorer health and higher rates of special health care needs were associated with underweight in young children, but with obesity in older children. Moreover, variations in typical BMI trajectory associated with normal development may have different implications for future health. For example, in a recent study (Rolland-Cachera & Peneau, 2013), researchers found that children who had low or normal BMI and then experienced an early adiposity rebound (point at which BMI stops
decreasing in early childhood and begins to increase), higher BMI was associated with high fat mass, and was more likely to be associated with health risks related to obesity. By adulthood, underweight is associated with increased mortality from non-cancer and non-cardiovascular disease causes, while obesity is associated with increased mortality from cardiovascular disease and obesity-related cancers (colon, breast, esophageal, uterine, ovarian, kidney and pancreatic), but not other cancers (Flegal et al., 2007).

1.2.3 Abnormal Bodyweight and SES. An association between low SES and obesity (Chivers, Parker, Bulsara, Beilin, & Hands, 2012; Jansen et al., 2013; Mustillo et al., 2003; Turer et al., 2013; Waring & Lapane, 2008) and underweight (Meyers et al., 2013; Waring & Lapane, 2008) has been found in a number of studies. Most of the research in developed countries has focused on the role of SES in risk of obesity. However, despite the number of studies that have investigated the association between obesity and SES, there is considerable variability in terms of the specific measures used to estimate SES, and which SES variables were found to be associated with obesity across these studies.

In one review on the association between poverty and obesity, Drewnowski and Specter (2004) discuss the positive association between poverty and prevalence of obesity, suggesting that low income is associated with an obesogenic diet due to the inverse relationship between energy density and energy cost. The authors point out that energy-dense foods such as refined grains, high fat foods, and food with added sugar, are more affordable than healthy diets consisting of fresh foods. This may increase the risk of obesity among families who are unable to meet their energy requirements through foods such as meats, fish, whole grains, fruits and vegetables, due to the cost of such foods.
When parental education is the measure of SES, studies have reported a negative relationship between parent education and prevalence of obesity (Chivers et al., 2012; Shields, 2006). The results of the 2004 Canadian Community Health Survey (Shields, 2006) suggest that the relationship between SES and obesity was clearest when examining highest level of education: children from families where the highest level of education achieved by a household member was high school-level education or less were more likely to be obese than those who had members of the household with post-secondary education. Interestingly, children from middle-income households were more likely to be obese than children from lower or higher income families.

Despite some conflicting results in terms of which SES factors may be associated with risk of obesity, overall research suggests a strong association between lower SES and prevalence of obesity. While it is less clear whether SES is associated with underweight in developed countries at this time, the association between SES and underweight found in some studies (Meyers et al., 2013; Waring & Lapane, 2008) suggest that there may be an association. As such, it was decided that SES would be an important variable to control for in the current study.

1.2.4 Abnormal Bodyweight and Sex. Males have generally been found to be at higher risk of both obesity (Tremblay & Willms, 2000; Turer et al., 2013; Waring & Lapane, 2008) and underweight (Meyers et al., 2013; Waring & Lapane, 2008), though there is research that reports higher rates of underweight among girls (Lazzeri et al., 2013), as well as no sex differences in prevalence of obesity (Mustillo et al., 2003).

Sex differences in prevalence of abnormal bodyweight may be at least partly due to differences in development. For instance, BMI curves show sex differences in BMI
trajectories that may be partly influenced by differences in pubertal timing among boys and girls (Cole et al., 2000). Ball and McCargar (2003) also point out that there are developmental differences for boys and girls that also directly influence bodyweight composition (e.g., percentage percent body fat versus lean muscle mass) and which may, in turn, influence health risks associated with abnormal bodyweight for males and females in childhood.

1.2.5 Abnormal Bodyweight and Comorbid Psychopathology. Most of the literature on social-emotional health outcomes has focussed exclusively on overweight/obesity rather than underweight. Research suggests that, relative to normal weight children, obese children have significantly higher odds of receiving a psychiatric referral (Turer et al., 2013), and are much more likely to experience social problems (Drukker, Wojciechowski, Feron, Mengelers, & Van Os, 2009; Erermis et al., 2004; Jansen et al., 2013; Pitrou, Shojaei, Wazana, Gilbert, & Kovess-Masféty, 2010), than normal weight children. Some studies have also found obesity to be associated with a higher risk of emotional problems, in general (Jansen et al., 2013; Turer et al., 2013) and depression and/or anxiety in particular (Erermis et al., 2004; Mustillo et al., 2003; Roberts & Duong, 2013; Waring & Lapane, 2008). However, in some of these studies, associations were found only for certain groups of children. For instance, two studies (Mustillo et al., 2003; Roberts & Duong, 2013) found the association to be significant only for boys. In the study by Mustillo et al. (2003), the associations among obesity and depression were also only significant for children who were chronically obese but not for children with adolescent-onset obesity.
In a recent review, Latzer and Stein (2013) point out that some studies, particularly those based on non-clinical samples, have not found an association between obesity and emotional problems such as depression and anxiety, (see review by Latzer & Stein, 2013) suggesting that comorbidity may differ between clinical and non-clinical populations. In another review Korczak, Lipman, Morrison and Szatmari (2013) suggest that the conflicting results found in several studies that have investigated sex differences in associations among obesity and depression may also be due in part to differences in ages assessed and assessment approaches used across studies.

Other studies have investigated associations among obesity and other types of comorbidities. For instance, several studies have found an association among obesity and behavior or conduct problems (Duarte et al., 2010; Erermis et al., 2004; Griffiths, Dezateux, & Hill, 2011; Turer et al., 2013), oppositional defiant disorder (Mustillo et al., 2003), and aggression (Erermis et al., 2004). Latzer and Stein (2013) also provide a review of several studies that have identified higher rates of eating disorders, and binge eating behavior more generally, in obese children. Interestingly, in the study by Erermis et al., researchers found that scores on the Eating Attitudes Test, a widely-used inventory of eating pathology, were higher for both the clinical obese group and non-clinical obese group, relative to the normal weight control group. Scores on this test of disordered eating were among the few that were elevated in both groups, suggesting that even non-clinical groups of obese children may be at higher risk of eating pathology.

As mentioned earlier, very few studies have investigated the mental health outcomes of children who are underweight in developed countries. In the few studies that have investigated this issue, most have not found underweight to be associated with
adverse mental health outcomes in children (Drukker et al., 2009; van Grieken et al., 2013; Waring & Lapane, 2008). However, several studies have found underweight children to be at higher risk for adverse psychosocial, cognitive and academic outcomes. For instance, underweight has been associated with maltreatment related to appearance (van Grieken, Renders, Wijtzes, Hirasing, & Raat, 2013), lower cognitive performance (Bisset, Fournier, Pagani, & Janosz, 2013), and learning and behavioral developmental disabilities (Phillips et al., 2014). Children who are underweight may also meet criteria for anorexia nervosa if their weight status is associated with symptoms such as calorie restriction, distorted body image, and fear of gaining weight (American Psychiatric Association, 2013).

Additional research is needed to help identify whether or not there is an association among underweight and other mental health problems in children, and the current study provided an important opportunity to examine the association among underweight and ADHD, specifically. Furthermore, given the lack of research regarding the potential relationship of other mental health problems to underweight, and the previous findings of an association among obesity and other mental health problems, including depression, anxiety and conduct problems, the current study controlled for emotional disorder/anxiety and conduct problems/physical aggression in adjusted models.

1.3 The Association between ADHD and Abnormal Bodyweight

1.3.1 History (Early Studies). The earliest studies investigating an association among bodyweight and ADHD were generally concerned with whether or not children with ADHD who were treated with stimulants might experience suppression of growth of either height and/or weight (see review by Spencer et al., 1996). However, researchers
eventually began to question whether delays in growth were restricted to ADHD children treated with stimulants and at least one study found evidence for a delayed tempo of growth in height in early, but not late, adolescence among children with ADHD (Spencer et al., 1996). Later, another study in this line of research also found evidence of growth suppression and altered fat metabolism (evidenced by higher percentages body fat and larger abdominal circumference) in children with ADHD relative to those without ADHD (Ptacek, Kuzelova, Paclt, Zukov, & Fischer, 2009). Furthermore, growth suppression in that study was not associated with ADHD medication, although medication use was associated with a lower percentage of body fat in children with ADHD.

In the first study to specifically examine an association between ADHD and obesity, Altfas (2002) reported a high prevalence of ADHD among adults receiving treatment for obesity and an association between ADHD and decreased weight loss among patients in treatment. Altfas (2002) hypothesized that impairments related to ADHD, including problems with attention and impulsivity, might contribute to the development of obesity, or that attentional mechanisms might somehow be associated with metabolism and regulation of the body’s energy resources that contribute to obesity. This study spurred a new line of research that sought to determine whether or not ADHD was associated with abnormal bodyweight in clinical populations of obese children, as well as ADHD populations and non-clinical populations.

These early studies prompted a surge of research that sought to replicate these early findings of an association between ADHD and abnormal bodyweight, particularly obesity. As those studies proliferated, researchers also began to develop hypotheses regarding the mechanisms that might underlie the associations that were continuing to be
found. Research into these mechanisms is critical to developing a better understanding of the nature of the relationship between ADHD and abnormal bodyweight and the etiology of both disorders. However, in line with the majority of the current research in this area the focus of present study is to help establish whether or not the associations between ADHD and abnormal bodyweight are systematic and robust. Therefore, the following sections will be concerned with examining the research into the association between ADHD and abnormal bodyweight that has been conducted to date.

Nevertheless, information regarding the issues that need to be addressed given that an association between ADHD and abnormal bodyweight has been found, and an overview of the hypotheses proposed to explain this association are useful in appreciating the complexities of the relationships being considered. For instance, although a statistical relationship between ADHD and abnormal bodyweight has been found, one must rule out alternate explanations for this association before concluding that this association is indicative of a genuine relationship between these conditions. At this time three of these potential alternative explanations can be ruled out: that the association is due to referral bias, error, or chance alone. A more detailed discussion of these issues is provided in Appendix A (“Issues to Address in Explaining the Association between ADHD and Abnormal Bodyweight”).

Having ruled out these potential explanations, the focus of explanatory efforts turns to developing hypotheses regarding the mechanisms that might underlie the association between ADHD and abnormal bodyweight. To date, several mechanisms have been hypothesized to help explain this relationship, most of which fall into one of three primary explanatory categories: ADHD and abnormal bodyweight arise from
common biological mechanisms, ADHD or ADHD-related symptoms contribute to the development of abnormal bodyweight, or obesity or obesity-related factors contribute to ADHD or symptoms of ADHD. For instance, shared genetic risk factors (see review by Albayrak et al., 2013), problems with dopaminergic functioning (Liu, Li, Yang, & Wang, 2008; Verbeken, Braet, Lammertyn, Goossens, & Moens, 2012) or sleep (Harvey, Murray, Chandler, & Soehner, 2011; Pulgarón, 2013) are among the proposed biological mechanisms proposed to underlie the association between ADHD and abnormal bodyweight, while impulsivity and inattention are among the ADHD-related mechanisms proposed to contribute to the development of obesity (for instance, see review by Cortese & Vincenzi, 2012). A more detailed discussion of these and other mechanisms proposed to underlie the association between ADHD and abnormal bodyweight are provided in Appendix B (“Mechanisms Proposed to Explain the Association between ADHD and Abnormal Bodyweight”).

1.3.2 Current Empirical Support for an Association between ADHD and Abnormal Bodyweight. Most studies to date have focussed on establishing whether or not the association between ADHD and abnormal bodyweight is systematic and robust. These studies comprise a critical component of research in this area, and findings are mixed and vary considerably among different types of studies and methodological approaches used. The following sections provide a review of the empirical support for an association between ADHD and abnormal bodyweight found to date, using clinical, community and population samples.
1.3.2.1 Studies based on clinical samples. Clinical studies based on ADHD samples
have been inconsistent in both their methodologies and their findings regarding an
association among ADHD and abnormal bodyweight. Some studies have compared
prevalence of abnormal bodyweight in a clinical sample to those of a reference
population (i.e., national norms). Of these, two (Holtkamp et al., 2004; Yang, Mao,
Zhang, Li, & Zhao, 2013) found a higher prevalence of obesity in their samples of
ADHD children relative to children in the reference population, while two (Curtin,
Bandini, Perrin, Tybor, & Must, 2005; Fliers et al., 2013) did not. Fliers et al. (2013)
found a lower prevalence of obesity among 5-9-year-old girls and a lower prevalence of
underweight among 5-9-year-old ADHD boys and 10-12-year-old ADHD girls,
compared to the general population. Of the other nine age/gender groups examined in that
study, there were no significant differences in prevalence of obesity in ADHD children
relative to children in the general population. It is also worth noting that in the study by
Curtin et al. (2005), researchers did find that prevalence of obesity was higher among
ADHD children who were not on medication than among ADHD children who were
taking medication.

Five other clinical studies have compared prevalence of abnormal bodyweight in
children with ADHD to that of a control group. Three of these studies did not find a
higher prevalence of obesity among children with ADHD relative to the control group
(Hanč et al., 2014; Hubel, Jass, Marcus, & Laessle, 2006; Pauli-pott, Albayrak,
Hebebrand, & Pott, 2010), and one (Dubnov-Raz, Perry, & Berger, 2011) found a lower
prevalence of obesity in the ADHD group. However, of these studies, two (Hubel et al.,
2006; Pauli-pott et al., 2010) excluded children on medication, two (Hanč et al., 2014;
Hubel et al., 2006) excluded females, and two (Dubnov-Raz et al., 2011; Hanč et al., 2014) excluded children with common comorbid disorders (e.g., mood disorders), thus limiting the generalizability of these findings, especially for clinical ADHD populations, in which all of these variables are associated with ADHD and abnormal bodyweight, as reviewed earlier.

The fifth study (Güngör, Celiloglu, Raif, Ozcan, & Selimoglu, 2013) avoided these methodological shortcomings. In that study, in addition to including a control group, and including children of both sexes, children with common comorbidities, and those taking medication for ADHD, researchers also examined several measures of obesity and underweight. These measures included BMI, weight-for-height (similar to BMI, but adjusted only for gender, not age), mean standardized weight and mean standardized height, as well as triceps and subscapular skin fold tests. Using weight-for-height, Güngör et al. (2013) found that the ADHD group had a significantly higher prevalence of both moderate and severe underweight (3.3% and 7.7%, respectively) compared to the control group (0.2 and 2.8% respectively). Likewise, the ADHD group had a significantly higher prevalence of obesity (24.8%) than the control group (18.9%). When using BMI, there were significant differences in proportion of ADHD found among different weight groups, but the significant differences were for the obese group rather than the underweight group. In fact, BMI was the only measure in which underweight was not significantly associated with ADHD, suggesting that future studies should consider using weight-for-height when investigating underweight in children.

Finally, while the methodology used by this study avoided many of the pitfalls common
to other clinical studies of ADHD children, researchers did not control for comorbidity or SES, which are potential confounds.

Two additional studies investigated the prevalence of ADHD in clinical samples of obese children. In the first (Erermis et al., 2004), researchers compared prevalence of ADHD among three groups: a clinical obese group, a non-clinical obese group, and a control group of non-obese children. They found a higher prevalence of ADHD in the clinically obese group (13.3%) compared to both the non-clinical obese group (3.3%) and the control group (3.3%). In the second study (Braet, Claus, Verbeken, & Van Vlierberghe, 2007), researchers sought to investigate symptoms of ADHD and ADHD prevalence among a clinical sample of obese children and a community control group. While they found group differences in symptoms of hyperactivity, impulsivity and inattention, they did not find prevalence of ADHD diagnosis to differ between groups. Also, when the sample was stratified by gender, researchers found that the association among obesity and symptoms of ADHD was only significant for boys. However, this study had some methodological issues, including not controlling for comorbidity and excluding underweight children from their sample, which may have influenced their findings.

1.3.2.2 Population and community studies. Although it is helpful to know whether or not there is a relationship between ADHD and abnormal bodyweight in clinical populations, such samples represent a small proportion of individuals and as previously discussed, studies on such samples are often plagued by methodological issues that make the interpretation of results difficult. Population and community studies both provide an opportunity to examine an association between ADHD and abnormal
bodyweight in non-clinical populations. Population studies may be considered a gold standard of non-clinical research, in that they afford the opportunity to investigate these associations in large samples that are representative of the populations from which they are derived. Community studies provide similar information, but are generally based on smaller samples and are generalizable only to other communities that are similar in sociodemographic composition to the original samples.

A number of population studies have found a significant association among ADHD and abnormal bodyweight in children. In some, ADHD has been associated with increased risk of obesity (Chen, Kim, Houtrow, & Newacheck, 2010; Erhart et al., 2012; Khalife et al., 2014; Kim, Mutyala, Agiovlasitis, & Fernhall, 2011; Phillips et al., 2014; van Egmond-Fröhlich, Widhalm, & de Zwaan, 2012; Waring & Lapane, 2008). In others, obesity has been found to be associated with increased odds of ADHD (Erhart et al., 2012; Halfon, Larson, & Slusser, 2013). In the study by Waring and Lapane (2008), ADHD was also found to be associated with increased odds of underweight.

In some of these studies, however, the association between ADHD and abnormal bodyweight was found only for specific groups. In some studies (Kim et al., 2011; Waring & Lapane, 2008) only children with ADHD who were not medicated had increased odds of obesity while in another study (Byrd, Curtin, & Anderson, 2013), researchers did not find ADHD to be associated with obesity in unmedicated children with ADHD, and actually found that children who were medicated had lower odds of obesity than non-ADHD children. In another study (van Egmond-Fröhlich et al., 2012), the association between ADHD and obesity was only significant for girls with ADHD, but not for boys. With respect to underweight, Kim et al. (2011) found that only children
with ADHD who were medicated had increased odds of underweight, and Erhart et al. (2012) found no association between ADHD and underweight.

In studies based on community samples, results were similar. For instance, several studies found ADHD to be associated with increased odds of obesity (Kim et al., 2014; Koshy, Delpisheh, & Brabin, 2011; Lam & Yang, 2007; Rojo, Ruiz, Domínguez, Calaf, & Livianos, 2006), while two others did not find such an association (Mustillo et al., 2003; Pitrou et al., 2010). It is worth noting, however, that the two community studies that did not find ADHD to be associated with obesity were investigations into associations among obesity and mental health problems in general, of which ADHD was one among several variables being investigated, and neither study controlled for medication use. Finally, only one community study examined ADHD and underweight, specifically, and no association was found (Kim et al., 2014).

All of the studies in this section investigated the relationship between ADHD symptoms and abnormal bodyweight based on continuous or dichotomous (i.e. “ADHD” based on cut-off scores on rating scales) variables. Neither of these approaches is ideal for identifying a non-linear relationship between ADHD symptom severity and abnormal bodyweight. It is possible that inconsistencies in study outcomes could be a result of a non-linear relationship between ADHD and underweight and/or obesity.

1.3.2.3 Summary of overall findings of studies on the association between ADHD and abnormal bodyweight. In general, population and community studies provide strong support for an association between ADHD and obesity in non-clinical populations, with most finding significant associations. Findings from clinical samples, on the other hand, are far more inconsistent. However, as previously discussed, most of
these studies suffer from significant methodological issues (e.g., absence of a control group and exclusion of children with comorbid disorders, females, and/or children on medication) that made it difficult to draw conclusions regarding an association between ADHD and abnormal bodyweight in typical clinical populations. Of the two studies that did not experience these methodological issues (Erermis et al., 2004; Güngör et al., 2013), both found a significant association between ADHD and obesity and one (Güngör et al., 2013) also found an association between ADHD and underweight. Unfortunately, no clinical studies have been conducted on prevalence of ADHD in underweight populations, specifically, and only two studies (Fliers et al., 2013; Güngör et al., 2013) have examined underweight in a clinical sample of ADHD children. Therefore, given the mixed findings on the potential association between ADHD and underweight in non-clinical samples, the status of this potential association is uncertain at this time.

1.3.3 Unresolved Issues in Current Research. There are still many unresolved issues in the research regarding the association between ADHD and abnormal bodyweight. What follows is a review of some of the key issues that have yet to be resolved, and which will be addressed in the present study.

1.3.3.1 Role of underweight in the association between ADHD and abnormal bodyweight. There is a dearth of research regarding the association between ADHD and underweight. It is not clear whether ADHD is associated specifically with obesity or with abnormalities in bodyweight more generally. Furthermore, given that some studies have found an association between ADHD and underweight, it is problematic that many studies have included underweight children in normal weight (or “non-obese”)}
comparison groups, as this may have weakened associations found between ADHD and obesity, and contributed to inconsistencies in outcomes across studies.

**1.3.3.2 Effect of definitions of ADHD on outcomes.** It is unclear to what extent the way in which ADHD variables are measured and defined influences outcomes. This issue might be particularly important given that the way in which ADHD is measured and defined in a study influences the interpretations that can be made based on the outcomes of that study. For instance, if ADHD is measured as a continuous variable, then no conclusions can be made regarding an association among ADHD, as a clinical diagnosis, and abnormal bodyweight. Instead, one may draw more general conclusions regarding a possible association between symptoms of ADHD and abnormal bodyweight. Moreover, many of the population studies conducted previously relied on either symptom scores or previous diagnoses in order to establish the presence of ADHD. However, a clinical diagnosis of ADHD is based on more than just symptom counts and therefore it remains unclear whether there is an association between ADHD, as a clinical entity, and abnormal bodyweight in non-clinical populations.

Additionally, few studies, particularly non-clinical studies, have investigated the association between abnormal bodyweight and specific ADHD symptom domains, such as hyperactivity/impulsivity and inattention. Therefore, it is still unclear to what extent specific dimensions of ADHD symptoms might be associated with abnormal bodyweight.

**1.3.3.3 Linearity of the association between ADHD and abnormal bodyweight.**

So far, studies on the association between ADHD and abnormal bodyweight have used either continuous and/or dichotomous (i.e. “ADHD” versus “non-ADHD”) approaches to defining ADHD, using statistical approaches that are limited to identifying linear
relationships. To date, there do not appear to be any studies that have investigated whether or not the association might actually be non-linear in nature.

1.3.3.4 Influence of covariates. As discussed earlier, SES, age, sex, medication use, and comorbid mood, anxiety and conduct problems have all been associated with ADHD and abnormal bodyweight in a number of previous studies. Given the variability in how these variables were addressed across studies, if at all, the extent to which they might influence the association between ADHD and abnormal bodyweight is still uncertain.

1.3.3.5 Influence of abnormal bodyweight on ADHD outcomes in non-clinical populations of children. While there are several clinical studies that address the potential association between ADHD and abnormal bodyweight in both ADHD and obese populations, most population and community studies have investigated this relationship from the perspective of bodyweight as the outcome variable. While this makes sense theoretically, given the number of ways in which ADHD symptoms could influence bodyweight, it is possible that the association between ADHD and abnormal bodyweight may differ when abnormal bodyweight is the predictor variable. For example, in one study (Erhart et al., 2012) that examined the relationship between ADHD and obesity from both perspectives, researchers found that the odds of ADHD for overweight children were more than twice those for normal weight children, while the odds of obesity for ADHD children were 70% higher than for non-ADHD children, suggesting that the increase in odds was greater when abnormal bodyweight was the predictor variable.
It is important to know whether and to what extent underweight and obese children in the general population might be at higher risk of experiencing ADHD or symptoms associated with ADHD. For instance, previous research has found that ADHD could hinder treatment of unhealthy bodyweight (for instance see Cortese & Castellanos, 2014), therefore effective screening of this population would be essential if children with abnormal bodyweight are, indeed, at significantly higher risk of ADHD.

1.3.3.6 Robustness of the association between ADHD and abnormal bodyweight in non-US populations. There have now been a number of population studies that have investigated the association between ADHD and abnormal bodyweight in children, however the majority of these population studies have been conducted on US populations. While a few others were carried out using population samples from Germany (Erhart et al., 2012; van Egmond-Frölich, 2012), Australia (Jansen et al. 2013), and Finland (Khalife et al., 2014), given the inconsistencies in outcomes across these studies, it is unclear whether or not differences in the characteristics of specific populations might result in differences in the associations found (or not) between ADHD and abnormal bodyweight. For instance, it is possible that the associations found in the United States are influenced by its obesity epidemic, its ethnic composition, or the absence of a universal healthcare system. Therefore, it remains to be seen whether the association between ADHD and abnormal bodyweight will be found in countries that differ from the United States on those characteristics. For instance, Canada has a very different ethnic composition and a universal health care, and at present there are no studies that have investigated the association between ADHD and abnormal bodyweight in Canadian children, specifically.
1.4 Rationale for Current Study

1.4.1 Goals and Objectives. The primary goal of the present cross-sectional study was to investigate whether ADHD is associated with increased risk of underweight and/or overweight (Objective 1), and whether underweight and obesity is associated with increased risk of ADHD (Objective 2), in a national sample of Canadian children. These objectives allow me to address several unresolved issues discussed above, including whether or not the associations found previously are robust, whether or not the outcomes found in previous population studies (conducted mostly on American populations) would be found in a Canadian sample of children, whether or not the association between ADHD and abnormal bodyweight is specific to obesity or pertains to underweight as well, and whether such an association changes depending on which variable is the predictor variable.

The secondary goal was to approach the abovementioned objectives using methods that would permit the remaining unresolved issues to be addressed. First, in order to clarify whether the association between ADHD and abnormal bodyweight is specific to ADHD as a clinical diagnostic entity (a taxon) or to symptoms associated with ADHD as a dimensional trait, three separate ADHD variables were created: one dichotomous variable that would approximate a clinical ADHD diagnosis, one continuous variable, and one multi-categorical symptom severity variable. The latter variable permitted examination of whether or not the association between ADHD and abnormal bodyweight might be non-linear in nature. Next, additional ADHD variables were created to help clarify the relationship between abnormal bodyweight and the symptom dimensions of hyperactivity/impulsivity and inattention. Finally, to clarify the
impact of several important confounds found in previous studies, age, sex, SES, medication use, emotional disorder, anxiety, and conduct problems, were included as covariates in adjusted models.

1.4.2 Predictions. Several predictions were made based on outcomes of previous research. First, it was predicted that there would be an association between ADHD and obesity in Canadian children, given the similarity of Canadian children to American children on many important factors, including prevalence of obesity, risk factors associated with ADHD and obesity in both populations, and because underweight children would not be included in the “normal weight” comparison group. Second, it was predicted that underweight would be associated with ADHD, particularly among medicated children. Third, it was predicted that the nature of this relationship would change, if only in magnitude, depending on how ADHD was measured. For instance, given the previous literature regarding the dimensional nature of ADHD, including heritability of ADHD symptoms across the spectrum of symptom severity, it was expected that the categorical ADHD variable would reveal elevated odds of obesity across ADHD symptom severity groups, and that the dichotomous variable would not be as strongly associated with obesity as the variables that allowed an examination of a range of ADHD symptoms. Finally, it was predicted that both hyperactive/impulsive and inattention symptoms would contribute to the association between ADHD and obesity, given that the two dimensions are usually highly correlated and that both dimensions have been associated with obesity in previous studies.
Chapter 2: Methods

2.1 Participants

The data for this study come from the National Longitudinal Survey of Children and Youth (NLSCY; Statistics Canada, 2007), a prospective long-term survey conducted by Statistics Canada and Human Resources and Skills Development Canada (HRSDC). The NLSCY was designed to examine factors that influence important aspects of child health and development, including social-emotional well-being and behaviour. To do so, the NLSCY followed Canadian children longitudinally from birth to early adulthood. Data collection was carried out at two-year intervals, beginning with cycle 1 in 1994-5, and continuing through cycle 8 in 2008-9.

Data from cycle 4 (2000-2001) was analysed for children between the ages of 4 and 11 years. Cycle 4 was the last cycle that included data for children 4 to 11 years of age without any gaps in age (Statistics Canada, 2003). The original cross-sectional sample for cycle 4 was comprised of children aged 0 to 17 years as of January 1, 2001 (Statistics Canada, 2003), selected to be representative of the Canadian population of civilians in Canada’s 10 provinces, excluding children living on Indian reserves, crown lands, in institutions, those whose families were full-time members of the Canadian Armed Forces, or were residing in some remote regions at the time of the survey (Statistics Canada, 2007). The sample was created by random sampling based on a stratified, multistage design, and is described in detail in documentation provided by Statistics Canada (Statistics Canada, 2003).
The data used for the present study were collected directly from the person most knowledgeable (PMK) about the child, typically the mother (97.2%), via computer-assisted personal interviews (Statistics Canada, 2007). Participation in the survey was voluntary, and the overall response rate was 83.2% (Statistics Canada, 2003). Since the NLSCY is a probability sample, cross-sectional weights were provided to ensure that the sample would be representative of the Canadian population at the time of data collection. Details regarding the procedures followed to create the cross-sectional weights are provided in detail by Statistics Canada (2003).

Following the recommendations of Statistics Canada (2003, p. 131), cross-sectional sampling weights were used to create adjusted weights for the final sample, such that the weights had a mean weight of one and a sum of weights equal to the sample size. This created a final weighted sample that maintained the same distributions as would have been obtained using the original sampling weights with the full sample, and helped prevent over-estimation of the significance of results obtained (Statistics Canada, 2003). Since the final sample was comprised of only those children for whom valid data were available for all measures (see below), weights were adjusted through the process of rescaling, wherein the original cross-sectional weight for each child was divided by the average cross-sectional weight of the sample.

Access to NLSCY data is restricted, and researchers must submit an application to the Canadian Initiative on Social Statistics (CISS) Access to Research Data Centres Program to access the microdata files (Statistics Canada, 2014). The RDC Program is a joint initiative of Statistics Canada, the Social Sciences and Humanities Research Council, and the Canadian Institutes of Health Research, which provides researchers with
access to such data. Approval for this project was obtained following a review of the project proposal by the RDC-Access Granting Committee in January, 2013. The ethics review board at the University of Toronto determined that this project did not require further approval.

2.2 Measures

2.2.1 ADHD Variables

2.2.1.1 ADHD scale. ADHD was measured as a continuous variable, based on scores on the Hyperactivity-Inattention Scale, which included items from the Ontario Child Health Study and the Montreal Longitudinal Survey (Statistics Canada, 2003). Questions relating to hyperactivity/impulsivity were: how often would you say that your child (1) can’t sit still or is restless? (2) is impulsive, acts without thinking? (3) has difficulty awaiting turn in games or groups? and (4) cannot settle on anything for more than a few minutes? Questions related to inattention were: how often would you say that your child (1) is easily distracted, has trouble sticking to any activity? (2) can’t concentrate, can’t pay attention for long? and (3) is inattentive? It should be noted that one hyperactivity/impulsivity question that was included in previous cycles (how often would you say that your child fidgets?) was removed from this scale for cycle 4, because respondents found it to be redundant.

Questions were answered on a 3-point likert scale, where 1=’never or not true,” 2=“sometimes or somewhat true,” and 3=“often or very true.” Responses were rescaled so that the lowest possible score was 0, indicating no evidence of the symptoms; the final scale score therefore ranged from 0 to 14. Cronbach’s Alpha was .82 for this scale, suggesting that it has a high level of reliability (Statistics Canada, 2003).
This scale was evaluated by Charach, Lin and To (2010), using current methylphenidate use, diagnosis of emotional disorder, and functional impairment as criteria by which to judge its sensitivity (accuracy in identifying children with ADHD as ADHD cases) and specificity (accuracy in correctly identifying children without ADHD as non-cases) in classifying children with ADHD. Results suggested that the scale can be used in population studies to identify children with clinically significant levels of ADHD symptoms, with higher cut-offs being associated with highly specific, but not very sensitive, identification of ADHD cases. Although the Charach et al. (2010) study was based on the scale as it was used in cycle 1 (including the redundant item removed in cycle 4), the same factor structure was used when re-assessing reliability of the scale in cycle 4 (Statistics Canada, 2003).

Finally, it should be noted that while the items included in this scale were validated (in the context of the original studies) using the third edition of the Diagnostic and Statistical Manual of Mental Disorders, as Charach et al. (2010) point out, the underlying constructs of ADHD are consistent with those identified in the fourth edition (American Psychiatric Association, 2000), including impulsivity, over-activity and inattention, and also remain consistent with the constructs identified in the fifth edition (American Psychiatric Association, 2013).

2.2.1.2 Categorical ADHD symptom severity. This variable was created specifically for this study using scores from the ADHD Scale variable to provide a categorical measure of symptom severity, and included 4 ratings: 0=no symptoms (scale score of 0), 1=mild symptoms (scale score between 1 and 5), 2=moderate symptoms (scale score between 6 and 9), and 3=severe symptoms (scale score of 10 or higher). The
percentile ranks associated with the cut-offs for these categories were 13.1, 26.4-73.5, 81.7-95.6, and 97.0, respectively (see Table 3 for exact proportion of sample in each category). These percentile ranks suggest that children in the “moderate” and “severe” groups are experiencing clinically significant levels of ADHD symptoms, while those in the “no” or “mild” groups are not, based on PMK-ratings.

Both the continuous measure of ADHD symptoms from the ADHD scale and this categorical ADHD symptom severity variable made it possible to examine the relationship between ADHD symptoms and abnormal bodyweight; however each has a potential advantage over the other. If the relationship between ADHD and abnormal bodyweight is linear, then keeping ADHD as a continuous variable provides the most information (Pasta, 2009). Conversely, if the relationship between ADHD and bodyweight is not linear, then considering ADHD symptoms categorically will allow for a better analysis of this more complex relationship (Pasta, 2009).

2.2.1.3 ADHD proxy diagnosis. To address the question of whether there is a relationship between ADHD as a diagnostic entity (versus symptoms of ADHD), a variable was needed that could identify cases that would most likely meet diagnostic criteria for ADHD. Since the NLSCY did not carry out diagnostic interviews, both scores on the ADHD scale and PMK responses to the question: “Does [your] child have any of the following long-term conditions: Attention Deficit Disorder?” were used to create an ADHD proxy diagnosis variable. For the ADHD scale, the cut-off recommended by Charach et al. (2010) was chosen to minimize the overall rate of error (i.e., false negatives and false positives). However, this cut-off needed to be adjusted to account for the removal of the one item in cycle 4. The resulting cut-off was a score of 10 on the
ADHD scale. Thus, any child with a score on the ADHD scale of 10 or higher was identified as likely having ADHD (ADHD Proxy Diagnosis=1/yes).

However, to avoid having another variable based only on ADHD symptom ratings, and to reduce the rate of false negatives associated with this approach (see Charach et al. for specific error rates), this threshold was lowered to a score of 6 (percentile rank 81.7) for children who were identified by the PMK as having been diagnosed with ADHD by a health professional. Thus, children who had been diagnosed in the community and who were still experiencing clinically significant symptoms of ADHD at the time of the study were included in the ADHD group. Conversely, those identified by the PMK as having been diagnosed with ADHD but whose current symptom count was less than 6 were not included in the ADHD group. These criteria helped to eliminate children who may have outgrown symptoms since being diagnosed or who may have been misdiagnosed in the community (though in the final sample, the prevalence was 0%).

Thus, children scoring greater than 10 on the ADHD scale, or children scoring greater than 6 who had already been diagnosed with ADHD by a health professional, were identified as likely having ADHD, and therefore received a proxy diagnosis of ADHD; all other children were identified as not having ADHD. In the final sample, the vast majority of children identified as using “Ritalin or similar” medications (99.5%), and all children who had been diagnosed by a health professional in the community, were classified as having a proxy diagnosis of ADHD, suggesting that this variable provides a good estimate of ADHD prevalence in this population. Furthermore, the overall prevalence of ADHD in this sample (5.3% based on this variable), is consistent with the
prevalence of ADHD in Canadian children (4.8%) estimated by Waddell, Offord, Shepherd, Hua, and McEwan (2002), based on their review of six studies that used large (N>1000) community samples, standardized assessment measures and multiple informants (e.g., both parent and teacher), and assessed functional impairment, as well as with the world-wide prevalence (5.2%) estimated in a meta-regression analysis by Polanczyk et al. (Polanczyk et al., 2007), based on DSM and ICD criteria.

2.2.1.4 Hyperactivity/impulsivity and inattention symptom severity variables.

Given that hyperactivity/impulsivity and inattention symptoms may each contribute to the relationship between ADHD and abnormal bodyweight, categorical variables were created that would provide a separate measure of hyperactivity/impulsivity and inattention symptoms based on scores on the hyperactivity/impulsivity and inattention questions from the ADHD Scale. Due to the unequal range of scores for each of these constructs, these variables were created by grouping scores into 3 categories: 0=no symptoms, 1=low-moderate symptoms, and 2=high symptoms. Cut-offs for the high symptoms groups were created using the scores on each scale that distinguished the 97th percentile. For the hyperactivity/impulsivity scale, this was a score of 6, and for the inattention scale, this was a score of 5. The no symptom group comprised children with a total score of 0 on the associated items, while those in the mild-moderate symptoms group included all other possible scores.

2.2.2 Bodyweight Classification Variable. The bodyweight classification variable created for this study was based on body mass index (BMI; weight in kilograms, divided by the square of height in metres). Since interpretation of BMI varies considerably during development based on both the age and gender of a child (Rolland-
Cachera et al., 1982), I chose to use the CDC BMI percentiles variable, which classifies children’s BMI into percentiles ranging from “less than the 3rd percentile” to the “97th percentile,” based on American height and weight data for children and youth. This original NLSCY variable was created using 2000 CDC growth charts, which provided BMI percentiles based on gender and age, with age divided into monthly intervals (Statistics Canada, 2005). Detailed information regarding the development of the 2000 CDC growth charts is available elsewhere (Kuczmarski et al., 2002). As discussed earlier, although the CDC growth charts are based on data from the US, estimates of obesity American children (10%) and Canadian children (8%) are similar (Shields, 2006).

The BMI percentile variable was then used to create a categorical weight variable that would allow for a comparison of both underweight and obese children to normal weight children such that children with BMI below the 5th percentile were classified as “underweight,” and those with BMI at or above the 95th percentile were classified as “obese,” while children with BMI at or above the 5th percentile and below the 95th percentile were classified as “normal weight.” These are the cut-offs recommended by the CDC to identify children whose bodyweight places them at higher nutritional risk and overall health risk (Kuczmarski et al., 2002). These cut-offs have also been used to identify underweight and/or obesity in children in other studies investigating the association of ADHD and bodyweight (for example, see Chen et al., 2010; Güngör et al., 2013; Kim et al., 2011; Phillips et al., 2014), allowing for a comparison of our findings with those of previous studies conducted with different populations.
2.2.3 Covariates

2.2.3.1 Age. Several age variables are available in the NLSCY, of which two were used. The first variable was effective age (in years), which was calculated as 2000 minus year of birth, and determined which set of questions would be used for specific children. In the current project, this variable was used to select the sample of 4 to 11 year-olds, as it ensured that the PMK for each child included in the final sample was administered the appropriate question sets. The second variable was actual age in years, which was the age variable used in analyses.

2.2.3.2 Sex. A recoded version of the available dichotomized sex variable was used, such that 0=male and 1=female.

2.2.3.3 SES. For the present project, the highest education of PMK or spouse variables were used to estimate SES, as education is indicative of not just the parent’s employment and income potential, but also of how parents may be more or less able to positively influence their children’s socioeconomic conditions (Galobardes, Lynch, & Smith, 2007). Furthermore, parent education has been associated with both ADHD (Sauver et al., 2004) and abnormal bodyweight (Shields, 2006) in previous studies. Given that the PMK of the majority of children (97.2%) was the biological or adoptive mother, and that mother’s education has been associated with child development and the quality of the home environment (Statistics Canada, 2003), and ADHD (Sauver et al., 2004), the mother’s highest education variable was used wherever possible. When mother’s highest education was not available, the PMK education variable was used; if this information was missing, the spouse’s highest education (as reported by the PMK) variable was used.
The final variable provided a scale of educational attainment, with scores ranging from 0 = no schooling through 11 = graduate degree completion.

2.2.3.4 Medication use. Two variables were used to control for the potential influence of both ADHD and non-ADHD medications used by children in this study: “ADHD medication” and “other medication”. Use of ADHD medications was determined by the PMK’s response to the question “Does he/she take any of the following medications on a regular basis: Ritalin or other similar medications?” Use of other medications, excluding asthma puffers, tranquilizers or nerve pills, and anti-convulsants or anti-epileptic pills, was determined by the PMK’s response to the question “Does he/she take any of the following medications on a regular basis: Other?” This would include children who were regularly taking antidepressants, antipsychotics, etc., which might be associated with ADHD, non-normal bodyweight, and other psychological problems, while allowing us to maintain adequate cell sizes in analyses.

2.2.3.5 Other psychological problems. Two other NLSCY variables were used in this study, to control for their potential influence on both ADHD and bodyweight status: the Emotional Disorder and Anxiety Scale (EDA Scale), and the Conduct Disorder and Physical Aggression Scale (CDPA Scale). The EDA Scale included 7 items from the Ontario Child Health Study. The CDPA Scale included 6 items taken from the Ontario Child Health Study and the Montreal Longitudinal Survey (Statistics Canada, 2003). As with the ADHD Scale, questions for these scales were answered on a 3-point likert scale, where 1=”never or not true,” 2=“sometimes or somewhat true,” and 3=“often or very true,” and responses were rescaled, so that the lowest possible score was 0, indicating no evidence of the symptoms. The EDA Scale scores ranged from 0 to 14, with a
Cronbach’s Alpha value of .74, while the CDPA Scale scores ranged from 0 to 12, with a Cronbach’s Alpha of .77 for children aged 4-11 years, in cycle 4 (Statistics Canada, 2003). It should also be noted that two items from previous cycles were removed from the EDA Scale in cycle 4 and all subsequent cycles (Statistics Canada, 2003).

2.3 Analytic Approach

2.3.1 Data cleaning. Of the 30,500 children in cycle 4, 13,700 were classified as 4 to 11 years of age, based on effective age. The final sample was comprised of all cases that had valid data on all variables to be used in analyses. Generally, data were assumed to be valid for each variable if:

1. Responses were provided for all variables used in analysis, or required to create the variables used in analysis, including age, gender, PMK or spouse highest education, BMI, ADHD scale, EDA scale, CDPA scale, ADHD and “other” medication use, and cross-sectional weight. For scale variables, this meant that valid responses were provided for each question included in the scale; no imputations were used.

2. Responses on basic variables such as birth year (used to calculate age) or gender were consistent with those provided at earlier cycles, or could be determined from previous responses, for children for whom data were available in previous cycles. For instance, if gender was recorded as male in cycles 1-3, but female in cycle 4, a correction would be made in cycle 4. However, if there were only two records available, and these were in conflict, the case was removed from analysis due to unknown gender.
3. BMI values, specifically, were flagged as possibly erroneous based on a 2-stage cleaning procedure, described below.

BMI values were screened through a cleaning process that included 2 stages. Height and BMI values were standardized, by creating z-scores for each age group in half-year increments. The resulting height and BMI z-scores were used to aid in the cleaning process, as follows.

In the first stage, height z-scores outside the +/−3 range were flagged for further review. Flagged height values were reviewed using data from previous cycles, wherever possible. If a flagged height was found to be consistent with height reported in previous cycles, the value was kept. If, however, there were inconsistencies across cycles that suggest the height reported in cycle 4 was likely an error (e.g., parent reported a height that was smaller than the height values reported in previous cycles, then the value was removed. If height was not reported in previous cycles for a flagged case, then BMI z-scores were checked; if BMI z-scores associated with the reported height value were beyond +/−2, then the height values were considered potential errors, and the cases were removed.

In the second stage, BMI values associated with a BMI z-score beyond +/−5 were removed, as suggested in the guidelines provided by the World Health Organization (World Health Organization, 2009). Although the WHO percentiles were not used in this study, these guidelines were provided to help reduce error in analysing nutritional survey data. However, given that parent-reported BMI information is more prone to error than measured values (Statistics Canada, 2005), these guidelines were considered to be lenient, and further cleaning was deemed necessary. Thus, the next step in this stage was
to examine BMI values associated with BMI z-scores beyond +/-4, then +/-3. BMI values associated with BMI z-scores beyond +/-4 were removed if other inconsistencies were found, including discrepancies in height, weight or BMI reported in cycle 4 relative values reported in previous cycles (for instance, height in cycle 4 less than height in cycle 3; large increase in weight, but little to no change in reported height). BMI values associated with a BMI z-score of +/-3 were removed if either (a) height at cycle 4 was less than height reported at a previous cycle, or (b) no change in height was reported and BMI reported in cycle 4 had change more than 10 points from last reported cycle. This cleaning process led to the removal of less than 1% of the sample.

2.3.2 Statistical Analysis. All analyses were conducted using weighted data (see above), using SPSS version 21 for Windows. The first step to analysing the data involved describing the data for the sample as a whole, and then by ADHD and weight status. All potentially confounding variables were summarized for the overall sample, by ADHD group, and by weight category. Group means were compared using one-way Analysis of Variance (ANOVA). In cases where the data did not meet the assumptions required for ANOVA, non-parametric tests were used, including the Mann-Whitney U-Test, and the Kruskal-Wallis Test. Post-hoc analyses were conducted using Fisher’s least significant difference to identify group means that were significantly different at the .05 level. For categorical variables, proportions were compared using Pearson Chi-Square test, and comparing column proportions to identify subsets of categories that differed significantly at the .05 level. The same approach was used to compare proportions of children in each weight category based on their ADHD status, and vice versa.
The formal analyses were conducted using different approaches depending on whether the dependent variable was continuous or categorical in nature. Analysis was approached in two stages, based on which of the primary variables (ADHD or abnormal bodyweight) was the dependent variable.

When abnormal bodyweight was the outcome variable, the question being addressed was: does ADHD influence the odds of underweight and obesity? To answer this question, a series of multinomial logistic regression analyses was run, with weight category as the dependent variable, and ADHD scale, ADHD symptom severity rating, ADHD proxy diagnosis, hyperactivity/impulsivity symptoms severity, then inattention symptom severity as predictors (each in a separate regression). These regressions were then repeated, controlling for potentially confounding variables.

When ADHD was the dependent variable, the research question was: is being underweight or obese associated with increased odds of ADHD? To answer this question, different analyses were run for each ADHD variable. For the first analysis, the ADHD scale was the dependent variable. Since ADHD scale scores were positively skewed, generalized linear modelling (GZLM) was used, with a gamma distribution and log link. GZLM extends linear modelling to model data that are not normally distributed, and may not meet the assumption of equality of variances required by other modelling approaches (O. Falenchuk, personal communication, May 16, 2013). The Gamma distribution is used in place of the normal distribution when data are positively skewed and consist of only positive values (SPSS Inc., 2010). The log link is likewise preferred when the assumption of normality is not met. Another advantage of using a gamma distribution in GZLM is
that it allows results to be expressed in terms of percent change in outcome based on the level of the predictor variable (O. Falenchuk, personal communication, April 23, 2014).

For the second set of analyses, the ADHD symptom severity rating was the dependent variable, thus a multinomial logistic regression approach was used, with the “no symptoms” group serving as the reference category. For the third analysis, ADHD proxy diagnosis was the dependent variable, so binary logistic regression was used with “no ADHD” as the reference group. Finally, for the fourth and fifth analyses, hyperactivity/impulsivity and inattention symptom severity were the dependent variables, and once again multinomial regression analyses were run, with “no symptoms” (hyperactivity/impulsivity or inattention, depending on which dependent variable was being used) serving as the reference groups.

All analyses were repeated with potentially confounding variables, including age, sex, SES, EDA scale and CDPA scale, entered into the models. Since statistical control for ADHD medications was not possible for this objective given that ADHD medication use is almost exclusively associated with ADHD status, these analyses were also repeated after removing children who were on ADHD medication. Given that “other medication” could include medications typically associated with ADHD (e.g., non-stimulants used to treat ADHD, medications used to treat side-effects associated with ADHD medications, etc.), children who were taking “other” medications were also removed. In order to facilitate the comparison of results across objectives 1 and 2, the first set of analyses were also repeated after excluding children on ADHD or “other” medications. Additionally, controlling for medication use in objective 1 using two different methods provided a
valuable opportunity for converging evidence of any associations found, as well as to observe the relative influence of each approach on outcomes found.

The results of all logistic regressions are expressed in terms of odds ratios (OR), or adjusted odds ratios (AOR) in the case of adjusted models (e.g. odds of underweight or obesity, relative to normal weight). Odds ratios indicate the change in odds of an outcome relative to the odds of the “reference group” outcome. For instance, in terms of the first objective, the odds ratios would indicate the odds of being underweight or obese rather than normal weight, given the identified ADHD outcome (for example, increase in ADHD score, having ADHD versus not having ADHD, and so forth). Adjusted odds ratios provide the same information, taking the influence of covariates into account by holding all but the variable of interest (e.g., ADHD) constant. In other words, the adjusted odds ratios tell us the change in odds of a specific outcome, compared to the reference outcome (e.g., the odds of being underweight versus normal weight) at a particular level of the predictor variable of interest (e.g., moderate ADHD symptoms, versus no ADHD symptoms), when variables such as age, set, and SES are held constant. An OR equal to one suggests no change in the odds, where a value less than one indicates a decrease in odds, and a value greater than one indicates an increase in odds. Thus, both OR and AOR provide an indication of the magnitude of the effect, if any, of the independent variable(s) on the outcome variable of interest; AOR does so while holding covariates constant.

Results from the GZLM analyses are expressed in terms of exponentiated beta (ExpB). When GZLM is based on a gamma distribution, ExpB indicates percent change in the dependent variable given the level of the independent variable. Since GZLM were
run when the dependent variable was the ADHD scale, ExpB indicates the percent
change in average ADHD score for children in the underweight or obese groups
compared to those in the normal weight group. Again, a value greater than one would
suggest that abnormal bodyweight (underweight or obese) is associated with an increase
in average the ADHD score, and a value less than one indicates that abnormal
bodyweight is associated with a decrease in ADHD score. Finally, results from the
analyses conducted on the sample after excluding children taking ADHD or “other”
medications are reported when they were substantively different than the results of the
original analyses.
Chapter 3: Results

3.1 Sample Characteristics

The final sample consisted of 10,200 children (see Table 1 for sample characteristics), after removing all cases with missing or invalid data on study variables. This final sample was compared to the original sample of all 4-11 year-olds (weighted samples; all descriptives and results that follow are based on weighted data), to determine whether the two samples were comparable with regards to key variables. Some small, but statistically significant differences were found. Independent samples t-tests revealed that the estimated SES of the final sample (M=6.31, SD=3.01) was slightly higher than that of the original sample (M=6.21, SD=3.05; p=.001), and that the average ADHD scale score (M=3.76, SD=2.98) of the final sample was slightly lower than that of the original sample (M=3.82, SD=3.02; p=.046). A Pearson Chi-square test also revealed that the proportion of males in the final sample was less than that of the original sample (50.5% versus 51.2%; p=.043).

In terms of differences between ADHD symptom severity groups, increasing symptom severity (indicated by membership in an increasingly severe symptom category) was associated with younger age, lower SES, higher EDA and CDPA scores, a higher proportion of males, and a higher proportion of children taking both ADHD and “other” medications. All group differences were significant at the p<.05 level except for: average age of moderate symptom group versus mild and severe symptom groups, and proportion of children taking “other” medications in the mild and moderate groups (see Table 1). Furthermore, children with a proxy diagnosis of ADHD had lower SES, were more likely
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Overall Sample</th>
<th>ADHD Proxy Diagnosis</th>
<th>P</th>
<th>ADHD Symptoms</th>
<th>P</th>
<th>Weight Classification</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
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<td>Mild</td>
</tr>
<tr>
<td>Age (years)</td>
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<td>8.19 (2.23)</td>
<td>8.29 (2.20)</td>
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<td>6.33 (3.01)</td>
<td>5.84 (3.00)</td>
<td>.000</td>
<td>6.7 (3.03)</td>
<td>6.3 (2.98)</td>
<td>6.1 (3.06)</td>
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<td>EDA Scale 9</td>
<td>2.24 (2.15)</td>
<td>2.14 (2.05)</td>
<td>4.10 (2.89)</td>
<td>.000</td>
<td>1.3 (1.66)</td>
<td>1.9 (1.90)</td>
<td>3.2 (2.29)</td>
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<td>CDPA Scale 10</td>
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<td>1.18 (1.62)</td>
<td>3.31 (2.94)</td>
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<td>0.4 (0.87)</td>
<td>1.0 (1.44)</td>
<td>2.1 (2.13)</td>
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<td></td>
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<tr>
<td>Male</td>
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<td>49.3%</td>
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<td>48.1%</td>
<td>59.3%</td>
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<tr>
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<td>50.7%</td>
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<td>0.2%</td>
<td>0.4%</td>
<td>2.4%</td>
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<td></td>
<td></td>
<td></td>
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<tr>
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<tr>
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<td>2.6%</td>
<td>5.2%</td>
<td>.000</td>
<td>1.7%</td>
<td>2.7%</td>
<td>3.2%</td>
</tr>
</tbody>
</table>

1 - Attention Deficit Hyperactivity Disorder; 2 - Based on CDC percentiles: underweight <5th percentile, obese ≥ 95th percentile; 3 - Based on one-way ANOVA; 4 - Based on Mann-Whitney U Test; 5 - Based on Kruskal-Wallis Test; 6 - Based on Pearson Chi-Square Test; 7 - Groups with different subscripts that are different letters within a row are significantly different at the p≤.05 level; 8 - Based on highest education of mother or PMK or spouse, if unavailable; 9 - Emotional disorder/anxiety scale; 10 - Conduct disorder/physical aggression scale
to be male, had higher EDA and CDPA scores, and were more likely to be taking both ADHD medications and “other” medications (see Table 1).

There were also significant group differences among the various weight groups (see Table 1). For instance, compared to both obese and normal weight children, underweight children had higher average EDA scores and were more likely to be taking ADHD medication, but less likely to be taking “other” medications. Underweight children were also younger than normal weight children, on average. Children in the obese group had lower SES scores and were significantly younger than the normal weight and underweight groups. Both underweight and obese children had higher average CDPA scores and were more likely to be male than children in the normal weight group.

3.2 Unadjusted Prevalence of Abnormal Weight by ADHD Classification

A significantly higher proportion of children in the ADHD group (based on proxy ADHD diagnosis) were underweight compared to non-ADHD children but the groups did not differ in proportion of obese children (see Table 2). In terms of symptom severity groups, children in the “no ADHD symptom” group had the lowest prevalence of obesity and children in the “severe ADHD symptom” group had the highest prevalence of underweight, relative to all other symptom groups. Children with moderate ADHD symptoms had a higher proportion of obese children relative to the mild ADHD symptom group. There were no other significant differences in proportion of underweight or obesity.
Table 2 – Proportion (%) of Children in Weight Categories in Overall Sample and by ADHD\(^1\) Classification

<table>
<thead>
<tr>
<th>Bodyweight Classification(^2)</th>
<th>Overall Sample</th>
<th>ADHD Proxy Diagnosis(^4)</th>
<th>ADHD Symptoms(^4)</th>
<th>(P)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No ADHD</td>
<td>ADHD</td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>10.7%</td>
<td>10.4%(^a)</td>
<td>15.9%(^b)</td>
<td>.000(^3)</td>
</tr>
<tr>
<td>Normal Weight</td>
<td>71.1%</td>
<td>71.4%(^a)</td>
<td>65.6%(^b)</td>
<td>.000(^3)</td>
</tr>
<tr>
<td>Obese</td>
<td>18.2%</td>
<td>18.2%(^a)</td>
<td>18.5%(^a)</td>
<td>11.8%(^a)</td>
</tr>
</tbody>
</table>

1 - Attention Deficit Hyperactivity Disorder; 2 - Based on CDC percentiles: underweight <5\(^{th}\) percentile, normal weight ≥5\(^{th}\) percentile to <95\(^{th}\) percentile; obese ≥ 95\(^{th}\) percentile; 3 – Overall significance of weight category by ADHD groups, based on Pearson Chi-Square Test; 4 - Groups with different subscripts that are different letters (within their row) are significantly different at the p<.05 level.
3.3 Mean ADHD Scores and Unadjusted Prevalence of ADHD by Weight Classification

Overall, both the underweight and obese groups had significantly higher average ADHD scale scores compared to the normal weight group (see Table 3). There was no statistically significant difference between the average ADHD scores of the underweight and obese groups. In terms of ADHD symptom severity, the underweight group had a significantly higher proportion of children with severe ADHD symptoms compared to the normal weight group, but a lower proportion of children with mild ADHD symptoms compared to both the normal weight and obese groups. The obese group, on the other hand, had a significantly higher proportion of children with moderate ADHD symptoms compared to the normal weight group, and a significantly lower proportion of children with no ADHD symptoms compared to both the normal weight and underweight groups.

Finally, the underweight group had a significantly higher proportion of children with a proxy ADHD diagnosis compared to both the normal weight and obese groups (see Table 3). There was no statistically significant difference in the proportion of children with a proxy ADHD diagnosis between the normal weight and obese groups.

3.4 Influence of ADHD on Odds of Abnormal Bodyweight (Objective 1)

All results for objective 1 (see Tables 4 and 5) are organized by ADHD variables. Results from the unadjusted models are provided first, followed by those from the adjusted models.
Table 3 – Descriptives for ADHD\(^1\) Variables for Overall Sample and by Weight Category

<table>
<thead>
<tr>
<th>ADHD Variable</th>
<th>Overall Sample</th>
<th>Weight Classification(^2, 4)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHG Scale</td>
<td>3.76 (2.98)</td>
<td>Underweight: 4.10 (3.22)(^a)</td>
<td>4.15 (2.90)(^a)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal: 3.61 (2.95)(^b)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Obese: 4.15 (2.90)(^a)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ADHD Symptom Severity</th>
<th>Proportion (%)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms</td>
<td>13.1%</td>
<td>13.2(^a)</td>
<td>14.2(^a)</td>
</tr>
<tr>
<td>Mild symptoms</td>
<td>60.5%</td>
<td>57.2(^a)</td>
<td>60.8(^b)</td>
</tr>
<tr>
<td>Moderate symptoms</td>
<td>22.1%</td>
<td>23.4(^{ab})</td>
<td>20.9(^b)</td>
</tr>
<tr>
<td>Severe symptoms</td>
<td>4.4%</td>
<td>6.2(^a)</td>
<td>4.1(^b)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ADHD Proxy Diagnosis</th>
<th>Proportion (%)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>94.7%</td>
<td>92.2(^a)</td>
<td>95.1(^b)</td>
</tr>
<tr>
<td>Yes</td>
<td>5.3%</td>
<td>7.8(^a)</td>
<td>4.9(^b)</td>
</tr>
</tbody>
</table>

\(^1\) - Attention Deficit Hyperactivity Disorder; \(^2\) - Based on CDC percentiles: underweight <5\(^{th}\) percentile, normal weight ≥5\(^{th}\) percentile to <95\(^{th}\) percentile; obese ≥ 95\(^{th}\) percentile; \(^3\) – Overall significance across groups, based on Pearson Chi-Square Test; \(^4\) - Groups with different subscripts that are different letters (within their row) are significantly different at the \(p<.05\) level.
Table 4 – Association of ADHD\textsuperscript{1} with Underweight and Obesity\textsuperscript{2, 3}

<table>
<thead>
<tr>
<th>ADHD Variable</th>
<th>Unadjusted Model</th>
<th></th>
<th>Adjusted Model\textsuperscript{4}</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Underweight</td>
<td>P</td>
<td>Obese</td>
<td>P</td>
</tr>
<tr>
<td></td>
<td>Underweight</td>
<td>P</td>
<td>Obese</td>
<td>P</td>
</tr>
<tr>
<td>ADHD Scale\textsuperscript{5}</td>
<td>1.06 (1.03-1.08)</td>
<td>\textit{.000} &amp; 1.06 (1.04-1.08)</td>
<td>\textit{.000}</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.02 (.99-1.04)</td>
<td>\textit{.247} &amp; 1.04 (1.02-1.06)</td>
<td>\textit{.000}</td>
<td></td>
</tr>
<tr>
<td>ADHD Symptom Severity\textsuperscript{6}</td>
<td>1.02 (.84-1.23)</td>
<td>\textit{.866} &amp; 1.69 (1.41-2.02)</td>
<td>\textit{.000}</td>
<td></td>
</tr>
<tr>
<td>Mild Symptoms</td>
<td>1.69 (1.41-2.02)</td>
<td>\textit{.000} &amp; .84 (.69-1.02)</td>
<td>\textit{.075}</td>
<td></td>
</tr>
<tr>
<td>Moderate Symptoms</td>
<td>2.08 (1.71-2.53)</td>
<td>\textit{.000} &amp; .86 (.68-1.09)</td>
<td>\textit{.222}</td>
<td></td>
</tr>
<tr>
<td>Severe Symptoms</td>
<td>1.92 (1.43-2.57)</td>
<td>\textit{.000} &amp; .96 (.67-1.38)</td>
<td>\textit{.824}</td>
<td></td>
</tr>
<tr>
<td>ADHD Proxy Diagnosis\textsuperscript{7}</td>
<td>1.65 (1.21-2.26)</td>
<td>\textit{.002} &amp; 1.34 (1.01-1.78)</td>
<td>\textit{.046}</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1.11 (.88-1.39)</td>
<td>\textit{.377} &amp; 1.37 (.99-1.90)</td>
<td>\textit{.058}</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.65 (1.30-2.11)</td>
<td>\textit{.000} &amp; 1.02 (.78-.132)</td>
<td>\textit{.901}</td>
<td></td>
</tr>
</tbody>
</table>

1 – Attention Deficit Hyperactivity Disorder; 2 - Based on multinomial logistic regression; reference group is normal weight; 3 – Bodyweight classification based on CDC percentiles: underweight <5\textsuperscript{th} percentile, normal weight ≥5\textsuperscript{th} to <95\textsuperscript{th} percentile, obese ≥ 95\textsuperscript{th} percentile; 4 – Adjusted for age, gender, SES, ADHD medication, other medication, emotional disorder/anxiety, and conduct disorder/physical aggression; 5 – NLSCY parent-rated ADHD symptom scale; 6- Categories based on scores on ADHD scale; reference group is “no ADHD symptoms;” 7 - Based on study-defined proxy ADHD diagnosis variable; reference group is “no ADHD.
Table 5 – Odds\(^1\) of Underweight and Obesity\(^2\) Associated with Low-Moderate and Severe Symptoms of Hyperactivity/Impulsivity (HI)\(^3\) and Inattention (IA)\(^4\)

<table>
<thead>
<tr>
<th>ADHD Variable</th>
<th>Odds Ratios (95% CI)</th>
<th>Adjusted Odds Ratios(^5) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Underweight</td>
<td>P</td>
</tr>
<tr>
<td>Level of HI Symptoms(^3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low-Moderate</td>
<td>1.17 (.98-1.39)</td>
<td>.075</td>
</tr>
<tr>
<td>Severe</td>
<td>2.05 (1.55-2.69)</td>
<td>.000</td>
</tr>
<tr>
<td>Level of IA Symptoms(^4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low-Moderate</td>
<td>1.20 (1.05-1.38)</td>
<td>.007</td>
</tr>
<tr>
<td>Severe</td>
<td>1.51 (1.13-2.02)</td>
<td>.005</td>
</tr>
</tbody>
</table>

1 – Based on multinomial logistic regression analysis; reference group is “normal weight;” 2 - Bodyweight classification based on CDC percentiles: underweight <5\(^{th}\) percentile, normal weight ≥5\(^{th}\) percentile to <95\(^{th}\) percentile, obese ≥ 95\(^{th}\) percentile; 3 – Categories based on scores on HI questions from NLSCY parent-rated ADHD scale; reference group is “no HI symptoms;” 4 – Categories based on scores on IA questions from NLSCY parent-rated ADHD scale; reference group is “no IA symptoms;” 5 – Adjusted for age, gender, SES, ADHD medication, other medication, emotional disorder/anxiety, and conduct disorder/physical aggression.
3.4.1 Unadjusted Models

3.4.1.1 ADHD scale as predictor variable. For each one-point increase in score on the ADHD scale (see Table 4), the odds of both underweight and obesity increased by 6% (exponentiated B [ExpB] = 1.06, 95% confidence interval [CI95] =1.03-1.08, p=.000, and ExpB = 1.06, CI95 = 1.04-1.08, p=.000, respectively).

3.4.1.2 Categorical ADHD symptom severity as predictor variable. Mild ADHD symptoms (see Table 4) were associated with a 69% increase in odds of obesity (OR = 1.69, CI95 = 1.41-2.02, p = .000), but no significant change in odds of underweight (OR = 1.02, CI95 = .84-1.23, p = .866). Moderate ADHD symptoms were associated with more than double the odds of obesity (OR = 2.08, CI95 = 1.71-2.53, p = .000), but did not significantly influence the odds of underweight (OR = 1.21, CI95 = .97-1.50, p = .090). Severe ADHD symptoms nearly doubled the odds of obesity (OR = 1.92, CI95 = 1.43-2.57, p = .000), and also increased odds of underweight by 65% (OR = 1.65, CI95 = 1.21-2.26, p = .002).

3.4.1.3 Proxy ADHD diagnosis as predictor variable. Having a proxy diagnosis of ADHD (see Table 4) increased odds of being underweight by 65% (OR = 1.65, CI95 = 1.30-2.11, p = .000), but did not significantly influence the odds of obesity (OR = 1.11, CI95 = .88-1.39, p = .377).

3.4.1.4 Hyperactive/impulsive and inattentive ADHD symptom severity as predictor variables. Low-moderate levels of hyperactivity/impulsivity symptoms (see table 5) were associated with a 77% increase in odds of obesity (OR = 1.77, CI95 = 1.52-2.07, p = .000), while severe hyperactivity/impulsivity symptoms were associated with
more than double the odds of both underweight (OR = 2.05, CI₉₅ = 1.55-2.69, p = .000) and obesity (OR = 2.10, CI₉₅ = 1.63-2.70, p = .000).

In terms of inattentive symptoms of ADHD (see table 5), low-moderate symptoms were associated with a 20% increase in odds of underweight (OR = 1.20, CI₉₅ = 1.05-1.38, p = .007), and a 43% increase in odds of obesity (OR = 1.43, CI₉₅ = 1.29-1.60, p = .000). Severe inattention symptoms were associated only with an increased risk of underweight (OR = 1.51, CI₉₅ = 1.13-2.02, p = .005).

3.4.2 Adjusted Models

3.4.2.1 ADHD scale as the predictor variable. In the adjusted model (see Table 4), higher ADHD scale scores continued to be associated with increased odds of obesity (AOR = 1.04, CI₉₅ = 1.02-1.06, p = .000), although the magnitude of the association decreased somewhat.

3.4.2.2 Categorical ADHD symptom severity as the predictor variable. Mild (AOR = 1.39, CI₉₅ = 1.16-1.67, p = .001), moderate (AOR = 1.63, CI₉₅ = 1.31-2.01, p = .000), and severe ADHD symptoms (AOR = 1.37, CI₉₅ = .99-1.90, p = .058) continued to be associated with increased odds of obesity (see table 4). Although the influence of severe symptoms was no longer statistically significant at the p<.05 level in this analysis, when the analysis was repeated after removing children on ADHD or “other” medications, the association became stronger and statistically significant (AOR = 1.43, CI₉₅ = 1.02-2.01, p = .040).
3.4.2.3 Proxy ADHD diagnosis as the predictor variable. ADHD proxy diagnosis (see table 4) also remained significantly associated with increased odds of underweight (AOR = 1.34, CI<sub>95</sub> = 1.01-1.78, p = .046), and not obesity (AOR = 1.02, CI<sub>95</sub> = .78-1.32, p = .901).

3.4.2.4 Hyperactive/impulsive and inattentive ADHD symptom severity as the predictor variables. Hyperactivity/impulsivity symptoms were associated only with increased risk of obesity, but not underweight (see table 5). Specifically, low-moderate symptoms of hyperactivity/impulsivity were associated with a 40% increase in odds of obesity (AOR = 1.40, CI<sub>95</sub> = 1.19-1.64, p = .000), and severe hyperactivity/impulsivity symptoms were associated with a 39% increase in risk of obesity (AOR = 1.39, CI<sub>95</sub> = 1.05-1.85, p = .021). In terms of inattentive symptoms (see table 5), only one association remained statistically significant in the adjusted model, which was that of low-moderate inattention symptoms with obesity (AOR = 1.30, CI<sub>95</sub> = 1.15-1.46, p = .000).

3.5 Influence of Abnormal Bodyweight on Odds of ADHD (Objective 2)

3.5.1 Unadjusted Models

3.5.1.1 Bodyweight as the predictor variable – effects of underweight. Being underweight (see table 6) was associated with an 11% increase in ADHD score (OR = 1.11, CI<sub>95</sub> = 1.06-1.15, p = .000), a 65% increase in odds of having severe ADHD symptoms (OR = 1.65, CI<sub>95</sub> = 1.21-2.26, p = .002), and a 65% increase in odds of having a proxy ADHD diagnosis (OR = 1.65, CI<sub>95</sub> = 1.30-2.11, p = .000). However, when children on ADHD or “other” medication were excluded, being underweight became associated with a 26% increase in odds of having moderate ADHD symptoms (OR = 1.26, CI<sub>95</sub> = 1.01-1.58, p = .044), and no longer had a significant impact on odds of
<table>
<thead>
<tr>
<th>Weight Classification</th>
<th>ADHD Scale&lt;sup&gt;3,4&lt;/sup&gt;</th>
<th></th>
<th>Severity of ADHD Symptoms&lt;sup&gt;5,6&lt;/sup&gt;</th>
<th></th>
<th>Proxy Diagnosis&lt;sup&gt;7,8&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exp B (95% CI)</td>
<td>P</td>
<td>Odds Ratio (95% CI)</td>
<td>P</td>
<td>Odds Ratio (95% CI)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Moderate Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Severe Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Unadjusted Model</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>1.11 (1.06-1.15)</td>
<td>.000</td>
<td>1.02 (.84-1.23)</td>
<td>.866</td>
<td>1.21 (.97-1.50)</td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>1.12 (1.08-1.15)</td>
<td>.000</td>
<td>1.69 (1.41-2.02)</td>
<td>.000</td>
<td>2.08 (1.71-2.53)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adjusted Model</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>1.02 (.98-1.05)</td>
<td>.365</td>
<td>.79 (.64-.96)</td>
<td>.018</td>
<td>.82 (.65-1.04)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>1.06 (1.03-1.09)</td>
<td>.000</td>
<td>1.37 (1.13-1.65)</td>
<td>.001</td>
<td>1.60 (1.29-1.98)</td>
</tr>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 – Attention Deficit Hyperactivity Disorder; 2 – Based on CDC percentiles, such that underweight is <5<sup>th</sup> percentile, and obese is >95<sup>th</sup> percentile; reference group is “normal weight;” 3 – NLSCY parent-rated ADHD symptom scale; 4 – Based on generalized linear model with gamma distribution and log link; 5 – Categories based on scores on ADHD scale; 6 – Based on multinomial logistic regression; reference group is “no symptoms;” 7 – Based on study-defined proxy ADHD diagnosis; 8 – Based on binary logistic regression; reference group is “No ADHD;” 9 – Adjusted for age, gender, SES, emotional disorder/anxiety, and conduct disorder/physical aggression.
having a proxy ADHD diagnosis (OR = 1.29, CI$_{95}$ = .96-1.73, p = .089).

Being underweight was also associated with more than twice the odds of having severe hyperactivity/impulsivity symptoms (see table 7; OR = 2.05, CI$_{95}$ = 1.55-2.69, p = .000), and 20% higher odds of low-moderate inattention symptoms (OR = 1.20, CI$_{95}$ = 1.05-1.38, p = .007), and a 51% increase in odds of experiencing severe inattention symptoms (OR = 1.51, CI$_{95}$ = 1.13-2.02, p = .005). Furthermore, when children on ADHD and “other” medications were excluded, underweight became associated with increased odds of low-moderate hyperactivity/impulsivity symptoms (OR = 1.20, CI$_{95}$ = 1.00-1.44, p = .047), but was no longer associated with increased odds of low-moderate (OR = 1.04, CI$_{95}$ = .91-1.18, p = .578) or severe inattention symptoms (OR = 1.28, CI$_{95}$ = .92-1.78, p = .144).

3.5.1.2 Bodyweight as the predictor variable – effects of obesity. Being obese was also associated with higher risk of ADHD (see Table 6). For instance, children in the obese group had a 12% increase in ADHD score (OR = 1.12, CI$_{95}$ = 1.08-1.15, p = .000). They also had 69% higher odds of experiencing mild ADHD symptoms (OR = 1.69, CI$_{95}$ = 1.41-2.02, p = .000), more than double the odds of experiencing moderate ADHD symptoms (OR = 2.08, CI$_{95}$ = 1.71-2.53, p = .000), and nearly double the odds of experiencing severe ADHD symptoms (OR = 1.92, CI$_{95}$ = 1.43-2.57, p = .000), relative to no ADHD symptoms. Obesity did not increase children’s odds of having a proxy diagnosis of ADHD (OR = 1.11, CI$_{95}$ = .88-1.39, p = .377).

In terms of hyperactive/impulsive and inattentive ADHD symptoms (see table 7), being obese was associated with a 77% increased odds of low-moderate
Table 7 – Odds of Low-Moderate and Severe Symptoms of Hyperactivity/Impulsivity (HI) and Inattention (IA) Associated with Bodyweight Classification

<table>
<thead>
<tr>
<th>Weight Classification</th>
<th>Severity of HI Symptoms</th>
<th></th>
<th></th>
<th></th>
<th>Severity of IA Symptoms</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low-Moderate</td>
<td>P</td>
<td>Severe</td>
<td>P</td>
<td>Low-Moderate</td>
<td>P</td>
<td>Severe</td>
<td>P</td>
</tr>
<tr>
<td>Underweight</td>
<td>1.17 (.98-1.39)</td>
<td>.075</td>
<td>2.05 (1.55-2.69)</td>
<td>.000</td>
<td>1.20 (1.05-1.38)</td>
<td>.007</td>
<td>1.51 (1.13-2.02)</td>
<td>.005</td>
</tr>
<tr>
<td>Obese</td>
<td>1.77 (1.52-2.07)</td>
<td>.000</td>
<td>2.10 (1.63-2.70)</td>
<td>.000</td>
<td>1.43 (1.29-1.60)</td>
<td>.000</td>
<td>.87 (.65-1.17)</td>
<td>.348</td>
</tr>
<tr>
<td>Underweight</td>
<td>.86 (.71-1.03)</td>
<td>.098</td>
<td>1.28 (.94-1.73)</td>
<td>.117</td>
<td>1.04 (.90-1.20)</td>
<td>.589</td>
<td>1.27 (.93-1.73)</td>
<td>.132</td>
</tr>
<tr>
<td>Obese</td>
<td>1.37 (1.16-1.61)</td>
<td>.000</td>
<td>1.36 (1.02-1.78)</td>
<td>.033</td>
<td>1.29 (1.15-1.45)</td>
<td>.000</td>
<td>.81 (.59-1.10)</td>
<td>.182</td>
</tr>
</tbody>
</table>

1 – Based on multinomial logistic regression analysis; 2 – Categories based on scores on HI questions from NLSCY parent-rated ADHD scale; reference group is “no HI symptoms;” 3 – Categories based on scores on IA questions from NLSCY parent-rated ADHD scale; reference group is “no IA symptoms;” 4 – Bodyweight classification based on CDC percentiles: underweight <5th percentile, normal weight ≥5th percentile to <95th percentile, obese ≥ 95th percentile; 5 - Adjusted for age, gender, SES, emotional disorder/anxiety, and conduct disorder/physical aggression.
hyperactivity/impulsivity symptoms (OR = 1.77, CI<sub>95</sub> = 1.52-2.07, p = .000), more than double the odds of having severe hyperactivity/impulsivity symptoms (OR = 2.10, CI<sub>95</sub> = 1.63-2.70, p = .000), and 43% higher odds of low-moderate inattention symptoms (OR = 1.43, CI<sub>95</sub> = 1.29-1.60, p = .000).

### 3.5.2 Adjusted Models

#### 3.5.2.1 Bodyweight as the predictor variable – effects of underweight.

In the adjusted models the outcome was somewhat different, particularly for the underweight group (see table 6). For instance, while being underweight was still associated with higher odds of having a proxy ADHD diagnosis (AOR = 1.46, CI<sub>95</sub> = 1.12-1.90, p = .000), it was no longer associated with higher ADHD scores (ExpB = 1.02, CI<sub>95</sub> = .98-1.05, p = .365), or severe ADHD symptoms (AOR = 1.04, CI<sub>95</sub> = .73-1.46, p = .845), and it became associated with decreased odds of having mild ADHD symptoms (AOR = .79, CI<sub>95</sub> = .64-.96, p = .018). However, when children on ADHD or “other” medication were excluded from analysis, being underweight was no longer significantly associated with any ADHD outcome variables, including mild ADHD symptoms (AOR = .98, CI<sub>95</sub> = .79-1.21, p = .839) or ADHD Proxy diagnosis (AOR = 1.15, CI<sub>95</sub> = .84-1.57, p = .401).

In terms of specific ADHD symptom domains, underweight was not significantly associated with either hyperactive/impulsive or inattentive symptoms of ADHD symptoms in any of the adjusted models (see table 7).
**3.5.2.2 Bodyweight as the predictor variable – effects of obesity.** The influence of obesity on ADHD was more consistent in the adjusted models (see Table 6). For instance, obesity continued to be associated with higher ADHD scores (ExpB = 1.06, CI_{95} = 1.03-1.09, p = .000), and obese children had 37% higher odds of experiencing mild ADHD symptoms (AOR = 1.37, CI_{95} = 1.13-1.65, p = .001), and 60% higher odds of having moderate ADHD symptoms (AOR = 1.60, CI_{95} = 1.29-1.98, p = .000). In addition to not being associated with increased odds of having a proxy ADHD diagnosis (AOR = 1.03, CI_{95} = .80-1.32, p = .849), obesity was also no longer significantly associated with higher odds of severe ADHD symptoms (AOR = 1.34, CI_{95} = .97-1.85, p = .073).

Obesity was associated with increased risk of hyperactivity/impulsivity and inattention symptoms in the adjusted models (see Table 7). Specifically, obesity was associated with increased risk of experiencing low-moderate hyperactivity/impulsivity symptoms by 37% (AOR = 1.37, CI_{95} = 1.16-1.61, p = .000), severe hyperactivity/impulsivity symptoms by 36% (AOR = 1.36, CI_{95} = 1.02-1.78, p = .033), and low-moderate inattention symptoms by 29% (AOR = 1.29, CI_{95} = 1.15-1.45, p = .000).
Chapter 4: Discussion

Several important patterns in the relationship between ADHD and abnormal bodyweight were identified in this nationally representative sample of Canadian children, and are reviewed in terms of the study objectives, below. For objective 1, results are discussed separately for each predictor variable, and for objective 2, results of the analyses in which bodyweight was the predictor variable are discussed for the underweight category, then for the obese category. Tables 8 and 9 provide a brief, simplified summary of the significant outcomes found for each objective.

4.1 Objective 1: Is ADHD associated with abnormal bodyweight?

4.1.1 ADHD Scale. The results of the present study suggest that when ADHD is measured as a continuous variable, it is significantly associated with increased odds of obesity, but not with odds of underweight. In fact, higher mean ADHD scores were consistently associated with higher odds of obesity in both unadjusted and adjusted models, even after excluding children on “ADHD medication” and “other” medications. Although there do not appear to be any studies that have investigated the relationship between ADHD scores as a continuous variable and underweight, two previous studies have investigated the relationship between ADHD scores and odds of obesity in non-clinical samples of children (Lam & Yang, 2007; van Egmond-Fröhlich et al., 2012). Our results are consistent with those of Lam and Yang (2007), who found that higher ADHD scores were associated with higher odds of obesity, but not with those of van Egmond-Fröhlich et al. (2012), who found this to be the case only for females, once they had controlled for potential confounds.
Table 8 – Summary\(^1\) of Objective 1 Outcomes\(^2\)

<table>
<thead>
<tr>
<th>ADHD Variables</th>
<th>Underweight</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD Scale</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Categorical ADHD Symptom Severity</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Mild</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Moderate</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proxy ADHD Diagnosis</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Hyperactive/Impulsive Symptom Severity</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Low-Moderate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inattentive Symptom Severity</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Low-Moderate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1: + indicates significant positive results; 2: Based on adjusted models conducted on overall sample

Table 9 – Summary\(^1\) of Objective 2 Outcomes\(^2\)

<table>
<thead>
<tr>
<th>Weight Category</th>
<th>ADHD Scale</th>
<th>Categorical ADHD Symptom Severity</th>
<th>Proxy ADHD Diagnosis</th>
<th>Hyperactive/Impulsive Symptom Severity</th>
<th>Inattentive Symptom Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
<td>Low-Moderate</td>
</tr>
<tr>
<td>Underweight</td>
<td></td>
<td>–</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

1: + indicates significant positive results, - indicates significant negative results; 2: Based on adjusted models conducted on overall sample
4.1.2 Categorical ADHD Symptom Severity. When measured categorically, ADHD symptom severity (based on symptom scores) was also associated with increased odds of obesity, but not with odds of underweight. Odds of obesity were consistently higher in both the unadjusted and adjusted models, with and without children on ADHD and “other” medications, for all levels of ADHD symptoms, relative to no symptoms.

Although there do not appear to be any previous studies that have investigated the association between ADHD symptom severity as a non-dichotomous, categorical variable and odds of abnormal bodyweight, the finding that ADHD symptom severity was not significantly associated with underweight is generally consistent with the results from studies that examined the relationship using ADHD as a dichotomous variable. For instance, neither the population study by Erhart et al. (2012), nor the community study by Kim et al. (2014), found ADHD to be associated with underweight when ADHD diagnosis was based solely on cut-off scores on a parent-report ADHD rating scale.

Likewise, the finding that ADHD symptoms were associated with higher risk of obesity in the present study is consistent with the results reported by Erhart et al. (2012). Although two other studies (Jansen et al., 2013; Pitrou et al., 2010) did not find such an association, it is interesting to note that both of those studies used a rating scale that did not assess symptoms of impulsivity, and both used combined underweight/normal weight and overweight/obese categories. Also, neither study controlled for medication use. These methodological issues may have influenced their results, and makes it more difficult to compare their findings to those of the present study.

By examining symptom severity in more than two categories, the present study was able to identify differences in odds of obesity associated with different levels
symptom severity. Specifically, there was a clear and consistent pattern in which, compared to children with no ADHD symptoms, those with moderate symptoms had the highest odds of obesity, while those with mild and severe symptoms had higher odds relative to children with no symptoms, but lower than that of those with moderate symptoms. Interestingly, after adjusting for covariates, the odds of obesity for those with mild symptoms (AOR = 1.39) were very similar to the odds for those with severe symptoms (AOR = 1.37), and this was also true after excluding children on ADHD and “other” medications (AOR = 1.44 and 1.43, respectively).

Thus, it would appear that as symptom severity increased, odds of obesity increased, but once symptoms reached the “severe” level, odds began to decline. One possible explanation is that underlying relationship between ADHD symptoms and obesity is different at the most severe level of symptomatology. Another possible explanation is that while ADHD symptoms generally increase risk of obesity, at the most severe level of severity other factors come into play that moderate that risk.

A recent study by Khalife et al. (2014) provides some support for both explanations. The authors found that hyperactivity and inattention in childhood were each independently associated with higher odds of abdominal obesity and/or obesity based on BMI in adolescence, while combined hyperactivity and inattention was not (Khalife et al., 2014). Given that overall symptom scores on an ADHD scale were lower for children with only hyperactive or inattentive symptoms, and highest for those with combined hyperactivity and inattention, this finding provides some support for the first explanation: more severe symptoms may impact ADHD differently than less severe symptoms.
Khalife et al. (2014) also found that while “probable ADHD,” hyperactivity, inattention, and CD in childhood were each significantly associated with one or more measures of obesity in adolescence, once these variables were analysed as “pure” constructs (i.e. CD without ADHD, and probable ADHD, hyperactivity and inattention without CD), results were no longer statistically significant. In the present study, there was a greater change in odds ratios for the children in the severe group, once we controlled for covariates including CD, than for the other ADHD symptom severity groups.

Thus, while severe symptoms were initially associated with higher odds of obesity (ranging from OR = 1.92 for the whole sample, to OR = 2.13 for the sample after excluding children on ADHD or “other” medication) than mild symptoms (OR = 1.69-1.76), once we controlled for confounds, including CD, odds of obesity were comparable for the severe symptom group (AOR = 1.37-1.43) than the mild symptom group (AOR = 1.39-1.44). Moreover, CDPA scores were higher, on average, for the severe ADHD group compared to all other ADHD symptom severity groups. Taken as a whole, these results are consistent with those of Khalife et al. (2014), and provide support for the second explanation: as ADHD symptoms become more severe, odds of obesity increase, but once at the most severe level other factors, such as CD, moderate that effect.

### 4.1.3 Proxy ADHD Diagnosis

In the present study, ADHD proxy diagnosis was associated with higher odds of underweight, but not obesity, after controlling for important confounds. This study is the second that has found a significant association between ADHD diagnosis and increased odds of underweight (see also Güngör et al., 2013). In two other population studies that investigated this relationship, one found an
association only for children who were taking ADHD medication (Waring & Lapane, 2008), while the other found no association (Phillips et al., 2014). However, in both of these studies, children were classified as having ADHD based on parent response to a question regarding whether the child had been diagnosed with ADHD by a health professional. Thus, children with high levels of ADHD symptoms who had not been diagnosed in the community would not have been included in their ADHD groups, and perhaps more problematically, would have been included in their non-ADHD comparison groups. It is possible that this approach to classifying children into ADHD and non-ADHD groups may have prevented researchers from detecting significant differences among children with ADHD. The implications of the way in which ADHD diagnosis was established in the present study, and how variable definitions may help explain the overall pattern of results found, is discussed at length in section 4.3.1.

Although the present study did not find a significant association between ADHD proxy diagnosis and risk of obesity, this is consistent with some previous research in which ADHD diagnosis was based on clinically-assessed ADHD diagnosis, rather than either a previous diagnosis by a health professional or cut-offs based on severity of scores on ADHD rating scales, but not both. For instance, in the population study by Byrd et al. (2013), researchers found that children with ADHD who were not medicated did not differ in their odds of obesity relative to non-ADHD children, while those who were medicated actually had lower odds of obesity than non-ADHD children. Likewise, similar studies based on community and clinical ADHD samples did not find clinical ADHD diagnosis to be associated with significantly increased risk of obesity (Fliers et al., 2013; Mustillo et al., 2003; Pauli-Pott, Neidhard, Heinzel-Gutenbrunner, & Becker,
2013), or actually found ADHD to be associated with lower risk of obesity in one of more stratified ADHD groups (Dubnov-Raz et al., 2011; Fliers et al., 2013). Although two clinical studies did find ADHD diagnosis to be significantly associated with obesity, it is notable that one did not have a control group (Yang et al., 2013), and one had a control group with very small numbers of controls in abnormal weight categories (Güngör et al., 2013).

4.1.4 Hyperactivity/Impulsivity and Inattention Symptom Severity. There was a significant association between both low to moderate and high hyperactivity/impulsivity, as well as low to moderate (but not high) inattention symptoms, and increased odds of obesity. Hyperactivity/impulsivity and inattention symptoms were not significantly associated with increased odds of underweight, after controlling for potential confounds. There do not appear to be any previous studies that have investigated the role of specific ADHD symptom dimensions on odds of underweight. However, a recent longitudinal study by Khalife et al. (2014) did investigate the role of ADHD symptoms of inattention and hyperactivity at 8 years of age in predicting obesity at 16 years of age. In that study, inattentive symptoms of ADHD at 8 years of age predicted obesity at 16 years of age, and while hyperactivity at age 8 did not predict obesity at age 16 when obesity was based on BMI, it did predict abdominal obesity at 16 years of age. Other longitudinal studies have also found that impulsive and inattentive (Cortese, Faraone, Bernardi, Wang, & Blanco, 2013; Fuemmeler, Østbye, Yang, McClernon, & Kollins, 2011), but not necessarily hyperactive (Cortese et al., 2013) ADHD symptoms in childhood are associated with increased odds of obesity in adulthood, in national samples from the US.
4.2 Objective 2: Is abnormal bodyweight associated with odds of ADHD?

4.2.1 Underweight. Overall, the findings in the present study suggest that underweight is significantly associated with higher odds of proxy ADHD diagnosis, but not with higher scores on the ADHD scale, or with increased odds of ADHD symptom severity.

The finding that underweight was not associated with ADHD scores (i.e. ADHD as a continuous variable), is consistent with the findings of Drukker et al. (2009), and with the other analyses in the present study, including the lack of association found between these variables in objective 1. The results of the analyses with ADHD symptom severity as a categorical variable, however, were less consistent across analyses. While most of the associations in the adjusted models were non-significant, there was one exception: underweight was associated with lower odds of mild ADHD symptoms.

These results should be interpreted cautiously, given the absence of other significant associations among underweight and ADHD symptom categories or ADHD scores in general, as well as the fact that this association was lost when children on ADHD medication were removed from the adjusted analysis. While the latter effect could suggest that medication use mediated the relationship between underweight and mild ADHD symptoms, further research is required to determine if these findings can be replicated in similar samples. Overall, the current study did not support the prediction that underweight would be associated with ADHD scores or ADHD symptom severity as a categorical variable, after controlling for potential confounds.

On the other hand, the present study found that underweight was associated with ADHD proxy diagnosis in the overall sample, consistent with the outcomes for the first
objective, in which bodyweight was the outcome variable. However, unlike the findings for the first objective, this was not the case once children on ADHD and “other” medications were removed from the analysis. This again raises the possibility that medication use may mediate the relationship between underweight and increased risk of ADHD diagnosis. Unfortunately, there do not appear to be other studies that have investigated the association of underweight and clinical ADHD diagnosis with underweight as the predictor variable, and further studies will be needed to help confirm these findings in similar populations, as well as to clarify the potential influence of medication use on this relationship.

Finally, in terms of an association between underweight and hyperactivity/impulsivity and inattention dimensions of ADHD specifically, the current study suggests that underweight does not significantly increase children’s odds of having symptoms in either of these dimensions. These results are consistent with those of a recent clinical study (Güngör et al., 2013), in which researchers found that there was no significant association between underweight and symptoms of hyperactivity. Unfortunately, that study did not investigate the dimensions of impulsivity or inattention, and replications of these findings in future studies are necessary to help confirm the results of the present study.

4.2.2 Obesity. In terms of obesity, the pattern of results is essentially the same as that found in the first objective. Specifically, obesity was associated with higher odds of ADHD across all ADHD outcome variables, except proxy ADHD diagnosis, which was not significant. Overall, these findings are consistent with those of Braet et al (2007), who found that odds of ADHD diagnosis did not differ among the overweight and control
groups, but that overweight children did have significantly higher scores than normal weight children on measures of ADHD symptoms of impulsivity and inattention.

In terms of specific outcome variables, there are a limited number of existing studies available for comparison. For instance, in the present study children who were obese had higher ADHD scores, on average, than normal weight children. Only two other studies, both based on community samples, examined the relationship between obesity and ADHD as a continuous variable. The present results are consistent with one (Davis et al., 2009), which examined this relationship in adults, but not with the other (Drukker et al., 2009), which investigated it in children and adolescents.

In the present study, obesity was also significantly associated with higher odds of mild and moderate levels of ADHD symptoms, but not severe symptoms, in the adjusted models. Although there do not appear to be other studies that have investigated the relationship between obesity and ADHD symptom severity using a non-dichotomous categorical ADHD variable, our results are generally consistent with previous studies that defined ADHD using symptom rating score cut-offs, in both population (Erhart et al., 2012) and community (Rojo et al., 2006) samples of children. However, in one other population study (Jansen et al., 2013), researchers did not find an association between ADHD and obesity based on cut-off scores on a rating scale. As discussed earlier, this study used a combined underweight/normal weight group and a measure of ADHD that does not assess symptoms of impulsivity, which may have made it more difficult to detect a relationship between these variables, and to compare their results with those of the present study.
By examining ADHD symptom severity as a non-dichotomous categorical variable, the present study was able to identify changes in odds of different levels of ADHD symptoms associated with obesity. For instance, in the adjusted models, obese children had the highest odds of having moderate ADHD symptoms and increased odds of mild symptoms in all models, while the odds of severe ADHD symptoms were significantly increased only in the unadjusted models. Once again it appears that covariates had a particularly strong influence on the relationship between obesity and ADHD among children with the most severe symptom scores. Further research is required in order to help clarify the nature of this relationship.

The fact that no significant association was found between obesity and ADHD proxy diagnosis was not surprising, given the results discussed previously. Unfortunately, there is little previous research in which obesity was the predictor variable, and ADHD diagnosis, based on previous diagnosis or clinical diagnosis, was the outcome variable. Our results differ from one such study (Halfon et al., 2013); however in that study ADHD was identified based on parent response to a question regarding whether their child had been previously diagnosed by a health professional, and therefore their control group included children with undiagnosed ADHD. Furthermore, the present study did not control for learning disabilities or developmental delay, which were among the disorders considered in that study, which was not ADHD-specific in its focus.

Finally, in terms of obesity and hyperactivity/impulsivity and inattention symptoms of ADHD, the current study found that obese children had higher odds of experiencing low-moderate and severe hyperactivity/impulsivity, and low-moderate inattention symptoms. These results are consistent with those of Braet et al. (2007), who
found that overweight children had higher scores on measures of impulsivity, hyperactivity and inattention compared to normal weight children.

4.3 Implications of Results

4.3.1 Theoretical and Research Implications

4.3.1.1 Underweight versus obesity and definition/measurement of ADHD. This study highlights the impact of using different approaches to measuring and defining ADHD variables on outcomes in research on the association between ADHD and abnormal bodyweight in a national sample of children. Perhaps most interesting was the finding that ADHD proxy diagnosis was associated with higher odds of underweight, but not obesity, after controlling for important covariates, while the opposite pattern was found when ADHD symptom severity variables were used. In the latter analyses, there was no significant association between ADHD symptom scores or ADHD symptom severity groups and underweight, but there was a consistent association with obesity, after controlling for confounds.

This raises some intriguing questions. First, why was there a significant association between ADHD proxy diagnosis and underweight in the absence of a significant association between moderate or severe symptoms of ADHD and underweight? Secondly, why was there not a significant association between ADHD proxy diagnosis and obesity when there was a consistent association between all ADHD symptom measures and obesity?

There are two key differences among these analyses, each of which is particularly relevant to one of the questions above, and each of which is related to the composition of the groups used in analysis. Specifically, for the analysis in which ADHD symptom
severity was the outcome of interest, children were grouped based solely on their scores on the ADHD scale, and children with varying levels of ADHD symptomatology were compared to children with no symptoms of ADHD. However, when ADHD proxy diagnosis was the outcome of interest, children were grouped based on both their scores on the ADHD scale and on whether they had been diagnosed with ADHD by a health professional. As a result, the ADHD proxy group included all children from the severe symptom group and the subset (4%) of children from the moderate ADHD symptom group (n = 2, 200) who had been diagnosed by a health professional in the community. Therefore, for the analyses in which proxy ADHD diagnosis was the outcome variable, the reference group included children with mild and moderate symptoms of ADHD, but who had not been diagnosed with ADHD in the community.

Thus, the first key difference between analyses involving the scale-based variables versus proxy diagnosis was the composition of the ADHD groups: children with specific levels of ADHD symptoms, versus children with severe symptoms plus children with moderate symptoms who were diagnosed with ADHD in the community. The second key difference was the composition of the comparison groups: children with no symptoms versus children with no, mild or moderate symptoms who had not been diagnosed with ADHD in the community.

The first key difference is most helpful for answering the first question, in which underweight is the outcome of interest. In this case, neither moderate nor severe ADHD symptoms were independently associated with increased risk of underweight, after controlling for covariates. On the other hand, having a proxy ADHD diagnosis was significantly associated with underweight, despite the above-mentioned overlap between
these groups. Therefore, it would seem that the unique characteristics of the ADHD proxy diagnosis group is likely key to the significant association found between ADHD and underweight. Specifically, it would appear that it was only by including the most impaired children (i.e., those with the most severe symptom scores and those who were impaired enough to have been diagnosed by a health professional) that we were able to detect an association between ADHD and underweight.

Furthermore, this ADHD group was significantly different from the reference group, which included children with both low and moderate levels of ADHD symptoms, suggesting that just having symptoms of ADHD was not enough to significantly increase odds of being underweight in this sample.

Conversely, odds of obesity were significantly associated with moderate and severe levels of ADHD symptoms, as well as mild. Therefore, it would seem that severe symptom ratings were not key to the relationship between ADHD and obesity in this sample. This leads to the second key difference in these analyses: differences among reference groups. As mentioned above, in the analysis with ADHD symptom severity as the predictor variable (and also for the hyperactivity/impulsivity and inattention symptom analyses), the reference group was children with no symptoms of ADHD (or hyperactivity/impulsivity or inattention symptoms).

Thus, in the analyses where ADHD proxy diagnosis was the outcome of interest, the reference group – children without a proxy diagnosis of ADHD – included children with no symptoms and mild symptoms, as well as those with moderate symptoms who had not been diagnosed with ADHD in the community. Given that mild and moderate symptoms contributed heavily to the association between ADHD and obesity (indeed
moderate ADHD symptoms were associated with the highest risk of obesity), the fact that no significant differences were found between this reference group and the ADHD proxy diagnosis group is no longer surprising. Indeed, the analysis of ADHD symptom severity and obesity clearly demonstrated that any level of ADHD symptoms significantly increased odds of obesity, relative to having no symptoms of ADHD.

This pattern of results is relevant to the question of whether or not the association between ADHD and abnormal bodyweight is a result of a relationship between ADHD symptoms or ADHD as a clinical, diagnostic entity. It would appear that, based on the results of the current study, the answer to that question is different depending on whether one is interested in underweight or obesity. In the case of underweight, it would appear that the association is with ADHD as a “clinical diagnosis,” as approximated by combining information about current symptoms based on scores on an ADHD scale and previous diagnosis by a health professional. Conversely, in the case of obesity, it would appear that the relationship is with symptoms of ADHD, and that even mild symptoms of ADHD increase odds of obesity and vice versa (i.e. obesity is associated with higher odds of experiencing even mild ADHD symptoms).

These findings highlight the importance of examining both underweight and obesity in studies examining the relationship between ADHD and abnormal bodyweight, as well as the potential risks associated with combining underweight and normal weight children into one reference group in studying the influence of ADHD on obesity, specifically. It also highlights the importance of including a variety of measures of ADHD in studies examining the association between ADHD and abnormal bodyweight. As discussed previously, research on the nature of the construct of ADHD suggests that it
is better understood as a dimensional construct, and it has been recommended that researchers routinely include both categorical and dimensional measures of ADHD in studies, given the clinical and research dilemmas posed by using either approach exclusively (see review by Coghill & Sonuga-Barke, 2012).

4.3.1.2 Linearity of the relationship between ADHD and abnormal bodyweight.

In addition to the issues already discussed, it should be noted that by analysing ADHD symptomatology as a non-dichotomous variable, the present study demonstrated the utility of examining different levels of ADHD symptoms to help clarify the nature of the relationship between abnormal bodyweight, particularly obesity, and ADHD symptom severity. Had this study only included a continuous ADHD variable, it would have appeared that the association between ADHD and obesity was linear, wherein as ADHD symptoms increased, so too did odds of obesity. However, by examining symptom severity as a categorical variable, the current study was able to identify a non-linear relationship between obesity and ADHD symptoms. More specifically, it revealed more of a curvilinear relationship, in which odds of obesity increased as symptoms increased from no- to moderate-symptom levels, but then declined at the severe-symptom level. Further studies are needed to replicate these findings, and to help clarify the nature of the relationship, including the influence of potential mediators and moderators, between obesity and underweight at various levels of ADHD symptom severity.

4.3.1.3 Influence of covariates on the relationship between ADHD and abnormal bodyweight. Another interesting result of the present study was the impact of the approach used to control for medication use on the relationship between ADHD and abnormal bodyweight. Generally, odds ratios between ADHD and abnormal bodyweight
were somewhat higher when children on ADHD and “other” medications were removed from the sample entirely than when medication use was controlled for by entering these variables as covariates in logistic regressions. Although this can be a sign of collinearity amongst covariates, the consistency in outcomes across analyses, including between objective 1 and 2 analyses (where medication use could not be entered as a covariate), suggests this is not the case.

Furthermore, with few exceptions, removing children on medication from analyses did not alter the overall significance of the results compared to including medication use as a covariate, suggesting that either method may be appropriate depending on the goals of the study at hand. An exception to this was that, in analyses in which underweight was the predictor and ADHD was the outcome variable, removing children taking medication had an impact on the significance of results in a several analyses. However, as a whole, the outcomes from these analyses showed less consistency than other analyses, and further research is necessary in order to gain insight into the reliability of the associations found, and the potential role of medication use as a mediator in such a relationship, should there be one.

**4.3.1.4 Impact of abnormal bodyweight on ADHD outcomes.** An important advantage of repeating the analyses after excluding children taking ADHD or “other” medication was that it allowed for a comparison of the relative magnitude of the association between ADHD and abnormal bodyweight across objectives 1 and 2. This comparison revealed that, after adjusted for potential confounds, odds ratios were highest when abnormal bodyweight was the outcome variable, and in the case of severe ADHD symptoms, it was the only significant adjusted odds ratio.
This suggests that the influence of ADHD on odds of abnormal bodyweight was greater than the influence of abnormal bodyweight on ADHD. This is consistent with the theories posited to account for the association between ADHD and abnormal bodyweight, which would suggest that, in general, ADHD symptoms more readily explain abnormal bodyweight than vice versa. Furthermore, there is some longitudinal research to support the hypothesis that ADHD more likely contributes to abnormal bodyweight than the other way around (e.g., see Khalife et al., 2014). On the other hand, while the magnitude of the association between ADHD and abnormal bodyweight was different depending on which was examined as the outcome variable, the overall pattern of results was generally consistent across analyses, and clearly demonstrated that children with abnormal bodyweight have higher odds of ADHD or ADHD-related symptoms than normal weight children.

It is also important to note the important influence of other covariates included in this study. For instance, in several analyses the association between ADHD and underweight was significant in the unadjusted models for several ADHD variables, particularly severe ADHD symptoms, and severe symptoms of hyperactivity/impulsivity and inattention, specifically. However, when key variables such as SES, parent education, and other psychological problems were entered as covariates, none of these associations remained significant. Furthermore, results of analyses involving ADHD symptom variables and obesity demonstrated that the impact of ADHD on odds of obesity were consistently lower when these covariates were controlled for, especially for children in severe ADHD symptom groups. Therefore the results of this study clearly demonstrate
the importance of controlling for these important confounds when examining the relationship between ADHD and abnormal bodyweight.

4.3.2 Clinical Implications

The results of the present study suggest that children who received a proxy diagnosis of ADHD had 34% higher odds of being underweight than children without ADHD, and children who were underweight had 46% higher odds of having a proxy diagnosis of ADHD. This represents a clinically meaningful increase in risk for both groups and suggests that it would be pertinent to screen underweight children for ADHD and to monitor the growth of children with ADHD, in order to identify children who may be at risk of developing problems with insufficient weight gain.

As discussed, the proxy diagnosis group consisted of children with the highest ADHD scores and/or who had been diagnosed with ADHD by a health professional, and therefore likely constituted the most impaired group. If more impaired children are, indeed, at higher risk of being underweight, careful consideration must be given to ensuring that treatment for ADHD does not negatively impact weight gain. On the other hand, the fact that the odds of underweight were actually higher for children with a proxy ADHD diagnosis who were not on medication suggests that medication, despite its potential for suppressing appetite, may not have a detrimental impact on underweight. This is consistent with several studies in which ADHD medications were not found to increase underweight in ADHD children (Biederman et al., 2003; Ptacek et al., 2009; Spencer et al., 1996).

In this study, obese children had 12% higher ADHD scores than normal weight children, and 60% higher odds of experiencing moderate ADHD symptoms, which reflect
borderline to clinically significant ADHD scores. This is clinically meaningful, given the potential social, emotional and health-related risks of obesity. Furthermore, given the potential for ADHD to have a detrimental impact on weight loss efforts (for instance, see review by Cortese, Comencini, Vincenzi, Speranza, & Angriman, 2013), these findings highlight the importance of routinely screening obese children for symptoms of ADHD, and for addressing ADHD symptoms in treatment planning for obesity-related concerns. Given that obese children in this sample actually had the highest odds of moderate ADHD symptoms, it may be particularly important to carefully assess need for treatment of ADHD in borderline and sub-threshold cases of ADHD in obese children.

For instance, it is possible that by identifying specific ADHD symptoms that may be problematic for a particular child, treatment for obesity could be tailored to reduce the negative impact of those symptoms on the child’s ability to adopt a healthy eating and lifestyle behaviors that help the child achieve a healthier bodyweight. Indeed, according to a recent review of research on the impact of ADHD on weight loss in obese children, (Cortese & Castellanos, 2014) preliminary evidence suggests that ADHD negatively impacts treatment of obesity and treating ADHD can lead to significantly improvement in weight loss treatment outcomes.

Furthermore, in the present study mild to moderate symptoms of ADHD were associated with 39% to 60% higher odds of obesity respectively, and for children who were not on ADHD or “other” medication, severe ADHD symptoms were also associated with 43% higher odds of obesity. This suggests that children with even mild symptoms of ADHD should be screened for obesity, and that routine monitoring of weight gain might be helpful in the early identification of children who may be at risk of developing weight
problems. Furthermore, these results also suggest that all children with ADHD symptoms may benefit from being screened for issues associated with poor diet and exercise habits; those who are having difficulties in these areas could be provided with nutritional counselling to help prevent problems with obesity from developing.

4.4 Limitations of the Current Study

One limitation of the present study was the use of parent-reported height and weight, rather than measured values. However, our estimates of underweight (10.7%) and obesity (18.2%) are not inconsistent with previous studies. For instance, Waring and Lapane (2008) found that 6.7% of US children were underweight, and 21% were obese, based on measured height and weight in a national sample, and Byrd et al. (2013) found an obesity prevalence of 18% in their national sample of US children, based on measured height and weight. On the other hand, the proportion of obese children in our sample exceeded those of Shields and Tremblay (2010), who found that 13% of Canadian children were obese, based on measured height and weight.

However, unless inaccurate reporting of height and weight is systematically related to the variables of interest in this study, errors in reporting would not be expected to alter the underlying associations found between key variables. Fortunately, while parent-reported height and weight data may not be as reliable as measured height and weight (Shields, Connor Gorber, Janssen, & Tremblay, 2011; Weden et al., 2013), some studies that have investigated the accuracy of parent-reported versus measured height and weight have not found that inaccurate reporting is consistently associated with child or parent characteristics, with the exception of child’s age. For instance, two studies have found that parent reported height and weight are more accurate for older children (Shields
et al., 2011; Weden et al., 2013), and most biased for younger children, particularly for children 2-5 years of age (Weden et al., 2013). For children 9-11 (Shields et al., 2011) and 9-12 years of age (Weden et al., 2013), on the other hand, parent-reported data were quite consistent with measured values. Given these findings, it is particularly important to control for age when investigating the association between ADHD and abnormal bodyweight, and this was done for all adjusted models in the current study.

Weden et al. (2013) also did not find gender, ethnicity or SES to be significantly associated with differences in parent-reported and measured height in the overall sample, although there was a statistically significant difference in parent report of extremely low heights among the lowest income parents. However, it was speculated that this was related to low income families having less access to routine health care, resulting in less accurate height estimates among those families; this would not be expected to be an issue for Canadian children, given Canada’s universal health care system.

Overall, it is expected that using parent-reported height and weight resulted in a higher prevalence of abnormal bodyweight in this national sample of Canadian children than would have been found based on measured data. However, it is also expected that errors in parent-reported height and weight would not have significantly influenced the estimates of association between ADHD and abnormal bodyweight. Indeed, the results of the present study are consistent with those of most of the other studies based on non-clinical populations of children population and community samples of children, which relied on measured height and weight, and which likewise found a significant association between ADHD and abnormal bodyweight (Erhart et al., 2012; Khalife et al., 2014;
Another limitation of the current study was the reliance on a proxy ADHD diagnosis in place of a thorough, clinical diagnosis. However, as discussed at length previously, by using a combination of information from scores on a reliable ADHD scale and previous diagnosis by a health professional in the community, the proxy ADHD diagnosis maximized specificity and increased sensitivity beyond that which would have been possible using only one of the two sources on information available.

It is notable that the results of the analyses based on the proxy ADHD diagnosis variable were consistent with the findings of studies in which a formal, clinical ADHD diagnosis was made (Byrd et al., 2013; Fliers et al., 2013; Mustillo et al., 2003; Pauli-Pott et al., 2013). Furthermore, as discussed earlier, the prevalence of ADHD in this sample (5.3%), based on the proxy ADHD diagnosis variable, was highly similar to that estimated by Waddell et al. (2002; 4.8%), where ADHD diagnosis was based on clinical criteria. Therefore, it would appear that the proxy ADHD diagnosis variable used in the present study provided a reasonable approximation to a clinical ADHD diagnosis for the purposes of classifying children with ADHD in this sample.

The categorical ADHD, hyperactive/impulsive and inattention symptom severity variables used in this study were based on symptom scores rather than other potential approaches to measuring severity. For instance, it might have been useful to include a similar variable, wherein severity categories were based on number of symptoms rated “often or very true.” However, given that there were only 7 questions in total, with only 3 and 4 pertaining to hyperactivity/impulsivity and inattention dimensions of ADHD
symptoms, respectively, such an approach would have been more difficult to implement in a useful way. For instance, the range for the ADHD dimension variables would have been highly restricted (for example, for hyperactivity/impulsivity the range would have been 0-3), and may have resulted in highly uneven distributions, with the majority of the sample having a score of 0. This, in turn, would have created problems for statistical analysis. Therefore, while it would have been ideal to be able to take other factors into account when creating symptoms severity variables, given these limitations, using symptom scores provided the best measure of symptom severity that could be used consistently across symptom severity variables.

Another limitation of this study was the approach to addressing the complexities of adjusting cross-sectional weights for the final sample, and the limitations of SPSS in handling sample weights, particularly in complex sampling designs. However, it is reassuring that the overall pattern of results were consistent across different approaches to analysis, including different statistical analyses, unadjusted and adjusted models, with and without children taking ADHD and “other” medications, based on ADHD or abnormal bodyweight as the outcome variable. Furthermore, when analyses were repeated using unweighted samples (data not shown), the overall pattern of results was consistent with those based on the weighted sample. Therefore, it appears that weighting issues did not significantly impact the present findings.

Finally, this was a cross-sectional study; as such, no inferences can be made regarding causality. Longitudinal studies suggest that there is a relationship between ADHD and bodyweight, but that it may change over time (e.g., see Schwartz et al.,
Further prospective, longitudinal research is necessary to clarify the mechanism underlying the association of ADHD and abnormal bodyweight.

4.5 Strengths of the Current Study

The present study had several notable strengths. First, it was based on a large, nationally representative sample of Canadian children. Second, it included children ages 4 to 11 years, covering a wide range of childhood ages, whilst minimizing the impact of measurement issues associated with younger ages for the outcome variables of interest. For instance, symptoms of ADHD are more difficult to distinguish from normal developmental behavioral differences prior to the age of 4 years (American Psychiatric Association, 2013), and parent estimates of bodyweight and height are less reliable for younger children (Weden et al., 2013).

ADHD was also measured in multiple ways, both continuously and categorically, using dichotomous and multiple-category approaches. This provided an opportunity to investigate both linear and non-linear statistical relationships amongst key variables, and to identify the relative importance of ADHD symptoms versus ADHD as a diagnostic entity. Furthermore, results were expressed in a way that allows for intuitive interpretation of the clinical importance of the associations, particularly those expressed in terms of odds ratios.

The use of the proxy ADHD variable, in the absence of data based on clinical diagnosis of ADHD, was another significant strength. By combining the high specificity of using a cut-off score of 10 on the ADHD scale, with the increased sensitivity of including children with lower scores whose symptoms were impairing enough to have received a diagnosis by a health professional, we were able to provide a much closer
approximation to clinically-assessed ADHD classification than has been achieved in many previous population studies, which were restricted to using only ADHD cut-off scores or reports of previous diagnoses in the community. As discussed at length, this was critical to detecting the association among ADHD and underweight, in particular.

Another strength of the current study was that covariates were entered simultaneously into analyses as opposed to being entered individually in a series of analyses, or through sample stratification. This allowed for statistical control of each variable in the presence of all the other covariates, and reduces the risk of type 1 errors associated with repeated analyses.

Another important strength of the present study was in the examination of both underweight and obesity, providing a more comprehensive examination of the relationship between ADHD and abnormal bodyweight. Few studies have investigated the association between ADHD and underweight, specifically, and this study is only the third (see also Güngör et al., 2013 and Waring & Lapane, 2008) to find a significant association among them.

Moreover, by investigating abnormal bodyweight as a categorical variable, the risk of missing a non-linear association between ADHD and bodyweight was minimized, and the outcomes were generally expressed in terms of odds ratios, which are easily interpreted and clinically relevant. Furthermore, given that “increased” or “decreased” BMI can have either a negative or positive connotation depending on which end of the BMI continuum a value falls (i.e. above or below “healthy”), a categorical variable seemed more appropriate to this investigation.
Furthermore, this study controlled for the use of ADHD and “other” medications using two separate approaches. In the first, medication use was controlled through statistical approaches, while in the second it was controlled by removing children who were taking ADHD or “other” medication from the sample. The first approach maximized power and generalizability by including the entire sample in analyses. However, this approach could not be used for Objective 2, and therefore the adjusted models for objective 1 and 2 could not be meaningfully compared. The second approach made it possible to compare the relative magnitude of the association between ADHD and abnormal bodyweight across objectives 1 and 2, albeit at the expense of generalizability.

4.6 Future directions

The outcomes of the present study suggest that the relationship between ADHD and abnormal bodyweight is complex, and that outcomes vary depending on a number of methodological issues. There is a need for additional research in this area, in which ADHD diagnosis is based on diagnostic assessment using current clinical criteria, and height and weight are measured. Furthermore, including ADHD symptom severity variables, particularly multi-categorical variables, would be useful in helping to clarify the association of various, clinically-relevant levels of ADHD symptomatology and abnormal bodyweight. Given the paucity of research into the association of ADHD and underweight, further research in this area is necessary to replicate the findings of the current study. Furthermore, future studies seeking to explore the relationship between ADHD and obesity, specifically, should avoid including underweight children in their reference groups, to avoid potential confounding.
The results of the current study also stress the importance of controlling for mood, anxiety, and conduct problems, as well as SES, age, sex, ADHD medication and other psychotropic medications when investigating the association of ADHD and abnormal bodyweight. Finally, it would also be helpful for future research to help clarify the potential impact of different approaches to classifying bodyweight on the identification of an association between ADHD and abnormal bodyweight, by using more than one of the more common approaches (CDC, WHO, or IOTF) within one study.

4.7 Conclusions

The results of this study suggest that ADHD diagnosis is associated with increased odds of underweight, while ADHD symptoms are associated with increased odds of obesity. Likewise, children who were underweight were found to have increased odds of having a proxy ADHD diagnosis, and obese children were found to have increased odds of experiencing mild to moderate symptoms of ADHD. Obese children were also found to have increased odds of experiencing low-moderate and severe symptoms of hyperactivity/impulsivity and low-moderate symptoms of inattention, specifically. The mixed pattern of results across ADHD measures suggests that it is important to consider ADHD categorically and dimensionally in order to fully understand the nature of the relationship between ADHD and abnormal bodyweight. In this sample, a dimensional approach was most effective for identifying the association between ADHD and obesity, while a dichotomous categorical approach (i.e. proxy ADHD diagnosis) was helpful in identifying the relationship between ADHD and underweight. Finally, by including a non-dichotomous categorical ADHD symptom severity variable, this study was able to identify a non-linear relationship between ADHD and obesity, such that
children with moderate symptoms of ADHD had the greatest increase in odds of obesity relative to children with no symptoms. This suggests that the association between ADHD and obesity may not be linear. Further studies are needed to confirm these findings.


Coghill, D., & Sonuga-Barke, E. J. (2012). Annual research review: Categories versus dimensions in the classification and conceptualisation of child and adolescent


Hartmann, A. S., Rief, W., & Hilbert, A. (2012). Laboratory snack food intake, negative mood, and impulsivity in youth with ADHD symptoms and episodes of loss of control eating. where is the missing link? *Appetite*, 58, 672-678.


Roberts, R., & Duong, H. (2013). Obese youths are not more likely to become depressed, but depressed youths are more likely to become obese. *Psychological Medicine, 43*, 2143-2151.


Appendix A: Issues to Address in Explaining the Association between ADHD and Abnormal Bodyweight

When two or more disorders are found to frequently co-occur, there are a variety of potential explanations that must be considered in trying to explain the associations among them. The first step in explaining such co-occurrences is to rule out the possibility that the associations are due to chance, error or bias (for example, see Schachar & Tannock, 1995).

**Association Due to Chance Alone**

When two disorders are found to be statistically associated with one another, one must first consider the possibility that the disorders are actually not related to one another, and that an association has been found due to chance alone. If an association between two disorders is due to chance alone, it is likely that additional studies would fail to replicate the findings of association. In the case of the association between ADHD and abnormal bodyweight, a number of studies have found a significant (i.e. not due to chance alone) association among ADHD and abnormal bodyweight, suggesting that the associations found to date are not likely due to chance alone.

However, a better approach to ruling out chance findings has been suggested (Caron & Rutter, 1991), in which prevalence of comorbidity found is compared to the expected prevalence of comorbidity. In that approach, expected prevalence is calculated as the product of the base rates of the individual disorders in the sample. If the observed prevalence is greater than the expected prevalence, then one can conclude that the comorbidity is not due to chance alone in that particular sample. As mentioned above, several studies have found significant associations between ADHD and abnormal
bodyweight. Using one of those studies as an example (Phillips et al., 2014), the base rates of underweight and obesity in the total sample were 3.82% and 14.22%, respectively. The base rate of ADHD was 8.9%. Therefore, the expected prevalence of comorbid ADHD and underweight, and ADHD and obesity were .34% and 1.27%, respectively. However, in that sample the observed prevalence of ADHD and underweight and ADHD and obesity were .45% and 1.6%, respectively, representing a 32% and 26% higher prevalence of co-occurrence of ADHD and underweight and obesity, respectively, than would be expected by chance alone, providing further evidence that these associations found are not due to chance alone.

**Association Due to Error**

An association found between two disorders could also be due to error related to measurement technique or statistical approaches. In order to rule out this explanation, one would need to demonstrate that the association between two disorders is robust to changes in measurement and analytic approaches. Studies that have found an association between ADHD and abnormal bodyweight have varied considerably in terms of how ADHD and abnormal bodyweight variables have been measured and defined, and the analytic approaches used to examine the relationship between them, therefore this explanation can also be ruled out at this time.

**Association due to Referral Bias**

Finally, two unrelated disorders could be also associated due to referral bias. In order to rule out this explanation, one would have to demonstrate that the association is evident in diverse populations, including non-clinical populations. Associations between
ADHD and abnormal bodyweight have been found in diverse clinical and non-clinical samples; therefore this explanation can also be ruled out at this time.

Having established that a statistical association found among two or more disorders is not due to chance, error, or referral bias, the task becomes one of trying to determine the mechanisms underlying their association. A number of mechanisms have been proposed to explain the association between ADHD and abnormal bodyweight, and are discussed in Appendix B.
Appendix B: Mechanisms Proposed to Explain the Association between ADHD and Abnormal Bodyweight

Given that ADHD and abnormal bodyweight are both highly complex, heterogeneous conditions, it is extremely difficult to identify untangle the possible pathways that could lead to an association among them. In doing so, one must take into consideration the myriad of factors influencing each disorder, as well as potential interactions among them, and how all such relationships could culminate to an observed association between the two disorders.

What follows is a review of some of the proposed mechanisms that have been hypothesised to underlie the association among ADHD and abnormal bodyweight, and the current research that supports or refutes them. However, given the highly complex nature of the potential pathways that might help explain the association among ADHD and abnormal bodyweight, the focus of this review will be primarily on those hypotheses that have garnered a good deal of support, or that touch on variables that are investigated in the current cross-sectional study. Where helpful, a brief review and a limited number of examples will be provided for other mechanisms explored in the literature to date.

Association Due to Common Underlying Etiology/Association Due to Common Biological Mechanisms

ADHD and abnormal bodyweight could be associated due to shared etiological mechanisms. For instance, it is possible that specific genetic variations give rise to both ADHD and abnormal bodyweight. There is some evidence that genes associated with the development of obesity are also associated with ADHD (see review by Albayrak et al., 2013). Variations in dopamine-related genes, including dopamine receptor genes, such as
DRD4 and DRD2, have been found in several studies of ADHD and obesity, suggesting a potential common etiological pathway underlying problems with dopaminergic functioning thought to be involved with ADHD and obesity (for instance, see review by Bazar, Yun, Lee, Daniel, & Doux, 2006). However, given the multifactorial nature of both obesity and ADHD, including epigenetic (i.e. complex interplay between genes and the environment) influences (Archer et al., 2011; Gluckman & Hanson, 2008), there is likely more than one pathway from genetic variations to ADHD and obesity.

ADHD and abnormal bodyweight could also share common underlying biological mechanisms that give rise to both disorders, or increase the risk of developing both disorders. A recent review by Cortese and Vincenzi (2012) provides a good overview of some of the biological mechanisms common to both ADHD and obesity, which could explain the association among them for some children. For example, the authors review evidence that suggests that both disorders can be influenced by problems associated with dopamine-regulated neurological processes. There are two primary theories regarding precisely how dopaminergic processes could underlie the association between ADHD and obesity, including “reward deficiency syndrome” (see review by Cortese & Vincenzi, 2012) or “dopamine deficit theory” (see review by Liu et al., 2008), and reward sensitivity (Verbeken et al., 2012). In the former two theories, problems with dopaminergic functioning results in decreased sensitivity to natural reward leads to engaging in behaviors, such as overeating and risk-taking behaviors, which increase dopamine and so increase the reward response. In the latter, increased sensitivity to reward promotes behaviors that are experienced as rewarding, such as eating highly palatable foods or engaging in risk taking behavior.
Interestingly, a recent study (Verbeken et al., 2012) provides support for both the reward sensitivity theory and the reward deficiency theory. In this study, researchers found that the relationship between reward sensitivity and BMI is curvilinear, with a peak in normal weight and overweight children, and lower reward sensitivity among children with lower and higher BMI. In that study, children who were overweight had high reward sensitivity, and authors speculated that this caused them to engage in overeating behavior because of the positive sense of reward they experience when doing so. On the other hand, obese children had lower reward sensitivity, and authors speculated that this led them to fail to experience high levels of natural reward, therefore triggering an overcompensation response, in which they engaged in a higher level of overeating to boost their experience of reward, resulting in higher BMI among these children. Further research in which eating behavior is also assessed is necessary in order to provide support for the theory that differences in reward sensitivity in these groups influenced eating behavior, and in turn, led to the development of overweight and obesity.

Another potential etiological pathway to both ADHD and abnormal bodyweight is through underlying sleep disorders. Sleep disorders have been associated with both obesity and ADHD (see reviews by Altevogt & Colten, 2006; Owens, 2005). For instance, in a recent study (Yurumez & Kilic, 2013), researchers found sleep problems to be 84% higher among ADHD children compared to controls, and that ADHD children who experienced sleep problems had more night waking compared to children with sleep problems in the control group. Given that clinical and experimental studies have described behavioral manifestations of sleepiness to include symptoms of hyperactivity, impulsivity and inattention (see review by Owens, 2005), it is theoretically possible that
sleep deprivation could cause a clinical presentation of ADHD, or mimic ADHD symptoms.

Other studies have found that childhood obesity is associated with decreased sleep time, decreased REM sleep, lower sleep efficiency, and higher rates of sleep apnea (see review by Pulgarón, 2013). Interestingly, in a study of obese adolescents (Cortese et al., 2007), researchers found a significant association among ADHD symptoms and excessive daytime sleepiness scores, suggesting that obese adolescents who exhibit symptoms of daytime sleepiness may be at higher risk of ADHD.

Although the nature of the relationship between ADHD, obesity and sleep disorders is not entirely clear, it is interesting to note that dopamine is associated with regulation of the sleep-wake cycle, and that the DRD2 receptor has been associated with REM sleep, in particular (see review by Harvey et al., 2011). Moreover, it is believed that the circadian and reward circuits are intricately related, suggesting a potential pathway by which sleep could be associated with both ADHD and obesity. It is also interesting to note that sleep problems are associated with other comorbid health conditions that are, themselves, associated with ADHD and/or obesity, including behavioral problems, mood and anxiety disorders, diabetes, and cardiovascular disease (Harvey et al., 2011).

**Obesity or Obesity-Related Mechanisms Cause or Contribute to ADHD or Symptoms**

It is also theoretically possible that biological mechanisms specific to obesity could influence ADHD, or alternately, cause symptoms associated with ADHD. For instance, binge eating is associated with obesity in some individuals, and could contribute
to behavioral manifestations of ADHD, including problems with organization, feelings of restlessness, and inattentiveness (see Cortese & Vincenzi, 2012 for a review). It is also possible that binge eating could cause biological changes that manifest as ADHD. For instance, some researchers have found that overeating can lead to a down-regulation of dopamine receptors, which could, in turn, induce the dysfunctions associated with ADHD (Bazar et al., 2006). On the other hand, a recent longitudinal study (Khalife et al., 2014) found that while childhood ADHD predicted adolescent obesity, childhood obesity did not predict adolescent ADHD, suggesting that obesity did not contribute to the development of ADHD in this large, national sample. Furthermore, Cortese and Vincenzi (2012) also point to conflicting evidence on the association among obesity and binge eating, including one study that found no difference in levels of ADHD symptoms between obese adults with and without symptoms of binge eating. Overall, there is not currently enough support to conclude that obesity or related factors play a role in the development of ADHD.

**ADHD or ADHD-Related Mechanisms Cause or Contribute to Abnormal Bodyweight**

It is also possible that biological mechanisms associated with ADHD contribute to the development of abnormal bodyweight. For instance, as discussed previously, some research suggests that ADHD may be associated with delayed tempo of growth and altered fat metabolism that results in shorter stature throughout early childhood, and higher proportion of body fat among children and adolescents with ADHD (Ptacek et al., 2009). A recent longitudinal study (Schwartz et al., 2014) that investigated BMI growth trajectories in children with and without ADHD provides some support for the idea that
ADHD may alter developmental patterns of growth in children. Specifically, in that study researchers found that children with ADHD who were not medicated demonstrated more rapid BMI growth relative to non-ADHD children after the age of 10 years, while those who were medicated showed slower BMI growth initially, but later experienced more rapid BMI growth than non-ADHD children, resulting in higher BMI in later adolescence.

**ADHD Symptoms Cause or Contribute to Abnormal Bodyweight**

It is also possible that the cognitive and behavioral manifestations of ADHD contribute to the development of obesity. A limited number of longitudinal studies provide some support for the theory that ADHD symptom dimensions, including impulsivity and inattention, may contribute to the development of obesity. For instance, one study (Fuemmeler et al., 2011) found higher odds of obesity among adults who reported experiencing three or more symptoms of inattention or hyperactivity/impulsivity in adolescence. In another study (Khalife et al., 2014), researchers found that inattention at the age of 8 years predicted obesity at 16 years of age, based on both BMI and abdominal obesity measures, while symptoms of hyperactivity predicted only abdominal obesity. The fact that hyperactivity was found to predict abdominal obesity, but not obesity based on BMI is not inconsistent with the study by Ptacek et al. (2009) discussed earlier, which suggested that hyperactivity may be associated with differences in how fat is metabolized and stored in children with ADHD, even in the absence of differences in sex, age and height-adjusted weight. The fact that the outcomes were somewhat different for symptoms of inattention versus hyperactivity in that study also raises the possibility
that specific symptom domains associated with ADHD could influence the development of obesity in different ways.

Cortese and Vincenzi (2012) review the possible roles of impulsivity and inattention in the development of obesity. In terms of impulsivity, for instance, they suggest that two components of impulsivity – deficient inhibitory control (problems with planning and self-monitoring) and delay aversion (difficulty putting off an action), could each contribute to over-eating behaviors, such as eating in the absence of hunger, making poor food choices without consideration for future consequences, preferring fast food over food that requires preparation, and so forth. Although no longitudinal studies have addressed the specific pathways through which impulsivity might contribute to the development of obesity, there are a few cross-sectional studies that have examined some of these factors.

For instance, these hypotheses are consistent with the results of a recent meta-analysis (Thamotharan, Lange, Zale, Huffhines, & Fields, 2013), in which researchers found that obese children were most impaired on disinhibition and decision-making aspects of impulsivity, relative to other dimensions of impulsivity. Another recent study (Fields, Sabet, & Reynolds, 2013) found that obese adolescents responded more impulsively on a measure of delay discounting (a dimension of impulsivity associated with impulsive decision-making), but did not differ from normal controls on a measure of behavioral disinhibition. Another recent study (Van Den Berg et al., 2011) found that obese children scored higher than normal weight and underweight children on the dimension of impulsivity/fun-seeking.
On the other hand, a study by Hartmann, Rief and Hilbert (2012) found that although children with ADHD consumed more calories than children with loss of control eating and normal controls in a laboratory snack situation, there were no group differences in general impulsiveness or problems with planning (a specific dimension of impulsivity). Thus, in this experimental situation there was no evidence that these dimensions of impulsivity influenced snacking in behavior in ADHD children. However, these results may not be generalizable to real-life snacking behavior.

In terms of inattention, Cortese and Vincenzi (2012) suggest that children with ADHD may have difficulty attending to internal hunger and satiety cues, or be distracted from eating when they are occupied by an engaging task. Theoretically, these problems could lead to underweight for children who do not later compensate for missed caloric intake, and obesity for other children, if they over-compensate for missed meals later in the day. There do not appear to be any studies that have investigated these theoretical pathways between inattention and the development of obesity, however, and further research is necessary to identify the way in which symptoms of inattention might be contributing to the development of obesity.

The potential role of hyperactivity in the development of abnormal bodyweight is less clear. It seems intuitive to hypothesise that hyperactivity could, through excess energy expenditure, potentially contributing to low bodyweight. However, some studies have found that children with ADHD (including those with symptoms of hyperactivity) engage in less physical activity than non-ADHD children (for instance, see Kim et al., 2011). It is more difficult to identify how hyperactivity could, in and of itself, contribute to obesity. Perhaps hyperactive children would be less inclined to sit still long enough to
eat healthy meals and might therefore rely more on snack food when hungry, since
snacks (particularly pre-packaged) could be eaten while continuing to engage in other
activities.

No longitudinal studies have examined hyperactivity in relation to abnormal
bodyweight independently of impulsivity, and the results from at least one cross-sectional
study (Güngör et al., 2013) appear to controvert an association between hyperactivity and
either underweight or obesity. In that study, while both underweight and obesity were
more prevalent among children with ADHD than non-ADHD controls, rates of
hyperactivity did not differ between the groups. On the other hand, Braet et al. (2007)
found higher levels of hyperactivity among overweight children, based on structured
clinical interview (though not based on parent ratings).

**Summary**

At this time there is considerable support for the role of dopamine-related genes
and dopaminergic functioning in the development of both obesity and ADHD, suggesting
a potential role for common underlying biological mechanisms in the association among
these disorders. There is also a significant amount of support for the role of sleep
pathology in the development of ADHD and obesity. However, it is possible that sleep
disorders in ADHD and obese children could also be associated with dopaminergic
processes, suggesting that sleep disorders could, themselves, arise out of an underlying
problem with dopaminergic functioning, either along with both ADHD and obesity, or
directly contributing to the development of ADHD and obesity.

There is also significant support for the role of ADHD symptoms in the
development of obesity, possibly mediated by eating behavior. Both impulsivity and
inattention appear to be of particular import to the association among ADHD and abnormal bodyweight, while the role of hyperactivity is less certain. Once again, however, it is difficult to disentangle the potential roles of common underlying dysfunctions and ADHD symptomatology in the association among ADHD and abnormal bodyweight.