Overweight and Obesity in Children with Autism Spectrum Disorders: Findings Consistent with Typically Developing Children

DISSERTATION

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By

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Abstract

Childhood overweight and obesity are considerable problems both in the United States and worldwide. These abnormal weight categories are often accompanied by increased physical and mental health complications including diabetes mellitus, cardiovascular issues, and depression. Most concerning, elevated body mass in childhood generally leads to elevated body mass in adulthood, which is associated with higher rates of morbidity and mortality. Youth with intellectual or developmental disabilities, such as autism spectrum disorder (ASD), appear to be at heightened risk for overweight and obesity due to high medication use, atypical eating habits, and sedentary behavior. Previous literature is mixed as to whether these children actually have higher prevalence rates of overweight and obesity, although the methodology associated with some studies has been subpar due to use of parent-derived height and weight, and small sample sizes.

This dissertation was designed to investigate the prevalence of abnormal weight in children with ASD, and to identify what variables were associated with elevated body mass. The sample comprised children from the United States and Canada who visited a hospital or clinic that was part of the Autism Treatment Network. In this sample, 32.9% of the children were overweight and 17.3% were obese, which was not significantly different from the rates of elevated body mass in typically developing children or from some previous studies of children with ASD.
Multiple hierarchical regression models were run to analyze the data from a variety of perspectives, while trying to avoid confounds such as prescribed medication, different Child Behavior Checklist (CBCL) age versions, and clinical site. The most successful model was called “Atheoretical Empiricism,” and it found that Asian heritage, high levels of paternal education, stimulant use, atomoxetine use, high scores of the Anxious/Depressed CBCL subscale, and having a pervasive developmental disorder – not otherwise specified (PDD-NOS) diagnosis were associated with lower BMI percentile. Hispanic heritage, SSRI use, alpha 2 agonist use, high scores on the Sleep Disordered Breathing subscale of the Children’s Sleep Habits Questionnaire, and elevated scores of the CBCL Aggressive Behavior subscale and Withdrawn/Depressed subscale were associated with higher BMI percentile (greater likelihood of being overweight or obese). The variance accounted for declined when the more specific theory-driven investigations were conducted. The model had a better fit for older children whose parents completed the 6-18 year CBCL version rather than younger children whose parents completed the 1.5-5 year version. When evaluated by specific ASD diagnosis, the model fit best for children with PDD-NOS. There were great variations between model fit across sites; data from two Northeastern sites accounted for more variance (13.5% for Site 23 and 17.1% for Site 2) than any of the previous manipulations. Although far less variance was accounted for than initially hoped, variance levels in this study were consistent with amounts from other investigations. This study confirmed that several of the predictors for overweight and obesity in the neurotypical population held true for children with ASD. Future directions for research and weight-related interventions were discussed.
This dissertation is dedicated to Fred and Nancy Grondhuis, my parents.
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CHAPTER 1: INTRODUCTION

Overweight (body mass index [BMI] or weight in kilograms divided by height in meters squared > 85th percentile) and obesity (BMI > 95th percentile; discussed further in Chapter 2) are considered nutritional disorders (Ito, 2006) and contribute to chronic health conditions that have far-reaching medical and psychological implications, including greater risk for diabetes mellitus, metabolic syndrome, orthopedic complications, cardiovascular problems, depression, low self-esteem, and cancer (Lobstein, Baur, & Uauy, 2004; Deckelbaum & Williams, 2001). These conditions are not inevitable when one becomes obese, but instead they increase the likelihood of physical and mental health complications, which become more serious with age. This makes childhood overweight and obesity potentially serious issues that can have significant effects on quality of life, morbidity, and mortality (e.g., Daniels, 2006; Deckelbaum & Williams, 2001; Freedman, Khan, Dietz, Srinivasan, & Berenson, 2001). This dissertation explores three issues related to BMI percentile: (a) the percentage of children with autism spectrum disorders (ASD) that exceed common weight thresholds, (b) the validity of a conceptual model for predicting weight status, and (c) the ability of elements of the model to predict BMI percentile extremes in children with ASD.

Youth with intellectual and developmental disabilities (IDD) are also affected by the population’s increase in weight status. Throughout this dissertation, weight status will be measured by BMI percentile, which corrects for the fact that BMI tends to change
in systematic ways across age for youth (i.e., percentiles were computed by months of age, taking maturity into account). Some research suggests that children with IDD are impacted to a greater extent than what is experienced by those who are neurotypical (Allison, Packer-Munter, Pietrobelli, Alfonso, & Faith, 1998; Hamilton, Hankey, Miller, Boyle, & Melville, 2007), and this difference may become apparent by three years of age (Emerson, 2008). This difference between youth with IDD and those who are typically developing may be due to disability-specific risk factors (e.g., increased medication use, syndrome specific symptoms) as well as increased behavioral, cognitive, or social difficulties (Grondhuis & Aman, 2013; Chen, Kim, Houtrow, & Newacheck, 2009). Research on weight issues in children and adolescents with IDD tends to be separated by specific disability (i.e., Down syndrome, cerebral palsy, intellectual disability; Grondhuis & Aman, 2013). That is, research tends to be conducted within each type of condition rather than spread across multiple conditions or diagnoses. This syndrome-specific research is preferable, as different conditions may have different risk profiles that could contribute to overall weight.

A 2010 study by Rimmer and colleagues used an online survey to collect information about weight status and disability. They listed overweight and obesity prevalence, respectively, for five conditions; autism: 42.5% and 24.6%, Down syndrome: 55.0% and 31.2%, non-specific intellectual disability: 27.2% and 12.4%, cerebral palsy: 18.8% and 4.0%, and spina bifida: 64.5% and 18.6%. The autism, Down syndrome, and spina bifida groups were more likely to be overweight, and autism and Down syndrome groups were more likely to be obese than children without disabilities. These differences
were statistically different from typically developing peers, who had rates of overweight and obesity of 28.8% and 13.0%, respectively. The study also looked at the prevalence of secondary conditions in combination with disability. For children with intellectual disabilities, those with comorbid hyperlipidemia, diabetes, early maturation, or a preoccupation with weight (possibly an indication of disordered eating behavior) were more likely to be overweight than their peers without disabilities. Only high blood pressure was associated with overweight in children with physical disabilities, but not developmental disabilities. This interplay helps to demonstrate that elevated weight status is not based on a single factor, such as diagnosis. Instead, there are multiple influences affecting an individual’s ultimate body mass.

Autism spectrum disorder (ASD) is among the most commonly diagnosed neurodevelopmental conditions, with estimates from the Centers for Disease Control and Prevention (CDC; 2012) indicating that 1 in 88 children are on the autism spectrum. More current estimates, still using DSM-IV criteria, suggested that prevalence is still on the rise, with 1 in 68 children affected (CDC, 2014). However, not all researchers accept CDC prevalence, with some regarding the estimates to be too high (Mandell & Lecavalier, 2014). This dissertation occurred at a time of transition in the field, as the new DSM-V was released in May 2013 and contains new terminology and diagnostic criteria. That said, the data for this project were collected through 2012, employing the criteria from DSM-IV-TR, and almost all literature in this review employed previous diagnostic criteria and terminology unless otherwise noted. Use of older terminology is often necessary, as this reflects the diagnostic criteria in use when the data were
collected. Failure to note the potential differences between autistic disorder, pervasive developmental disorder – not otherwise specified (PDD-NOS), and Asperger disorder could overlook important subject differences, regardless of what one thinks of the diagnostic and terminological shift.

Despite the high rates of occurrence, the literature on obesity in children and adolescents with ASD is sparse. Curtin and colleagues (2010) reported that 30.4% of their sample with autism were obese compared to 23.6% of children without autism, although the finding was not statistically significant ($p = .075$). While there may be differences between those with and without ASD, there are a multitude of methodological limitations in the available ASD weight literature. These limitations include a failure to report overweight (the obesity precursor) and persistent use of parent-reported data for diagnosis, height, and weight, which historically have been unreliable. Other small-scale studies (Curtin, Bandini, Perrin, Tybor, & Must, 2005; Xiong et al., 2009; Rimmer, Yamaki, Lowry, Wang, & Vogel, 2010; Memari, Kordi, Ziaee, Mirfzeli, & Setoodeh, 2012) have also found that overweight and obesity are common problems for children with ASD, although with limited information about risk factors and correlates that may cause altered weight status. We do know from Memari and colleagues’ (2012) study that obesity in Iranian children with ASD was associated with age (older children tended to be heavier than younger children), sex (girls were more likely to be heavier than boys), and socioeconomic variables (positive association between family income and BMI percentile). Evans et al. (2012) found that, unexpectedly, high fruit and vegetable consumption in children with ASD was associated with increased BMI. While this is a
start, investigation into correlates and risk factors for obesity in youngsters with ASD is an area in great need of further investigation.

Obesity is highly researched because it is thought of as a public health crisis, although how that research has been conducted has changed over the decades. Friedman and Brownell (1995) characterized “generations” of studies. The first generation employed less than desirable methodology, often with only single variables measured, and it failed to find consistent psychological correlates. The second generation of research, reported to be upcoming at the time of their manuscript publication in the mid-1990s, was beginning to use risk factor models that ultimately would identify individuals who were most likely to be affected by obesity. They concluded by recommending an even more progressive third generation that would focus on the causal pathways between obesity and other problem areas. This is where the current literature appears to be, namely with an emphasis on mechanisms of weight gain relative to weight outcomes.

Many people have come forth with conceptual models with which to frame their obesity research programs. Most existing models for children tend to be of the concentric circle ecological variety reminiscent of Bronfenbrenner’s work (1979). Conceptualizations such as these can account for many of the areas that play a role in childhood obesity, including genetics, familial characteristics, community, and societal influences. It can also help identify the perceived causes of childhood obesity such as quality of nutrition and physical activity.

One such model, by Davison and Birch (2001; see Figure 1), focused on whether the vulnerability was due to (a) child characteristics and risk factors (e.g., age, gender,
dietary intake), (b) parenting style and family characteristics (e.g., child feeding practices, parent weight status, family TV viewing), and/or (c) community, demographic, and societal characteristics (such as ethnicity, neighborhood crime rates, accessibility to recreational facilities). They used their “model” as a guide for demonstrating that overweight and obesity in childhood is complex problem that needs to account for multiple factors from a variety of sources. Their model was not put forth as a formal structure for empirical validation or refutation, and, overall, it seems too complex for statistical analysis. I was not able to find empirical work that supported or challenged the
model. Harrison and colleagues (2011) took this viewpoint further by proposing six levels of influence (cell, child, clan, community, country, and culture) and five “zones” that related to different opportunities, resources, and practices (not pictured due to small, illegible typeface). This is also a conceptualization of how weight might be examined in youth today: broadly, across multiple domains, and in detail. Both of these two models were intended to account for childhood variability, although other models specific to infants (Tabacchi, Giammanco, La Guardia, & Giammanco, 2007) and adolescents (Neumark-Sztainer, 2005) also exist.

As these models were intended to inform future research practices, rather than be testable models in and of themselves, I selected what I considered to be the most salient features and then added further variables based on my reading of the literature on youth with disabilities. I took five variables from both models: a) weight status/BMI percentile, b) ethnicity, c) socioeconomic status (SES), d) sedentary behavior, and e) physical activity. I then took one additional variable (sufficient sleep) from the Harrison and colleagues model (2011). The new variables added to my IDD-specific model (see Figure 2) included psychopathology, compromised self-regulation, medication use, nutrition complications, and intelligence quotient. The arrows presented in the figure refer to the directionality of the relationships believed to exist. Single directional arrows imply that one variable (such as ASD) has an effect on the variable that it points towards. Therefore, ASD has an effect on all of the variables in the middle column (including additional psychopathology, sleep problems, etc.). The double-headed arrows imply a bidirectional relationship where both variables have an effect on one another. An
example from the typically developing literature could be if one has trouble with
depression it could lead to increases in appetite. This could, in turn, cause an increase in
weight, which could further increase depressive symptoms due to the social stigma that
can be associated with excess weight.

A testable conceptualization of obesity in children with developmental
disabilities, principally autism, would be advantageous for the ASD community. The
presence of a disability adds another layer to an already complex set of factors that
contribute to weight status stemming from an array of sources. Such a model would
enable investigators to (a) highlight areas that perhaps were not taken into consideration
for those who are neurotypical, (b) explore correlates that may be more detrimental to this group of individuals that goes above and beyond what is experienced by the typically developing population, and (c) identify areas where interventions could be implemented.

Researchers often use very large, nation-wide data sets to assess weight issues (Ogden, Yanovski, Carroll, & Flegal, 2007). This is because data are needed from multiple geographic locations to derive prevalence rates that can be generalized to the nation as a whole, rather than people who reside in specific counties or states. Focusing this analysis on youth with ASD takes the current research enterprise in a new direction, away from one based solely on neurotypical children. There are multiple data sets available that are focused on ASD, although each has specific goals. The National Database for Autism Research (ndar.nih.gov) is a platform where medical centers having ASD samples submit their data using a uniform set of demographic, clinical, psychological, and educational data. The Interactive Autism Network (IAN; ianproject.org) allows individuals with an ASD diagnosis or their families to answer online questionnaires about symptoms and life experiences. Two considerable flaws in this database are that the families are self-selected (and therefore could be non-representative of the ASD population at large) and that data are not filtered or verified through professionals. Nobody has absolute certainty that these individuals even have an autism diagnosis. The Autism Genetic Resource Exchange (AGRE; agre.autismspeaks.org) was designed for families with at least two children on the spectrum, and gathers information about clinical, medical, and genetic history. AGRE includes data from 1,700 families worldwide, but similar to IAN, the families are self-
selected. Because these data sets have goals that were not directly aligned with this project and may include parent-reported medical variables (such as height, weight, and diagnosis), the Autism Treatment Network (ATN) was used.

The ATN is a large network of clinical centers that was developed by the national foundation Autism Speaks, and is a group of 17 medical centers in the United States (n = 15) and Canada (n = 2) that provide initial diagnoses and follow-up care to children with ASD and their families. They use an interdisciplinary approach that, at minimum, includes psychologists, pediatricians, neurologists, and psychiatrists. Each ATN site administers a standardized set of instruments to assess the children. This battery includes physical assessments, cognitive testing, autism-specific testing, and an array of parent completed forms to assess adaptive behavior and psychopathology. Additional information about the ATN can be found on the website http://www.autismspeaks.org/science/resources-programs/autism-treatment-network.

The ATN data set did not include measures of compromised self-regulation or physical activity/sedentary behavior. I condensed the model further to include only variables that could be tested (see Figure 3). Although this is only a partial model, it should still shed light on whether these variables play a role for altered weight status in children with ASD. All constructs initially listed in Figure 2, regardless of inclusion in the final model, will be discussed below to summarize their relevance and to emphasize their importance in the literature to both ASD and weight issues.
Additional Psychopathology was broken down into depression, disruptive behavior disorders, anxiety, and ADHD. The Medications category was split into several medication classes (e.g., stimulants, atypical antipsychotics).
Psychopathology

Youngsters with ASD are often impacted with co-occurring psychopathology (Simonoff et al., 2008; Lainhart, 1999). It appears that anxiety disorders, attention deficit hyperactivity disorder (ADHD), and mood disorders (such as depression) are the most common for this population (Lainhart, 1999), although many other psychological conditions have been reported as co-occurring (e.g., Gadow, DeVincent, & Schneider, 2008). Each psychological disorder tends to have its own prevalence data for rates of obesity and overweight, which may contribute to the overall rate of childhood obesity in youth with ASD.

The connection between psychopathology and altered weight has been established in the neurotypical adult literature (Puder & Munsch, 2010) but is less established for neurotypical children and adolescents (Mustillo et al., 2003). This smaller pool of research may be due to a divergence between the emotions experienced by children and the child’s ability to verbalize these feelings (Reeves, Postolache, & Snitker, 2008). If so, this problem with expressing emotions would only increase in difficulty when working with youth with developmental disabilities, including those with ASD. Another difficulty with the literature on psychopathology and obesity is that many professionals conceptualize the model as unidirectional. That is, obesity causes a maladaptive emotional state, such as depression, as opposed to depression causing obesity (Dockray, Susman, & Dorm, 2009; Goodman & Whitaker, 2002). In my model, the potential bidirectional relationship between these two constructs is deliberately represented. More
researchers are starting to embrace a bidirectional approach and I believe this is more accurate.

Depression

The literature on psychopathology and obesity in neurotypical children has been inconsistent (Pine, Goldstein, Wolk, & Weissman, 2001). For instance, a study assessing predictors of adult BMI found that both childhood BMI and childhood depression were significantly associated with adult weight (Pine et al., 2001). In an earlier project, the same researchers also found that conduct disorder also predicted higher adult BMI, and that major depression failed to be significant when controlling for conduct symptoms (Pine, Cohen, Brook, & Coplan, 1997). A different investigation that compared obese and normal-weight children from a community sample also found that overweight children were more likely to demonstrate depressive symptomatology (Goncalves, Silva, & Antunes, 2012).

Mustillo and colleagues (2003) conducted a longitudinal study that evaluated obesity and psychiatric conditions in typically developing children between the ages of 9 and 16 years. They found that depression was more common in boys who were chronically obese (that is, obese most of their lives) than non-obese peers, and that oppositional defiant disorder was more likely in chronically obese children regardless of sex. In this study, they did not find any associations between obesity and bulimia nervosa, conduct disorder, anxiety, ADHD, or substance use. Depression was listed as one of the main comorbidities associated with obesity by Dietz and Robinson (2005), and depression could be associated with a large number of symptoms (sleep problems,
sedentary behaviors, altered food intake) that these two conditions share (Reeves et al., 2008). Given the social deficits children with ASD experience, it seems very possible that these youth may be comforting themselves with food, thereby causing them to be overweight or obese, rather than feeling stigmatized and depressed because of their weight. **Given the available literature presented, I hypothesized depression to be positively associated with BMI percentile.**

*Anxiety and Oppositional Defiant Disorder*

The literature on obesity and psychological conditions other than depression in neurotypical children is even more limited. Anxiety disorders have also been inconsistently linked to obesity in typically developing youth (Britz et al., 2000; Mustillo et al., 2003), although obesity and externalizing behavioral problems seem to be associated with one another more consistency. Mamun and colleagues (2009) found that children with behavioral problems, like those with oppositional defiant disorder at age 4 or 5 years, were more likely to be obese by the time they reached young adulthood. This risk for obesity later in life was more noticeable in those with behavior problems during childhood than those with behavior problem onset during adolescence (Mamun et al., 2009). **The established connection between behavior problems, such as oppositional defiant disorder, and weight led me to hypothesize that disruptive behavior problems would be positively associated with BMI percentile.** Also, due to the scarcity of literature on anxiety and weight, I viewed this analysis as exploratory.
ADHD and Compromised Self-Regulation

ADHD has a complicated relationship with abnormal weight. Children who are medication naïve, or not previously medicated for their mental health problems, tend to have elevated body mass compared to their non-ADHD peers, probably due to problems with inhibitory control (Cortese et al., 2008; Faraone, Biederman, Morley, & Spencer, 2008). Because ADHD is most commonly treated with stimulant medications (methylphenidate [Concerta], amphetamine [Adderall], etc.), this relationship reverses for children who are not medication naïve, which will be discussed further in the medications section. In non-medicated children with ADHD, 36% were overweight and 23% were obese (Curtin et al., 2005). This was not significantly different when compared to the values put forth for typically developing children by the National Health and Nutrition Examination Survey (NHANES, a common reference population) with 31% of children being overweight and 16% being obese (Curtin et al., 2005). Given the existing literature, I hypothesized that ADHD would be positively associated with BMI percentile. That being said, it also seemed likely that many children with ADHD in this sample would be taking stimulant medications to manage their ADHD symptoms. If this occurred, signal would likely be “cancelled” for ADHD alone and would instead be attributed to the medication, which would be negatively associated with BMI percentile. This is discussed in further detail starting on p. 18. While additional variables such as prior medication history or duration of medication treatment would have been valuable additions to these analyses, I only had access to current medications, which was admittedly imprecise.
Compromised self-regulation (which includes impulsiveness) is associated with psychopathology. For instance, ADHD comprises symptoms of inattention, impulsiveness, and overactivity. However, poor self-regulation can also occur without being severe enough to meet diagnostic criteria for ADHD. Central to compromised self-regulation are features such as inability to inhibit desires, problems overriding impulses, and impaired delay of gratification (Stice, Spoor, Ng, & Zald, 2009). Children with ASD appeared to demonstrate some deficits in inhibitory control in a study by Christ and colleagues (2007). Youth with ASD were able to perform as well as their typical peers on the Stroop task (reading names of colors presented in font colors that do not necessarily match), although they demonstrated deficits on a flanker interference task where they had to identify whether letters were “flanked,” or next to, similar letters on either side.

Obesity in children has been linked to dysregulation in the hypothalamus and hippocampus, brain areas that are involved with monitoring motivation, cognitive control, and impulsivity (Bruce et al., 2010). Bruce et al. (2010) investigated brain change pre- and post-meals, and found that children were highly responsive to food-based stimuli, which could increase motivation for food and quantity of food consumed. Problems with eating behaviors have been a recurring theme for people with abnormal body mass, as demonstrated by binge eating being linked to higher rates of impulsivity among some obese children (Puder & Munsch, 2010). Logically, one might expect more severely compromised self-regulation to be associated with higher weight. This potential hypothesis cannot be tested because the ATN has no direct measure of self-regulation.
Summary

Ultimately, we know that various forms of psychopathology have an established relationship with abnormal weight status. As previously mentioned, conditions such as depression, ADHD, anxiety, compromised self-regulation, and others can alter inhibitory control or appetite, leading to changes in body mass. Individuals with ASD experience these conditions relatively frequently (Matson & Nebel-Schwalm, 2007), which emphasizes the need to be cognizant of the relationships of these conditions with overweight and obesity. Each of these disorders and/or traits leads to hypotheses about psychological conditions in relation to extremes of weight.

Medications

Many children with ASD are treated with medication to address behavioral or emotional problems or treat comorbid medical issues (Grondhuis & Aman, 2013). Medications are almost always accompanied by drug-specific side-effect profiles, although adverse event presentations in individuals may be highly variable. Although side effects can be mild in nature, they do have the ability to be problematic. In the case of altered weight status, medications can diminish or increase appetite, alter metabolic functioning, or change food preferences (Bernstein, 1987), although, not all studies have found relationships between medications in general and weight status in persons with intellectual disability (Lin, Yen, Li, & Wu, 2005).

Atypical antipsychotics are commonly prescribed to youth with ASD to mitigate irritability and aggressive behaviors, among other things, and this class of psychotropic drug has been highly associated with weight gain (Nasrallah, 2003). The change in body
mass has been correlated with decreased metabolic rate combined with increased appetite (and thus, increased caloric intake) and decreased physical activity (possibly from sedating effects; Baptista, 1999). Each drug formulation has its own side effect profile, which makes some atypical antipsychotics (e.g., clozapine and olanzapine) more prone to increases in body mass than others (e.g., ziprasidone and quetiapine; Nasrallah, 2003). Risperidone and aripiprazole are the only two compounds that have been granted Food and Drug Administration (FDA) approval for treating disruptive behaviors in autistic children (www.fda.gov). Numerous studies have found that risperidone is associated with moderate weight gain that does not appear to be related to dose (Nasrallah, 2003). There was also a relationship between risperidone use in children with autism and comorbid intellectual disability and weight gain across all age groups (Hellings, Zarcone, Crandall, Wallace, & Schroeder, 2001). Aripiprazole has exhibited mixed results; one meta-analysis found that it caused relatively lower increases in body mass (equivalent to haloperidol) than most other atypical antipsychotic drugs in the majority of short-term studies (Jody et al., 2002), and another study demonstrated gains of 1.0 to 1.2 kg over eight weeks (Marcus et al., 2009). Based on the existing literature, I hypothesized that atypical antipsychotics would be positively associated with BMI percentile in this study.

Psychostimulants, the most common drug class for ADHD management, were previously mentioned as compounds that could have an effect on weight. To elaborate, this class of drug is associated with slight weight loss due to appetite reduction, although this was not always found (Faraone et al., 2008), and weight change appears to stabilize
over time. Stimulants can also cause changes in growth rate, and delays in expected height can be seen in children for up to two years after medication has been stopped (Faraone et al., 2008). This shift in growth can understandably alter the ratio between weight and height that is evaluated through BMI. Children with ADHD who receive stimulant medications often have lower body mass when compared to their non-ADHD peers not prescribed stimulant medication (Cortese et al., 2008; Faraone et al., 2008).

One study from Curtin and colleagues (2005) indicated that only 16% of children medicated with stimulants in their sample were overweight and 6% were obese, although they did not compare these values to reference data. Atomoxetine is a non-stimulant medication (selective norepinephrine reuptake inhibitor) also prescribed for ADHD symptoms (Buitelaar et al., 2008). This compound has a history of weight loss at treatment onset, which may or may not be maintained with continued pharmacotherapy (Wernicke & Kratochvil, 2002). Given the existing research, I hypothesized that stimulant medications and atomoxetine would be inversely associated with BMI percentile in this study.

Medications such as valproic acid, used for epilepsy and bipolar disorder, have also been associated with increased weight profiles (Wirrel, 2003; Fava, 2000), as have alpha 2 agonists, antihistamines, and selective serotonin reuptake inhibitors (SSRIs; Malone, 2005; Khan, Jain, Soltys, & Takahashi, 2012). I hypothesized that all of the compounds listed in the previous sentence would be positively associated with BMI percentile in the model.

Sleep Problems
Disordered sleep is known to occur at a greater rate in children with ASD than in neurotypical children (Hollway & Aman, 2011). It appears that there are many factors that contribute to this relationship, including internalizing or externalizing behavior difficulties, anxiety, autism symptom severity, medical conditions, and medication use (Hollway & Aman, 2011). Compared to youth with normal weight, adolescents who are overweight tend to have more disrupted sleep in which individuals wake multiple times throughout the night. They also have shorter sleep duration, as 49% of the overweight sample reported fewer than eight hours of sleep during week nights (Beebe et al., 2007). These children were also at higher risk for sleep-disordered breathing, such as apnea (Beebe et al., 2007; Flint et al., 2007). Adolescents further appear to suffer from daytime sleepiness, which can undermine the amount or duration of physical activities in which the youngster would otherwise engage (Gupta, Mueller, Chan, & Meininger, 2002). Sleep deprivation (Spiegel, Tasali, Penev, & Van Cauter, 2004) as well as a decrease in hours spent sleeping was connected to increased hunger (Tremblay & Chaput, 2008), which can perpetuate the cycle of obesity and insomnia. Short sleep duration in early childhood (Reilly et al., 2005) and adulthood (Chaput et al., 2009) has been identified as impacting metabolism, which in turn may affect weight status (Tremblay & Chaput, 2008). Therefore, I hypothesized that all elevated sleep problems would be positively associated with BMI percentile.

Nutrition

Food is half of the energy equation (calories consumed versus calories expended) that controls weight. Typically developing children often derive eating behaviors and
preferences from their parents through modeling and/or training. With close to two-thirds the adult population in the United States overweight and one-third obese (subsumed within the overweight category), there is a good chance that parents are not passing on ideal dietary behaviors. An important aspect of childhood nutrition is that some eating problems are developmentally appropriate for preschool-aged youngsters (Kuhl, Clifford, & Stark, 2012). These typical behaviors can include strict food preferences, rejection of new items, and tantrums with parental noncompliance to child demands (e.g., crying because parents will not give them the ice cream they wanted for dinner; Schreck, Williams, & Smith, 2004). While children are usually able to outgrow these tendencies during the elementary school years, many children with ASD do not, and these problem behaviors can persist throughout the lifespan.

Matson and colleagues (2009) looked at feeding problems and found that children with autism were more likely to “eat too much” and experience weight gain when compared to typically developing children and to children with a disability that was not an ASD. They also found that both children with autism and with PDD-NOS were more likely to prefer certain smells or textures of food or eat a restricted amount of food when compared to children who were either typically developing or diagnosed with a non-spectrum disability. Food selectivity and fussiness have been thought to be associated with inadequate nutritional intake (Bandini et al., 2010) and underweight (Hendy, Williams, Riegel, & Paul, 2010). This may seem counterintuitive, given that at least one study (Rimmer et al., 2010) reported higher rate of overweight and obesity seen in this population coupled with the large prevalence of abnormal food behaviors. One study of
children being treated in a feeding clinic (Hendy et al., 2010) found that fussiness (based on a score from the Child Eating Behavior Questionnaire; Wardle, Guthrie, Sanderson, & Rapoport, 2001) in children with autism was negatively associated with the range of different foods consumed and positively correlated with the use of meals prepared, especially for the child following food refusal. These findings were similar to what was found in children with developmental disabilities other than autism and those without developmental disabilities (Hendy et al., 2010).

One study (Lindsay et al., 2006) demonstrated that children with ASD, being treated with risperidone, consumed diets that were adequate in terms of macronutrients including fats, carbohydrates, and protein. There were variations in vitamin and mineral intake, with 45% of the sample (N = 20) having inadequate calcium, 40% receiving inadequate vitamin K, 30% inadequate pantothenic acid, and 35% inadequate intake of vitamin D (Lindsay et al., 2006). These micronutrients are now believed to play a role in regards to weight status. An overview of obesity risk factors demonstrated that inadequate intake of micronutrients (such as calcium, vitamins B_{12}, C, and E) predicted an increased tendency to be overweight (Tremblay & Chaput, 2008).

One study found that children with ASD were more likely to consume sweetened beverages (such as juice) and snack foods and less likely to ingest fruits and vegetables when compared to their typically developing peers (Evans et al., 2012). Interestingly, higher consumption of fruits and vegetables or fruits alone was positively correlated with body mass in their sample. That is, the more fruits or vegetables eaten by children, the heavier the child was likely to be (Evans et al., 2012). This is counter to previous
literature that indicated that higher produce intake was associated with lower levels of body mass (Epstein et al., 2012).

Applied behavior analysis (ABA) is the leading psychosocial treatment option available to persons with ASD, and a salient feature of this intervention is positive reinforcement for desirable behaviors (McIlvane et al., 2011). A common form of reinforcement is through edible rewards – chips, candies, etc. (Bandini, Curtin, Hamad, Tybor, & Must, 2005). Although these reinforcers likely do not comprise a substantial portion of a child’s daily food intake, the use of calorically dense foods as reinforcers cannot be ignored as a possible contributor to weight issues.

Physical Activity and Sedentary Behavior

While literature on energy expenditure in youth with IDD is limited, it is known that adults with DD tend to engage in more sedentary activities than adults without a disability (Havercamp, Scandlin, & Roth, 2004). One reason why overweight and obese youth with disabilities (such as autism) spend less time on physical activity could be because more of their days are occupied with behavior interventions (e.g., ABA) and specific therapies (e.g., physical, occupational, speech) than is the case in neurotypical children. There are a limited number of hours in the day, and there is less time to encourage children to engage in physical activity when parts of the day are consumed by various therapies. With the exception of physical therapy, the majority of the daily activities in the life of a child or adolescent with an ASD are performed when seated, which allows for minimal energy expenditure (Chen et al., 2010).
Physical activity, or lack thereof, has a defined relationship with obesity, since a failure to expend all of the calories consumed leads to excess body mass (Reeves et al., 2008). We do know that youngsters with intellectual disability demonstrate poorer performance on fitness assessments when compared to their peers with average IQ (Frey & Chow, 2006), which could complicate participation in physical activities that would burn calories and help maintain a healthy weight. The amount of physical activity at 3 years of age was a better predictor of BMI at 6 years than was diet (Jago, Baranowski, Baranowski, Thompson, & Greaves, 2005). One study (Pulkki-Raback, Eloavaino, Kivimaki, Raitakari, & Keltikangas-Jarvinen, 2005) found that children with deficits in peer socialization were more likely to be sedentary in adulthood, possibly because many activities engaged in while alone (watching television, using the computer, etc.) are usually performed while sitting. This has interesting ramifications, because problems with social behavior is a core feature of ASD and may help to account for part of the increased risk for abnormal weight status in such children. **This leads to the logical hypothesis that increased levels of sedentary behavior would be positively associated with BMI percentile.** Unfortunately, the ATN data base did not include indices of sedentary behavior or exercise expenditure, so I was not able to address this hypothesis.

**Intelligence and Adaptive Behavior**

Around 70% of individuals with DSM-IV autistic disorder have comorbid intellectual disability, although this number is lower when the entire spectrum is taken into account (Fombonne, 2003). There is limited research available using the new DSM-5 ASD criteria, but at least one article reported a negative correlation between IQ and
comorbid psychopathological symptoms in children with ASD (Tureck, Matson, Cervantes, & Konst, 2014). More time needs to elapse before the field can expect an update on ID prevalence in youth with ASD using the new criteria.

It has been suggested that level of intellectual functioning may be a more valid way of differentiating between the ASD subtypes (Lecavalier, Snow, & Norris, 2011), which underscores the importance of IQ level in youngsters. Intellectual disability, by definition, includes deficits in adaptive behavior, and youth with ASD tend to display characteristic profiles on measures of adaptive behavior (Paul et al., 2004). For example, children with autistic disorder tend to score lower than their peers with PDD-NOS on abstract concepts, relating experiences, socialization, coping, and daily living skills.

There is limited research available about intelligence quotient (IQ) or adaptive behavior and how it corresponds to obesity, particularly in children. One study (Moran et al., 2005) evaluated adults with intellectual disability and found that those with severe intellectual disability had lower rates of obesity when compared to other intellectual disability groups, possibly due to the likelihood for this group to be in residential facilities and have restricted access to food. Individuals with mild intellectual disability did not appear to have higher rates of obesity than the typically developing population without such a disability (Moran et al., 2005). A different study by Rimmer and Yamaki (2005) found that American adults with intellectual disabilities were much more likely to be overweight than their typical intelligence peers. In the sample of Rimmer and Yamaki (2005), 60.6% of people with intellectual disability were classified as obese (compared to
30.5% in the general population), and they were 2.5 times more likely to have extreme obesity with a BMI greater than 40.

In the typically developing population, it appears that intelligence is only loosely related to adult obesity. A Danish study (Halkjaer, Holst, & Sorensen, 2003) found that intelligence and education were both inversely related to adult obesity. Once IQ scores were adjusted for educational achievements, intelligence failed to predict later obesity. A study from the United Kingdom reported that lower IQ scores in childhood were associated with greater rates of obesity and general weight gain in adulthood (Chandola, Deary, Blane, & Batty, 2006). These findings were mediated by both education and a healthy lifestyle. Child education may not be a viable factor in a sample of children with ASD, as many do not engage in conventional schooling and therefore amount of education may not have the same relationship between education and weight.

A meta-analysis of studies that evaluated levels of intelligence and weight found that obese school-aged children had lower full scale and performance IQs than did non-obese children (Yu, Han, Cao, & Guo, 2010). They also found that the available studies suggested a significant difference between full-scale IQ in severely (defined as Weight ÷ Ideal Weight > 1.5) obese children when compared to normal-weight children, but no statistical difference between those with mild or moderate obesity (defined as Weight ÷ Ideal Weight = 1.2-1.5. The studies included in this review were primarily from China (17 articles) or the United Kingdom (6 articles); there were no papers based on data from the United States. I looked for information on the IQ ranges that were studied and was unable to determine this. The large majority of studies (n = 17) were written in Chinese
and, of the remainder, the IQ range was either not reported or was not standardized in a meaningful way ($M \neq 100$, $SD \neq 15$). For example, the Moray House Tests (Scottish Council for Research in Education, 1933) were mental tests that do not correspond readily to commonly used IQ tests that are frequently used in the developmental disabilities field (Deary, Whiteman, Starr, Whalley, & Fox, 2004). **For this dissertation, I hypothesized that IQ would be inversely related to BMI percentile.**

**Socioeconomic Status (SES)**

The prevalence rates of ASD across SES have been a recent topic of inquiry. It appears that there is a positive correlation between rates of diagnosed autism and income level. Families who make more money are more likely to have a child diagnosed with an ASD (Maenner, Arneson, & Durkin, 2009; Durkin et al., 2010). Most have attributed this relationship to ascertainment bias: the more parental education and wealth, the higher the likelihood of an accurate diagnosis of ASD (Wing, 1980). This relationship still needs to be verified and validated across geographic regions.

SES has been identified as a major contributor to the obesity problem in America. Children coming from families who are in lower income brackets or who have less education tend to be more obese. Individuals in the lower income brackets typically purchase lower quality foods that are relatively inexpensive (but highly caloric), have fewer recreation areas for physical activities close to the home, and engage in more sedentary behaviors. Children, in particular, tend to be passive consumers of energy-dense and nutritionally void foods while watching television, which often takes up hours each day (Ebbeling, Pawlak, & Ludwig, 2002). This pattern of eating behavior combined
with physical inactivity results in excessive calories that are not metabolized. It should then come as no surprise that such children gain weight at a rapid pace. Chronically obese children or those with childhood obesity (that dissipated during adolescence) were twice as likely to be from families having at least one parent who did not graduate from high school (Mustillo et al., 2003).

Household income does not affect typically developing men and women in similar ways. Women with higher income tend to be healthier than lower-income women, while men tend to become increasingly more overweight and obese with wealth (Mandal & Chern, 2011). This gender difference seems to be the case in typically developing children as well as adults. A recent study from the United Kingdom found that, by age 7 for boys and age 4 for girls, there was a socioeconomic pattern that differentiated BMI (Howe et al., 2011). Children of mothers with college degrees had lower BMIs than children of mothers who completed less education based upon the British testing system (Ordinary and Advanced Level exams; Howe et al., 2011). Furthermore, childhood BMI was negatively correlated with occupational extrapolated income (estimated income based on job title and responsibilities) in the same study, which essentially confirmed the result using projected salary (Howe et al., 2011). It has also been found that typically developing women who are obese make less money than their non-obese peers, which reinforces the notion that people with lower income are more likely to be obese. However, this general finding did not hold in the United States for men and women of African American decent (Gortmaker, Must, Perrin, Sobol, & Dietz, 1993). In summary, children of college-educated women tend to have lower body
weight than children of women who did not obtain a college education, adult women in the higher SES brackets tend to be lighter than women in the lower SES brackets, while men in the higher SES brackets tend to be heavier than men in the lower SES brackets. 

For these reasons, I hypothesized that higher rates of parental education (this study’s proxy for SES) would be inversely associated with BMI percentile. No prediction was made about the effect of the sex of the subjects.

Ethnicity

Ethnicity appears to have a strong relationship with obesity. One study by Fontaine and colleagues (2003) found that, on average, Caucasian women lost an average of 8 years, Caucasian men lost 13 years, African American women lost 5 years, and African American men lost 20 years of their life expectancy from being severely obese (adult BMI > 35). Ethnicity also has a definitive link with SES (Freedman et al., 2007). Freedman and colleagues (2007) used the NHANES data to compare ethnicity, BMI, and family income in typically developing children and found that body mass dropped with higher SES for Caucasians and Mexican Americans, but that BMI percentile increased for African American children as familial income rose. These findings were most pronounced for children between 6 and 18 years of age, as opposed to those who were between the ages of 2 and 5 years (Freedman et al., 2007). Alternatively, people of Asian descent have historically been lighter than peers from other ethnicities (Lauderdale & Rathouz, 2000). Given the array of literature available on ethnicity and abnormal weight status, I hypothesized that being African American or Hispanic would be positively
associated with BMI percentile, while being of Asian heritage would be inversely associated with BMI percentile.

Conclusions

Childhood overweight and obesity are serious conditions that could very much affect the trajectory of an individual’s psychological and physical health in adulthood. These overweight and obesity conditions often force people with child onset obesity to face an uphill weight battle, across the lifespan, which is very difficult to win. The population shift to greater overall body mass can distort perceptions about what weight should be considered “normal.” Parents may be less likely to recognize weight problems in their children if they cannot also see it in themselves (Burke, Heiland, & Nadler, 2010). Additionally, in youth with disabilities, overweight seems to augment the impact of their disability (Chen et al., 2010). Although this is understandable, it can be dangerous to disregard weight and the potentially serious associated health conditions (Grondhuis & Aman, 2013).

At this time, the majority of trials aimed at prevention of obesity in typically developing children have failed to demonstrate consistent results (Stice, Shaw, & Marti, 2006). We must approach this topic from a new standpoint that acknowledges both risk factors for obesity in general and specific problem areas for individuals with ASD. Altered weight can be attributed to an array of mechanisms, and it is not usually possible to pinpoint a single defining cause. Properly identifying risk factors for obesity both in childhood and later, in adulthood, has been described as a key to prevention (e.g., Chaput
et al., 2009; Reilly et al., 2005; Dietz, 2000). The other key, of course, is to respond to the presence of these risks.

Hypotheses

Youth with ASD and their families face many obstacles. Physiological and psychological problems associated with abnormal weight should not be additional complications introduced into the lives of these individuals, and researchers should be identifying ways to maximize health.

The primary goals of this dissertation were to a) determine a prevalence rate for overweight and obesity in youth with ASD from a large and geographically diverse dataset that was collected by medical and health care professionals, and b) identify what variables appear to be most related to BMI percentile in an effort to determine where future interventions should focus their attention. These interventions could reduce negative health outcomes and diminish the associated economic burden of caregivers. Prevention of abnormal body mass has been hailed as the “only realistic solution” to the obesity problem that currently has deleterious effect on society (Lobstein et al., 2004). Being able to identify what characteristics are most common among those with excessive weight, particularly within specialty populations, can allow us to tackle this problem both on the individual level, through education, and at the community level, with public policy programs. Although a model in and of itself will not be able to reduce the obesity problem that children with ASD face, this dissertation will contribute additional information to the sparse weight literature in ASD and enable us to proceed with improved preventative measures.
I posited the following hypotheses (summarized from within the Introduction) based on my theoretical model (Figure 3) in combination with the previously presented existing literature.

1. The theoretical model proposed will be found to be fundamentally valid based on predictions of BMI percentile outcome.
2. Children with ASD will have significantly higher BMI percentiles than the general population determined by reference to the CDC norms.
3. African American heritage, Hispanic heritage, depression, disruptive behavior disorders, SSRI use, atypical antipsychotic use, mood stabilizer use, and sleep disordered breathing will be positively associated with BMI centile.
4. Asian heritage, stimulant use, atomoxetine use, IQ, and adaptive behavior will be negatively associated with BMI centile.
5. Parental education level will be inversely associated with BMI centile.
CHAPTER 2: METHODS

Overweight and Obesity Classifications

Although imperfect, BMI is an affordable and commonly used measurement of weight status. Child weight evaluations are more complicated than assessments for adults since they need to take growth trajectories into account (see Appendix A for CDC growth trajectories for boys and girls), and therefore percentiles derived from BMI-based curves are used to compare children to their peers. That being said, the subsequent terminology is used inconsistently (Flegal, Ogden, Wei, Kuczmarski, & Johnson, 2001) and, although there are suggested rules, there appears to be a fair amount of confusion within the literature that was reviewed for this project.

*Overweight* in childhood is defined as being greater than the 85\textsuperscript{th} percentile on the CDC growth curves but below the 95\textsuperscript{th} percentile, whereas *obesity* is classified as being above the 95\textsuperscript{th} percentile. Despite this distinction, some articles claim to be using CDC criteria, when they in fact list that overweight is a BMI above the 85\textsuperscript{th} percentile without excluding those above the 95\textsuperscript{th} percentile cut off (Rimmer et al., 2010). Others tend to present the overweight and obesity findings together (e.g. “The prevalence rate for overweight and obesity is 37.2\%”), sometimes in combination with the correct overweight and obesity prevalence rates described early (Wang & Lobstein, 2006; Ogden et al., 2006; De, Small, & Baur, 2008). Still others appear to have conflicting criteria by listing different cut-offs throughout their manuscript (Curtin et al., 2005; Ogden &
This confusion in reporting can be seen when the authors attempted to compare their results to other researchers who used different weight classification criteria (Memari et al., 2012). Without clear definitions of categories, and adherence to them, it is very difficult to compare one study’s results to another.

The CDC perpetuates this confusion by offering materials for schools and other large groups that calculate overweight as greater than the 85th percentile without the 95th percentile cutoff (Children’s BMI Group Calculator – Metric Version, 2010). If matters were not complicated enough, starting in 2007 the nomenclature shifted from “at risk for overweight” to “overweight” for the 85th percentile cut off, and from “overweight” to “obese” for the 95th percentile cutoff (Ogden & Flegal, 2010). This essentially means that the term “overweight” could refer to any number of weight distinctions in recent literature, and a careful review of the cutoffs criteria is warranted. I emailed Dr. Cynthia Ogden from the CDC to clarify what would be considered “best practice” for overweight figures since there appears to be considerable flux with the definition of the overweight category (personal communication, August 19, 2013; see Appendix B). She stated that I could present the findings either way (between the 85th and 95th percentiles OR greater than the 85th percentile), and as such, I conformed to the most popular way of presenting overweight, which is overweight defined as simply exceeding the 85th percentile without excluding those above the 95th percentile cutoff. Please assume that this is the way that all research throughout this dissertation is presented unless otherwise specified. That is, overweight will incorporate those above the 85th percentile (including those above the 95th percentile) and obese will include subjects whose weight exceeds the 95th percentile.
The most recent prevalence estimates indicated that 31.7% of children in the United States are overweight and that 16.9% are obese (based on data collected between 2007-2008; Ogden, Carroll, Curtin, Lamb, & Flegal, 2010).

Instruments

The ATN used a battery of measures that was supposed to be administered to every child whose data were included in the data set. Of the complete database, only a minority of measures were suitable to the model and were included for analyses. The complete list of measures can be found in Appendix C. Although there was no required training per se to become members of the ATN, institutions under consideration for ATN membership had to meet several criteria for ATN funding. According to the packet for the 2014 application cycle, sites needed to have existing programs for the diagnosis and treatment of ASDs and be part of academic health centers or hospitals. This should have helped to ensure a level of competency for both the centers and personnel prior to approval (http://www.autismspeaks.org/docs/sciencedocs/atn/atn_full_application_rfa_5-2-13.pdf). Additionally, the lead autism specialists who completed the exams had to participate in a webinar that explained specific ATN procedures, although there was no protocol at the time when the data were collected for this dissertation for standardized assessment of height and weight (B.L. Handen, Principal Director of University of Pittsburgh site, personal communication, July 19, 2013; D. Coury, Medical Director of ATN, personal communication, July 22, 2013).
Child Behavior Checklist (CBCL)

The CBCL (Achenbach & Rescorla, 2001) is a behavioral rating scale used to assess psychopathology in children ages 1.5-18 years, inclusive. It is broken down into two age categories, one for children ages 1.5-5 years, and another for 6-18 years of age. Although these two versions assess many similar constructs, there are important age variations (e.g., the scale for older children included a Conduct Problems subscale whereas the younger version did not). The CBCL contains 118 statements about child problem behaviors that are completed by the parent or caregiver. Although the scale comes in parent, teacher, and self-report versions, only the parent-report version was used in this study. The empirically derived questions align to various criteria that are divided into problem scales (Internalizing, Externalizing, and Total Problems), syndrome scales (Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, Aggressive Behavior, Emotional Symptoms, and Pervasive Developmental Problems), and DSM-oriented scales (Affective Problems, Anxiety Problems, Somatic Problems, Attention Deficits/Hyperactivity Problems, Oppositional Defiant Problems, and Conduct Problems).

Subscale alphas were reported for the 1.5-5 year rating form ranged from .66 (Anxious/Depressed) to .92 (for both Aggression and Externalizing) in a sample of typically developing children (Pandolfi, Magyar, & Dill, 2009). Pandolfi and colleagues (2009) also evaluated the measure with an ASD sample and found comparable results, with the exception of the Somatic Complaints subscale that was .80 in the typical children and only .49 in those with ASDs. Embregts (2000) assessed the psychometric
characteristics using a sample of 42 children with intellectual disability and found that they were not as robust as they were when a typically developing sample was used. For this study, depression was assessed using the Withdrawn/Depressed syndrome subscale (rather than Anxious/Depressed), whereas anxiety was assessed using the DSM-oriented Anxiety Problems subscale (again, rather than Anxious/Depressed). The Anxious/Depressed subscale was given lower priority because it did an inadequate job of disentangling the two variables of interest (i.e., items reflecting both anxiety and depression are contained in this subscale). Disruptive behavior disorders were assessed using the Conduct Problems and the Oppositional Defiant Disorder subscales (whichever was more appropriate given child age). ADHD was assessed using the DSM-oriented ADHD Problems subscale. Subscale T-scores were used rather than raw values so comparisons could be made across age versions (Achenbach & Rescola, 2001). Because the number of items varied across subscales in the two versions, this forced the use of T-scores to achieve continuity from the younger to older scale versions.

*Vineland Adaptive Behavior Scales-II (Vineland-II)*

The Vineland-II (Sparrow, Cicchetti, & Balla, 2005) is an individually administered adaptive behavior assessment designed to examine functional skills in four domains: Communication, Socialization, Daily Living Skills, and Motor Skills (only included for those at or under the age of 6 years) normed from birth to 90 years of age. There is an optional Maladaptive Behavior section to help quantify problem areas. There are multiple versions of the instrument, including a Survey Interview Form, an Expanded Interview Form, Parent/Caregiver Rating Form, and a Teacher Rating Form. In order to
minimize caregiver and personnel time burden, the Survey Interview Form, which is the shortest of the interview formats, was used. The instrument norms were established using a sample of over 3,000 people across the age range (Sparrow et al., 2005).

The previous version of this measure had been evaluated for persons with ASDs. The Vineland Adaptive Behavior Scale’s (Sparrow, Balla, & Cicchetti, 1984) Adaptive Behavior Composite Score was found to be significantly and negatively correlated (r = -.49; de Bildt, Kraijer, Sytema, & Minderaa, 2005) with scores received on the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000). Thus, higher levels of adaptive behavior were associated with fewer autism symptoms (or lower overall symptom severity). The Vineland-II provides an Adaptive Behavior Composite Score, which is an estimate of overall adaptive behavior. In this study this composite score was used for the Adaptive Behavior component when assessing the overall weight model.

**Intelligence Assessments**

A variety of intelligence tests were administered at the discretion of the site personnel, due to the heterogeneous presentation of these children. *Almost all children had data from only one intelligence test available.* The choice of what test to administer could be based on a variety of factors, including (but not limited to) language ability, time available for testing, and/or child cooperativeness. While the majority of intelligence assessments produce equivalent results in typically developing children (e.g., Roid & Miller, 1997; Roid, 2003), this may not be the case when assessing youth with ASD (Grondhuis & Mulick, 2012). For youngsters with ASDs, the tests’ language demands may have a significant impact on a child’s IQ score. For instance, Grondhuis
and Mulick (2012) found that children with ASDs received higher scores using nonverbal measures, such as the Leiter International Performance Scale, Revised, than using verbal assessments such as the Stanford-Binet Intelligence Scales, 5th Edition. It is regrettable and a limitation that not all children who were included in my sample were assessed using the same scale. Nevertheless, I concluded that there was more benefit from having some estimate of IQ than from discounting IQ entirely or excluding participants. If participants had more than one IQ test in the ATN, priority was given in the order the tests are presented below. That is, if there was a Stanford-Binet available then it would be used, and if not then a WISC, and so on.

*Stanford-Binet Intelligence Scales, 5th Edition*

The Stanford-Binet Intelligence Scales is a verbal intelligence measure that offers a Full-Scale IQ, Abbreviated IQ, Nonverbal IQ, and Verbal IQ (Roid, 2003). The Nonverbal section, which is a new addition to the 5th edition of the instrument, does not require verbal answers, although the questions are still presented verbally and require language comprehension. Five subtests are administered for each of the Verbal and Nonverbal sections. These include subtests on Fluid Reasoning, Knowledge, Quantitative Reasoning, Visual Spatial Processing, and Memory. The scale has excellent split-half reliability: .91 on Abbreviated IQ, .95 on Nonverbal IQ, .96 on Verbal IQ, and .98 on Full-Scale IQ. The vast majority, 1,741 (66.7%) of children in the current study received the Stanford-Binet.
Wechsler Intelligence Scales for Children (WISC)

This assessment for children ages 6 years 0 months to 16 years 11 months includes 15 subscales (Coding, Picture Concepts, Block Design, Digit Span, Similarities, Letter-Number Sequencing, Vocabulary, Matrix Reasoning, Comprehension, Picture Completion, Symbol Search, Cancellation, Arithmetic, Information, and Word Reasoning). The subscales are combined to create a Verbal Comprehension Index, Perceptual Reasoning Index, Processing Speed Index, and Working Memory Index, in addition to the Full-Scale IQ (Wechsler, 2004a). Not all subtests are needed to compute the different Indices or IQ score.

The WISC-IV was standardized on a sample of 2,200 children from across the country. The measure has high internal consistency, with split-half reliability coefficients for the five indices or IQ score ranging from .88 (Processing Speed) to .97 (Full-Scale IQ). Children with autism also had high reliability on the subtests; all scores were above .88 (Letter-Number Sequencing), with the exception of Digit Span Forwards (.77; Wechsler, 2004b). Typically developing children scored similarly on the Stanford-Binet and WISC (Roid, 2003). There were 56 (2.1%) children who were tested using the WISC.

Wechsler Preschool and Primary School Inventory-III (WPPSI)

The WPPSI is an intelligence test that is designed for children ages 2 years 6 months to 7 years 3 months (Wechsler, 2002a). It comprises 14 subtests that include Block Design, Matrix Reasoning, Information, Vocabulary, Picture Concepts, Word Reasoning, Symbol Search, Coding, Comprehension, Similarities, Picture Completion,
Object Assembly, Picture Naming, and Receptive Vocabulary. These subtests can be combined to create Verbal IQ, Performance IQ, Processing Speed Quotient (only for those at or above 4 years of age), General Language Composite, and Full Scale IQ, which was the measure used in this project. Not all subtests need to be completed to derive the possible IQ and composite scores. This instrument was standardized on a sample of 1,700 children from around the country (Weschsler, 2002a). The IQ and composite scores had high internal consistency, ranging from .89 for the Processing Speed Quotient to .96 for the Full-Scale IQ score. The split-half reliability was especially strong for those with autism, as all subscale reliabilities were over .94 (Weschsler, 2002b). The WPPSI is thought to be more appropriate for children who may have impaired cognitive abilities and are at least 6 years of age (Wechsler, 2002a). Although there is substantial overlap between the WPPSI and WISC, they are not identical instruments. There were only 30 (1.1%) children in the ATN data base who received the WPPSI.

**Leiter International Performance Scale-Revised (Leiter-R)**

The Leiter-R is an entirely nonverbal measure where instructions are given in pantomime, and youth respond by completing a task or pointing to an answer (Roid & Miller, 1997). This is a common assessment in children with ASDs, and it appears that children on the spectrum tend to receive higher scores on this nonverbal assessment when compared to the Stanford-Binet (Grondhuis & Mulick, 2013). The Leiter-R is separated into two batteries that include 10 subscales each. The Visualization and Reasoning Battery includes the following subscales: Design Analogies, Form Completion, Repeated Patterns, Classification, Matching, Sequential Order, Picture Context, Figure Ground,
Paper Folding, and Figure Rotation. The subscales of the Visualization and Reasoning battery are combined to obtain a composite IQ. The Attention and Memory Battery subscale includes Forward Memory, Backward Memory, Attention Sustained, Visual Coding, Spatial Memory, Attention Divided, Delayed Pairs, Associated Pairs, Immediate Recognition, and Delayed Recognition. The Attention and Memory subscales are not included in the Full Scale composite score but instead are used to evaluate deficits in attention or memory. Subtests are administered based on the age of the person being evaluated. The Leiter-R has norms for individuals aged 2 years 9 months to 20 years 11 months based on a representative sample of 1,719 subjects (Roid, Pomplun, & Martin, 2009). There were 79 (3.0%) children who were assessed using the Leiter-R.

*Mullen Scales of Early Learning*

The Mullen Scales of Early Learning (Mullen, 1995) is a measure of cognitive functioning that can be used from birth to 68 months of age. It comprises a Gross Motor Scale (only used for those up to 33 months of age), Visual Reception Scale, Fine Motor Scale, Receptive Language Scale, and Expressive Language Scale. Children with ASD tend to score significantly lower than their typically developing peers in all four Mullen scales. It appears that this difference may be related to increased off-task (inattentive) behavior, in which children with ASDs engage significantly more (Akshoomoff, 2006). The Early Learning Composite is produced by summing the T-score of the subscales to acquire a standard score that evaluates the general cognitive functioning of the children tested (Mullen, 1995). The ATN sample was quite young, and as such, 704 (27.0%) children were assessed using the Mullen.
**Children’s Sleep Habits Questionnaire (CSHQ)**

The CSHQ is a retrospective parent-completed questionnaire that contains 45 items that investigate sleep behavior (Owens, Spirito, & McGuinn, 2000). Ratings are made with respect to the previous week. The CSHQ data were assigned to their appropriate subscales, which included Bedtime Resistance (6 items), Sleep Onset Delay (1 item), Sleep Duration (3 items), Sleep Anxiety (4 items), Night Waking (3 items), Parasomnias (7 items), Sleep Disordered Breathing (3 items), and Daytime Sleepiness (8 items). The data were then compiled into the Total Sleep Disturbance Score, which is a composite value of all 33 items. In a community sample of school-aged children, the CSHQ demonstrated mediocre internal consistency (.68), and the variable subscale alphas ranged from .36 (substandard) for parasomnias to .70 (barely adequate) for bedtime resistance. In a clinical sample of school-aged children from the same study, the CSHQ performed better, with internal consistency (.78), and the variable subscale alphas ranged from .44 (mediocre) for night wakings to .93 (robust) for sleep disordered breathing. Although this measure has mixed psychometric characteristics, it appears to be comparable or better than other available sleep instruments (Spruyt & Gozal, 2011). The CSHQ is one of the most widely used assessments of sleep in children with autism (e.g., Sounders et al., 2009; Goldman et al., 2009), and given that sleep is an important variable of interest, it warranted being included. A copy of the measure’s scoring algorithm is available in Appendix D.
Height, Weight, and BMI

A physician or nurse practitioner conducted a physical and neurological assessment on each child during his or her initial visit. Information taken from this evaluation included the following: (a) height (in inches and centimeters, as the data-capture system accepted both standard and metric), (b) weight (in pounds and kilograms), (c) age at time of assessment, and (d) sex. These variables were used as part of an Excel macro from the CDC to calculate child body mass index (BMI) and BMI percentile for later analyses (Children’s BMI Group Calculator – Metric Version, 2010).

Nutritional Problems

Nutritional problems were assessed on a physician-completed form called “Diagnosis and Problems.” This form was designed to assess everything from minor and transitory complaints (e.g., headaches) to severe (e.g., neurofibromitosis) physical problems as well as mental health issues (e.g., depression). The questions that assessed nutritional difficulties included the following: feeding difficulties, pica, food intolerance, eating disorder not otherwise specified, other nutritional problems, or changes to feeding or nutritional practices in the last week to three months (see Appendix E).

Medication

Medications were listed by name, and parents endorsed whether or not the child was taking a given medication and for what purpose. This was simply a binary option where each medication listed was recorded in a Yes/No fashion. An “Other” category was also available if an agent was used but not listed in the array of choices given. The name of the substance was supposed to be recorded, although this information was not
always provided. Medications were recoded into their drug classes for analysis. The categories used were (a) stimulants, (b) atomoxetine, (c) selective serotonin reuptake inhibitors (SSRIs), (d) anticonvulsants, (e) antihistamines, (f) melatonin, (g) atypical antipsychotics, (h) alpha 2 agonists, and (i) other. If the information from the “other” drug category included the name of the medication, I investigated the drug class and decided if it truly belonged in the “other” category or if it would not be more correct to move it to a previously defined category. If the drug name was not included or was not previously represented, it stayed in the new “other” category. Dosage was not analyzed.

Diagnosis

A diagnosis form was completed by study personnel that specified what type of ASD was identified, as well as any other comorbid conditions. The ASD diagnosis was based on the ADOS (Lord et al., 2000), a semi-structured, play-based child observation designed to elicit autistic behaviors, as well as DSM-IV-TR diagnostic criteria (APA et al., 2004). Based on the available information, the interdisciplinary team assigned a diagnosis for the child (if applicable). Should the child be evaluated and found not to have an ASD, the child’s data were excluded from the database. Those diagnoses were recorded as autistic disorder (1), Asperger disorder (2), and PDD-NOS (3) to approximate severity of diagnosis from lowest level of functioning to better functioning.

Demographics

Information about birth date, sex, ethnicity, and parental education levels were recorded on the ATN’s Demographics form.
Participants

The study participants were 2,610 children and adolescents with ASD who were seen at one of the 17 Autism Treatment Network (ATN) locations in the United States and Canada. Table 1 lists all 17 ATN sites and it indicates how many subjects came from each site. Table 2 lists demographic characteristics of the total sample.

The subjects ranged in age from 22 to 217 months, or just under 2 years of age to 18 years of age. The average age was 76.15 months, or 6.25 years, with a standard deviation

<table>
<thead>
<tr>
<th>Name</th>
<th>Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arkansas Children’s Hospital</td>
<td>149</td>
</tr>
<tr>
<td>Baylor College of Medicine</td>
<td>103</td>
</tr>
<tr>
<td>Children’s Hospital of Philadelphia</td>
<td>34</td>
</tr>
<tr>
<td>Cincinnati Children’s Hospital</td>
<td>363</td>
</tr>
<tr>
<td>Columbia University Medical Center</td>
<td>67</td>
</tr>
<tr>
<td>Kaiser Permanente San Jose ASD Center</td>
<td>161</td>
</tr>
<tr>
<td>Kennedy Krieger Institute</td>
<td>96</td>
</tr>
<tr>
<td>Mass General Hospital</td>
<td>104</td>
</tr>
<tr>
<td>Nationwide Children’s Hospital</td>
<td>43</td>
</tr>
<tr>
<td>Oregon Health and Science University</td>
<td>217</td>
</tr>
<tr>
<td>Toronto ATN Site*</td>
<td>199</td>
</tr>
<tr>
<td>University of Alberta</td>
<td>5</td>
</tr>
<tr>
<td>University of Colorado, Denver</td>
<td>194</td>
</tr>
<tr>
<td>University of Missouri</td>
<td>300</td>
</tr>
<tr>
<td>University of Pittsburgh Medical Center</td>
<td>272</td>
</tr>
<tr>
<td>University of Rochester</td>
<td>246</td>
</tr>
<tr>
<td>University of Washington</td>
<td>2</td>
</tr>
<tr>
<td>Vanderbilt University Medical Center</td>
<td>55</td>
</tr>
</tbody>
</table>

* Toronto ATN Site included Holland Bloorview Kids Rehab, Surrey Place Centre, and Hospital for Sick Children
of 40.06 months (3.34 years). There were 2,204 (84.4%) males and 406 (15.6%) females in the sample. Ethnically, 1,963 (75.2%) children were Caucasian, 221 (8.5%) were African American, 188 (7.2%) were Hispanic, 158 (6.1%) were of Asian descent, and 80 (3.1%) were listed as Other if they did not fall into one of the aforementioned categories. Specific ASD subtypes included 1,685 (64.6%) participants with Autistic Disorder, 667 (25.6%) with PDD-NOS, and 258 (9.9%) with Asperger Disorder. A plurality of mothers of the children in the sample had completed some college ($n = 817, 31.3$%), while fathers were most likely to have earned bachelor’s degrees ($n = 644, 24.7$%). Additional information about parental education levels can be found in Table 2.

Table 3 presents the frequency of medication use, which was taken by 1,123 (43%) subjects in the sample. Medication use ranged from only a single drug per subject ($n = 605, 23.2$%) up to as many as seven medications per individual ($n = 2, 0.1$%). The catch-all category of Other was used most frequently ($n = 663, 24.3$%) and included an array of agents such as aspirin, complementary and alternative medicines (CAM treatments), diabetes treatments, and any drugs that were not specifically listed on the medication sheet. Although there was a space where investigators could identify the medication taken, this was not always used. This prevented more refined identification for a subset of individuals who reported using additional medications, but the actual substance was not recorded. Table 4 shows the mean subscale scores for the CBCL and CSHQ, the mean full-scale IQ score, and the mean Adaptive Behavior Composite score for the Vineland-II.
Table 2. Demographic Information for the Autism Treatment Network Sample

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>N</strong></td>
<td><strong>%</strong></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>2,204</td>
<td>84.4</td>
</tr>
<tr>
<td>Females</td>
<td>406</td>
<td>15.6</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>1,963</td>
<td>75.2</td>
</tr>
<tr>
<td>African American</td>
<td>221</td>
<td>8.4</td>
</tr>
<tr>
<td>Hispanic</td>
<td>188</td>
<td>7.2</td>
</tr>
<tr>
<td>Asian American</td>
<td>158</td>
<td>6.1</td>
</tr>
<tr>
<td>Other</td>
<td>80</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>DSM-IV Diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autistic Disorder</td>
<td>1,685</td>
<td>64.6</td>
</tr>
<tr>
<td>PDD-NOS</td>
<td>667</td>
<td>25.5</td>
</tr>
<tr>
<td>Asperger Syndrome</td>
<td>258</td>
<td>9.9</td>
</tr>
<tr>
<td><strong>Mother Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 8th Grade</td>
<td>18</td>
<td>.7</td>
</tr>
<tr>
<td>Some high school</td>
<td>73</td>
<td>2.8</td>
</tr>
<tr>
<td>High school diploma</td>
<td>413</td>
<td>15.8</td>
</tr>
<tr>
<td>Some college</td>
<td>817</td>
<td>31.3</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>720</td>
<td>27.6</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>421</td>
<td>16.1</td>
</tr>
<tr>
<td>Absent or no info</td>
<td>148</td>
<td>5.7</td>
</tr>
<tr>
<td><strong>Father Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 8th Grade</td>
<td>19</td>
<td>.7</td>
</tr>
<tr>
<td>Some high school</td>
<td>91</td>
<td>3.5</td>
</tr>
<tr>
<td>High school diploma</td>
<td>512</td>
<td>19.6</td>
</tr>
<tr>
<td>Some college</td>
<td>592</td>
<td>22.7</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>644</td>
<td>24.7</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>432</td>
<td>16.5</td>
</tr>
<tr>
<td>Absent or no info</td>
<td>320</td>
<td>12.3</td>
</tr>
<tr>
<td></td>
<td><strong>Mean</strong></td>
<td><strong>SD</strong></td>
</tr>
<tr>
<td>Age in months</td>
<td>76.15</td>
<td>40.06</td>
</tr>
</tbody>
</table>

*Note.* PDD-NOS = Pervasive Developmental Disorder – Not Otherwise Specified
Other (in Ethnicity) included people who identified as something different than Caucasian, African American, Hispanic, or Asian American.
Table 3. Medication Use and Frequency (Alphabetical)

<table>
<thead>
<tr>
<th>Medication Class</th>
<th>N</th>
<th>% of total sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha 2 Agonists</td>
<td>156</td>
<td>6.0</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>227</td>
<td>8.7</td>
</tr>
<tr>
<td>Atomoxetine</td>
<td>49</td>
<td>1.9</td>
</tr>
<tr>
<td>Atypical Antipsychotics</td>
<td>150</td>
<td>5.7</td>
</tr>
<tr>
<td>Melatonin</td>
<td>263</td>
<td>10.1</td>
</tr>
<tr>
<td>Mood Stabilizers</td>
<td>62</td>
<td>2.4</td>
</tr>
<tr>
<td>Other</td>
<td>663</td>
<td>24.3</td>
</tr>
<tr>
<td>SSRIs</td>
<td>151</td>
<td>5.8</td>
</tr>
<tr>
<td>Stimulants</td>
<td>274</td>
<td>10.5</td>
</tr>
<tr>
<td>Using 0 Medications</td>
<td>1487</td>
<td>57.0</td>
</tr>
<tr>
<td>Using 1 Medication</td>
<td>605</td>
<td>23.2</td>
</tr>
<tr>
<td>Using 2 Medications</td>
<td>254</td>
<td>9.7</td>
</tr>
<tr>
<td>Using 3 Medications</td>
<td>153</td>
<td>5.9</td>
</tr>
<tr>
<td>Using 4 Medications</td>
<td>81</td>
<td>3.1</td>
</tr>
<tr>
<td>Using 5 Medications</td>
<td>24</td>
<td>0.9</td>
</tr>
<tr>
<td>Using 6 Medications</td>
<td>4</td>
<td>0.2</td>
</tr>
<tr>
<td>Using 7 Medications</td>
<td>2</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Note. SSRIs = Selective Serotonin Reuptake Inhibitors; In computing percent of total sample, the numerator was the number of children taking each drug and the denominator was the total sample.
Table 4. Scores on CBCL, CSHQ, IQ tests, and Vineland-II, $N = 2,610$

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CBCL ($T$ scores)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affective Problems</td>
<td>62.64</td>
<td>9.16</td>
<td>50-95</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td>61.19</td>
<td>10.39</td>
<td>50-100</td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>60.80</td>
<td>9.93</td>
<td>50-100</td>
</tr>
<tr>
<td>Anxious/Depressed</td>
<td>58.64</td>
<td>9.99</td>
<td>50-100</td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>62.03</td>
<td>8.43</td>
<td>50-83</td>
</tr>
<tr>
<td>Emotion Problems*</td>
<td>62.04</td>
<td>10.39</td>
<td>50-100</td>
</tr>
<tr>
<td>CD/ODD</td>
<td>60.03</td>
<td>9.18</td>
<td>50-91</td>
</tr>
<tr>
<td>Pervasive Development Problems*</td>
<td>73.10</td>
<td>9.22</td>
<td>50-98</td>
</tr>
<tr>
<td>Rule Breaking Behavior**</td>
<td>56.65</td>
<td>6.58</td>
<td>50-84</td>
</tr>
<tr>
<td>Social Problems**</td>
<td>64.64</td>
<td>8.71</td>
<td>50-98</td>
</tr>
<tr>
<td>Somatic Complaints</td>
<td>59.32</td>
<td>8.41</td>
<td>50-96</td>
</tr>
<tr>
<td>Thought Problems**</td>
<td>68.54</td>
<td>8.63</td>
<td>50-92</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>67.88</td>
<td>10.27</td>
<td>50-100</td>
</tr>
<tr>
<td><strong>CSHQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedtime Resistance</td>
<td>9.10</td>
<td>3.28</td>
<td>6-18</td>
</tr>
<tr>
<td>Sleep Onset Delay</td>
<td>1.73</td>
<td>0.79</td>
<td>1-3</td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>4.30</td>
<td>1.70</td>
<td>3-9</td>
</tr>
<tr>
<td>Sleep Anxiety</td>
<td>6.12</td>
<td>2.07</td>
<td>4-12</td>
</tr>
<tr>
<td>Night Waking</td>
<td>5.07</td>
<td>0.89</td>
<td>3-9</td>
</tr>
<tr>
<td>Parasomnias</td>
<td>9.57</td>
<td>2.12</td>
<td>7-19</td>
</tr>
<tr>
<td>Sleep Disordered Breathing</td>
<td>3.46</td>
<td>0.92</td>
<td>3-9</td>
</tr>
<tr>
<td>Daytime Sleepiness</td>
<td>13.42</td>
<td>2.62</td>
<td>8-23</td>
</tr>
<tr>
<td>Total Sleep Disturbance</td>
<td>49.69</td>
<td>8.03</td>
<td>36-83</td>
</tr>
<tr>
<td><strong>IQ Scores</strong></td>
<td>77.70</td>
<td>25.54</td>
<td>20-148</td>
</tr>
<tr>
<td><strong>Adaptive Behavior Composite Score</strong></td>
<td>71.69</td>
<td>13.89</td>
<td>20-150</td>
</tr>
</tbody>
</table>

*Obtained only in 1.5-5 version of CBCL ($n = 1,464$); **Obtained only in 6-18 version of CBCL ($n = 1,084$); the alternative version of the CBCL does not have a similar subscale. Clinical cut-off for these subscales are scores at or above 70.
Analyses

Cleaning and Compiling the Data

Analyses were conducted using IBM SPSS Statistics 19 and the SPSS companion AMOS (Analysis of MOment Structures), designed to analyze mean and covariance structures (IBM Corp, 2010). The first step was to make sure that each case was only used once. Although the data set started with 6,684 individual entries, this number included multiple entries from many subjects. The data were reduced due to multiple patient visits, and only data from the first recorded visit were retained. While this typically resulted in the data from Visit 1 being used, there were times when the IQ testing, for example, was not completed until Visit 2 in which case Visit 2 IQ data were used. Data preparation included the following steps: (a) preparing the outcome variable file by deleting duplicate cases, (b) cleaning separate data files by eliminating all subjects who were not included in the outcome variable file or other variables of interest, and (c) combining all files together by matching patient identification numbers. Not every subject had all measures of interest entered into the database, and they were eliminated so a complete data set would result.

There were substantial data missing from the CSHQ. A MCAR (Missing Completely At Random) test was run to determine whether the data were missing in a systematic way or by chance (Little, 1988). The statistic, $\chi^2 (2807, N = 2864) = 3668.71$, $p < .001$, suggested that the data were missing in a methodical fashion. This could be due to a variety of factors, such as respondents regularly ignoring questions that perhaps were sensitive to them, and could have skewed these analyses. I excluded subjects with
missing cases, resulting in the final sample size of 2,610. There were occasionally missing data for the CBCL scores (roughly 10 cases per subscale for the entire sample). Because they were missing at random and affected so few subjects, the subscale average was used.

Prevalence Rates

Prevalence rates for weight categories (underweight, normal weight, overweight, and obesity) were derived by first calculating the BMI score and its corresponding percentile based on CDC algorithms. Then I examined the frequency distributions for BMI percentiles. Pearson’s chi-squared tests were then run to compare the frequencies within demographic groups (sex, age, and ethnicity).

Factor Analysis of Nutritional Variables

Another question addressed whether the nutritional variables could be reduced down to a small number of latent variables. Were these nutritional variables connected in a substantive way, they could be used as a proxy of nutritional health. Exploratory factor analyses were run in SPSS AMOS using the Graphics interface. Exact graphic models can be found in Appendix F. An exploratory factor analysis with this program requires that only a single regression weight is fixed to 1 (as indicated by a 1 on the regression line) per given factor, forcing that variable alone to load onto the factor and allowing the remaining variables to have no such constraints (IBM Technote, 2007a). The data were analyzed using one- and two-factor models, with all ten nutritional deficit variables (see Appendix E). The graphical approach of the software requires more manipulation for these exploratory procedures than other software, but the intentional combination of a
single fixed factor and the remaining factors not being fixed made this factor analytic approach exploratory for these nutritional variables rather than confirmatory. Although this statistical software may not be as common as some other options for factor analysis, it was reported as one of several sound software packages in an authoritative review of exploratory factor analysis in developmental disability literature (Norris & Lecavalier, 2010) and used the well-known extraction parameter of maximum likelihood (Norris & Lecavalier, 2010; Browne & Cudeck, 1992).

**Structural Equation Modeling/Path Analysis**

The analysis to test the questions in this dissertation was originally conceptualized as a structural equation model that would highlight the direct dependencies among the variables included within the model. Structural equation models have latent (or unobserved) variables, such as factors derived from factor analysis. Because the factor analysis ultimately did not produce viable factors and there were no latent variables to include, as is necessitated by structural equation modeling, I tried to use a path analysis which does not require latent variables. This would still focus on the direct dependencies of the variables and would create regression weights to describe the relationships between the variables of interest and the outcome measure of BMI centile.

**Hierarchical Multiple Linear Regression**

Path analyses are generally considered to be extensions of multiple regression analyses because they combine a series of hypothesized relationships between variables (Lleras, 2005). Ultimately, there were not enough significant connections between the variables of interest to warrant the path analysis approach. Subsequently, a series of
multiple regressions, or the individual parts of the path analysis, was used. Specifically, a hierarchical multiple linear regression model was used to assess the individual contributions of several variables (e.g., the Vineland Adaptive Behavior Composite score, intelligence test score, CBCL symptoms and problem subscales, and medication use in predicting weight status as defined by BMI percentile). Hierarchical regression models have variables entered in a series of blocks, which allows one to see if each new group of variables adds variance to the prediction produced by the previous blocks. Blocks were initially determined by expectations based on past research and my correlation matrix. When the model based upon theory and prior expectations did not perform as planned, all possible variables were entered and then compared based on medication use, CBCL subscale, ASD subtype, and site. This deliberate testing of all viable models was an exercise to evaluate the data from every possible angle and leave no stone unturned.
CHAPTER 3: RESULTS

Prevalence Rates

As previously discussed, children are considered underweight if they are below the 5th percentile, overweight if they are above the 85th percentile, and obese if they are above the 95th percentile. Weight classification prevalence rates are presented in Table 5. For the entire sample of 2,610 subjects, 118 (4.5%) children were underweight, 859 (32.9%) were overweight, and 452 (17.3%) were obese based on weight and height. This means that 1,633 (62.6%) children in the sample had normal weight for their heights. When compared to typically developing children and adolescents (Ogden et al., 2006), neither the rate of overweight (32.9% of this ASD sample vs. 33.6% in the typically developing sample; $\chi^2[1, N = 2610] = .397, p = .264$) nor obesity (17.3% in this ASD sample vs. 17.1% in the typically developing sample; $\chi^2[1, N = 2610] = .049, p = .412$) in children with ASDs were significantly different. When these figures were contrasted with other children with ASD from a previous study (Curtin et al., 2005), there was also no difference in prevalence of overweight or obesity ($\chi^2[1, N = 2610] = .003, p = .956$).

I was also interested in exploring whether these children were statistically similar in their weight classifications when evaluated by different demographic categories. There was no difference in weight classifications between the 2,204 males and the 406 females in the sample, $\chi^2(3, N = 2610) = .949, p = .814$. I then split the data set by age using 5 years as the median approximation closest to a whole year. The differences in weight status
between the 1,170 younger children (60 months of age or younger) and the 1,440 older children (61 months of age or older) approached, but did not reach significance, as \( \chi^2(3, N = 2610) = 7.535, p = .056; \) the older group had slightly more subjects falling into the higher weight categories. Finally, the data were analyzed based on ethnic breakdown, and there was a statistical difference in weight classification given membership in ethnic categories, \( \chi^2(12, N = 2610) = 27.214, p = .007. \) On average, subjects of African American and Hispanic heritage were more likely to be overweight. Please refer to Table 5 for additional weight classification details.

*Factor Analysis of Nutrition Variables*

An exploratory factor analysis was run through SPSS AMOS using the ten

### Table 5. Prevalence Rates of Different Weight Classifications Presented for the Complete Sample, Sex, Age, and Ethnic Group

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Underweight(%)</th>
<th>Normal(%)</th>
<th>Overweight(%)</th>
<th>Obese(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Sample (2,610)</strong></td>
<td>118 (4.5)</td>
<td>1,633 (62.6)</td>
<td>859 (32.9)</td>
<td>452 (17.3)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
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</tr>
<tr>
<td>Males (2,204)</td>
<td>100 (4.5)</td>
<td>1,375 (62.4)</td>
<td>729 (33.1)</td>
<td>379 (17.2)</td>
</tr>
<tr>
<td>Females (406)</td>
<td>18 (4.4)</td>
<td>258 (63.5)</td>
<td>130 (32.0)</td>
<td>73 (18.0)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
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</tr>
<tr>
<td>Under 5 yrs (1,170)</td>
<td>46 (3.9)</td>
<td>765 (65.4)</td>
<td>359 (30.7)</td>
<td>188 (16.1)</td>
</tr>
<tr>
<td>Above 5 yrs (1,440)</td>
<td>72 (5.0)</td>
<td>868 (60.3)</td>
<td>500 (34.7)</td>
<td>264 (18.3)</td>
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<tr>
<td><strong>Ethnicity</strong></td>
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<tr>
<td>Caucasian (1963)</td>
<td>90 (4.6)</td>
<td>1,253 (63.8)</td>
<td>620 (31.6)</td>
<td>327 (16.6)</td>
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<tr>
<td>African-Amer. (221)</td>
<td>12 (5.4)</td>
<td>120 (54.3)</td>
<td>89 (40.3)</td>
<td>51 (23.1)</td>
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<tr>
<td>Hispanic (188)</td>
<td>4 (2.1)</td>
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<td>84 (44.6)</td>
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<td>Asian (158)</td>
<td>10 (6.3)</td>
<td>104 (65.8)</td>
<td>44 (27.8)</td>
<td>19 (12.0)</td>
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<tr>
<td>Other (80)</td>
<td>3 (3.8)</td>
<td>55 (68.8)</td>
<td>22 (27.5)</td>
<td>11 (13.7)</td>
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</tbody>
</table>
nutritional problem variables (see Appendix E) to determine whether these items were assessing a single underlying theme, two separate factors, or no latent variables.

Ostensibly, these variables appeared to be connected to an underlying construct. The data set did not have any other nutritional variables and this seemed like a valuable opportunity to evaluate nutritional constructs of interest. A correlation matrix of the nutritional variables can be found in Table 6. Possible factors were evaluated in multiple ways. The one-factor model using all ten variables was not a good fit. The comparative fit index (CFI) was 0.478, where greater than 0.90 is considered adequate fit and greater than 0.95 is considered good fit. The root mean square error of approximation (RMSEA) was 0.299, where values greater than 0.10 indicate unacceptable fit, 0.08 – 0.10 suggest mediocre fit, 0.05 – 0.08 indicate reasonable fit, and less than 0.05 suggests a close model.

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<td>8</td>
<td>66</td>
<td>70</td>
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</table>

*Note: Values rounded to three digits, decimals omitted. Variables are as follows: 1 = Feeding Difficulty, 2 = Pica, 3 = Other Nutritional Problem, 4 = Eating Disorder, 5 = Food Intolerance, 6 = Feeding Disorder, 7 = Feeding Different in Last Three Months, 8 = Feeding Different in Past Week, 9 = Other Nutritional Problem in Last Three Months, 10 = Other Nutritional Problem in Past Week*
fit. A two-factor version was modeled using the output from the one-factor model, where the negatively loading variables were moved to a new factor. This two-factor model was also not a good fit. The CFI for this model version was 0.478 and the RMSEA was 0.291. Given the failure to identify factors in the one- and two-factor models and the weak correlations between the nutritional variables, I abandoned the factor analysis and I continued the analyses without any nutritional variables.

*Multiple Linear Regression of Variables Associated with BMI Percentile*

I then undertook analyses with a multiple linear regression model to determine which variables were contributing to BMI percentile and the extent to which they were contributing. I used a hierarchical-type of model so that different variables could be entered into blocks to see whether they accounted for variance above and beyond the previous variables. A correlation matrix with all available variables in the dataset is available in Table 7. This table was included to better visualize the bivariate relationships between the individual variables. When looking at the table it is important to remember that the column labeled “1” represents BMI percentile, which was the outcome variable for these analyses. In addition, the correlations of each potential predictor with all other predictors are shown in the matrix. Looking across the table at how the variables were associated with one another helped to inform why some of the results were found.

Block 1 comprised demographic variables including age in months, sex, ethnicity, and parental education. This block provided a significant model fit showing that
Table 7. Correlation matrix with all variables

The following matrix using numbers to represent variables to conserve space. Below is the list of which number corresponds to what variable. In the matrix, values are rounded to three digits, with decimals and zeroes omitted. Anything less than three digits should be assumed to have leading zeroes. Thus, if a value says “17,” for example, it should be read as 0.017, while “8” should be read as 0.008.

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
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<td>1. BMI percentile</td>
<td>23. Sleep Anxiety</td>
</tr>
<tr>
<td>2. Age</td>
<td>24. Night Waking</td>
</tr>
<tr>
<td>3. Sex</td>
<td>25. Parasomnias</td>
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<td>5. African American</td>
<td>27. Daytime Sleepiness</td>
</tr>
<tr>
<td>6. Asian</td>
<td>28. Affective Problems</td>
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<td>8. Other Ethnicity</td>
<td>30. Anxiety Problems</td>
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<td>9. Mother’s education</td>
<td>31. Anxious/Depressed</td>
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<td>10. Father’s education</td>
<td>32. ADHD Problems</td>
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<td>11. stimulant</td>
<td>33. Emotional Problems</td>
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<td>12. antihistamine</td>
<td>34. Disruptive Behavior Disorders</td>
</tr>
<tr>
<td>13. melatonin</td>
<td>35. Rule Breaking Behavior</td>
</tr>
<tr>
<td>14. atomoxetine</td>
<td>36. Social Problems</td>
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<tr>
<td>15. SSRI’s</td>
<td>37. Somatic Problems</td>
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<tr>
<td>16. alpha 2 agonists</td>
<td>38. Thought Problems</td>
</tr>
<tr>
<td>17. anticonvulsants</td>
<td>39. Withdrawn/Depressed</td>
</tr>
<tr>
<td>18. atypical antipsychotics</td>
<td>40. IQ score</td>
</tr>
<tr>
<td>19. other drug</td>
<td>41. Adaptive Behavior Composite Score</td>
</tr>
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<td>20. Bedtime Resistance</td>
<td>42. ASD Subtype</td>
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<td>21. Sleep Onset Delay</td>
<td>(continued)</td>
</tr>
<tr>
<td>22. Sleep Duration</td>
<td></td>
</tr>
</tbody>
</table>

59
Table 7. Correlation matrix with all variables (continued)

|     | 1   | 2     | 3   | 4   | 5   | 6   | 7   | 8   | 9   | 10  | 11  | 12  | 13  | 14  | 15  | 16  | 17  | 18  | 19  | 20  | 21  |
|-----|-----|-------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| x1  | -   |       |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x2  | -1  |       |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x3  | 6   | -17   |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x4  | -26 | 92    | -24 |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x5  | 35  | -15   | 41  | -530|     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x6  | -50 | -92   | 7   | -443| -77 |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x7  | 76  | -56   | 3   | -486| -85 | 71  |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x8  | -36 | 5     | -20 | -308| -54 | -45 | -49 |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x9  | -3  | 53    | 5   | -41 | 96  | -50 | -90 |     |     |     |     |     |     |     |     |     |     |     |     |     |
| x10 | -36 | 25    | 23  | -128| -112| -31 | -95 | 641 |     |     |     |     |     |     |     |     |     |     |     |     |
| x11 | -75 | 311   | -39 | 77  | -32 | -61 | -27 | 17  | 28  | -21 |     |     |     |     |     |     |     |     |     |     |
| x12 | 47  | 19    | 11  | -14 | 31  | -17 | 12  | -8  | 28  | 11  | -10 |     |     |     |     |     |     |     |     |     |
| x13 | 52  | 122   | -6  | 17  | 8   | -37 | 1   | -7  | 29  | 2   | 36  | 774 |     |     |     |     |     |     |     |     |
| x14 | -55 | 165   | 3   | 20  | 19  | -15 | -28 | 9   | 2   | -5  | 26  | -33 | 1   |     |     |     |     |     |     |     |
| x15 | 44  | 304   | -29 | 81  | -40 | -56 | -37 | -5  | 43  | 60  | 168 | 44  | 104 | 63  |     |     |     |     |     |     |
| x16 | 33  | 165   | 0   | 9   | 5   | -30 | -7  | 22  | 3   | -29 | 168 | 13  | 83  | 37  | 119 |     |     |     |     |     |
| x17 | 35  | 187   | 5   | 42  | -31 | -41 | 12  | -14 | 10  | -20 | 80  | 1   | 190 | 86  | 86  | 83  |     |     |     |     |
| x18 | 25  | 194   | -19 | 68  | -4  | -56 | -56 | -5  | 3   | -15 | 121 | -1  | 248 | 63  | 180 | 115 | 150 |     |     |     |
| x19 | -3  | 61    | -14 | 24  | -3  | -11 | -28 | 4   | 61  | 48  | 52  | 234 | 311 | -1  | 44  | 15  | 98  | 69  |     |     |
| x21 | -16 | 23    | 31  | -35 | 23  | 3   | 14  | 26  | -64 | -92 | 47  | 9   | -14 | 39  | 17  | 32  | 5   | 2   | 22  | 292 |

(continued)
|     | 1   | 2   | 3   | 4   | 5   | 6   | 7   | 8   | 9   | 10  | 11  | 12  | 13  | 14  | 15  | 16  | 17  | 18  | 19  | 20  | 21  |
|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| x22 | -7  | 54  | 42  | -22 | 65  | -37 | -9  | 15  | -39 | -95 | 68  | 53  | 55  | 37  | 19  | 111 | 22  | 65  | 81  | 293 | 430 |
| x23 | 13  | -70 | 1   | -23 | -33 | 50  | 9   | 27  | -10 | -66 | -42 | 30  | 24  | 12  | -27 | 10  | 34  | 11  | 56  | 732 | 208 |
| x24 | 24  | -57 | -49 | -51 | 30  | -3  | 52  | 5   | -29 | -61 | -39 | 24  | 25  | -15 | -2  | 23  | -3  | 11  | 40  | 260 | 43  |
| x25 | 42  | -152| -14 | 18  | -6  | -64 | 8   | 42  | -102| -133| -17 | 77  | 73  | -16 | 16  | 57  | 22  | 16  | 93  | 275 | 175 |
| x26 | 83  | 7   | 4   | -54 | 51  | 2   | 24  | 13  | -67 | -75 | -37 | 58  | 69  | 7   | 7   | 45  | 36  | 33  | 67  | 165 | 77  |
| x27 | -1  | 63  | -63 | 1   | 12  | -43 | 14  | 22  | -40 | -83 | 39  | 49  | 74  | 20  | 21  | 64  | 26  | 52  | 84  | 187 | 211 |
| x28 | 21  | 102 | 47  | 48  | -17 | -72 | 10  | -8  | -50 | -80 | 82  | 38  | 62  | 15  | 77  | 113 | 49  | 109 | 58  | 244 | 309 |
| x29 | -12 | 35  | 1   | 6   | 1   | -17 | 5   | -2  | -15 | -14 | 39  | 7   | 24  | 22  | 19  | 26  | 27  | 58  | 37  | -5  | 40  |
| x30 | -36 | 21  | -3  | 7   | 4   | -15 | 6   | -12 | -1  | -20 | 52  | -15 | -17 | 8   | -11 | 20  | 4   | 24  | 1   | 7   | 1   |
| x31 | -34 | 251 | 12  | 50  | -19 | -52 | -21 | 10  | -27 | -48 | 109 | 41  | 46  | 23  | 161 | 55  | 35  | 80  | 22  | 120 | 139 |
| x32 | -9  | 164 | 31  | 40  | 32  | -85 | -39 | 24  | -93 | 115 | 216 | 23  | 69  | 86  | 77  | 119 | 40  | 158 | 51  | 103 | 139 |
| x33 | -47 | 115 | 65  | 91  | -4  | -94 | -66 | 21  | -72 | -100| 67  | 13  | 25  | 5   | 84  | 75  | 30  | 66  | 36  | 124 | 165 |
| x34 | 12  | 99  | 55  | 51  | 20  | -90 | -46 | 34  | -107| -114| 130 | 26  | 74  | 35  | 58  | 97  | 41  | 156 | 27  | 140 | 147 |
| x35 | 19  | -52 | 66  | -23 | 26  | -18 | -20 | 30  | -73 | -64 | 123 | 13  | 34  | -1  | 38  | 103 | 30  | 129 | 38  | 139 | 93  |
| x36 | 43  | 179 | 36  | 50  | -86 | -40 | 1   | 23  | -71 | -68 | 113 | 7   | 33  | 7   | 46  | 33  | 73  | 41  | -17 | 83  | 132 |
| x37 | 26  | 25  | 7   | 28  | -41 | -75 | -4  | 30  | -55 | -70 | 5   | 72  | 75  | -20 | 40  | 30  | 19  | 43  | 116 | 135 | 163 |
| x38 | 27  | 71  | 22  | 93  | -52 | -118| 14  | -26 | -33 | -14 | 72  | 32  | 75  | 56  | 105 | 128 | 79  | 166 | 53  | 170 | 262 |
| x39 | 40  | -228| 5   | -5  | -19 | -12 | 43  | -3  | -80 | -73 | -47 | -6  | -17 | -59 | -65 | -15 | -40 | -22 | 10  | 130 | 131 |
| x41 | -31 | -117| -19 | 57  | -53 | 4   | -47 | 8   | 125 | 118 | -7  | 10  | -53 | 1   | -14 | -82 | -107| -114| -52 | -74 | -124 |
| x42 | -70 | 85  | 53  | 84  | -43 | -73 | -29 | 2   | 45  | 36  | 70  | 60  | 25  | 56  | 20  | 7   | -18 | -11 | 2   | -68 | -80 |
Table 7. Correlation matrix with all variables (continued)

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demographic factors significantly predicted BMI percentile, where $F(8, 2601) = 3.855, p < .001$, although it accounted for less than 1% of variance in the sample (adjusted $R^2 = .009$). Significant negative predictors within Block 1 included being of Asian descent ($\beta = -.043, p = .032$) and father’s education ($\beta = -.051, p = .047$). The only positive predictor of BMI centile was Hispanic ethnicity ($\beta = .072, p < .001$). A statistical summary of the variables included in the hierarchical regression can be found in Table 8, while an overview of the model itself can be found in Table 9.

Block 2 comprised medications that were significantly correlated, either positively or negatively, with BMI centile, which included stimulants, antihistamines, melatonin, and SSRIs. This did not include medications that failed to have significant correlations with BMI percentile in this sample. While medications that were excluded could have had literature that supported their connection to weight, such as the case with atypical antipsychotics, the purpose of this particular analysis was to attempt to identify those variables that were associated with BMI percentile in this sample. The addition of these variables accounted for a significant but very small change in the $R^2 (.012, F[4, 2597] = 8.032, p < .001)$ and the overall model that included both Block 1 and Block 2 was significant, $F(12, 2597) = 5.275, p < .001$. The amount of variance accounted for (adjusted $R^2$) by these two Blocks was .019. The stimulant category was a significant negative predictor of BMI centile ($\beta = -.089, p < .001$) and SSRIs were a significant positive predictor of BMI centile ($\beta = .058, p = .005$). Block 3 consisted of CSHQ subscales. The only two subscales out of a possible eight that were significantly correlated with BMI percentile were Parasomnias and Sleep Disordered Breathing.
### Table 8. Summary of hierarchical regression analysis predicting BMI centile

<table>
<thead>
<tr>
<th>Block 1 - Demographics</th>
<th>$B$</th>
<th>SE $B$</th>
<th>$p$</th>
<th>$\beta$</th>
<th>Partial Correlation</th>
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<tbody>
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<td>Age (months)</td>
<td>.000</td>
<td>.015</td>
<td>.984</td>
<td>.000</td>
<td>.000$^1$</td>
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<td>.006</td>
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<td>3.338</td>
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<td>.125</td>
<td>.031</td>
<td>.030</td>
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<td><strong>.032</strong></td>
<td><strong>-.043</strong></td>
<td><strong>-.042</strong></td>
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<tr>
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<td><strong>2.329</strong></td>
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<td><strong>.072</strong></td>
<td><strong>.071</strong></td>
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<tr>
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<td>-.031</td>
<td>-.031</td>
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<td>.531</td>
<td>.109</td>
<td>.041</td>
<td>.031</td>
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<tr>
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<td><strong>.047</strong></td>
<td><strong>-.051</strong></td>
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<th>$p$</th>
<th>$\beta$</th>
<th>Partial Correlation</th>
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<td><strong>2.042</strong></td>
<td>&lt;.001</td>
<td><strong>-.089</strong></td>
<td><strong>-.085</strong></td>
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<td>.775</td>
<td>.009</td>
<td>.006</td>
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<td>Melatonin</td>
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<td>3.143</td>
<td>.193</td>
<td>.040</td>
<td>.026</td>
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<td><strong>SSRIs</strong></td>
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<td><strong>2.681</strong></td>
<td><strong>.005</strong></td>
<td><strong>.058</strong></td>
<td><strong>.056</strong></td>
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<table>
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<th>Block 3 – Sleep Problems</th>
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<td>Parasomnias</td>
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<td>.012</td>
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<td><strong>Sleep Disordered Breathing</strong></td>
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<td><strong>.684</strong></td>
<td><strong>.001</strong></td>
<td><strong>.069</strong></td>
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<table>
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<tr>
<th>Block 4 - Comorbid Psychopathology</th>
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<th>$p$</th>
<th>$\beta$</th>
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<td>.088</td>
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<td>-.034</td>
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<td>ADHD Problems</td>
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<td>.088</td>
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<td>-.028</td>
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<td>.080</td>
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<td>.012</td>
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<td>Withdrawn/Depressed</td>
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<td>.050</td>
<td>.915</td>
<td>-.002</td>
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$^1$ Age did not reach statistical significance, which on the surface is interesting since the categorical split between younger and older children in the prevalence rate analysis was trending towards significance. The prevalence analysis was nonparametric in nature, and was only classificatory. This regression analysis used age to predict BMI percentile above and beyond what was already taken into account from BMI percentile. The differences in results could also be due to the groups starting at different places along the weight continuum (younger children should be inherently lighter than older children given their physical development), thus possibly masking results that may be present. Alternatively, it is also possible that the data were non-linear in nature.
Table 9. Overview of hierarchical regression analysis predicting BMI centile

<table>
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<tr>
<th>Block</th>
<th>( R^2 )</th>
<th>Adjusted ( R^2 )</th>
<th>( \Delta R^2 )</th>
<th>Change ( F(p) )</th>
<th>Overall ( F(p) )</th>
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<td>.009</td>
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<td>3.855 (&lt;.001)</td>
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<td>.024</td>
<td>.019</td>
<td>.012</td>
<td>8.032 (&lt;.001)</td>
<td>5.275 (&lt;.001)</td>
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<td>Block 3</td>
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<td>.024</td>
<td>.005</td>
<td>7.292 (.001)</td>
<td>5.585 (&lt;.001)</td>
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<td>.025</td>
<td>.003</td>
<td>1.770 (.132)</td>
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<td>Block 5</td>
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<td>.026</td>
<td>.001</td>
<td>1.481 (.228)</td>
<td>4.418 (&lt;.001)</td>
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</table>

These variables also accounted for a significant change in \( R^2 (.005, F[2, 2595] = 7.292, p = .001) \), and the overall model that included Blocks 1, 2, and 3 was significant, \( F(14, 2595) = 5.585, p < .001 \). With this Block included, the adjusted \( R^2 \) indicated that the amount of variance accounted for was 2.4%. Only Sleep Disordered Breathing was a significant predictor of BMI centile (\( \beta = .069, p = .001 \)).

Block 4 included the comorbid psychological conditions as measured by the CBCL that were predicted to be related to weight status: Anxiety Problems, ADHD Problems, any DBD, and Depressed/Withdrawn. The change between the 3\(^{rd}\) and 4\(^{th}\) Block was not significant, \( F(4, 2591) = 1.770, p = .132 \), indicating that there was not a significant difference in the amount of variance predicted (adjusted \( R^2 = .025 \)), although the overall model was still significant, \( F(18, 2591) = 4.742, p < .001 \). None of the variables in Block 4 were significant predictors. Block 5 included IQ score and the Adaptive Behavior Composite Score. The change between Block 4 and Block 5 also was
not significant, $F(2, 2589) = 1.481, p = .228$, which again means that there was not a
difference in the amount of variance predicted. Neither of the variables in this block
were significant predictors. Although the model with all Blocks included was significant,
$F(20, 2589) = 4.418, p < .001$, the variables only accounted for 2.6% of the total
variance.

Associations Emerging from Atheoretical Analyses

The initial hierarchical multiple linear regression model accounted for only a very
small amount of variance, 2.6%, which was much less than hoped. It was possible that
the model was restricted by including only those variables that were previously supported
by literature or significantly correlated with the BMI centile outcome. In an effort to be as
thorough as possible, I made a strategic move to analyze the data from all viable
perspectives, even at the risk of Type I error, to ensure that every angle was evaluated. In
the first of these analyses, all potential variables (including the previously discounted
ASD subtype) were incorporated into the model. These results hereafter will be called
the “atheoretical empiricism” model, and can be seen in Table 10 (for the summary) and
Table 11 (for the overview). They will also be discussed below.

In this new model, the first block of variables was identical to the previous model.
That is, it included age in months, sex, ethnicity, and parental education. Being Hispanic
was positively associated with BMI centile ($\beta = .071, p < .001$), while those who were
Asian ($\beta = -.043, p = .030$) or had greater rates of paternal education ($\beta = -.051, p = .047$)
were significantly more likely to weigh less. This overall model was statistically
Table 10. Summary of atheoretical empiricism hierarchical regression analysis predicting BMI centile

<table>
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<th></th>
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<th>β</th>
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<td>.051</td>
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<td>3.982</td>
<td>.132</td>
<td>.031</td>
<td>.030</td>
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<td>Atypical Antipsychotics</td>
<td>2.297</td>
<td>2.825</td>
<td>.416</td>
<td>.018</td>
<td>.016</td>
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<tr>
<td>Other Medication</td>
<td>-1.051</td>
<td>1.284</td>
<td>.413</td>
<td>-.017</td>
<td>-.016</td>
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<tr>
<td><strong>Block 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sleep Problems</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedtime Resistance</td>
<td>.209</td>
<td>.286</td>
<td>.464</td>
<td>.023</td>
<td>.014</td>
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<tr>
<td>Sleep Onset Delay</td>
<td>-.516</td>
<td>.839</td>
<td>.539</td>
<td>-.014</td>
<td>-.012</td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>-.358</td>
<td>.412</td>
<td>.385</td>
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<tr>
<td>Sleep Anxiety</td>
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<td>.438</td>
<td>.600</td>
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<td>-.010</td>
</tr>
<tr>
<td>Night Waking</td>
<td>.180</td>
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<td>.796</td>
<td>.005</td>
<td>.005</td>
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<tr>
<td>Parasomnias</td>
<td>.298</td>
<td>.326</td>
<td>.362</td>
<td>.021</td>
<td>.018</td>
</tr>
<tr>
<td><strong>Sleep Dis. Breathing</strong></td>
<td>2.336</td>
<td>.688</td>
<td>.001</td>
<td>.071</td>
<td>.067</td>
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<tr>
<td>Daytime Sleepiness</td>
<td>-.234</td>
<td>.249</td>
<td>.346</td>
<td>-.020</td>
<td>-.019</td>
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</tbody>
</table>

(continued)
Table 10. Summary of atheoretical empiricism hierarchical regression analysis predicting BMI centile (continued)

| Block 4 | Comorbid Psychopathology |  |  |  |  |  |  |  |
|---------|--------------------------|---|---|---|---|---|---|
|         |                          | $B$ | $SE\ B$ | $p$ | $\beta$ | Partial Correlation |
|         | Affective Problems       | .060 | .109 | .580 | .018 | .011 |
|         | Aggressive Behavior      | **.285** | **.137** | **.038** | **.097** | **.041** |
|         | Anxiety Problems         | -.109 | .059 | .064 | -.036 | -.037 |
|         | Anxious/Depressed        | **-.315** | **.086** | **.001** | **-.096** | **-.072** |
|         | ADHD Problems            | -.127 | .093 | .173 | -.035 | -.027 |
|         | DBD                      | .124 | .140 | .374 | .038 | .018 |
|         | Somatic Complaints       | .032 | .088 | .715 | .009 | .007 |
|         | Withdrawn/Depressed      | **.141** | **.071** | **.048** | **.048** | **.039** |

| Block 5 | IQ Score                 | -.026 | .028 | .363 | -.022 | -.018 |
|         | Adaptive Behavior Composite | .012 | .051 | .820 | .005 | .004 |

| Block 6 | ASD Subtype              | **-.1.964** | **.730** | **.007** | **-.056** | **-.052** |

**Note.** ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers significant, $F(8, 2601) = 3.840, p < .001$, and the variance accounted for (adjusted $R^2$) was .009.

Block 2 included all medications (stimulants, antihistamines, melatonin, atomoxetine, SSRIs, alpha 2 agonists, anticonvulsants, atypical antipsychotics, and other medications), and both the change in variance accounted for (change in $R^2 = .019$, $F[9, 2592] = 5.668, p < .001$) and the overall model (adjusted $R^2 = .024$, $F[17, 2592] = 4.837, p < .001$) were significant. The SSRIs ($\beta = .054, p = .009$) and alpha 2 agonists ($\beta = .042,$
were both positive predictors of BMI centile. Stimulants (β = -.098, p < .001) and atomoxetine (β = -.061, p = .002) were negative predictors.

The variables added in Block 3 included all of the sleep variables. These were all of the CSHQ subscales and included Bedtime Resistance, Sleep Onset Delay, Sleep Duration, Sleep Anxiety, Night Waking, Parasomnias, Sleep Disordered Breathing, and Daytime Sleepiness. As in the first regression model, only Sleep Disordered Breathing was a significant predictor of BMI centile (β = .071, p = .001). The change in accounted variance (change in $R^2 = .006, F[8, 2584] = 2.164, p = .027$) was significant, as was the overall regression model with all three blocks ($F[25, 2584] = 3.993, p < .001$).

Block 4 included the CBCL comorbid psychopathology variables (Affective Problems, Anxiety Problems, Anxious/Depressed, Aggressive Behaviors, ADHD Problems, DBD, Somatic Complaints, and Withdrawn/Depressed).

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>$F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
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<td>.009</td>
<td>---</td>
<td>---</td>
<td>3.840 (&lt;.001)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.031</td>
<td>.024</td>
<td>.019</td>
<td>5.668 (&lt;.001)</td>
<td>4.837 (&lt;.001)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.037</td>
<td>.028</td>
<td>.006</td>
<td>2.164 (.027)</td>
<td>3.993 (&lt;.001)</td>
</tr>
<tr>
<td>Block 4</td>
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<td>.034</td>
<td>.009</td>
<td>3.006 (.002)</td>
<td>3.773 (&lt;.001)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.046</td>
<td>.033</td>
<td>.000</td>
<td>.414 (.661)</td>
<td>3.579 (&lt;.001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.049</td>
<td>.036</td>
<td>.003</td>
<td>7.966 (.005)</td>
<td>3.710 (&lt;.001)</td>
</tr>
</tbody>
</table>
Anxious/Depressed subscale was a significant negative predictor of BMI ($\beta = -0.067$, $p = 0.001$), while Withdrawn/Depressed ($\beta = 0.040$, $p = 0.039$) and Aggressive Behavior ($\beta = 0.097$, $p = 0.038$) were significant positive predictors. The change in the added variance (change in $R^2 = 0.009$, $F[7, 2577] = 3.006$, $p = 0.002$) and the overall model ($F[32, 2577] = 3.773$, $p < 0.001$) were both statistically significant, with the total variance accounted for (adjusted $R^2$) being 0.034.

Block 5 included IQ score and Adaptive Behavior Composite Score. Neither were significant predictors of BMI centile, and the change in variance accounted for by adding the block was not significant either (change in $R^2 = 0.000$, $F[2, 2575] = 0.414$, $p = 0.661$) leaving the total variance accounted for by all variables up to this point at 3.3%. The overall model was still significant ($F[34, 2575] = 3.579$, $p < 0.001$).

Block 6 added in the new variable of ASD subtype (which included autistic disorder, Asperger disorder, and PDD-NOS), which was a significant predictor of BMI centile ($\beta = -0.056$, $p = 0.007$). The change in variance predicted by adding in this factor was significant (change in $R^2 = 0.003$, $F[1, 2574] = 7.966$, $p = 0.005$), as was the overall model fit ($F[35, 2574] = 3.710$, $p < 0.001$). This relationship indicates that subjects with autistic disorder were more likely to be heavier than subjects with PDD-NOS. This may indicate that the increased severity associated with autistic disorder could be connected with higher BMI percentile. The inclusion of all the variables elevated the overall amount of variance accounted for from 2.6% in the previous model, to 3.6%.
Analyses with Confounding Medications Removed

Only a minimal amount of variance was accounted for after adding in all appropriate variables, which made me wonder if the variables were confounded with one another. Specifically, I was concerned about children who were taking more than one psychopharmaceutical agent whose side effects could counteract one another. For example, stimulants are associated with reducing appetite and therefore may lower body mass while atypical antipsychotics often increase body weight due to excessive appetite. Children who were taking both types of medications could be providing conflicting data. In an effort possibly to clarify findings, children who were on multiple medications were examined more in depth. If the child was taking either a stimulant or atomoxetine (both compounds associated with lower body mass) and an atypical antipsychotic, SSRI, alpha 2 agonist, anticonvulsant, or antihistamine, then they were dropped from the analysis. This reduced the data size by 146 children, for a sample of 2,464. The same exploratory multiple regression was then performed, and results for the summary and overview can be found in Tables 12 and 13.

Block 1, with the demographic variables previously mentioned, accounted for .8% of the variance (adjusted \( R^2 \)), which was statistically significant (\( F[8, 2455] = 3.559, p < .001 \)), although slightly less variance was predicted than in the previous model. There were some similarities in the variables that were significant, as Hispanic ethnicity (\( \beta = .067, p = .001 \)) and paternal education (\( \beta = -.058, p = .031 \)) were still associated with BMI centile. Being of Asian descent, which was significant in the previous model, was very close to significant in this version (\( \beta = -.040, p = .052 \)), while maternal education
Table 12. Summary of hierarchical regression analysis predicting BMI centile with confounding medications removed (n = 2,464)

<table>
<thead>
<tr>
<th>Block 1</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Partial Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Age (months)</td>
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<td>.016</td>
<td>.997</td>
<td>.000</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
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<td>.907</td>
<td>-.002</td>
<td>.002</td>
<td></td>
</tr>
<tr>
<td>African American</td>
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<td>2.202</td>
<td>.098</td>
<td>.034</td>
<td>.033</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>-4.944</td>
<td>2.546</td>
<td>.052</td>
<td>-.040</td>
<td>-.039</td>
<td></td>
</tr>
<tr>
<td><strong>Hispanic</strong></td>
<td><strong>7.830</strong></td>
<td><strong>2.370</strong></td>
<td><strong>.001</strong></td>
<td><strong>.067</strong></td>
<td><strong>.067</strong></td>
<td></td>
</tr>
<tr>
<td>Other Ethnicity</td>
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<td>3.550</td>
<td>.136</td>
<td>-.030</td>
<td>-.030</td>
<td></td>
</tr>
<tr>
<td><strong>Father Education</strong></td>
<td><strong>1.068</strong></td>
<td><strong>.545</strong></td>
<td><strong>.050</strong></td>
<td><strong>.052</strong></td>
<td><strong>.040</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Mother Education</strong></td>
<td><strong>-9.78</strong></td>
<td><strong>.453</strong></td>
<td><strong>.031</strong></td>
<td><strong>-0.58</strong></td>
<td><strong>-0.44</strong></td>
<td></td>
</tr>
</tbody>
</table>

| Block 2                          |          |          |          |          |          |                    |
| Medications                      |          |          |          |          |          |                    |
| **Stimulants**                   | **-7.809**| **2.684**| **.004** | **-0.61**| **-0.59**|                    |
| Antihistamines                   | 4.931    | 3.820    | .197     | .045     | .026     |                    |
| Melatonin                        | .320     | 3.939    | .935     | .003     | .002     |                    |
| **Atomoxetine**                  | **-13.340**| **5.574**| **.017** | **-0.48**| **-0.48**|                    |
| SSRIs                            | 4.838    | 3.278    | .140     | .031     | .030     |                    |
| **Alpha 2 Agonists**             | **6.277**| **3.144**| **.046** | **.041** | **.040** |                    |
| Anticonvulsants                  | 9.110    | 4.906    | .063     | .039     | .038     |                    |
| Atypical Antipsychotics          | 3.547    | 3.328    | .287     | .024     | .022     |                    |
| Other Medication                 | 1.000    | 1.317    | .448     | -.016    | -.015    |                    |

| Block 3                          |          |          |          |          |          |                    |
| Sleep Problems                   |          |          |          |          |          |                    |
| Bedtime Resistance               | .127     | .291     | .662     | .014     | .009     |                    |
| Sleep Onset Delay                | -.762    | .864     | .378     | -.020    | -.018    |                    |
| Sleep Duration                   | -.306    | .425     | .471     | -.017    | -.015    |                    |
| Sleep Anxiety                    | -.128    | .454     | .778     | -.009    | -.006    |                    |
| Night Waking                     | .258     | .708     | .716     | .008     | .007     |                    |
| Parasomnias                      | .351     | .337     | .297     | .024     | .021     |                    |
| **Sleep Dis. Breathing**         | **2.183**| **.702** | **.002** | **.067** | **.063** |                    |
| Daytime Sleepiness               | -.153    | .256     | .551     | -.013    | -.012    |                    |

(continued)
Table 12. Summary of hierarchical regression analysis predicting BMI centile with confounding medications removed (n = 2,464; continued)

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>p</th>
<th>β</th>
<th>Partial Correlation</th>
</tr>
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<tbody>
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<td><strong>Block 4</strong></td>
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<td></td>
<td></td>
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<td></td>
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<tr>
<td>Comorbid Psychopathology</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Affective Problems</td>
<td>.059</td>
<td>.112</td>
<td>.599</td>
<td>.018</td>
<td>.011</td>
</tr>
<tr>
<td><strong>Aggressive Behavior</strong></td>
<td><strong>.308</strong></td>
<td><strong>.140</strong></td>
<td><strong>.028</strong></td>
<td><strong>.105</strong></td>
<td><strong>.045</strong></td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>-.106</td>
<td>.061</td>
<td>.082</td>
<td>-.035</td>
<td>-.035</td>
</tr>
<tr>
<td><strong>Anxious/Depressed</strong></td>
<td><strong>-.306</strong></td>
<td><strong>.089</strong></td>
<td><strong>.001</strong></td>
<td><strong>-.093</strong></td>
<td><strong>-.070</strong></td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>-.107</td>
<td>.095</td>
<td>.264</td>
<td>-.029</td>
<td>-.023</td>
</tr>
<tr>
<td>DBD</td>
<td>-.157</td>
<td>.143</td>
<td>.272</td>
<td>-.047</td>
<td>.022</td>
</tr>
<tr>
<td>Somatic Complaints</td>
<td>.025</td>
<td>.090</td>
<td>.779</td>
<td>.007</td>
<td>.006</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>.110</td>
<td>.073</td>
<td>.133</td>
<td>.037</td>
<td>.030</td>
</tr>
<tr>
<td><strong>Block 5</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IQ Score</td>
<td>-.023</td>
<td>.029</td>
<td>.437</td>
<td>-.019</td>
<td>-.016</td>
</tr>
<tr>
<td>Adaptive Beh. Composite</td>
<td>-.004</td>
<td>.052</td>
<td>.933</td>
<td>-.002</td>
<td>-.002</td>
</tr>
<tr>
<td><strong>Block 6</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASD Subtype</td>
<td><strong>-1.945</strong></td>
<td><strong>.748</strong></td>
<td><strong>.009</strong></td>
<td><strong>-0.56</strong></td>
<td><strong>-0.53</strong></td>
</tr>
</tbody>
</table>

*Note. ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.*

became significantly associated with BMI centile \((\beta = .052, p = .050)\).

Block 2 had a change in \(R^2\) of .017 \((F[9, 2446] = 4.707, p < .001)\) and included all of the medications, albeit in their new, hopefully less, confounded form. As with the previous model, both stimulants \((\beta = -.061, p = .004)\) and atomoxetine \((\beta = -.048, p = .017)\) were negatively associated with BMI centile. In this block SSRIs appeared to undergo the greatest change, as they were significantly associated with BMI in the previous model, and now failed to do so \((\beta = .031, p = .140)\). Alpha 2 agonists, which
were previously non-significant, became significant and positively associated with BMI centile ($\beta = .041, p = .046$). The model with Blocks 1 and 2 was significant (adjusted $R^2 = .022, F[17, 2446] = 4.190, p < .001$).

Block 3, which included the CSHQ sleep variables, marginally failed to reach statistical significance in the amount of change added to the model (change in $R^2 = .006, F[8, 2438] = 1.933, p = .051$). As in the previous model, only Sleep Disordered Breathing was a significant predictor of BMI centile ($\beta = .067, p = .002$). The overall model still predicted a significant amount of variance (adjust $R^2 = .025, F[25, 2438] = 3.476, p < .001$).

Comorbid psychopathologies were again added into Block 4, and significantly accounted for a very small amount of variance change in the model (change in $R^2 = .008, F[4, 2444] = 2.514, p < .05$).

Table 13. Overview of hierarchical regression analysis predicting BMI centile with confounding medications removed (n = 2,464)

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>$Adjusted R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
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<td>.008</td>
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<td>---</td>
<td>3.559 ($&lt;.001$)</td>
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<tr>
<td>Block 2</td>
<td>.028</td>
<td>.022</td>
<td>.017</td>
<td>4.707 ($&lt;.001$)</td>
<td>4.190 ($&lt;.001$)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.034</td>
<td>.025</td>
<td>.006</td>
<td>1.933 (.051)</td>
<td>3.476 ($&lt;.001$)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.042</td>
<td>.029</td>
<td>.008</td>
<td>2.514 (.010)</td>
<td>3.256 ($&lt;.001$)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.043</td>
<td>.029</td>
<td>.000</td>
<td>.409 (.644)</td>
<td>3.092 ($&lt;.001$)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.045</td>
<td>.031</td>
<td>.003</td>
<td>6.761 (.009)</td>
<td>3.201 ($&lt;.001$)</td>
</tr>
</tbody>
</table>
As in the previous model, the CBCL Anxious/Depressed subscale was a significant negative predictor of BMI centile ($\beta = -.085, p = .002$) while Aggressive Behavior maintained a positive association with the outcome variable ($\beta = .105, p = .028$). The Withdrawn/Depressed subscale was no longer significant in this model version ($\beta = .039, p = .116$). The overall amount of variance accounted for was still significant (adjusted $R^2 = .029, F[32, 2431] = 3.256, p < .001$).

Block 5 added in the IQ score as well as the Adaptive Behavior Composite Score and did not significantly change the amount of variance accounted for (change in $R^2 = .000, F[2, 2429] = .409, p = .644$), although the overall model still was significant (adjusted $R^2 = .027, F[34, 2429] = 3.092, p < .001$). As with the previous model, neither variable (IQ score $\beta = -.019, p = .437$; Adaptive Behavior Composite Score $\beta = -.002, p = .933$) was significantly associated with BMI centile.

Finally, Block 6 was added into the model to investigate ASD subtype. It remained a significant predictor in the model ($\beta = -.056, p = .009$). The amount of change in the variance from adding this predictor was significant (change in $R^2 = .003, F[1, 2428] = 6.761, p = .009$), as was the overall model (adjusted $R^2 = .030, F[35, 2428] = 3.201, p < .001$). The amount of variance accounted for by this model without confounding drugs was 3.0%, which is even less than the 3.3% of variance accounted for in the previous model with all available variables included.
Analyses Conducted in Subjects With One Medication or Less

The disappointing performance of the model without confounding medications made me wonder if perhaps I was not being restrictive enough. Although I tried to eliminate cases who took medications that may be interacting with one another, it is possible they still were doing so because children could be on more than one medication as long as they had the same weight-altering effects. For instance, a child could be taking both an SSRI and an atypical antipsychotic, which might be cancelling any existing signal. To try to clarify this issue further, I restricted this analysis to children who were only taking a single medication or who were not currently taking medications. To see a summary of the hierarchical regression using this sample ($n = 2094$) please refer to Table 14 and for an overview of the results, Table 15. As this analysis did not reveal anything new, I have not summarized it in narrative form. Suffice it to say here that essentially the same variables emerged as significant. Attempts to simplify the medication relationships did not prove productive. Indeed, the amount of variance predicted, 1.8%, became smaller.
Table 14. Summary of hierarchical regression analysis predicting BMI centile with one medication or less (n = 2,094)

<table>
<thead>
<tr>
<th>Block 1</th>
<th>Demographics</th>
<th></th>
<th></th>
<th></th>
<th>Partial</th>
<th>Correlation</th>
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<tbody>
<tr>
<td>B</td>
<td>SE B</td>
<td>p</td>
<td>β</td>
<td>Correlation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (months)</td>
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<td>.018</td>
<td>.582</td>
<td>-.012</td>
<td>.012</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
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<td>-.005</td>
<td>-.005</td>
<td></td>
</tr>
<tr>
<td>African American</td>
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<td>2.399</td>
<td>.215</td>
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<td>.027</td>
<td></td>
</tr>
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<td>-.049</td>
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<tr>
<td>Hispanic</td>
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<td>.056</td>
<td>.055</td>
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(continued)
Table 14. Summary of hierarchical regression analysis predicting BMI centile with one medication or less (n = 2,094; continued)

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<td>Withdrawn/Depressed</td>
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*Note.* ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.
Table 15. Overview of hierarchical regression analysis predicting BMI centile with one medication or less (n = 2,094)

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<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
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<td>.008</td>
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<td>3.060 (.002)</td>
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<td>Block 2</td>
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<td>.011</td>
<td>.007</td>
<td>1.896 (.057)</td>
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<tr>
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<td></td>
<td></td>
<td>1.896 (.057)</td>
<td>2.483 (.001)</td>
</tr>
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<td>Block 3</td>
<td>.024</td>
<td>.012</td>
<td>.005</td>
<td>1.314 (.232)</td>
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<td>.016</td>
<td>.008</td>
<td>2.001 (.043)</td>
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<td></td>
<td></td>
<td></td>
<td>2.078 (&lt;.001)</td>
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<tr>
<td>Block 5</td>
<td>.031</td>
<td>.015</td>
<td>.000</td>
<td>1.12 (.894)</td>
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<td></td>
<td></td>
<td>1.961 (.001)</td>
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<td>.018</td>
<td>.003</td>
<td>6.283 (.012)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>2.089 (&lt;.001)</td>
</tr>
</tbody>
</table>

Other Investigations

Although the analyses performed did elicit statistically significant results, the amount of variance accounted for across the various models was clearly disappointing, despite the fact that the data were deliberately over-analyzed with likelihood that I was violating Type I error. This prompted further investigation to ascertain that mistakes were not made or data problems were not overlooked. The first step was to double-check the data with a specific focus on the BMI centile outcome variable. As previously discussed, this figure was computed using the CDC’s algorithm by imputing date of birth, date of visit, weight, height, and sex. There was room for error because of the computational aspect, and I recalculated these aspects of the dataset and found them to be correct. This ensures that the primary outcome (BMI percentile) was accurately computed.
Next, the data were examined for multicollinearity, which occurs when variables in a multiple regression are highly intercorrelated, and therefore interfere with the model’s ability to determine the individual contribution of variables to the outcome variance. This was examined using the variance inflation factor (VIF), where a score greater than 5 indicates that there is likely multicollinearity and a score greater than 10 signifies definite multicollinearity. All scores for this data set were less than 2, with the exception of melatonin and alpha 2 agonists, which had a VIF of 3.2. This suggests that multicollinearity was not an issue.

**Transformations**

Although skewness and kurtosis were not a problem for the vast majority of variables, many of the distributions were still considered abnormal (with a slight, nonsignificant positive skew) after visual scans. To mitigate effects of this abnormal distribution, two different transformations that could address positive skew were performed on the few variables that were most distorted (ADHD Problems, Daytime Sleepiness, and BMI centile). The first conversion was a square root transformation. This transformation did not account for more variance than the atheoretical empiricism model (both had an adjusted $R^2$ of .033). A full table of complete results can be found in Appendix G. Next, a Log10 transformation was performed on the same variables mentioned previously. Not only did this not improve the amount of variance for which the model could account, but it reduced it. The final amount of variance was 2.6%, as compared to 3.3% from the atheoretical empiricism model. Again, this analysis can be found in Appendix G, with full results.
CBCL Version

Age may not have been a significant predictor in the previously discussed models, but I thought it could be possible that there was more predictive information in the CBCL than was evident in the analyses. This was because, in the model originally tested, the CBCL subscales were limited to only those common to the 1.5-5 year form and the 6-18 year form. I could not include Emotional Symptoms, Pervasive Developmental Problems, Rule-Breaking Behavior, Social Problems, and Thought Problems in any of the previous models because they were only evaluated on one version of the CBCL, rather than both. This could have prevented effects of potentially important subscales from showing up. Eventually, this led to an unintentional, but informative, outcome.

The first step was to divide the sample by age into the respective CBCL versions. Although this should have been straightforward, it was discovered that 38 families were incorrectly given the 1.5-5 year CBCL when their child was at least 6 years of age, while 24 families were incorrectly given the 6-16 year CBCL when their child was under 6 years of age. Collectively, these 38 plus 24 (62 total) reflect an error rate of 2.3%. These incorrectly administered assessments were not due to a single or few negligent sites, and rather were evenly distributed across the locations. These 62 CBCLs were not included in the following analyses.

For the younger 1.5-5 year CBCL group, there were a total of 1,464 subjects. This group’s model did not do a good job of accounting for variance (adjusted $R^2 = .026$, $F[38, 1423] = 2.027, p < .001$) although it was statistically significant. In this model, age ($\beta = .067, p = .011$), stimulants ($\beta = -.058, p = .033$), Withdrawn/Depressed ($\beta = .095, p = .056$),
.046), and ASD subtype ($\beta = -0.072, p = 0.010$) were all significant predictors of BMI. Anticonvulsants, which had not proved to be significant in any prior model, just missed significance in this iteration ($\beta = 0.053, p = 0.052$). Interestingly, other variables that were previously significant, such as ethnicity, paternal education, other medications (e.g., SSRIs), and sleep disturbances, failed to do so in this model. Please refer to Table 16 for a summary and Table 17 for an overview of this analysis.

There were 1,084 older children who were evaluated using the 6-18 year CBCL. This group did a better job of accounting for variance (adjusted $R^2 = 0.070, F[38, 1043] = 3.099, p < 0.001$) and was a statistically significant model. For these children, African American ethnicity ($\beta = 0.084, p = 0.007$), Hispanic ethnicity ($\beta = 0.101, p = 0.001$), stimulants ($\beta = -0.126, p < 0.001$), atomoxetine ($\beta = -0.094, p = 0.002$), alpha 2 agonists ($\beta = 0.064, p = 0.037$), and Sleep Disordered Breathing ($\beta = 0.112, p < 0.001$) were significant predictors of BMI centile. Additionally, so were Aggressive Problems ($\beta = 0.187, p = 0.018$), Anxious/Depressed ($\beta = -0.115, p = 0.009$), ADHD Problems ($\beta = -0.103, p = 0.011$), Somatic Complaints ($\beta = 0.082, p = 0.032$), IQ ($\beta = -0.089, p = 0.022$), Adaptive Behavior Composite Score ($\beta = 0.074, p = 0.047$), and ASD subtype ($\beta = -0.070, p = 0.030$). Although there were fewer children in this older CBCL category, it appears as though their scores in this were more highly correlated with BMI centile than the scores of the younger children. Please refer to Table 18 for a complete summary and Table 19 for an overview of this analysis.
Table 16. Summary of hierarchical regression analysis predicting BMI centile with only children with CBCL 1.5-5 year version (n = 1,464)

<table>
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<tr>
<th>Block</th>
<th>B</th>
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<th>β</th>
<th>Partial Correlation</th>
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(continued)
Table 16. Summary of hierarchical regression analysis predicting BMI centile with only children with CBCL 1.5-5 years version (n = 1,464; continued)

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<td></td>
<td>Somatic Complaints</td>
<td>-.139</td>
<td>.121</td>
<td>.254</td>
<td>-.039</td>
<td>-.030</td>
</tr>
<tr>
<td></td>
<td><strong>Withdrawn/Depressed</strong></td>
<td><strong>.287</strong></td>
<td><strong>.139</strong></td>
<td><strong>.040</strong></td>
<td><strong>.098</strong></td>
<td><strong>.054</strong></td>
</tr>
</tbody>
</table>

| Block 5 | IQ Score | -.002 | .039 | .960 | -.002 | -.001 |
|         | Adaptive Beh. Composite | -.082 | .063 | .198 | -.039 | -.034 |

| Block 6 | ASD Subtype | -2.474 | .953 | .010 | -.072 | -.069 |

*Note.* ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.
Table 17. Overview of hierarchical regression analysis predicting BMI centile with only children with CBCL 1.5-5 years version (n = 1,464)

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.012</td>
<td>.006</td>
<td>---</td>
<td>---</td>
<td>2.177 (.027)</td>
</tr>
<tr>
<td>2</td>
<td>.023</td>
<td>.012</td>
<td>.011</td>
<td>1.850 (.055)</td>
<td>2.009 (.009)</td>
</tr>
<tr>
<td>3</td>
<td>.028</td>
<td>.011</td>
<td>.005</td>
<td>.933 (.488)</td>
<td>1.664 (.021)</td>
</tr>
<tr>
<td>4</td>
<td>.046</td>
<td>.022</td>
<td>.017</td>
<td>2.592 (.004)</td>
<td>1.943 (.001)</td>
</tr>
<tr>
<td>5</td>
<td>.047</td>
<td>.022</td>
<td>.001</td>
<td>1.007 (.365)</td>
<td>1.892 (.001)</td>
</tr>
<tr>
<td>6</td>
<td>.051</td>
<td>.026</td>
<td>.004</td>
<td>6.735 (.010)</td>
<td>2.027 (&lt;.001)</td>
</tr>
</tbody>
</table>
Table 18. Summary of hierarchical regression analysis predicting BMI centile with only children with CBCL 6-18 years version (n = 1,084)

<table>
<thead>
<tr>
<th>Block 1</th>
<th>Demographics</th>
<th></th>
<th></th>
<th></th>
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</tr>
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<td>B</td>
<td>SE B</td>
<td>p</td>
<td>β</td>
<td>Partial Correlation</td>
<td></td>
</tr>
<tr>
<td>Age (months)</td>
<td>.034</td>
<td>.029</td>
<td>.229</td>
<td>.037</td>
<td>.037</td>
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</tr>
<tr>
<td>Sex</td>
<td>-1.234</td>
<td>2.657</td>
<td>.642</td>
<td>-.014</td>
<td>-.014</td>
<td></td>
</tr>
<tr>
<td><strong>African American</strong></td>
<td><strong>10.041</strong></td>
<td><strong>3.697</strong></td>
<td><strong>.007</strong></td>
<td><strong>.084</strong></td>
<td><strong>.083</strong></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>-7.811</td>
<td>5.052</td>
<td>.122</td>
<td>-.047</td>
<td>-.047</td>
<td></td>
</tr>
<tr>
<td><strong>Hispanic</strong></td>
<td><strong>13.108</strong></td>
<td><strong>3.972</strong></td>
<td><strong>.001</strong></td>
<td><strong>.101</strong></td>
<td><strong>.100</strong></td>
<td></td>
</tr>
<tr>
<td>Other Ethnicity</td>
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<td>.263</td>
<td>-.034</td>
<td>-.034</td>
<td></td>
</tr>
<tr>
<td>Mother Education</td>
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<td>.840</td>
<td>.440</td>
<td>.029</td>
<td>.024</td>
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<tr>
<td>Father Education</td>
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<td>.660</td>
<td>.336</td>
<td>-.036</td>
<td>-.029</td>
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<table>
<thead>
<tr>
<th>Block 2</th>
<th>Medications</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
<td>p</td>
<td>β</td>
<td>Partial Correlation</td>
<td></td>
</tr>
<tr>
<td><strong>Stimulants</strong></td>
<td><strong>-9.633</strong></td>
<td><strong>2.358</strong></td>
<td><strong>&lt;.001</strong></td>
<td><strong>-.126</strong></td>
<td><strong>-.124</strong></td>
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</tr>
<tr>
<td>Antihistamines</td>
<td>-1.099</td>
<td>5.247</td>
<td>.834</td>
<td>-.010</td>
<td>-.006</td>
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</tr>
<tr>
<td>Melatonin</td>
<td>4.726</td>
<td>4.988</td>
<td>.344</td>
<td>.052</td>
<td>.029</td>
<td></td>
</tr>
<tr>
<td><strong>Atomoxetine</strong></td>
<td><strong>-14.604</strong></td>
<td><strong>4.676</strong></td>
<td><strong>.002</strong></td>
<td><strong>-.094</strong></td>
<td><strong>-.096</strong></td>
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</tr>
<tr>
<td>SSRIs</td>
<td>5.213</td>
<td>2.990</td>
<td>.082</td>
<td>.054</td>
<td>.053</td>
<td></td>
</tr>
<tr>
<td><strong>Alpha 2 Agonists</strong></td>
<td><strong>6.571</strong></td>
<td><strong>3.148</strong></td>
<td><strong>.037</strong></td>
<td><strong>.064</strong></td>
<td><strong>.064</strong></td>
<td></td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>2.060</td>
<td>4.737</td>
<td>.664</td>
<td>.014</td>
<td>.013</td>
<td></td>
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<tr>
<td>Atypical Antipsychotics</td>
<td>2.831</td>
<td>3.430</td>
<td>.409</td>
<td>.028</td>
<td>.025</td>
<td></td>
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<tr>
<td>Other Medication</td>
<td>.665</td>
<td>2.156</td>
<td>.758</td>
<td>.010</td>
<td>.009</td>
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</table>

<table>
<thead>
<tr>
<th>Block 3</th>
<th>Sleep Problems</th>
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<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE B</td>
<td>p</td>
<td>β</td>
<td>Partial Correlation</td>
<td></td>
</tr>
<tr>
<td>Bedtime Resistance</td>
<td>.187</td>
<td>.479</td>
<td>.696</td>
<td>.018</td>
<td>.012</td>
<td></td>
</tr>
<tr>
<td>Sleep Onset Delay</td>
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<td>.430</td>
<td>-.027</td>
<td>-.024</td>
<td></td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>-.231</td>
<td>.658</td>
<td>.726</td>
<td>-.013</td>
<td>-.011</td>
<td></td>
</tr>
<tr>
<td>Sleep Anxiety</td>
<td>-.160</td>
<td>.671</td>
<td>.811</td>
<td>-.011</td>
<td>-.007</td>
<td></td>
</tr>
<tr>
<td>Night Waking</td>
<td>-1.107</td>
<td>1.237</td>
<td>.371</td>
<td>-.028</td>
<td>-.028</td>
<td></td>
</tr>
<tr>
<td>Parasomnias</td>
<td>.284</td>
<td>.550</td>
<td>.605</td>
<td>.017</td>
<td>.016</td>
<td></td>
</tr>
<tr>
<td><strong>Sleep Dis. Breathing</strong></td>
<td><strong>3.976</strong></td>
<td><strong>1.118</strong></td>
<td><strong>&lt;.001</strong></td>
<td><strong>.112</strong></td>
<td><strong>.109</strong></td>
<td></td>
</tr>
<tr>
<td>Daytime Sleepiness</td>
<td>.144</td>
<td>.386</td>
<td>.709</td>
<td>.012</td>
<td>.011</td>
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</table>

(continued)
Table 18. Summary of hierarchical regression analysis predicting BMI centile with only children with CBCL 6-18 years version (n = 1,084; continued)

<table>
<thead>
<tr>
<th></th>
<th>$B$</th>
<th>$SE$</th>
<th>$p$</th>
<th>$\beta$</th>
<th>Partial Correlation</th>
</tr>
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<tbody>
<tr>
<td><strong>Block 4</strong></td>
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<tr>
<td>Comorbid Psychopathology</td>
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<td></td>
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<td></td>
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<td>Affective Problems</td>
<td>.189</td>
<td>.197</td>
<td>.329</td>
<td>.052</td>
<td>.030</td>
</tr>
<tr>
<td><strong>Aggressive Behavior</strong></td>
<td>.611</td>
<td>.259</td>
<td>.018</td>
<td>.187</td>
<td>.073</td>
</tr>
<tr>
<td>Anxiety Problems</td>
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<td>.095</td>
<td>.711</td>
<td>-.011</td>
<td>-.011</td>
</tr>
<tr>
<td>Anxious/Depressed</td>
<td>-.363</td>
<td>.139</td>
<td>.009</td>
<td>-.115</td>
<td>-.081</td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>-.404</td>
<td>.159</td>
<td>.011</td>
<td>-.103</td>
<td>.078</td>
</tr>
<tr>
<td>DBD</td>
<td>.490</td>
<td>.286</td>
<td>.087</td>
<td>-.137</td>
<td>.053</td>
</tr>
<tr>
<td><strong>Somatic Complaints</strong></td>
<td>.305</td>
<td>.142</td>
<td>.032</td>
<td>.082</td>
<td>.066</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>.079</td>
<td>.135</td>
<td>.561</td>
<td>.023</td>
<td>.018</td>
</tr>
<tr>
<td>Rule Breaking Behavior</td>
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<td>.229</td>
<td>.815</td>
<td>.011</td>
<td>.007</td>
</tr>
<tr>
<td>Social Problems</td>
<td>.186</td>
<td>.149</td>
<td>.210</td>
<td>.052</td>
<td>.039</td>
</tr>
<tr>
<td>Thought Problems</td>
<td>-.018</td>
<td>.149</td>
<td>.904</td>
<td>.005</td>
<td>-.004</td>
</tr>
<tr>
<td><strong>Block 5</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IQ Score</td>
<td>-.104</td>
<td>.045</td>
<td>.022</td>
<td>-.089</td>
<td>-.071</td>
</tr>
<tr>
<td><strong>Adaptive Beh. Composite</strong></td>
<td>.174</td>
<td>.087</td>
<td>.047</td>
<td>.074</td>
<td>.061</td>
</tr>
<tr>
<td><strong>Block 6</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASD Subtype</td>
<td>-2.597</td>
<td>1.185</td>
<td>.029</td>
<td>-.070</td>
<td>-.068</td>
</tr>
</tbody>
</table>

*Note.* ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.
Table 19. Overview of hierarchical regression analysis predicting BMI centile with only children with CBCL 6-18 years version (n = 1,084)

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.025</td>
<td>.017</td>
<td>---</td>
<td>---</td>
<td>3.373 (.001)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.058</td>
<td>.043</td>
<td>.033</td>
<td>4.193 (.001)</td>
<td>3.850 (.001)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.073</td>
<td>.051</td>
<td>.015</td>
<td>2.119 (.030)</td>
<td>3.318 (.001)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.094</td>
<td>.063</td>
<td>.021</td>
<td>2.233 (.011)</td>
<td>3.016 (.001)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.100</td>
<td>.067</td>
<td>.006</td>
<td>3.288 (.038)</td>
<td>3.043 (.001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.104</td>
<td>.070</td>
<td>.004</td>
<td>4.801 (.029)</td>
<td>3.099 (.001)</td>
</tr>
</tbody>
</table>

**ASD Subtype**

ASD subtype was significant in many of the previous models, which could indicate that these distinctions, even though outlived with the release of the DSM-V, could have weight implications. The data set was split based on diagnostic categories, and Block 6, which previously included ASD subtype, was no longer included. There were 1,685 children with Autistic Disorder. When data from these children were included in the existing atheoretical empiricism model, $F(34, 1650) = 2.472, p < .001$. This is, of course, less than the amount of variance that was determined in the previous model version. An overview of the model details can be found in Table 20. There were 258 children with Asperger Disorder, and when their model was run it produced an adjusted $R^2$ of -.028. This
negative value indicates that the data were a poor fit for this existing model or the sample size was too small for the desired analysis (IBM Technote, 2007b; Peasnell, Pope, & Young, 2000; $F[34, 222] = .797, p = .786$).

When only children with PDD-NOS were included in the sample ($n = 667$) the model accounted for 5.1% of the variance, $F(34, 632) = 2.026, p < .001$. While this is more than what the atheoretical empiricism model determined, it was still not as high as desired. Out of curiosity, I decided to run the model again while excluding the children with Asperger disorder since the model did not perform well when confined to them. This combination of children with autistic disorder and PDD-NOS ($n = 2352$) increased the amount of variance accounted for (adjusted $R^2 = .038, F[34, 2317] = 3.634, p < .001$) over the atheoretical empiricism model, but it was less than the PDD-NOS model alone. Model details for all ASD subtypes can be found in Table 20.
Table 20. Overview of hierarchical regression analyses predicting BMI centile by ASD diagnosis

<table>
<thead>
<tr>
<th></th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autistic Disorder (n = 1,685)</strong></td>
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<td></td>
</tr>
<tr>
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<td>.007</td>
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<td>---</td>
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<td>.022</td>
<td>.020</td>
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<td>3.200 (&lt;.001)</td>
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<td>.029</td>
<td>.007</td>
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<tr>
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<td>.030</td>
<td>.001</td>
<td>1.242 (.289)</td>
<td>2.472 (&lt;.001)</td>
</tr>
<tr>
<td><strong>Asperger Disorder (n = 258)</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Block 1</td>
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<td>.006</td>
<td>---</td>
<td>---</td>
<td>1.190 (.306)</td>
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<tr>
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<td>.017</td>
<td>.479 (.888)</td>
<td>.803 (.689)</td>
</tr>
<tr>
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<td>-.039</td>
<td>.008</td>
<td>.262 (.977)</td>
<td>.616 (.925)</td>
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<tr>
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<td>-.026</td>
<td>.040</td>
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<td>.804 (.769)</td>
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<td>Block 5</td>
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<td>-.028</td>
<td>.006</td>
<td>.716 (.490)</td>
<td>.797 (.786)</td>
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<tr>
<td><strong>Pervasive Developmental Disorder-Not Otherwise Specified (n = 667)</strong></td>
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</tr>
<tr>
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<td>.026</td>
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<td>.053</td>
<td>.040</td>
<td>3.094 (&lt;.001)</td>
<td>3.188 (&lt;.001)</td>
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<td>.925 (.495)</td>
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<td>.051</td>
<td>.001</td>
<td>.178 (.837)</td>
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</tbody>
</table>

(continued)
Table 20. Overview of hierarchical regression analyses predicting BMI centile by ASD diagnosis (continued)

<table>
<thead>
<tr>
<th></th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
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<td>---</td>
<td>3.997 (&lt;.001)</td>
</tr>
<tr>
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<td>.023</td>
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<td>5.262 (&lt;.001)</td>
</tr>
<tr>
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<td>.007</td>
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<td>4.274 (&lt;.001)</td>
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<td>.008</td>
<td>2.430 (.013)</td>
<td>3.843 (&lt;.001)</td>
</tr>
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<td>Block 5</td>
<td>.052</td>
<td>.000</td>
<td>.240 (.787)</td>
<td>3.634 (&lt;.001)</td>
</tr>
</tbody>
</table>

Site Differences

The last area of exploration looked at whether there were site differences. Given that the ATN is a network of 17 academic, research, and medical institutions in the United States and Canada, it would be surprising if the associations in their data sets were identical to one another. In fact, previously published literature has shown regional differences in a variety of settings, even when procedural uniformity was assumed (Lord et al., 2012). For instance, Lord and colleagues (2012) evaluated data from 12 university-based autism providers (not associated with the ATN). In this study, all sites used identical gold standard instruments for diagnosis (in this case, the ADOS and Autism Diagnostic Interview-Revised; Lord, Rutter, & LeCouteur, 1994). The sites were not reliable in assigning similar diagnoses, likely due to inconsistent weighting of diagnostic
information, such as IQ or verbal ability. Thus, analysis of sites in this study was
designed to determine if differences were present between the sites. Should site
difference exist, it could help to address the lackluster amount of variance accounted for
in previous analyses performed. Site was added into the atheoretical empiricism model
as Block 7, and it did account for a small but significant amount of change (change in
\(R^2 = .001, F[1, 2574] = 3.932, p = .047\)), indicating that there were likely site differences.
The overall model with site as a predictor was also significant (adjusted \(R^2 = .036, F[36,
2573] = 3.691, p < .001\)). Only the 11 sites that had more than 100 participants each were
included in hopes that there would be enough power to uncover differences. Sites 1, 12
and 28 produced negative adjusted \(R^2\)'s. These negative values, while uncommon,
suggest that the model was either a poor fit or the sample size was still too small to detect
results (IBM Technote, 2007b; Peasnell et al., 2000). Site 1 had 217 participants, Site 12
had 300 subjects, and Site 28 had 199. These were larger samples than for other sites that
did fit the model well, which suggests that perhaps it was not the sample size that was an
issue. One site just had mediocre performance with the atheoretical empiricism model.
Site 4 \((n = 103)\) had an adjusted \(R^2\) of .017 \((F[34, 68] = 1.035, p = .441)\) which, not
surprisingly, failed to reach statistical significance. Please refer to Table 21 for an
overview of these analyses by site.

Quite a few sites appeared to perform slightly better than the atheoretical
empiricism model because of the amount of variance for which they accounted, but failed
to reach statistical significance. These included Site 24 \((n = 362, \text{adjusted } R^2 = .042,
F[36, 325] = 1.444, p = .053)\), Site 21 \((n = 149, \text{adjusted } R^2 = .058, F[34, 114] = 1.259, p\)
= .184), Site 22 (n = 194, adjusted $R^2 = .063, F[35, 158] = 1.357, p = .104$), and Site 26 (n = 161, adjusted $R^2 = .066, F[33, 127] = 1.335, p = .128$). Site 27 (n = 245), on the other hand, performed only somewhat better than the atheoretical empiricism model, but did reach significance. The adjusted $R^2$ was .067 ($F[36, 208] = 1.486, p = .047$).

Interestingly, two sites were able to account for much more variance than previous models. Site 2, which only had 106 subjects, accounted for more than 16.0% of model variance ($F[35, 70] = 1.557, p = .058$). Although this is a great deal more than the amount of variance accounted for in the atheoretical empiricism model, it was only trending rather than achieving significance outright. It was statistically significant when only three (adjusted $R^2 = .179, F[25, 80] = 1.916, p = .015$) or four blocks (adjusted $R^2 = .171, F[33, 72] = 1.655, p = .039$) were included in the model. Site 23 had 272 participants and analysis of its data accounted for the most variance. Site 23 was statistically significant using the full six-block atheoretical empiricism model. The data from this site were able to account for 13.5% of the outcome variance in the model ($F[35, 236] = 2.178, p < .001$), which is over four times the amount that was accounted for previously.

Correlations between the significant variables for these two sites and the remaining sample can be found in Table 22. This table enables us to examine the relationships between variables within each “type” of site. For instance, age was positively correlated with BMI percentile at Site 2 (.128) and negatively correlated with BMI percentile at Site 23 (-.174, significant at $p = .004$), with close to zero correlation between age and BMI percentile for the remaining sites. Throughout the table, Site 2
appeared to be different from Site 23 and the remaining samples. For instance, at Site 2, Sleep Onset Delay was positively related to BMI percentile (.122), while Site 23 (-.049) and the remaining samples (-.017) had negative correlations. At Site 2, Sleep Anxiety was negatively correlated with BMI percentile (-.121) while Site 23 and the remainder of the sample were correlated almost zero with BMI percentile. A similar relationship was found for the Withdrawn/Depressed CBCL subscale, where a positive correlation with BMI percentile (.171) was found for Site 2; the remaining sample and Site 23 were very close to zero correlation (Site 23: -.022, remaining sample: .040).

The average BMI centiles for Site 2, for Site 23, and for the remaining sample can be found in Table 23. Site 2 continued to perform differently from Site 23 and the remaining sample. For example, the males at Site 2 had an average BMI centile of 67.07%, which was higher than Site 23 (60.56%) and the remaining sample (63.66%). The females at Site 2 had lower average BMI percentile (55.58%) than at Site 23 (64.85%) or the remaining sample (64.33%). Similar patterns of Site 2 performing differently from Site 23 and the remaining sample can be seen for most medications (with the exception of SSRIs and anticonvulsants). This is especially true for the stimulant medications, where Sites 2 and 23 were significantly different ($p = .023$). The other variables that demonstrated significant differences between Sites 2 and 23 were Asian ethnicity ($p < .001$), Hispanic ethnicity ($p = .032$), and father’s education at the high school diploma level ($p = .029$). No other variables reached statistical significance between the sites, and BMI percentiles did not differ much for ASD subtypes.
I also looked at purely descriptive statistics for these two sites and the remaining sample, and these tables can be found in Appendix H. Chi-squared and MANOVA tests revealed the following differentiation of the groups: Site 2 had fewer parents endorse items on the Withdrawn/Depressed subscale, Site 23 had a different ethnic distribution and paternal education distribution than Site 2 or all other sites, and both Site 2 and 23 had fewer children with Autistic Disorder than the remaining sites. There were also differences related to medication use, which can be seen in Appendix H.

I wondered whether there could be age differences between these sites and the rest of the Autism Treatment Network, since sites with older samples would likely perform better than sites with younger children. This proved not to be the case for the mean ages, as Site 2 (average age of 80 months, 6.7 years) and Site 23 (average age of 81 months, 6.8 years) were very typical of all ATN sites, with an average age range of 61-93 months (mean of 76 months, 6.3 years). These ages were not significantly different, $F(2, 2985) = 1.888, p = .151$. On further exploration, we found that there was a difference in terms of age distribution between these two sites and the remainder of the sample ($\chi^2 [4, N = 2610] = 12.05, p = .017$). The remainder of the sample was used for these comparisons. The ages were split at less than 6 years of age, between 6 and 10 years of age, inclusive, and greater than 10 years of age. These results can be seen in Table 24. Site 2 had substantially more children in the “middle” age group than did the group of “all other” sites. Site 23, on the other hand, had a slightly higher proportion of the oldest children than did “all other” sites.
Next, I explored whether there were differences in the distribution of the ASD subtypes between these sites and the remainder of the sample. I compared the three ASD subtypes (autism, PDD-NOS, and Asperger disorder) against site, and significant differences were observed ($\chi^2 [4, N = 2610] = 36.18, p < .001$). Site 2 had more children diagnosed with PDD-NOS (57 children; 53.7%) than autistic disorder (43 children; 40.6%) or Asperger disorder (6 children; 5.7%). This was in contrast to Site 23 and the remaining sites, which had more children with autistic disorder than any other condition. The results for the ASD subtype breakdown can be found in Table 25.
Table 21. Overview of hierarchical regression analyses predicting BMI centile by ATN site

<table>
<thead>
<tr>
<th>Site 1, n = 217</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.048</td>
<td>.011</td>
<td>---</td>
<td>---</td>
<td>1.298 (.246)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.077</td>
<td>-.002</td>
<td>.029</td>
<td>1.263 (.265)</td>
<td>1.071 (.380)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.123</td>
<td>.008</td>
<td>.046</td>
<td>1.071 (.380)</td>
<td>1.071 (.380)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.142</td>
<td>-.013</td>
<td>.019</td>
<td>.917 (.520)</td>
<td>.917 (.520)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.144</td>
<td>-.022</td>
<td>.002</td>
<td>.869 (.651)</td>
<td>.869 (.651)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.166</td>
<td>-.002</td>
<td>.022</td>
<td>4.740 (.031)</td>
<td>.994 (.487)</td>
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</table>

<table>
<thead>
<tr>
<th>Site 2, n = 106</th>
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<tbody>
<tr>
<td>Block 1</td>
<td>.182</td>
<td>.114</td>
<td>---</td>
<td>---</td>
<td>2.690 (.010)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.231</td>
<td>.082</td>
<td>.049</td>
<td>.627 (.771)</td>
<td>1.554 (.095)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.375</td>
<td>.179</td>
<td>.144</td>
<td>2.297 (.029)</td>
<td>1.916 (.015)</td>
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<tr>
<td>Block 4</td>
<td>.431</td>
<td>.171</td>
<td>.057</td>
<td>.899 (.522)</td>
<td>1.655 (.039)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.440</td>
<td>.160</td>
<td>.009</td>
<td>.559 (.575)</td>
<td>1.573 (.054)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.448</td>
<td>.160</td>
<td>.008</td>
<td>.990 (.323)</td>
<td>1.557 (.058)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Site 4, n = 103</th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.095</td>
<td>.018</td>
<td>---</td>
<td>---</td>
<td>1.233 (.289)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.148</td>
<td>-.010</td>
<td>.053</td>
<td>.673 (.714)</td>
<td>.936 (.532)</td>
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<tr>
<td>Block 3</td>
<td>.255</td>
<td>.025</td>
<td>.106</td>
<td>1.391 (.214)</td>
<td>1.110 (.353)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.342</td>
<td>.042</td>
<td>.088</td>
<td>1.165 (.332)</td>
<td>1.138 (.320)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.353</td>
<td>.030</td>
<td>.011</td>
<td>.570 (.568)</td>
<td>1.092 (.372)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.354</td>
<td>.017</td>
<td>.001</td>
<td>.115 (.736)</td>
<td>1.050 (.423)</td>
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</tbody>
</table>

(continued)
Table 21. Overview of hierarchical regression analyses predicting BMI centile by ATN site (continued)

<table>
<thead>
<tr>
<th>Site 12, n = 300</th>
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<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Adjusted $R^2$</td>
<td>$\Delta R^2$</td>
<td>Change $F(p)$</td>
<td>Overall $F(p)$</td>
</tr>
<tr>
<td>Block 1</td>
<td>.011</td>
<td>.011</td>
<td>---</td>
<td>---</td>
<td>.421 (.908)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.040</td>
<td>-.017</td>
<td>.029</td>
<td>.947 (.484)</td>
<td>.699 (.803)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.073</td>
<td>-.012</td>
<td>.032</td>
<td>1.199 (.300)</td>
<td>.862 (.659)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.100</td>
<td>-.011</td>
<td>.027</td>
<td>1.016 (.424)</td>
<td>.899 (.630)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.113</td>
<td>-.005</td>
<td>.013</td>
<td>1.865 (.157)</td>
<td>.960 (.537)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.113</td>
<td>-.009</td>
<td>.000</td>
<td>.000 (.983)</td>
<td>.930 (.588)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Site 21, n = 149</th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Adjusted $R^2$</td>
<td>$\Delta R^2$</td>
<td>Change $F(p)$</td>
<td>Overall $F(p)$</td>
</tr>
<tr>
<td>Block 1</td>
<td>.048</td>
<td>-.006</td>
<td>---</td>
<td>---</td>
<td>.888 (.528)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.156</td>
<td>.053</td>
<td>.107</td>
<td>2.099 (.040)</td>
<td>1.522 (.101)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.209</td>
<td>.056</td>
<td>.053</td>
<td>1.040 (.410)</td>
<td>1.364 (.139)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.255</td>
<td>.049</td>
<td>.046</td>
<td>.893 (.525)</td>
<td>1.239 (.205)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.257</td>
<td>.036</td>
<td>.002</td>
<td>.181 (.834)</td>
<td>1.160 (.277)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.280</td>
<td>.058</td>
<td>.023</td>
<td>3.676 (.058)</td>
<td>1.259 (.184)</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Site 22, n = 194</th>
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<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Adjusted $R^2$</td>
<td>$\Delta R^2$</td>
<td>Change $F(p)$</td>
<td>Overall $F(p)$</td>
</tr>
<tr>
<td>Block 1</td>
<td>.034</td>
<td>-.008</td>
<td>---</td>
<td>---</td>
<td>.802 (.602)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.198</td>
<td>.010</td>
<td>.064</td>
<td>1.389 (.196)</td>
<td>1.120 (.338)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.125</td>
<td>-.005</td>
<td>.027</td>
<td>.657 (.728)</td>
<td>.960 (.523)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.232</td>
<td>.073</td>
<td>.107</td>
<td>2.778 (.007)</td>
<td>1.462 (.065)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.237</td>
<td>.068</td>
<td>.006</td>
<td>.572 (.565)</td>
<td>1.404 (.084)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.237</td>
<td>.063</td>
<td>.000</td>
<td>.031 (.859)</td>
<td>1.357 (.104)</td>
</tr>
</tbody>
</table>

(continued)
Table 21. Overview of hierarchical regression analyses predicting BMI centile by ATN site (continued)

<table>
<thead>
<tr>
<th></th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Site 23, n = 272</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Block 1</td>
<td>.082</td>
<td>.054</td>
<td>---</td>
<td>---</td>
<td>2.924 (.004)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.167</td>
<td>.111</td>
<td>.085</td>
<td>2.896 (.003)</td>
<td>2.999 &lt; .001</td>
</tr>
<tr>
<td>Block 3</td>
<td>.215</td>
<td>.135</td>
<td>.048</td>
<td>1.883 (.063)</td>
<td>2.698 &lt; .001</td>
</tr>
<tr>
<td>Block 4</td>
<td>.232</td>
<td>.126</td>
<td>.017</td>
<td>.651 (.734)</td>
<td>2.179 &lt; .001</td>
</tr>
<tr>
<td>Block 5</td>
<td>.240</td>
<td>.127</td>
<td>.008</td>
<td>1.180 (.309)</td>
<td>2.125 (.001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.250</td>
<td>.135</td>
<td>.011</td>
<td>3.295 (.071)</td>
<td>2.178 &lt; .001</td>
</tr>
</tbody>
</table>

| **Site 24, n = 362** |       |                |              |               |               |
| Block 1 | .028  | .005           | ---          | ---           | 1.249 (.269)  |
| Block 2 | .069  | .023           | .041         | 1.689 (.090)  | 1.492 (.095)  |
| Block 3 | .101  | .034           | .033         | 1.522 (.148)  | 1.514 (.057)  |
| Block 4 | .135  | .048           | .034         | 1.611 (.121)  | 1.554 (.030)  |
| Block 5 | .138  | .045           | .003         | .502 (.606)   | 1.490 (.041)  |
| Block 6 | .138  | .042           | .000         | .001 (.976)   | 1.444 (.053)  |

| **Site 26, n = 161** |       |                |              |               |               |
| Block 1 | .062  | .012           | ---          | ---           | 1.247 (.276)  |
| Block 2 | .115  | .023           | .053         | 1.241 (.284)  | 1.251 (.241)  |
| Block 3 | .172  | .033           | .057         | 1.188 (.311)  | 1.238 (.224)  |
| Block 4 | .246  | .065           | .074         | 1.579 (.137)  | 1.357 (.122)  |
| Block 5 | .255  | .061           | .009         | .784 (.459)   | 1.318 (.141)  |
| Block 6 | .265  | .066           | .010         | 1.676 (.198)  | 1.335 (.128)  |

(continued)
Table 21. Overview of hierarchical regression analyses predicting BMI centile by ATN site (continued)

<table>
<thead>
<tr>
<th></th>
<th>( R^2 )</th>
<th>( Adjusted R^2 )</th>
<th>( \Delta R^2 )</th>
<th>( Change F(p) )</th>
<th>( Overall F(p) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>( .041 )</td>
<td>( .009 )</td>
<td>---</td>
<td>---</td>
<td>( 1.267 (.261) )</td>
</tr>
<tr>
<td>Block 2</td>
<td>( .078 )</td>
<td>( .009 )</td>
<td>( .037 )</td>
<td>( 1.012 (.431) )</td>
<td>( 1.132 (.324) )</td>
</tr>
<tr>
<td>Block 3</td>
<td>( .132 )</td>
<td>( .033 )</td>
<td>( .054 )</td>
<td>( 1.695 (.101) )</td>
<td>( 1.331 (.142) )</td>
</tr>
<tr>
<td>Block 4</td>
<td>( .177 )</td>
<td>( .049 )</td>
<td>( .045 )</td>
<td>( 1.458 (.174) )</td>
<td>( 1.379 (.093) )</td>
</tr>
<tr>
<td>Block 5</td>
<td>( .190 )</td>
<td>( .054 )</td>
<td>( .013 )</td>
<td>( 1.627 (.199) )</td>
<td>( 1.401 (.079) )</td>
</tr>
<tr>
<td>Block 6</td>
<td>( .205 )</td>
<td>( .067 )</td>
<td>( .015 )</td>
<td>( 3.802 (.053) )</td>
<td>( \textbf{1.486 (.047)} )</td>
</tr>
</tbody>
</table>

Site 27, \( n = 245 \)

| Block 1 | \( .019 \) | \( -.022 \) | --- | --- | \( .456 (.886) \) |
| Block 2 | \( .043 \) | \( -.047 \) | \( .024 \) | \( .510 (.866) \) | \( .479 (.960) \) |
| Block 3 | \( .082 \) | \( -.050 \) | \( .039 \) | \( .926 (.497) \) | \( .621 (.920) \) |
| Block 4 | \( .135 \) | \( -.038 \) | \( .053 \) | \( 1.255 (.270) \) | \( .780 (.797) \) |
| Block 5 | \( .140 \) | \( -.045 \) | \( .005 \) | \( .445 (.670) \) | \( .756 (.834) \) |
| Block 6 | \( .163 \) | \( -.024 \) | \( .023 \) | \( \textbf{4.423 (.037)} \) | \( .874 (.675) \) |
Table 22. Pearson correlations with BMI centiles associated with variables predictive of for the sites (2, 23) that demonstrated good model fit and the remaining sample

<table>
<thead>
<tr>
<th>Variable</th>
<th>Site 2 (n = 106)</th>
<th>Site 23 (n = 272)</th>
<th>All Others (n = 2232)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months)</td>
<td>.128</td>
<td>-.174**</td>
<td>.019</td>
</tr>
<tr>
<td>Sleep Onset Delay</td>
<td>.111</td>
<td>-.049</td>
<td>-.017</td>
</tr>
<tr>
<td>Sleep Anxiety</td>
<td>-.129</td>
<td>.011</td>
<td>.019</td>
</tr>
<tr>
<td>Parasomnias</td>
<td>.128</td>
<td>-.038</td>
<td>.048*</td>
</tr>
<tr>
<td>Sleep Disordered Breathing</td>
<td>.056</td>
<td>.116</td>
<td>.080**</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td>-.190</td>
<td>-.032</td>
<td>-.009</td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>-.087</td>
<td>.034</td>
<td>-.043*</td>
</tr>
<tr>
<td>Anxious/Depressed</td>
<td>-.037</td>
<td>-.106</td>
<td>-.025</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>.166</td>
<td>-.022</td>
<td>.042*</td>
</tr>
</tbody>
</table>

*Note:*  
* indicates p < .05  
** indicates p < .01
Table 23. Average BMI centile associated with variables predictive of weight for the sites that demonstrated good model fit and the remaining sample

<table>
<thead>
<tr>
<th></th>
<th>Site 2 (n = 106)</th>
<th>Site 23 (n = 272)</th>
<th>All Others (n = 2322)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>67.07% (88)</td>
<td>60.56% (224)</td>
<td>63.66% (1892)</td>
</tr>
<tr>
<td>Female</td>
<td>55.58% (18)</td>
<td>64.85% (48)</td>
<td>64.33% (340)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>64.94% (84)</td>
<td>58.94% (228)</td>
<td>63.59% (1651)</td>
</tr>
<tr>
<td>African American</td>
<td>59.10% (5)</td>
<td>76.95% (30)</td>
<td>65.59% (186)</td>
</tr>
<tr>
<td>Asian</td>
<td>32.72% (5)*</td>
<td>82.62% (4)*</td>
<td>57.63% (149)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>87.71% (9)*</td>
<td>66.40% (8)*</td>
<td>71.26% (171)</td>
</tr>
<tr>
<td>Other Ethnicity</td>
<td>66.50% (3)</td>
<td>34.35% (2)</td>
<td>58.07% (75)</td>
</tr>
<tr>
<td>Father’s Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent or No Info</td>
<td>76.94% (9)</td>
<td>69.98% (20)</td>
<td>60.34% (291)</td>
</tr>
<tr>
<td>Less than 8th Grade</td>
<td>88.50% (1)</td>
<td>N/A (0)</td>
<td>57.03% (18)</td>
</tr>
<tr>
<td>Some high school</td>
<td>48.40% (2)</td>
<td>66.38% (5)</td>
<td>74.36% (84)</td>
</tr>
<tr>
<td>High school diploma</td>
<td>82.97% (15)*</td>
<td>66.11% (81)*</td>
<td>69.43% (416)</td>
</tr>
<tr>
<td>Some college</td>
<td>67.79% (16)</td>
<td>58.60% (43)</td>
<td>62.50% (533)</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>62.13% (41)</td>
<td>56.47% (81)</td>
<td>62.69% (522)</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>52.23% (22)</td>
<td>59.46% (42)</td>
<td>61.32% (368)</td>
</tr>
<tr>
<td>Stimulant</td>
<td>65.60% (11)*</td>
<td>38.51% (31)*</td>
<td>58.99% (232)</td>
</tr>
<tr>
<td>Antihistamine</td>
<td>76.19% (11)</td>
<td>59.24% (20)</td>
<td>69.04% (201)</td>
</tr>
<tr>
<td>Melatonin</td>
<td>78.57% (11)</td>
<td>61.30% (30)</td>
<td>69.31% (222)</td>
</tr>
<tr>
<td>Atomoxetine</td>
<td>64.20% (1)</td>
<td>42.11% (19)</td>
<td>57.19% (29)</td>
</tr>
<tr>
<td>SSRIs</td>
<td>71.39% (8)</td>
<td>56.48% (20)</td>
<td>71.07% (123)</td>
</tr>
<tr>
<td>Alpha 2 Agonists</td>
<td>73.68% (9)</td>
<td>57.68% (18)</td>
<td>69.46% (129)</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>72.68% (8)</td>
<td>65.04% (13)</td>
<td>71.09% (45)</td>
</tr>
<tr>
<td>Atypical Antipsychotics</td>
<td>61.61% (7)</td>
<td>58.58% (22)</td>
<td>68.63% (121)</td>
</tr>
<tr>
<td>Other Medication</td>
<td>58.70% (35)</td>
<td>59.10% (85)</td>
<td>64.51% (515)</td>
</tr>
<tr>
<td>Autistic Disorder</td>
<td>71.37% (43)</td>
<td>67.10% (134)</td>
<td>65.06% (1508)</td>
</tr>
<tr>
<td>Asperger Syndrome</td>
<td>61.69% (6)</td>
<td>58.23% (31)</td>
<td>60.38% (221)</td>
</tr>
<tr>
<td>PDD-NOS</td>
<td>52.89% (57)</td>
<td>46.95% (107)</td>
<td>61.36% (503)</td>
</tr>
</tbody>
</table>

*Note: The first numeral in each column is BMI percentile. The sample sizes are in parentheses

* sites different at \( p < .01 \)

Sex: \( \chi^2 (2, N = 2,610) = 1.114, p = .573 \)

Ethnicity: \( \chi^2 (8, N = 2,610) = 38.052, p < .001 \)

Father’s Ed: \( \chi^2 (12, N = 2,610) = 11.274, p = .490 \)

Medication: \( \chi^2 (16, N = 2,610) = 7.335, p = .966 \)

ASD Subtype: \( \chi^2 (4, N = 2,610) = 1.626, p = .804 \)
Table 24. Age distribution frequency by ATN site.

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Site 2 (%)</th>
<th>Site 23 (%)</th>
<th>All Others (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6 years</td>
<td>48 (45.3)</td>
<td>151 (55.5)</td>
<td>1290 (57.8)</td>
</tr>
<tr>
<td>6-10 years</td>
<td>42 (39.6)</td>
<td>68 (25.0)</td>
<td>594 (26.6)</td>
</tr>
<tr>
<td>≥ 10 years</td>
<td>16 (15.1)</td>
<td>53 (19.5)</td>
<td>348 (15.6)</td>
</tr>
<tr>
<td>Total sample size</td>
<td>106 (100)</td>
<td>272 (100)</td>
<td>2232 (100)</td>
</tr>
</tbody>
</table>

*Note.* $\chi^2 (4, N = 2610) = 12.05, p = .017.$

Table 25. ASD subtype distribution by ATN site

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Site 2 (%)</th>
<th>Site 23 (%)</th>
<th>All Others (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autistic Disorder</td>
<td>43 (40.6)</td>
<td>134 (49.3)</td>
<td>1508 (67.6)</td>
</tr>
<tr>
<td>Asperger Disorder</td>
<td>6 (5.7)</td>
<td>31 (11.4)</td>
<td>221 (9.90)</td>
</tr>
<tr>
<td>PDD-NOS</td>
<td>57 (53.7)</td>
<td>107 (39.3)</td>
<td>503 (22.5)</td>
</tr>
<tr>
<td>Total sample size</td>
<td>106 (100)</td>
<td>272 (100)</td>
<td>2232 (100)</td>
</tr>
</tbody>
</table>

*Note.* $\chi^2 (4, N = 2610) = 36.18, p < .001.$
Outcome Variable Inquiries

One last investigation was conducted in an attempt to identify why the amount of variance accounted for was consistently so low. This involved the actual outcome variable. I was unable to locate previous literature that attempted to identify risk factors for children using BMI percentile as the outcome measure of interest. The childhood overweight and obesity literature has been limited in comparison to adult weight literature, so it may not be surprising that BMI percentile was not used, but I believed that it warranted further examination. Some studies (e.g., Dockray et al., 2009) used either BMI alone (later adjusting for age) or BMI z scores, which is closely related to BMI percentile, and I looked at both of these possible outcome measures.

On the surface, it appears as though the analysis using BMI alone accounted for a large amount of variance when compared with the analyses previously discussed. The overall amount of variance account for was 23.6% ($F[1, 2572] = 23.424, p < .001$), and included all of the previous blocks discussed (demographics, medication, sleep problems, comorbid psychopathology, IQ score/adaptive behavior, and ASD subtype). However, I needed to add a separate new first block for age. This was because BMI alone only evaluates the relationship between height and weight, and does not take into account growing children who rapidly alter the ratio between those two constructs through normal development. Age alone accounted for 19.2% of the adjusted $R^2$ ($F[1, 2607] = 622.348, p < .001$), which then meant that the remaining variables accounted for an additional 4.4% of variance. While this additional variance was significant, it was not substantially different from what previous models were able to achieve. The use of BMI alone as the
outcome variable may not be able to drastically change what variables are important in weight in these children with ASDs. However, this demonstrates that a known predictor performed exactly as expected. This boosts confidence that the statistical model could and did perform as expected. For a visual depiction of how age impacts BMI in children, refer to the CDC growth charts for boys and girls in Appendix A. The summary and overview of this analysis can be found in Tables 26 and 27.

The final analysis conducted used BMI z-score as the outcome variable. This metric is typically used to evaluate change in weight over a period of time, and it is popular throughout the existing literature, particularly when discussing change in weight due to a given treatment (e.g., Faraone et al., 2008). This version performed very closely to the atheoretical empiricism model, as it was able to account for 3.8% of variance \((F[36, 2572] = 3.900, p < .001)\) as opposed to the original 3.6%. The variables themselves were also very similar in regards to whether they were significantly associated with the outcome of BMI z-score in this iteration, or BMI percentile in the earlier versions. Only being of Asian descent, use of alpha 2 agonists, aggressive behavior, and Withdrawn/Depressed scores on the CBCL failed to reach levels of significance in this z-score analysis, as compared with the atheoretical empiricism model. The other eight (Hispanic descent, father education, stimulants, atomoxetine, SSRIs, sleep disordered breathing, Anxious/Depressed CBCL subscale, and ASD subtype) were the same across both models, and no new variables reached significance in the z-score analysis. The specific details about this model, including summary and overview, can be seen in Tables 28 and 29.
Summary

To recap, these analyses revealed the following:

1) Prevalence rate calculations revealed that 118 (4.5%) children in the sample were underweight, 1,633 (62.6%) were normal weight, 859 (32.9%) were overweight, and 452 (17.3%) were obese. These largely reflect patterns found in the general population.

2) Validity of the theoretical model in the entire sample: this was established insofar as aspects of the model were supported statistically, but with the significant limitation that only small amounts of variance were predicted. This is considered at length in the Discussion.

3) The atheoretical approach provided greater ability to predict, but added variance was minor.

4) There was a lack of effect when children taking concomitant medications were eliminated.

5) The results were examined by site and it appeared that Sites 2 and 23 were statically different from the other sites included in the sample. When I focused on these sites alone I was able to account for much more variance than in the remaining sites.

6) Findings for outcome variable analyses, particularly when age was not controlled from the outset (as is the case in BMI percentiles and BMI z-scores), caused age to become a very strong predictor of outcome.
Table 26. Summary of hierarchical regression analysis predicting BMI unadjusted for age

<table>
<thead>
<tr>
<th>Block 1</th>
<th></th>
<th>B</th>
<th>SE B</th>
<th>p</th>
<th>β</th>
<th>Partial Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months)</td>
<td></td>
<td>14.559</td>
<td>.140</td>
<td>&lt; .001</td>
<td>.439</td>
<td>.439</td>
</tr>
<tr>
<td>Block 2</td>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td>.185</td>
<td>.180</td>
<td>.304</td>
<td>.018</td>
<td>.020</td>
</tr>
<tr>
<td>African American</td>
<td></td>
<td>.715</td>
<td>.238</td>
<td>.003</td>
<td>.054</td>
<td>.059</td>
</tr>
<tr>
<td>Asian</td>
<td></td>
<td>-.140</td>
<td>.277</td>
<td>.614</td>
<td>-.009</td>
<td>-.010</td>
</tr>
<tr>
<td>Hispanic</td>
<td></td>
<td>.772</td>
<td>.255</td>
<td>.002</td>
<td>.054</td>
<td>.059</td>
</tr>
<tr>
<td>Other Ethnicity</td>
<td></td>
<td>-.629</td>
<td>.383</td>
<td>.101</td>
<td>-.029</td>
<td>-.032</td>
</tr>
<tr>
<td>Mother Education</td>
<td></td>
<td>.071</td>
<td>.058</td>
<td>.220</td>
<td>.028</td>
<td>.024</td>
</tr>
<tr>
<td>Father Education</td>
<td></td>
<td>-.149</td>
<td>.048</td>
<td>.002</td>
<td>-.072</td>
<td>-.061</td>
</tr>
<tr>
<td>Block 3</td>
<td>Medications</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulants</td>
<td></td>
<td>-.933</td>
<td>.224</td>
<td>&lt; .001</td>
<td>-.077</td>
<td>-.081</td>
</tr>
<tr>
<td>Antihistamines</td>
<td></td>
<td>-.251</td>
<td>.385</td>
<td>.514</td>
<td>-.019</td>
<td>-.013</td>
</tr>
<tr>
<td>Melatonin</td>
<td></td>
<td>.809</td>
<td>.388</td>
<td>.037</td>
<td>.066</td>
<td>.041</td>
</tr>
<tr>
<td>Atomoxetine</td>
<td></td>
<td>-1.997</td>
<td>.482</td>
<td>&lt; .001</td>
<td>-.073</td>
<td>-.081</td>
</tr>
<tr>
<td>SSRI</td>
<td></td>
<td>.889</td>
<td>.295</td>
<td>.003</td>
<td>.056</td>
<td>.059</td>
</tr>
<tr>
<td>Alpha 2 Agonists</td>
<td></td>
<td>.324</td>
<td>.281</td>
<td>.249</td>
<td>.021</td>
<td>.023</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td></td>
<td>.839</td>
<td>.435</td>
<td>.054</td>
<td>.036</td>
<td>.038</td>
</tr>
<tr>
<td>Atypical Antipsychotics</td>
<td></td>
<td>.424</td>
<td>.308</td>
<td>.170</td>
<td>.026</td>
<td>.027</td>
</tr>
<tr>
<td>Other Medication</td>
<td></td>
<td>.015</td>
<td>.140</td>
<td>.916</td>
<td>.002</td>
<td>.002</td>
</tr>
<tr>
<td>Block 4</td>
<td>Sleep Problems</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bedtime Resistance</td>
<td></td>
<td>.045</td>
<td>.031</td>
<td>.147</td>
<td>.040</td>
<td>.029</td>
</tr>
<tr>
<td>Sleep Onset Delay</td>
<td></td>
<td>-.036</td>
<td>.091</td>
<td>.692</td>
<td>-.008</td>
<td>-.008</td>
</tr>
<tr>
<td>Sleep Duration</td>
<td></td>
<td>.007</td>
<td>.045</td>
<td>.870</td>
<td>.003</td>
<td>.003</td>
</tr>
<tr>
<td>Sleep Anxiety</td>
<td></td>
<td>-.063</td>
<td>.048</td>
<td>.189</td>
<td>-.035</td>
<td>-.026</td>
</tr>
<tr>
<td>Night Waking</td>
<td></td>
<td>-.179</td>
<td>.076</td>
<td>.018</td>
<td>-.043</td>
<td>-.046</td>
</tr>
<tr>
<td>Parasomnias</td>
<td></td>
<td>-.015</td>
<td>.036</td>
<td>.666</td>
<td>-.009</td>
<td>-.008</td>
</tr>
<tr>
<td>Sleep Dis. Breathing</td>
<td></td>
<td>.371</td>
<td>.075</td>
<td>&lt; .001</td>
<td>.092</td>
<td>.097</td>
</tr>
<tr>
<td>Daytime Sleepiness</td>
<td></td>
<td>.020</td>
<td>.027</td>
<td>.461</td>
<td>.014</td>
<td>.015</td>
</tr>
</tbody>
</table>

(continued)
Table 26. Summary of hierarchical regression analysis predicting BMI unadjusted for age (continued)

<table>
<thead>
<tr>
<th>Block 5</th>
<th>Comorbid Psychopathology</th>
<th>B</th>
<th>SE B</th>
<th>p</th>
<th>β</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Affective Problems</td>
<td>.018</td>
<td>.012</td>
<td>.121</td>
<td>.045</td>
<td>.031</td>
</tr>
<tr>
<td></td>
<td>Aggressive Behavior</td>
<td>-.001</td>
<td>.006</td>
<td>.892</td>
<td>-.002</td>
<td>-.003</td>
</tr>
<tr>
<td></td>
<td>Anxiety Problems</td>
<td>-.004</td>
<td>.006</td>
<td>.507</td>
<td>-.011</td>
<td>-.013</td>
</tr>
<tr>
<td></td>
<td>Anxious/Depressed</td>
<td>-.022</td>
<td>.009</td>
<td>.019</td>
<td>-.055</td>
<td>-.046</td>
</tr>
<tr>
<td></td>
<td>ADHD Problems</td>
<td>-.015</td>
<td>.010</td>
<td>.110</td>
<td>-.035</td>
<td>-.031</td>
</tr>
<tr>
<td></td>
<td>DBD</td>
<td>-.005</td>
<td>.009</td>
<td>.556</td>
<td>-.013</td>
<td>-.012</td>
</tr>
<tr>
<td></td>
<td>Somatic Complaints</td>
<td>.019</td>
<td>.010</td>
<td>.051</td>
<td>.042</td>
<td>.039</td>
</tr>
<tr>
<td></td>
<td>Withdrawn/Depressed</td>
<td>.017</td>
<td>.008</td>
<td>.028</td>
<td>.047</td>
<td>.043</td>
</tr>
</tbody>
</table>

| Block 6 | IQ Score                 | -.009| .003  | .005  | -.059| -.055        |
|         | Adaptive Beh. Composite  | -.003| .006  | .557  | -.012| -.012        |

| Block 7 | ASD Subtype              | -.240| .079  | .002  | -.056| -.060        |

*Note.* ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.
Table 27. Overview of hierarchical regression analysis predicting BMI unadjusted for age

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change F(p)</th>
<th>Overall F(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.193</td>
<td>.192</td>
<td>---</td>
<td>---</td>
<td>622.348 (&lt;.001)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.204</td>
<td>.202</td>
<td>.012</td>
<td>5.407 (&lt; .001)</td>
<td>83.445 (&lt; .001)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.224</td>
<td>.219</td>
<td>.019</td>
<td>7.204 (&lt; .001)</td>
<td>43.926 (&lt; .001)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.234</td>
<td>.226</td>
<td>.010</td>
<td>4.186 (&lt;.001)</td>
<td>31.503 (&lt;.001)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.241</td>
<td>.231</td>
<td>.007</td>
<td>3.044 (.002)</td>
<td>24.755 (&lt; .001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.244</td>
<td>.234</td>
<td>.003</td>
<td>5.757 (.003)</td>
<td>23.755 (&lt; .001)</td>
</tr>
<tr>
<td>Block 7</td>
<td>.247</td>
<td>.236</td>
<td>.003</td>
<td>9.169 (.002)</td>
<td>23.424 (&lt; .001)</td>
</tr>
</tbody>
</table>
Table 28. Summary of hierarchical regression analysis predicting BMI z-score

<table>
<thead>
<tr>
<th>Block 1</th>
<th></th>
<th></th>
<th></th>
<th>Partial Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (months)</td>
<td>.000</td>
<td>.001</td>
<td>.955</td>
<td>.001</td>
</tr>
<tr>
<td>Sex</td>
<td>.006</td>
<td>.064</td>
<td>.926</td>
<td>.002</td>
</tr>
<tr>
<td>African American</td>
<td>.177</td>
<td>.085</td>
<td>.037</td>
<td>.041</td>
</tr>
<tr>
<td>Asian</td>
<td>-.185</td>
<td>.099</td>
<td>.060</td>
<td>-.037</td>
</tr>
<tr>
<td><strong>Hispanic</strong></td>
<td><strong>.346</strong></td>
<td><strong>.091</strong></td>
<td>&lt; .001</td>
<td><strong>.075</strong></td>
</tr>
<tr>
<td>Other Ethnicity</td>
<td>-.210</td>
<td>.136</td>
<td>.124</td>
<td>-.030</td>
</tr>
<tr>
<td>Mother Education</td>
<td>.036</td>
<td>.021</td>
<td>.081</td>
<td>.044</td>
</tr>
<tr>
<td><strong>Father Education</strong></td>
<td><strong>-.043</strong></td>
<td><strong>.017</strong></td>
<td><strong>.011</strong></td>
<td><strong>-.065</strong></td>
</tr>
</tbody>
</table>

| Block 2                          |          |          |       |                     |
| Medications                      |          |          |       |                     |
| **Stimulants**                   | **-.357**| **.080** | < .001| **-.092**          |
| Antihistamines                   | .048     | .137     | .715  | .012                |
| Melatonin                        | .111     | .139     | .425  | .028                |
| **Atomoxetine**                  | **-.574**| **.172** | .001  | **-.066**          |
| **SSRIs**                        | **.268** | **.105** | **.011**| **.053**          |
| Alpha 2 Agonists                 | .169     | .100     | .092  | .034                |
| Anticonvulsants                  | .214     | .155     | .168  | .028                |
| Atypical Antipsychotics          | .098     | .110     | .372  | .019                |
| Other Medication                 | -.031    | .050     | .531  | -.013               |

| Block 3                          |          |          |       |                     |
| Sleep Problems                   |          |          |       |                     |
| Bedtime Resistance               | .011     | .011     | .328  | .030                |
| Sleep Onset Delay                | -.033    | .033     | .315  | -.022               |
| Sleep Duration                   | -.015    | .016     | .359  | -.021               |
| Sleep Anxiety                    | -.006    | .017     | .721  | -.011               |
| Night Waking                     | -.010    | .027     | .705  | -.008               |
| Parasomnias                      | .015     | .013     | .253  | .026                |
| **Sleep Dis. Breathing**         | **.106** | **.027** | < .001| **.083**            |
| Daytime Sleepiness               | -.009    | .010     | .349  | -.020               |

(continued)
Table 28. Summary of hierarchical regression analysis predicting BMI z-score (continued)

<table>
<thead>
<tr>
<th>Block 4: Comorbid Psychopathology</th>
<th>B</th>
<th>SE B</th>
<th>p</th>
<th>β</th>
<th>Partial Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affective Problems</td>
<td>.007</td>
<td>.004</td>
<td>.099</td>
<td>.054</td>
<td>.032</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td>-.001</td>
<td>.002</td>
<td>.655</td>
<td>-.009</td>
<td>-.009</td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>-.004</td>
<td>.002</td>
<td>.058</td>
<td>-.037</td>
<td>-.037</td>
</tr>
<tr>
<td><strong>Anxious/Depressed</strong></td>
<td><strong>-.012</strong></td>
<td><strong>.003</strong></td>
<td><strong>&lt; .001</strong></td>
<td><strong>-.093</strong></td>
<td><strong>-.070</strong></td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>-.002</td>
<td>.003</td>
<td>.608</td>
<td>-.013</td>
<td>.010</td>
</tr>
<tr>
<td>DBD</td>
<td>.003</td>
<td>.003</td>
<td>.315</td>
<td>.025</td>
<td>.020</td>
</tr>
<tr>
<td>Somatic Complaints</td>
<td>.002</td>
<td>.003</td>
<td>.656</td>
<td>.011</td>
<td>.009</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>.005</td>
<td>.003</td>
<td>.081</td>
<td>.042</td>
<td>.034</td>
</tr>
</tbody>
</table>

| Block 5: IQ Score               | -.001| .001 | .470 | -.017| -.014               |
| Adaptive Beh. Composite         | -.001| .002 | .727 | -.008| -.007               |

| Block 6: ASD Subtype            | **-.082**| **.028** | **.004** | **-.060**| **-.057**          |

*Note.* ASD subtype indicates that youth with PDD-NOS diagnoses were lighter than peers.
Table 29. Overview of hierarchical regression analysis predicting BMI z-score

<table>
<thead>
<tr>
<th>Block</th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.014</td>
<td>.011</td>
<td>---</td>
<td>---</td>
<td>4.524 (&lt; .001)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.031</td>
<td>.025</td>
<td>.017</td>
<td>5.148 (&lt; .001)</td>
<td>4.885 (&lt; .001)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.040</td>
<td>.031</td>
<td>.009</td>
<td>3.067 (.002)</td>
<td>4.324 (&lt; .001)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.048</td>
<td>.036</td>
<td>.008</td>
<td>2.743 (.005)</td>
<td>3.959 (&lt; .001)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.049</td>
<td>.036</td>
<td>.000</td>
<td>.521 (.594)</td>
<td>3.761 (&lt; .001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.52</td>
<td>.038</td>
<td>.003</td>
<td>8.379 (.004)</td>
<td>3.900 (&lt; .001)</td>
</tr>
</tbody>
</table>
CHAPTER 4: DISCUSSION

In the writer’s view, three outcomes were the most interesting aspects of this dissertation, and they revolve around the similarities between children with ASD and typically developing children in regards to abnormal weight. Specifically, these issues were (a) the consistency in overweight and obesity prevalence rates between the ATN’s ASD sample and previously published results in typically developing children, (b) the similarity of significant predictors of BMI percentile through the various models that were tested and variables that were statistically significant in previously published literature, and (c) the similarities in the magnitude of variance that was accounted for in this study in comparison to previous literature. Other results will also be discussed, but the majority of this section will focus on the three outcomes mentioned above.

Prevalence Rates

One goal of this dissertation was to evaluate abnormal weight status in children with ASD by exploring BMI derived from a large data set with measures of weight and height gathered by medical personnel. Other key assessments of interest were also explored for their predictive value. The prevalence rates of the abnormal weight classes in this sample of children with ASD were not significantly different from typically developing children for either overweight (ASD = 32.9% vs. typically developing = 33.6%) or obesity (ASD = 17.3 vs. typically developing = 17.1%; Ogden et al., 2006). The rates of overweight and obesity were also not statistically different from another

Other investigators found higher rates of overweight and obesity; specifically, 42.5% of children with autism were overweight and 24.6% were obese (Rimmer et al., 2010). Nevertheless, both the data from this dissertation and from previous studies (Curtin et al., 2005; Ogden et al., 2006) failed to demonstrate that children with ASD were more overweight or obese than typically developing children. I predicted that these youngsters might have increased weight status compared to CDC normative data due to a high frequency of medication use and limited time or interest in physical activity (MacDonald, Espistio, & Ulrich, 2011; Grondhuis & Aman, 2013). However, this did not occur to the extent that it made them heavier than children who are on a normal developmental trajectory. That being said, abnormal weight is still a serious issue for children with ASD. These children face many more problems (e.g., developmental, behavioral, medical) than typically developing youth (Gray et al., 2012). Regardless of whether overweight or obesity are more of an issue, when they occur they still need to be addressed within the scope of regular treatment. Abnormal weight can cause medical and psychological problems above and beyond what an individual otherwise faces. These issues extend beyond greater rates of morbidity and mortality associated with overweight.

**Factor Analysis of Nutrition Variables**

It made sense from a theoretical perspective that many nutritional constructs (including food intolerance, feeding difficulties, and pica) would be related to one another and that they would, therefore, be connected in a latent factor model. However,
this was not the case. The nutritional deficits variables (see Appendix E) did not load onto a one-factor model or a two-factor model, suggesting that they were not tapping any underlying constructs. This is not entirely surprising since the variables included were simply items on a diagnostic assessment form, rather than a cohesive measure designed to address specific nutrition problems such as picky/compulsive eating or specialty diets (e.g., gluten-free, casein-free diets). The correlations between these nutritional variables were very small (see Table 6) and made emergence of a latent variable or variables unfeasible. It also suggests that the data gathered on these nutritional variables were essentially unrelated. The ATN may benefit from adding a standardized measure (e.g., Child Nutrition Questionnaire, Wilson, Magarey, & Mastersson, 2008; Food Symptom Association Questionnaire, Carlson, Moore, Tsai, Shulman, & Chumpitazi, 2014) that explicitly evaluates presence and/or severity of nutritional or gastrointestinal variables.

Multiple Linear Regression Models

Although I had a single, primary analysis that was based on findings from the literature, it did not perform as expected and I ran multiple other analyses. These were purely exploratory and the intent was to analyze the data from all possible perspectives. I realize that I probably introduced some Type I Error by doing so, and none of the exploratory analyses should be viewed as definitive. These variables will be evaluated to see how well they performed over multiple analyses. The regression models were run in an attempt to determine what variables were most responsible for abnormal weight in the youth with ASD who were included in this sample. Table 30 illustrates that some variables of interest were more consistently able to reach significance over the course of
Table 30. Summary of significant variables in all analyses

<table>
<thead>
<tr>
<th>Variable</th>
<th>Original Analysis</th>
<th>Theoretical Analysis</th>
<th>Confounding Meds Removed One Medication or Less</th>
<th>CBCL 1-5 Version</th>
<th>CBCL 6-18 Version</th>
<th>BMI unadjusted for age</th>
<th>BMI z-scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>African American</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Asian</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Hispanic</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Other Ethnicity</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Mother Education</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Father Education</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Stimulants</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Melatonin</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Atomoxetine</td>
<td></td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>SSRIs</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Alpha 2 Agonists</td>
<td></td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Night Waking</td>
<td></td>
<td></td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Sleep Disordered Breathing</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td></td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Anxious/Depressed</td>
<td></td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Somatic Complainstes</td>
<td></td>
<td></td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>IQ Score</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Adaptive Behavior Composite</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>ASD Subtype</td>
<td></td>
<td></td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
</tbody>
</table>

Note: Y = significant in analysis; N = not significant in analysis. Variables that never reached significance in any analyses were excluded. These include sex, antihistamines, anticonvulsants, atypical antipsychotics, other medications, Bedtime Resistance, Sleep Onset Delay, Sleep Duration, Sleep Anxiety, Parasomnias, Daytime Sleepiness, Affective Problems, Anxiety Problems, and Disruptive Behavior Disorders. Shaded areas indicate that variable was not included in the analysis.
different models. On the other hand, quite a few variables never reached significance. The latter included sex, antihistamines, anticonvulsants, atypical antipsychotics, other medications, Bedtime Resistance, Sleep Onset Delay, Sleep Duration, Sleep Anxiety, Parasomnias, Daytime Sleepiness, Affective Problems, Anxiety Problems, and Disruptive Behavior Disorders. As previously mentioned, many of the significant findings throughout the multiple analyses were consistent with previously published literature. This is important, as it is essential to be able to identify ways in which children with ASD are different from typically developing children and in what ways they are similar. These similarities will be pointed out below in the order they were included in the analyses. I will only discuss variables that were significant in three or more analyses.

There were also several demographic variables that were significant in these hierarchical regression models that were expected based on the literature. Age was significant in the model that specifically looked at those who completed the CBCL for the younger age range (1.5-5 years). Age also reached significance in the analysis that used BMI alone, but this effect disappeared once age was controlled through use of percentiles. In the first CBCL analysis, age was associated with greater BMI percentile, which indicated that preschoolers’ age (up until the 5 year 11 month point) was positively but weakly associated with greater weight status. This could be due to the fact that as children age, the more opportunity they have to vary from their birth weight and to develop habits, such as prolonged inactivity and consumption of fast foods, that may be detrimental to their health and well-being. With age, they will also likely have higher rates of medication use, which we know can influence overall body mass.
Seven of the eight models demonstrated that children who were Hispanic were more likely to have elevated BMI percentile. Deckelbaum and Williams (2001) reported that Mexican-American children were more likely to be overweight than peers of other ethnic backgrounds. In three of the models, children who were of Asian heritage generally had low body weight in relation to height, which is consistent with earlier findings that identified Asian subjects as lighter on average (Lauderdale & Rathouz, 2000). African American heritage was only significantly associated with greater BMI percentile in two analyses. While the directionality of the relationship was consistent with previously published findings (e.g., Dietz and Robertson, 2005), it was not as robust as expected. Speculatively, this project may have drawn individuals, including African Americans, who were atypical of the general population (with higher education, SES, etc.), and therefore the results may not have followed the same ethnic expectations.

Father’s education level was negatively associated with BMI centile in five of the analyses, which could be reflective of two possible phenomena. First, more advanced education generally leads to greater prosperity and a larger take-home paycheck (Beaton, 1975). These individuals may be able to provide higher quality foods to their children (fruits, vegetables, lean meats), which may help them to maintain a healthier body weight. The lowest rating on this scale was assigned when there was no father present. This designation was associated with children from single-family homes being heavier than those who had two parents present, as was found in previous literature (Costello, Keeler, & Angold, 2001). This could be due to possible income constraints associated
with single parenting (e.g., inability to afford nutritious meals), limited time for leisure
activities that would encourage more physical activity, or effects of stress.

The variable that was most associated with weight in these analyses was stimulant
medication use, as it predicted lower BMI percentile in all eight analyses. It is well
established that stimulant use can suppress appetite, which can lead to reduction in body
weight (Cortese et al., 2008; Faraone et al., 2008). Unfortunately, the duration of
medication use was not available in the data set. Children with ADHD but who are
medication naïve have been shown to have higher body mass than children from the
general population who do not have ADHD (Cortese et al., 2008; Faraone et al., 2008).
This could have important weight implications if the majority of the sample was either
new to stimulant treatment or had been taking the medication for years (i.e., there could
be cancellation due to medication in some but not others due to variable duration of use).
Access to duration of stimulant treatment would enable more sophisticated exploration of
the effects of stimulant drugs, and would help with the interpretation of study findings.

The use of SSRIs was significantly associated with BMI centile in four of the
analyses, indicating that children who took SSRIs were heavier than their peers who did
not take those medications. This finding is consistent with the existing literature and is in
line with SSRI side effect profiles that list increase in appetite and weight gain as
possible adverse events (Ferguson, 2001). The neurotransmitter serotonin has been
connected to autism pathophysiology. Some believe serotonin dysregulation to be related
to some of the so-called “core features” of the disorder, as well as some of the associated
conditions (e.g., aggression, mood problems; Froehlich, 2011). SSRIs can be prescribed
for a number of reasons, although research on the efficacy of SSRIs for these children is mixed. A review of the literature found that there was no obvious improvement with SSRIs in the core symptoms, although a reduction in restrictive and repetitive behaviors was seen in some studies (Froehlich, 2011; Soorya, Kiarashi, & Hollander, 2008). That being said, a definitive study of the SSRI citalopram discounted such a therapeutic effect (King et al., 2009). A reduction in these potentially intrusive behaviors would be quite beneficial, if indeed they occur. However, doctors should be cognizant of weight gain as a possible side effect, especially if their patients have other risk factors for elevated weight status.

The remaining drug classes, including melatonin, atomoxetine, and alpha 2 agonists, were significant in fewer analyses than either the stimulants or the SSRIs. Melatonin was significant in two analyses (one of which was the BMI alone), atomoxetine was significant in three analyses (again, one of which was BMI alone), and alpha 2 agonists were significant in three analysis. The atomoxetine results from this study are also consistent with previously published literature. That is, children who took atomoxetine were more likely to be lighter than their peers who did not take atomoxetine (e.g., Wernicke & Kratochvil, 2002). There is some thought that these initial weight losses are temporary and that individuals will, with time, return to their previous weight (Wernicke & Kratochvil, 2002). As duration of drug exposure was unknown in this project, I cannot comment on the previously reported time phenomenon.

Alpha 2 agonists reached significance in three of the analyses performed. Children in this study who were prescribed alpha 2 agonists were heavier than their peers
who did not take the medication, which is in line with some of the existing literature. One open-label study of guanfacine in children and adolescents with traumatic stress related symptoms (Conner et al., 2013) indicated that a small but statistically significant increase in weight between baseline and the sixth week of treatment. Several other studies (e.g., Palumbo et al., 2008; Kollins et al., 2011; Jain, Segal, Kollins, & Khayrallah, 2011) did not report weight changes, so it is unknown whether the findings of Conner and colleagues (2013) would be replicated in other samples. More research is needed in this area to be able to state definitively whether this drug requires closer monitoring for weight-related side effects.

Another variable that was consistently associated with BMI percentile was Sleep Disordered Breathing. This variable was significant in seven of the models. This is also not surprising, as there is a well-established relationship between weight and problems such as sleep apnea and snoring that are assessed using the CSHQ subscale. There is a strong likelihood that disordered breathing during sleep did not cause subjects to have higher levels of body mass. Instead, it is likely that there was a reciprocal relationship, where people developed these respiratory conditions because of their excess weight. One of the causes of sleep apnea is excess fat of the neck resting on the windpipe, blocking the flow of air (Daniels, 2006). Hence, the relationship might reflect the effect of excess weight on sleep instead of the other way around. Night Wakings was the only other sleep variable measured with the CSHQ that was significant in a single analysis. That version was the BMI alone, without being adjusted for age. Although this does demonstrate that there may be certain underlying factors that make heavier children more likely to wake
repeatedly throughout the night (most notably the presence of sleep disordered breathing), these findings were not consistent enough to warrant further investigation.

The Aggressive Behavior subscale of the CBCL reached significance in four of the analyses. Although we know that aggression is a common feature in children with ASD (Farmer & Aman, 2011), it is not frequently associated with obesity and overweight in neurotypical samples. Farmer and Aman (2011) reported that children with ASD are frequently aggressive, but in a way that does not imply malicious intent. Instead, they appear to have rather limited awareness of behavioral norms and they tend to engage in *impulsive* (reactive) aggression. At least one study in neurotypical children observed a connection between overweight and obesity and high rates of bullying behaviors associated with aggression. In addition, there was a link between elevated weight status and increased victimization *from* bullies (Janssen, Craig, Boyce, & Pickett, 2004). The current study’s finding of aggression associated with BMI centile in children with ASD could be due to either (a) larger children attempting to reach their goals through physical intimidation (proactive aggression; Farmer & Aman, 2011) or (b) larger children reacting to others’ bullying behaviors, as the CBCL does not differentiate between types of aggressive behavior.

Anxious/Depressed, a CBCL subscale that was previously excluded in the initial analysis because of the nonspecific nature of the composite (i.e., both anxiety and depression are included), was significant in six of the models in which it was subsequently included. Interestingly, the results indicated that higher scores on the Anxious/Depressed subscale were associated with *lower* overall weight status. This
negative relationship is in contrast to previous literature showing that individuals with anxiety or depression were more likely to be overweight or obese (Goodman & Whitaker, 2002; Dockray et al., 2009). Since individual CBCL items were not available to enhance interpretation, future research is needed to confirm whether this finding is attributed more to the anxiety questions or the depression questions belonging to this subscale.

The CBCL Withdrawn/Depressed subscale was also statistically associated with weight percentile in four analyses. Children who scored higher on the Withdrawn/Depressed subscale were more likely to be overweight. This is in agreement with existing literature that documented this pattern in both typically developing adults and adolescents (Blaine, 2008), although the relationship can be confusing. It is difficult to say whether depression precedes weight gain, whether weight gain occurs first and depression follows, or if the process is dynamically and intricately intertwined. Both the ADHD problems subscale and the Somatic Complaints subscales were significant in one analysis, and for both it was within the CBCL 6-18 year version. This finding may suggest that age plays a meaningful role. The overall sample had more younger children (n = 1,464) than older children (n = 1,084) and those younger participants may be diluting any true signal in the analyses.

The last variable to be significantly associated with BMI centile was ASD subtype, and this was found in all seven analyses in which it was included. Children with autistic disorder were more likely to have elevated body mass than youngsters with PDD-NOS. This implies that children with more severe symptoms were more likely to be heavier than children who displayed fewer autistic traits. Many autistic symptoms
(including most restricted interests, impairments in social interaction, being withdrawn) may be associated with sedentary behavior. Previous literature has demonstrated that children with autism appear to engage in more sedentary than active behavior, as recorded by accelerometers (MacDonald et al., 2011).

Of the variables that failed to reach significance in any of the possible models, the biggest surprise was that of atypical antipsychotics. There is a considerable literature that reported significant and substantial positive associations between prescription of antipsychotics and weight gain (Correll, 2007; Stigler, Potenza, Posey, & McDougle, 2004; Wetterling, 2001). This null finding became less surprising once it was clear that the correlation between BMI percentile and atypical antipsychotics was small but positive, while the medication was highly correlated with other pharmacotherapies taken by children in this sample (see Table 7, starting on p. 59).

Additionally, it is intriguing that the disruptive behavior disorder subscale also did not predict weight status. This is in contrast to previous literature that found neurotypical children with oppositional defiant disorder, one of the two disruptive behavior disorder conditions, were more likely to be obese (Mustillo et al., 2003). My model was based in large part on prior research, and it did identify some predictors of overweight and obesity in children with ASD, although – obviously – not all elements of the model performed as hoped.

ATN Site

One of the most interesting findings of this dissertation concerned the site differences. When site was added into a block in the existing atheoretical empiricism
model, it was statistically significant (change in $R^2 = .001$, $F[1, 2574] = 3.932, p = .047$). This indicates that the sites were performing differently from one another. Indeed, there were very large differences in the amounts of variance that could be accounted for at the site level. These analyses only included sites that had more than 100 subjects, which I felt was appropriate due to the large number of variables included in the model. Nevertheless, sample size may have been inadequate at some sites that were included, as at least three produced negative adjusted $R^2$s, indicating either poor model fit or highly underpowered samples regardless of the 100-subject minimum (IBM Technote, 2007b; Peasnell et al., 2000).

More interesting than the sites that did not perform well (or those who performed similarly to the atheoretical empiricism model) were the sites that did a superior job in accounting for variance. At Site 2, the model accounted for 17.1% of the variance when only demographics, medications, sleep disturbance variables, and comorbid psychopathology were included in the model ($p = .039$). The overall model that included all variables failed to reach significance ($p = .058$) when the other two blocks (cognitive variables [IQ score and adaptive behavior composite score] and ASD subtype) were included in the model. At Site 23, the model accounted for 13.5% of the variance using all six blocks ($p < .001$) and 12.6% of the variance when using the same four blocks that were significant in Site 2 ($p < .001$).

What makes these sites so different from the others that were included in the ATN? It could be a geographic distinction as both of these sites were in the northeastern United States, but this is likely not the case. One other site (Site 27) was also in the same
region, and the model was not useful in predicting weight differences for this site.

Perhaps it has more to do with research practices. These sites may have been more experienced at research in general, which may include having an ingrained tradition for quality practices. Their levels of experience may have made them better at subject recruitment, either in volume of subjects or diagnostic accuracy — that is, how well the subjects’ symptoms aligned with the diagnoses assigned. They may have been better at filtering out questionable study participants who did not belong in the clinical cohort. The sites may also have done a better job in collecting quality data (greater precision, fewer missing data, fewer transcription errors, and so forth).

Finally, based on the chi-square analyses (reported in Tables 24 and 25) we know that these sites had statistically different distributions for both age and ASD subtype. For the age analyses, we know that Site 2 had almost equal rates of young children and “middle” aged children (between 6 and 10 years of age), with fewer older children. This is in contrast to Site 23, which had close to equal numbers of middle aged children and older children. This “surplus” of children over the age of 6 years found in Site 2 or the remainder of the sample may help explain why Site 2 was able to account for more variance, similar to the way in which the older CBCL group was able to account for more variance than the younger CBCL group.

The ASD subtype breakdown was also statistically different across sites. Both Site 2 and Site 23 had greater proportions of children diagnosed with PDD-NOS than did the remaining sites (Site 2, 53.7%; Site 23, 39.3%; All Others, 22.5%). Also, Site 2 had more children diagnosed with PDD-NOS than autistic disorder (PDD-NOS, n = 57 vs.
autistic disorder, \( n = 43 \). Site 23 did not have more diagnosed with PDD-NOS, but their distribution was closer to equal (PDD-NOS, \( n = 107 \) vs. autistic disorder, \( n = 134 \)) compared to the remaining sites (PDD-NOS, \( n = 503 \) vs. autistic disorder, \( n = 1508 \)).

The ASD subtype breakdown (discussed earlier; also summarized in Table 22) demonstrated that those with PDD-NOS tended to account for more model variance (5.1%) than the children with autistic disorder (3.0%). This is most likely related back to symptom severity; PDD-NOS allows for a greater range of scores due to greater variation in presentation, thus increasing ability to predict variance (Cohen, Cohen, West & Aiken, 2003). Although this is not a large difference, it may help explain why the data from Sites 2 and 23 accounted for more variance in BMI percentile, and it may indicate where we need to look in future studies to maximize prediction.

The disparities seen could be due to human error. Site 2, for example, started off with 320 possible unique cases available, but between multiple visits from the same patients or missing data, eventually only wound up with 106 in the final sample. It is possible that they even did a poor job of administering the required measures to families, perhaps inadvertently filtering out less-than-perfect, but still acceptable, subjects. These sites seem not to have many performance similarities, as demonstrated by the BMI centile averages by variable reported in Table 23. This is particularly true for the ethnicity variables, which varied up to 50+ centile points between sites. Alternatively, perhaps the sites themselves had nothing to do with the variations observed and perhaps the finding was spurious and due to multiple testing. In the context of this dissertation I thought it
was important to be as thorough as possible, although I recognize that I could have violated Type I error.

_Outcome Variable Inquiries_

The last analyses conducted focused on the outcome variable itself, BMI percentile, and addressed the question of whether this was an appropriate choice given that there were other viable options from which to choose. This was accomplished using two different possible outcome variables. The analyses using BMI z-score as the outcome variable was very similar to the atheoretical empiricism model, as it was only able to account for an additional 0.2% of variance. Since z-scores also take age into account, this was not surprising, but was a good exercise to test that the analyses were behaving as expected.

More interesting was the amount of variance that was able to be accounted for using only BMI alone, rather than BMI percentile. This overall model was able to account for 23.6% of variance, as compared to the original amount of 3.6%. As was previously discussed, the primary benefit of using BMI percentile instead of other possible outcome variables is BMI percentile’s ability to account for not only height and weight, but also to take age into account and then rank where children are in comparison to their peers. BMI alone does not incorporate age into its metric. Once age was included in the analysis, only 4.4% of variance remained to be accounted for by demographics, medications, additional psychopathology, sleep disturbances, cognitive and adaptive functioning, and ASD subtype. The additional 4.4% of variance, although
greater than the 3.6% that the atheoretical empiricism model accounted for, is likely a product of different variables entering the model at slightly different stages.

While these outcome variable inquiries were not able to uncover definitive answers to the issue at hand, I found it encouraging that the analyses behaved as expected when using BMI alone. Even though results of this analysis contributed little new knowledge, it gives credence to the data, as they performing as anticipated.

Relation to Existing Literature

The final piece mentioned at the beginning of this Discussion section highlighted the similarities between magnitude of this study’s findings and what has been published. When I began this project, I felt that my model would be effective in predicting children’s BMI percentile. After running my analyses, it became clear that the model was deficient in predicting substantial amounts of variance for BMI percentile. This led me to examine the feasibility of predicting weight status from clinical and demographic variables. Therefore, I conducted a new and fresh evaluation of the literature to examine how successful other investigators have been in light of my own struggles accounting for variance.

I searched on combinations of the following terms: “children,” “child,” “weight,” “overweight,” “obese,” “obesity” unrestricted for time of publication. I looked into some of the published studies on weight in children that used multiple variables to predict weight outcomes and reported useful statistics that could be computed into $R^2$ (variance accounted for; Card, 2012) to determine whether my study was alone in accounting for small variance or if this was a more common problem that resulted through reporting $p$
values instead of amount of variance accounted for. I was able to identify 24 studies (see Table 31) that reported statistics on weight outcomes compared to variables of interest. In many cases, these researchers used logistic regression instead of multiple regression, which produces an odds ratio instead of an $R^2$. There are manipulations that can be done with odds ratios to estimate what the $R^2$ may be, but these require explicit knowledge of degrees of freedom, which most of the articles did not provide.

For the articles that either did report the amount of variance accounted or provided data that could be transformed into such a statistic, it appeared that, they too, found equally small predicted variance. The right column of Table 31 reveals the range of predicted variance, which extended from essentially zero to a high of 41% (Dockray et al., 2008). Most $R^2$s were exceedingly small. Age repeatedly failed to account for large amounts of BMI percentile variance (.2% in De et al., 2008; .8% in Mikulovic et al., 2011), and even medication use in boys was only able to account for 3.9% of variance (Memari et al., 2012). When multiple variables were evaluated simultaneously, results similar to my own were obtained. Rimmer and colleagues (2010) looked at how disability status (autism, cerebral palsy, ID, etc.), sex, and a host of demographic variables were able to predict BMI percentile and found that it was only able to account for 4.2%.
<table>
<thead>
<tr>
<th>Study</th>
<th>Original Statistic Reported</th>
<th>Estimated Variance Accounted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bandini et al., 2005</td>
<td>odds ratios</td>
<td>No df listed, unable to compute $R^2$</td>
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</table>
| Beebe et al., 2007           | $F$, odds ratios            | Sleep actigraph x BMI% $R^2 = .057$◊  
                          |                             | Sleep survey x BMI% $R^2 = .049$†  
                          |                             | Self-report x BMI% $R^2 = .019$ |
| Chandola et al., 2006        | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Chaput et al., 2009          | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Curtin et al., 2010          | odds ratios                 | No df listed, unable to compute $R^2$                           |
| De et al., 2008              | $R$                         | Age x BMI% $R^2 = .002$                                          |
| Dockray et al., 2009         | $R^2$                       | Model with age, pubertal stage, physical activity, depression x BMI alone $R^2 = .41$ (girls), .28 (boys)* |
| Egan et al, 2013             | $\chi^2$                   | Diagnosis x BMI% $R^2 = .029$†  
                          |                             | Adaptive Functioning x BMI $R^2 = .005$                              |
| Emerson & Robertson, 2010    | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Goncalves et al., 2012       | $F$                         | Self-concept x BMI% $R^2 = .061$◊  
                          |                             | Anxiety x BMI % $R^2 = .010$                                     
                          |                             | Depression x BMI% $R^2 = .017$*                                   |
| Goodman & Whitaker, 2002     | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Guo et al., 2002             | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Halkjaer et al., 2003        | odds ratios                 | No df listed, unable to compute $R^2$                           |
| Hendy et al., 2010           | $R$                         | Meal Fussiness x BMI% $R^2 = .007$                             
                          |                             | Special Meal x BMI% $R^2 = .114$*                                |

(continued)
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<tr>
<th>Study</th>
<th>Original Statistic Reported</th>
<th>Estimated Variance Accounted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hernandez et al., 1998</td>
<td>$\chi^2$</td>
<td>Gender x BMI$% R^2 = .001$&lt;br&gt;Ses x BMI$% R^2 = .003$&lt;br&gt;Behavior Probs x BMI$% R^2 = .001$</td>
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<tr>
<td>Kim et al., 2011</td>
<td>odds ratios</td>
<td>No df listed, unable to compute $R^2$</td>
</tr>
<tr>
<td>Lam &amp; Yang, 2007</td>
<td>$F, \chi^2$</td>
<td>ADHD x BMI$% R^2 = .127$&lt;br&gt;Age x BMI$% R^2 = .003$&lt;br&gt;Sex x BMI$% R^2 = .023\dagger$&lt;br&gt;Father Ed x BMI$% R^2 = .012\dagger$&lt;br&gt;Mother Ed x BMI$% R^2 = .012\dagger$&lt;br&gt;Snoring x BMI$% R^2 = .021\dagger$</td>
</tr>
<tr>
<td>Memari et al., 2012</td>
<td>$R$</td>
<td>ASD Severity x BMI$% R^2 = .042\dagger$&lt;br&gt;Meds (girls) x BMI$% R^2 = .255*$&lt;br&gt;Meds (boys) x BMI$% R^2 = .039$&lt;br&gt;Ses x BMI$% R^2 = .076\dagger$</td>
</tr>
<tr>
<td>Mikulovic et al., 2011</td>
<td>$\chi^2$</td>
<td>Age x BMI$% R^2 = .008$&lt;br&gt;Parent Weight x BMI$% R^2 = .023\dagger$</td>
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<tr>
<td>Mustillo et al., 2003</td>
<td>$F$, odds ratios</td>
<td>No df listed, unable to compute $R^2$</td>
</tr>
<tr>
<td>Reilly et al, 2005</td>
<td>odds ratios</td>
<td>No df listed, unable to compute $R^2$</td>
</tr>
<tr>
<td>Rimmer et al., 2010</td>
<td>$F$, odds ratios</td>
<td>Disability, sex, demographics x BMI $R^2 = .042\dagger$</td>
</tr>
<tr>
<td>Waring &amp; Lapane, 2008</td>
<td>odds ratios</td>
<td>No df listed, unable to compute $R^2$</td>
</tr>
<tr>
<td>Yamaki et al., 2011</td>
<td>$\chi^2$</td>
<td>Sex x BMI$% R^2 = .023\dagger$&lt;br&gt;ethnicity x BMI$% R^2 = .003$&lt;br&gt;Age x BMI$% R^2 = .016$&lt;br&gt;Parent Ed x BMI$% R^2 = .076$</td>
</tr>
</tbody>
</table>

*Note.* Significance mentioned is what is recorded in the original article.<br>* = $p < .05$<br>$\dagger = p < .01$<br>$\diamond = p < .001$<br>No symbol indicates analysis failed to reach significance.
Readers should note that one study, by Dockray and colleagues (2009), was able to account for a substantially greater amount of variance than any other included in the table. These researchers accounted for 41% of variance in girl and 28% in boys when evaluating age, pubertal stage, levels of physical activity, and depression, in a hierarchical model similar to what I did. Although this seems impressive, the analysis was run using BMI alone, which failed to adjust for age, rather than BMI percentile (see Appendix A for a visual depiction of BMI as a function of age). They produced a similar outcome to mine when I ran the analysis using BMI alone, and the vast majority of their variance was due to age being included in the analysis instead of being controlled from the outset. Thus, the large variance reported (41% and 28%) was due to age association with BMI rather than the identification of novel relationships. In essence, this was a “non-finding,” since we already know that age plays such a large role in determining BMI in children.

Studies that were able to be transformed into $R^2$ and compared to my own results demonstrated similarly small variance values that were just as clinically modest as my own but, like my study, also were able to establish statistical significance. Reporting statistically significant results without stating how much variance (or similar metric) they are able to account for could be a disservice to the entire field. This may well create the impression that we are currently able to explain far more than is the case. Thus, researchers should be required to report not only relevant probability levels, but also the amount of variance accounted for in the model. Needless to say, publication of BMI outcomes without age correction in children should not be permitted.
Theoretical Model Interpretation

The framework adopted for this dissertation (Figure 5) was influenced by conceptual models of weight status and personal characteristics (Davison & Birch, 2001; Harrison et al., 2011), by supporting evidence from the available scientific literature, and the constraints of an existing data set. Although this model was derived to be testable and was based on previously reported connections between body mass and variables of interest, the model did not perform as expected and failed to account for adequate amounts of variance.

There are a couple of plausible reasons why this model did not perform as planned. Regardless of how much thought and planning went into carefully constructing a model that had empirical support, there could be a major flaw in the model or important elements could be missing. There could be other factors necessary for evaluating increased risk for abnormal weight than were included under the existing schema, or possibly too many extraneous variables that prevented the key variables from emerging. Weight issues appear to be a complex and multifaceted etiology. In this instance, clinical and demographic variables did not appear sufficient to predict substantial outcome.

Study Strengths

Much of the literature relating to children with ASD and weight status up to this point has been limited in terms of sample size and measurement procedures (e.g., use of parental report for height, weight, diagnosis). This project addressed both of these issues. The sample size comprised 2,610 children, which makes it larger than any other weight-related analysis conducted within children with ASD. Furthermore, the sample was
derived from the Autism Treatment Network where height and weight were collected by professionals, as opposed to being derived by parent report. The data were also collected from multiple sites around North America, making the sample fairly representative of the United States and Canada. As previously discussed, data bases that comprise different geographic locations and are run by clinicians are desirable for epidemiological weight-related research (Ogden et al., 2007). Having a nationwide (or in this case, “continent-wide”) network from which to evaluate data was a strength because the sample was large and fairly representative of children with ASD in the US and Canada.

Study Limitations

Although nationwide databases are currently considered one attractive and productive way to evaluate weight-related relationships, this data set was not of my own creation, which limited the variables I was able to investigate. Some important constructs that I would have liked to include in my model (physical vs. sedentary activity, for example) were not available in the database. Furthermore, I had to parse out missing or mishandled data (as in the case of excluding cases with missing CSHQ data). Some of the variables, such as the BMI centile conversions, needed to be done manually. This could have introduced human error, although I took great care to avoid miscalculations.

It is also possible, given the site differences, that there were varying practices at ATN sites. This could have resulted in variable integrity of the data, although I know of no way to resolve whether this was the case. Written standardized procedures for collecting data (be it weight and height or questionnaires) and for entering data into the database would increase confidence in the overall quality of the data, although there
would still be no definitive way to know if these procedures were being followed. Employing independent study monitors to periodically check that sites were following proper protocols would enhance the implementation of study procedures. Finally, although BMI is the usual standard for human weight research, it does have shortcomings as well. These include not being able to differentiate between different masses within the body (fat, muscle, bone, water, etc.) and the problematic nature of child growth, which necessitates that percentile construct be implemented. Hence, supplementation of the BMI data with other “weight” metrics, such as skin-fold thickness, would have enriched the database.

When I realized that the data were not producing the type of results that I was expecting in my initial analysis, I altered my focus in favor of analyzing the variables exhaustively. This deliberate over-testing was with the goal of not missing something important that was hidden in the data in some way that I had not previously conceptualized. Although some members in the field opt to control for multiple tests using statistical corrections (such as a Bonferroni correction), I did not use such a procedure, knowing full well that some analyses could result in spurious findings.

The final and largest limitation is the conceptualization of weight. This dissertation was based on previously published research and reflects the majority of the current knowledge in the field about weight. It is a very large postulation that we truly understand what factors play into weight, how to assess weight across the lifespan, and how to mitigate or reverse the effects of abnormal weight given the unacceptable psychological and medical consequences that may follow. The heterogeneity in the
literature demonstrates that there are a variety of individual variables that could be held accountable, but we lack a satisfactory comprehensive solution to the problem at hand. It is entirely possible that we are not correct in the way we are conceptualizing weight and its correlates, and if that is the case, then this project was headed in the wrong direction from its inception. Although it is not a pleasant thought to believe that much of the field’s knowledge up until this point about predicting weight and risky variables that may lead to future weight problems is incorrect, it bears stating as a potential limitation given the inability to solve the weight riddle.

Conclusions and Future Directions

One of the main reasons for pursuing this project was to identify risk variables that would be productive to target for intervention or prevention programs. These might be employed for either children in general or weight-related health complications in children with ASD. It is well documented in the literature that children who are overweight or obese are more likely than others to become adults with similar weight problems or have higher rates of morbidity and mortality (Wright, Parker, Lamont, & Craft, 2001; Dietz, 1997; Freedman et al., 2004). It seems especially important to identify risk factors for obesity within this specialty population to help identify children who are already significantly disadvantaged. While the study did not identify powerful risk variables, it did confirm some of the findings that we were aware of from the existing literature with typically developing children, and it was able to achieve statistically significant results similar to other previously published works. This similarity between children and adolescents with ASD and typically developing youth is important, in and of
It demonstrates that although these groups tend to have great differences in how they react to the external world, social situations, and life in general, in the case of weight-related issues, there may be fewer differences than previously believed.

As mentioned at the beginning in this section, the message of this project became one of “similarities between the ATN’s ASD sample and previously published weight literature.” The prevalence rates of overweight and obesity were not statistically different between the ASD group and the typically developing reference sample despite considerable statistical power. The results were also statistically similar between this ATN sample and the ASD sample from Curtin and colleagues (2005).

Consistent with previous research, we know that ethnicity and parental education played a role in the weight status of children with ASD. As predicted, various psychotropic medication also influenced overall body mass. Sleep Disordered Breathing was the only sleep disturbance variable to emerge consistently in the models, but we do not know the exact direction of the relationship.

This study also confirmed that comorbid psychopathology can play a role in weight status, albeit not to the extent that was expected. Children with aggressive behavior, and depressed or withdrawn behavior (as defined by the CBCL subscales) were more likely to be heavier. Children who were anxious or depressed were more likely to be lighter than their peers.

This investigation was conceptualized as a preliminary examination into what factors play a role in abnormal weight in hopes that it may help formulate a more treatment-oriented program to address these weight concerns. Future studies would
provide a service by going beyond what was available here and exploring the additional areas identified in the conceptual model presented in the Introduction. These include variables addressing (a) nutritional intake, (b) physical activity or sedentary behavior, and (c) compromised self-regulation. This could be accomplished through the addition of a portable device that could regularly ask parents or caregivers (over a period of a day, week, month, etc.) what foods their children consumed and what physical activities the children participated in. It will be interesting to see if the inclusion of these additional variables leads to better prediction, as it should offer a better profile from which specific goals can be written.

Finally, it is worth noting that a disproportionate part of the study sample was found to have very high BMI percentile. This is demonstrated in Appendix I (p. 172), where a large number of subjects were found to be within the bar reflecting the highest BMI percentiles. One question that naturally arises is whether there were clinical and demographic variables that distinguished these extreme cases from the remaining participants in the histogram. Another question revolves around whether there were important subgroups among the children comprising the very highest percentiles. Although there was not scope in this dissertation to explore these inquires, they would be an interesting avenue for future exploration.

This study also highlights the role that age may play when evaluating abnormal weight. The only true analysis where this variable became statistically significant was that using the CBCL 6-18 year version. This suggests that younger children may be
protected by the fact that they had less time to deviate from their expected weight categories.

Though it is encouraging that children with ASD do not experience higher rates of overweight and obesity in comparison to their typically developing peers, both groups are well above the CDC’s assumptions of 15% of children in the overweight category and 5% of children in the obese weight category. Overweight and obesity are complicated issues due to their multifaceted nature, but helping children with ASD to avoid the psychological and physical hardships that accompany the conditions is ample incentive to investigate further. We know that future interventions with individuals with ASD need to focus on preventing additional weight gain. This is preferred over striving to lose weight, except in the case of extreme obesity or severe health complications (e.g., Hamilton et al., 2007). This will allow children to maintain their normal growth trajectory in regards to height, which should reduce their overall BMI (from growing taller but not gaining more weight above their starting amount) without the added stress of intense diet and exercise programs. Future treatment programs need to be accessible to parents and caregivers and be easy to implement. These families already have the added burden of having a child with a disability who likely requires specialized therapies and interventions, so weight reduction programs need to be straightforward and easy to integrate into everyday life. We as professionals, caregivers, researchers, and members of the IDD community need to acknowledge that weight is not just an issue for the mainstream in our society, but rather, that it is a problem for people from all walks of life including those with ASD. Therefore, we should be doing everything we can to help these people, including making
health promotion programs available to children with ASD and their families. Abnormal weight status has become the norm for our country, but it must not become the standard for children with disabilities such as ASD.
REFERENCES

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Centers for Disease Control and Prevention. (2012). CDC estimates 1 in 88 children has been identified as having an autism spectrum disorder [Press release]. http://www.cdc.gov/media/releases/2012/p0329_autism_disorder.html


deficit/hyperactivity disorder: I. Efficacy and tolerability outcomes. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*(2), 180-188.


Appendix A: CDC Growth Chart for Boys and Girls

2 to 20 years: Boys

<table>
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<tr>
<th>NAME</th>
<th>RECORD #</th>
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<table>
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<tr>
<th>Age (Years)</th>
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<th>Weight (kg)</th>
<th>Stature (cm)</th>
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*To calculate BMI: Weight (kg) + Stature (cm) + Stature (cm) x 10,000 or Weight (lb) + Stature (in) + Stature (in) x 703

Published May 30, 2000 (modified 10/16/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

Safer, Healthier People.
Sabrina Grondhuis <grondhuis.1@buckeyemail.osu.edu>  Mon, Aug 5, 2013 at 9:18 AM
To: cogden@cdc.gov
Hello Dr. Ogden,

I am working on my dissertation about weight in youth with autism spectrum disorders and have a clarifying question for you about your presentation of overweight in some of your publications (Ogden et al., 2002; Ogden et al., 2006; Ogden et al., 2010). You state that overweight is when a child's weight is between the 85th and 95th percentile, but then consistently present overweight and obesity rates together ( > 85th percentile) in tables rather than make the distinction of overweight alone. Is this for convenience or some other methodology that I am overlooking?

I would like my presentation of findings to be consistent with the available literature, and recognize that these values are being shown in a variety of ways. If you could shed some light as to why you chose the combined presentation rather than overweight and obesity separately I would appreciate it.

Thank you for your time,
Sabrina Grondhuis

Ogden, Cynthia L. (CDC/OSELS/NCHS)  Mon, Aug 19, 2013 at 2:08 PM
To: Sabrina Grondhuis <grondhuis.1@buckeyemail.osu.edu>

You can present any way you like
either overweight or obese (>=85th percentile)
Or overweight between 85th and 95th
And obese (>=95)

Cynthia L. Ogden, PhD, MRP
Nutritional Epidemiologist and Analysis Branch Chief
National Health and Nutrition Examination Survey (NHANES)
CDC/National Center for Health Statistics/3311 Toledo Road Room 4415/Hyattsville, MD COgden@cdc.gov
Appendix C: Instrument List for the ATN

Below is a list of measures that are supposed to be completed for every child whose data gets incorporated into the ATN database. Measures marked with an asterisk were used in this dissertation. For additional information about what sections are collected, what specifically is recorded into the database, please see the following link. http://www.autismspeaks.org/docs/sciencedocs/atn/as-atn_assesment_battery.pdf

Autism Diagnostic Observation Schedule – 2 (ADOS)*
DSM-IV Symptom Checklist*
Mullen Scales of Early Learning*
Bayley Scales of Infant Development, 3rd Edition
Stanford-Binet Intelligence Scales, 5th Edition*
Differential Ability Scales II (DAS II)
Wechsler Preschool and Primary Scale of Intelligence, 3rd Edition (WPPSI-III)*
Wechsler Intelligence Scale for Children – 4th Edition (WISC-IV)*
Wechsler Abbreviated Scale of Intelligence (WASI)*
Leiter International Performance Scale-Revised (Leiter-R)*
Vineland Adaptive Behavior Scale – II (VABS-II)*
Child Behavior Checklist (CBCL)*
Aberrant Behavior Checklist (ABC)
Pediatric Quality of Life (PedsQL)
Clinical Evaluation of Language Fundamentals, 4th Edition (CELF-IV)
Pre-school Clinical Evaluation of Language Fundamentals, 2nd Edition (CELF P2)
Preschool Language Scale, 5th Edition (PSL-V)
Oral and Written Language Scales: Listening Comprehension and Oral Expression Scales (OWLS)
Appendix D: Children’s Sleep Habits Questionnaire (CSHQ) Subscale Items

1. **Bedtime Resistance** (6 items)
   - Goes to bed at same time (1) (R)
   - Falls asleep in own bed (3) (R)
   - Falls asleep in other’s bed (4)
   - Needs parent in room to sleep (5)
   - Struggles at bedtime (6)
   - Afraid of sleeping alone (8)

2. **Sleep Onset Delay** (1 item)
   - Falls asleep in 20 minutes (2) (R)

3. **Sleep Duration** (3 items)
   - Sleeps too little (9)
   - Sleeps the right amount (10) (R)
   - Sleeps same amount each day (11) (R)

4. **Sleep Anxiety** (4 items)
   - Needs parent in room to sleep (5)
   - Afraid of sleeping in the dark (7)
   - Afraid of sleeping alone (8)
   - Trouble sleeping away (21)

5. **Night Waking** (3 items)
   - Moves to other’s bed in night (16)
   - Awakes once during night (24)
   - Awakes more than once (25)

6. **Parasomnias** (7 items)
   - Wets the bed at night (12)
   - Talks during sleep (13)
   - Restless and moves a lot (14)
   - Sleepwalks (15)
   - Grinds teeth during sleep (17)
   - Awakens screaming, sweating (22)
   - Alarmed by scary dream (23)

7. **Sleep Disordered Breathing** (3 items)
   - Snores loudly (18)
   - Stops breathing (19)
   - Snorts and gasps (20)

8. **Daytime Sleepiness** (8 items)
   - Wakes by himself (26) (R)
   - Wakes up in negative mood (27)
   - Others wake child (28)
   - Hard time getting out of bed (29)
   - Takes long time to be alert (30)
   - Seems tired (31)
   - Watching TV (32)
   - Riding in car (33)

**Total Sleep Disturbance Score (33 items)**

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*Note:* Some items (R) should be reversed in scoring, so that a higher score reflects more disturbed sleep behavior. Number in parentheses refers to CSHQ item number.

*Note:* The Total Sleep Disturbance Score consists of all 33 subscale items instead of 35 (although items 5 and 9 are on both the Bedtime Resistance and Sleep Anxiety scales, they should be included only once in the total score).

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Appendix E: Questions Used for Latent Nutritional Deficits Variable

From Diagnosis and Problems (Dx1), which is completed by medical personnel (RN, NP, physician, etc.). Answer choices include: No, Yes, or Unsure and questions are given as “Has your child experienced __________.”

1. D1FEEDIF: Feeding difficulty
2. D1PICA: Pica
3. D1OTNUTR: Other nutrition problem
4. D1EATDON: Eating Disorder – Not Otherwise Specified
5. D1FOOINT: Food intolerance
6. D1FEEDO: Feeding Disorder
7. D1FED3MO: Feeding different in the last three months
8. D1FEEDWE: Feeding different in the past week
9. D1OTNU3M: Other nutrition problem in the last three months
10. D1OTNUWE: Other nutrition problem in the past week
Appendix F: Factor Models

Factor Analysis: One Factor Model with all 10 Variables
Factor Analysis: Two Factor Model with all 10 Variables
### Appendix G: Transformation Results

*Summary of hierarchical regression analysis predicting BMI centile with square root transformations*

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Note: * indicates that this variable, along with the BMI centile outcome variable, was transformed using the square root transformation

Overview of hierarchical regression analysis predicting BMI centile with square root transformations

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Summary of hierarchical regression analysis predicting BMI centile with Lg10 transformation

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Note: * indicates that this variable, along with the BMI centile outcome variable, was transformed using the Lg10 transformation

Overview of hierarchical regression analysis predicting BMI centile with Lg10 transformation

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<thead>
<tr>
<th></th>
<th>$R^2$</th>
<th>Adjusted $R^2$</th>
<th>$\Delta R^2$</th>
<th>Change $F(p)$</th>
<th>Overall $F(p)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td>.009</td>
<td>.005</td>
<td>---</td>
<td>---</td>
<td>2.794 (.004)</td>
</tr>
<tr>
<td>Block 2</td>
<td>.024</td>
<td>.018</td>
<td>.016</td>
<td>4.626 (&lt;.001)</td>
<td>3.780 (&lt;.001)</td>
</tr>
<tr>
<td>Block 3</td>
<td>.032</td>
<td>.023</td>
<td>.008</td>
<td>3.002 (.004)</td>
<td>3.567 (&lt;.001)</td>
</tr>
<tr>
<td>Block 4</td>
<td>.036</td>
<td>.024</td>
<td>.004</td>
<td>1.454 (.169)</td>
<td>3.043 (&lt;.001)</td>
</tr>
<tr>
<td>Block 5</td>
<td>.037</td>
<td>.024</td>
<td>.001</td>
<td>.771 (.463)</td>
<td>2.909 (&lt;.001)</td>
</tr>
<tr>
<td>Block 6</td>
<td>.039</td>
<td>.026</td>
<td>.002</td>
<td>6.009 (.014)</td>
<td>3.003 (&lt;.001)</td>
</tr>
</tbody>
</table>
### Appendix H: Descriptive Characteristics for Site 2, Site 23, and Remaining Sample

Mean values of variables predictive of weight for the sites (2, 23) that demonstrated good model fit and the remaining sample; mean (standard deviation)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Site 2 (n = 106)</th>
<th>Site 23 (n = 272)</th>
<th>All Others (n = 2,232)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (months)</td>
<td>79.83 (33.35)</td>
<td>80.76 (44.00)</td>
<td>75.38 (39.77)</td>
</tr>
<tr>
<td>Sleep Onset Delay</td>
<td>1.57 (0.72)</td>
<td>1.76 (0.80)</td>
<td>1.74 (0.80)</td>
</tr>
<tr>
<td>Sleep Anxiety</td>
<td>6.33 (2.17)</td>
<td>6.20 (2.08)</td>
<td>6.10 (2.06)</td>
</tr>
<tr>
<td>Parasomnias</td>
<td>9.43 (1.95)</td>
<td>9.54 (2.03)</td>
<td>9.58 (2.14)</td>
</tr>
<tr>
<td>Sleep Disordered Breathing</td>
<td>3.62 (1.02)</td>
<td>3.43 (0.89)</td>
<td>3.46 (0.92)</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td>63.36 (10.36)</td>
<td>61.31 (10.53)</td>
<td>61.06 (10.36)</td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>60.24 (8.89)</td>
<td>60.20 (10.26)</td>
<td>61.01 (10.01)</td>
</tr>
<tr>
<td>Anxious/Depressed</td>
<td>58.35 (9.45)</td>
<td>58.03 (9.13)</td>
<td>58.73 (9.32)</td>
</tr>
<tr>
<td>Withdrawn/Depressed</td>
<td>64.13 (9.67)*</td>
<td>67.73 (10.07)</td>
<td>68.08 (10.30)</td>
</tr>
</tbody>
</table>

*Note:* Sleep Onset Delay range was 1-3.
Sleep Anxiety range was 4-12.
Parasomnias range was 7-19.
Sleep Disordered Breathing Range was 3-9.
For the comorbid psychopathology, the values are in t-scores.
* = p < .001
Percentage of variables predictive of weight for the sites that demonstrated good model fit and the remaining sample

<table>
<thead>
<tr>
<th></th>
<th>Site 2 (n = 106)</th>
<th>Site 23 (n = 272)</th>
<th>All Others (n = 2,232)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>82.70% (86)</td>
<td>82.40% (224)</td>
<td>84.80% (1892)</td>
</tr>
<tr>
<td>Female</td>
<td>17.30% (18)</td>
<td>17.60% (48)</td>
<td>15.20% (340)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>79.25% (84)</td>
<td>83.82% (228)</td>
<td>74.00% (1652)</td>
</tr>
<tr>
<td>African American</td>
<td>4.72% (5)</td>
<td>11.03% (30)</td>
<td>8.30% (186)</td>
</tr>
<tr>
<td>Asian</td>
<td>4.72% (5)</td>
<td>1.47% (4)*</td>
<td>6.70% (149)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>8.49% (9)</td>
<td>2.94% (8)*</td>
<td>7.70% (171)</td>
</tr>
<tr>
<td>Other Ethnicity</td>
<td>2.83% (3)</td>
<td>0.74% (2)*</td>
<td>3.30% (74)</td>
</tr>
<tr>
<td>Father’s Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent or No Info</td>
<td>8.49% (9)</td>
<td>7.35% (20)**</td>
<td>13.00% (290)</td>
</tr>
<tr>
<td>Less than 8\textsuperscript{th} Grade</td>
<td>0.94% (1)</td>
<td>N/A (0)</td>
<td>0.80% (18)</td>
</tr>
<tr>
<td>Some high school</td>
<td>1.90% (2)</td>
<td>1.84% (5)</td>
<td>3.80% (84)</td>
</tr>
<tr>
<td>High school diploma</td>
<td>14.15% (15)</td>
<td>29.78% (81)**</td>
<td>18.70% (417)</td>
</tr>
<tr>
<td>Some college</td>
<td>15.09% (16)</td>
<td>15.81% (43)**</td>
<td>23.90% (533)</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>38.68% (41)**</td>
<td>29.78% (81)</td>
<td>23.40% (522)</td>
</tr>
<tr>
<td>Graduate degree</td>
<td>20.75% (22)</td>
<td>15.44% (42)</td>
<td>16.50% (368)</td>
</tr>
<tr>
<td>Stimulant</td>
<td>9.43% (10)</td>
<td>11.40% (31)</td>
<td>10.40% (232)</td>
</tr>
<tr>
<td>Antihistamine</td>
<td>10.37% (11)</td>
<td>7.40% (20)</td>
<td>9.00% (201)</td>
</tr>
<tr>
<td>Melatonin</td>
<td>10.37% (11)</td>
<td>11.00% (30)</td>
<td>9.90% (222)</td>
</tr>
<tr>
<td>Atomoxetine</td>
<td>0.94% (1)</td>
<td>7.00% (19)**</td>
<td>1.30% (29)</td>
</tr>
<tr>
<td>SSRIs</td>
<td>7.55% (8)</td>
<td>7.40% (20)</td>
<td>5.50% (123)</td>
</tr>
<tr>
<td>Alpha 2 Agonists</td>
<td>8.49% (9)</td>
<td>6.60% (18)</td>
<td>5.70% (129)</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>7.54% (8)**</td>
<td>4.80% (13)**</td>
<td>2.00% (45)</td>
</tr>
<tr>
<td>Atypical Antipsychotics</td>
<td>6.60% (7)</td>
<td>8.10% (22)</td>
<td>5.40% (121)</td>
</tr>
<tr>
<td>Other Medication</td>
<td>33.70% (35)**</td>
<td>32.40% (85)**</td>
<td>24.10% (515)</td>
</tr>
<tr>
<td>Autistic Disorder</td>
<td>40.56% (43)**</td>
<td>49.30% (134)**</td>
<td>67.60% (1510)</td>
</tr>
<tr>
<td>Asperger Syndrome</td>
<td>5.66% (6)</td>
<td>11.40% (31)</td>
<td>9.90% (220)</td>
</tr>
<tr>
<td>PDD-NOS</td>
<td>53.77% (57)**</td>
<td>39.30% (107)**</td>
<td>22.50%</td>
</tr>
</tbody>
</table>

*Note:* The first numeral in each column is the percentage of participants in that group. The sample sizes for each group are in parentheses

*: \( p = .002 \)

**: \( p < .001 \)
Appendix I: Variable Histograms

Histogram: Age in Months

Skewness: 1.031 (Standard Error = 0.048)
Kurtosis: 0.396 (Standard Error = 0.096)
Skewness: -0.586 (SE = 0.048)

Kurtosis: -0.986 (SE = 0.096)
Skewness: 0.831 (SE = 0.048)

Kurtosis: 0.110 (SE = 0.096)
Histogram: CSHQ Bedtime Resistance

Skewness: 0.834 (SE = 0.048)
Kurtosis: =0.512 (SE = 0.096)
Skewness: 0.519 (SE = 0.048)
Kurtosis: -1.249 (SE = 0.096)
Skewness: 1.107 (SE = 0.048)

Kurtosis: 0.030 (SE = 0.096)
Skewness: 0.867 (SE = 0.048)
Kurtosis: -0.013 (SE = 0.96)
Skewness: 0.937 (SE = 0.048)

Kurtosis: 2.373 (SE = 0.096)
Histogram: CSHQ Parasomnias

Skewness: 0.972 (SE = 0.048)

Kurtosis: 0.980 (SE 0.096)
Skewness: 2.638 (SE = 0.048)

Kurtosis: 8.274 (SE = 0.096)
Skewness: 0.667 (SE = 0.048)
Kurtosis: 0.043 (SE = 0.096)
Histogram: CBCL Affective Problems

Skewness: 0.385 (SE = 0.048)

Kurtosis: 0.604 (SE = 0.096)
Skewness: 0.987 (SE = 0.048)
Kurtosis: 0.658 (SE = 0.096)
Histogram: CBCL Anxiety Problems

Skewness: 0.596 (SE = 0.048)
Kurtosis: -0.353 (SE = 0.096)
Skewness: 1.113 (SE = 0.048)

Kurtosis: 0.723 (SE = 0.096)
Skewness: 0.234 (SE = 0.048)

Kurtosis: -1.019 (SE = 0.096)
Skewness: 0.805 (SE = 0.074)

Kurtosis: -0.021 (SE = 0.147)
Histogram: CBCL Emotional Problems

Skewness: 0.808 (SE = 0.063)

Kurtosis: 0.460 (SE = 0.127)
Histogram: CBCL Oppositional Behavior

Skewness: 0.644 (SE = 0.048)

Kurtosis: -0.765 (SE = 0.096)
Histogram: CBCL Somatic Complaints

Skewness: 0.762 (SE = 0.048)

Kurtosis: 0.063 (SE = 0.096)
Skewness: -0.353 (SE = 0.074)

Kurtosis: -0.506 (SE = 0.147)
Skewness: 0.599 (SE = 0.074)

Kurtosis: 0.064 (SE = 0.147)
Skewness: -0.292 (SE = 0.063)

Kurtosis: -0.033 (SE = 0.126)
Histogram: CBCL Rule-Breaking Behavior

Skewness: 1.026 (SE = 0.074)

Kurtosis: 0.282 (SE = 0.147)
Appendix I: Variable Scatterplots

Scatterplot: BMI Centile and Age
Scatterplot: BMI Centile and Ethnicity

- White
- Black
- Asian
- Hispanic
- Other
Scatterplot: BMI Centile and Paternal Education

BMI/centile

MALEED
Scatterplot: BMI Centile and Stimulants
Scatterplot: BMI Centile and Antihistamines
Scatterplot: BMI Centile and Melatonin
Scatterplot: BMI Centile and SSRIs
Scatterplot: BMI Centile and Alpha 2 Agonists
Scatterplot: BMI Centile and Atypical Antipsychotics
Scatterplot: BMI Centile and CBCL Affective Problems
Scatterplot: BMI Centile and CBCL Aggressive Behavior
Scatterplots: BMI Centile and Anxiety Problems
Scatterplots: BMI Centile and CBCL Anxious/Depressed
Scatterplot: BMI Centile and CBCL Emotional Problems
Scatterplot: BMI Centile and DBD (CBCL Conduct and ODD subscales combined)
Scatterplot: BMI Centile and CBCL Social Problems
Scatterplot: BMI Centile and CBCL Somatic Complaints
Scatterplot: BMI Centile and CBCL Thought Problems
Scatterplot: BMI Centile and CBCL Withdrawn/Depressed
Scatterplot: BMI Centile and CSHQ Bedtime Resistance
Scatterplot: BMI Centile and CSHQ Sleep Onset Delay
Scatterplot: BMI Centile and CSHQ Sleep Duration
Scatterplot: BMI Centile and CSHQ Sleep Anxiety
Scatterplot: BMI Centile and CSHQ Night Waking
Scatterplot: BMI Centile and CSHQ Daytime Sleepiness
Scatterplot: BMI Centile and IQ Score
Scatterplot: BMI Centile and Adaptive Behavior Composite Score