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Does Growth in Childhood ADHD and Depression Symptoms Predict Binge-Eating During Adolescence

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DOES GROWTH IN CHILDHOOD ADHD AND DEPRESSION SYMPTOMS PREDICT BINGE-EATING DURING ADOLESCENCE?

by

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Dedication

I would like to dedicate this document to children who experience mental health symptoms that interfere with their daily functioning, especially children with ADHD and depression symptoms. My hope is that research endeavors such as this one can contribute to the understanding of how and why youth experience these symptoms and accompanying impairment. Through the accumulation and application of this knowledge, I hope that we can identification of effective strategies to prevent and reduce the impact mental health symptoms have on outcomes later in life.

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Abstract

Binge-eating disorder (BED) refers to experiencing a loss of control while eating high quantities of food in a short period of time. A serious public health concern, BED is highly comorbid with other psychological disorders and increases risk for obesity and other health concerns, such as metabolic disorder and diabetes. Little is known about what mental health symptoms contribute to the development of BED for children and adolescents. Research with adults indicates that two strong predictors of binge-eating behavior include impulsivity and depression, and these symptom areas may contribute to BED for youth, as well. In the current study, I evaluated the extent to which ADHD symptoms, depression symptoms, and these symptoms together predict binge-eating symptoms.

Further, the degree to which developmental patterns of symptom severity for both ADHD and depression symptoms remain consistent from childhood to adolescence is also unclear. Although current research indicates that ADHD is a chronic, lifelong behavioral disorder, some research indicates that some youth may display onset of ADHD as late as 12 years of age and other findings indicate that some children display remission of symptoms as they reach adolescence. Thus, the degree to which ADHD remains a consistent diagnosis across childhood and adolescence appears unclear. Likewise, the correspondence between childhood and adolescent symptoms of depression also is unclear. Risk for depression escalates during adolescence, particularly for girls, but the degree to which those who develop clinical levels of depression symptoms in

adolescence demonstrated sub-threshold depression symptoms in childhood is unclear. To evaluate these questions, I evaluated the degree to which childhood symptom severity was consistent with adolescent symptom severity or transitioned to different levels of symptom severity.

Data for these analyses came from the Johns Hopkins Field Trial, a longitudinal study on school-based prevention programs (N = 678; age at initial assessment: M = 6.2, SD = .34; 46.8% female; 86.8% African-American; 63.4% received free or reduced lunch). To assess this study's hypotheses, teacher-reported ADHD symptoms and child self-reported depression symptoms assessed at four time points in each developmental period (childhood: fall and spring of first grade, second grade, and third grade; adolescence: sixth, seventh, eighth, and ninth grades) represented the initial level and change in ADHD and depression symptoms. Independent latent class analyses were used to identify the fewest groupings that best represent the individual differences in the intercepts and slopes of ADHD and depression symptoms during childhood and adolescence. These four latent class models were then combined within a transition model to identify the extent to which childhood symptoms classes predicted adolescent symptom classes. Symptom classes from each latent class model were then used to predict binge-eating symptoms in tenth grade.

Latent class growth modeling with ADHD and depression symptoms in childhood and adolescence indicated that three classes best fit childhood ADHD symptoms and adolescent depression symptoms, whereas two classes best fit childhood depression symptoms and adolescent ADHD symptoms. The full transition model resulted in two classes for childhood and adolescent depression and adolescent ADHD symptoms,

whereas three classes continued to fit childhood ADHD symptoms the best. Both ADHD and depression symptoms displayed strong correspondence from childhood and adolescence, although ADHD and depression symptom classes did not predict each other across development periods.

Childhood and adolescent ADHD symptom classes but not depression symptom classes predicted 10th grade binge-eating behaviors. The "high" ADHD symptoms class from childhood had higher binge-eating symptoms than the "increasing moderate" or "low" childhood ADHD symptoms classes. During adolescence, the "high" ADHD symptoms class also displayed more binge-eating symptoms than the "low" adolescent ADHD symptoms class. Further, childhood and adolescent ADHD symptom classes interacted, where binge-eating behaviors were highest for those who were members of both the "high" childhood and "high" adolescent ADHD symptoms classes.

These results indicate that elevated impulsivity and inattention symptoms create vulnerability for binge-eating behaviors. Further research is necessary to identify the mechanisms that contribute to increased binge-eating for children and adolescents with high ADHD symptoms.

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List of Abbreviations

AIC.....Aikake Information Criteria

ADHD..... Attention Deficit/Hyperactivity Disorder

ADHD1..... Childhood Attention Deficit/Hyperactivity Disorder

ADHD2..... Adolescent Attention Deficit/Hyperactivity Disorder

BIC.....Bayesian Information Criteria

BED..... Binge-Eating Disorder

BN..... Bulimia Nervosa

AN..... Anorexia Nervosa

BLRT .....Bootstrapped Likelihood Ratio Test

CFA.....Confirmatory Factor Analysis

DEP .....Depression

DEP1 .....Childhood Depression

DEP2 .....Adolescent Depression

GBG .....Good Behavior Game Intervention

FCI .....Family-Centered Intervention

LMR-LRT .....Lo-Mendell-Rubin Likelihood-Ratio Test

MLR.....Maximum Likelihood Robust Estimation

SRMR .....Standardized Root Mean Square Residual

RMSEA..... Root Mean Square Error of Approximation

Chapter 1: Introduction

Binge-eating disorder (BED) refers to experiencing a loss of control while eating high quantities of food in a short period of time. Although the age of onset usually occurs around 25 years of age, BED affects 1% to 3% of children and adolescents (Ackard, Neumark-Sztainer, Story, & Perry, 2003). It is a serious public health concern given that it is highly comorbid with other psychological disorders for approximately 78.9% of individuals with BED (Hudson, Hiripi, Pope, & Kessler, 2007; Wilfley et al., 2000). Further, children who just display BED symptoms rather than meeting criteria for the disorder experience significant risk for obesity and metabolic disease in adulthood (Hasler et al., 2004; Hudson et al., 2010; Tanofsky-Kraff et al., 2006; Tanofsky-Kraff et al., 2009; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). BED also complicates health problems associated with obesity for children. Obese children and adolescents with BED display less weight loss during treatment and increased health problems compared to those who are obese but without BED (Tanofsky-Kraff et al., 2012; Wonderlich et al., 2009). Despite the serious mental and physical health outcomes associated with BED, few studies have used prospective designs to evaluate the factors that contribute to the development of BED and binge-eating during childhood or adolescence.

Research with adults indicates that two strong predictors of binge-eating behavior include impulsivity and depression (Goosens, Braet, Verbeken, Decaluwé, & Bosmans, 2011; Hartmann, Czaja, Rief, & Hilbert, 2010; Tanofsky-Kraff et al., 2012). Given that impulsivity is a primary symptom of Attention Deficit/Hyperactivity Disorder (ADHD;

American Psychiatric Association (APA), 2000), youth with ADHD might be at greater risk for binge-eating than those without ADHD. Affecting about 7% to 10% of school age children and adolescents (Froehlich et al., 2007), ADHD is associated with deficits in reward processing (Anokhin, Golosheykin, Grant, & Heath, 2011; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009) and difficulty coping with negative affect (Seymour et al., 2012). Given that reward processing deficits and intolerance of negative affect figure prominently in theories of how binge-eating develops (Headt-Matt & Keel, 2011; Dawe & Loxton, 2004), these deficit areas may create vulnerability for binge-eating for children and adolescents with ADHD symptoms.

Depression is the most commonly comorbid disorder with BED with nearly 50% of children and adolescents with BED also meeting criteria for a depression diagnosis (Shisslak et al., 2006). Thus, it seems likely that depression would play an important role in the development of BED and several studies also indicate that depression predicts increases in BED symptoms (Goossens et al., 2011; Skinner, Haines, Austin, & Field, 2012; Spoor et al., 2006; Tanofsky-Kraff et al., 2011).

It is also possible that individuals with both ADHD and depression symptoms display a greater risk for engaging in BED behaviors than those with only ADHD or depression symptoms. Nearly 20–30% of children with ADHD meet criteria for depression, and adolescents with ADHD appear to experience a 5.5 times greater risk of developing a depressive disorder than adolescents without ADHD (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Difficulty coping with negative emotions may contribute to the link between ADHD and depression (Seymour et al., 2012) and lead adolescents with ADHD to impulsively engage in maladaptive behaviors, such as binge-

eating, to relieve the distress of negative emotions. Given the role of negative affect in binge-eating and the increased risk of developing depression for children and adolescents with ADHD, ADHD and depression symptoms may together increase risk for binge-eating more than either symptom area alone.

In the proposed study, I evaluated the extent to which ADHD symptoms, depression symptoms, and these symptoms together predict binge-eating symptoms. To assess these associations, data from the Johns Hopkins Field Trial were used to evaluate a latent growth class transition model of ADHD and depression symptoms during childhood and adolescence and the degree to which symptoms classes predicted binge-eating behaviors. Four separate latent class growth models were evaluated identifying the development of ADHD and depression symptoms in childhood (e.g., fall of first grade, spring of first grade, second grade, and third grade) and adolescence (e.g., sixth through ninth grade). These four latent class growth models were joined in a latent transition model to identify the extent to which childhood symptom classes predicting membership in adoelscent symptom classes. Finally, symptom classes from the four latent growth models were used in separate analyses to predict binge-eating behaviors from tenth grade.

The introduction of this prospectus provides a review of the literature to identify the support for these associations in previous research. This review begins with discussion about the symptoms, prevalence, health outcomes, and psychiatric comorbidities associated with binge-eating. After describing theories that explain how binge-eating develops, literature regarding the link between ADHD and binge-eating symptoms is presented followed by research findings regarding the role of depression in the development of binge-eating. I then present research findings that explain and support

the possibility that ADHD and depression symptoms together have a stronger effect on binge-eating than either symptom area alone. The proposed study section details the research questions that are addressed in the proposed study as well as the expected results. In the methods section, I present the known characteristics of the participants, study procedures, and measures relevant to the proposed study and describe the analytic plan. The results of model development and findings that resulted from the model are presented in the results section and these findings are reviewed in the context of previous research in the discussion section. Figures and tables relevant for each section are reported at the end of each section. Appendices contain the questionnaires used to collect primary constructs for this study.

1.1 Binge-Eating Disorder among Children and Adolescents

According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; APA, 2000), diagnostic criteria for Binge-Eating Disorder require that a binge-eating episode occurs within a window of two hours that involves consumption of a significant amount of food and the experience of lacking control over eating during this period of time. The individual must also display three or more of the following symptoms related to binge-eating: eating faster than usual, feeling uncomfortably full, overeating when not physically hungry, overeating only when alone due to embarrassment, feelings of disgust, guilt, shame, or depressed mood after overeating, and noticeable distress about overeating. Currently, individuals must binge at least two days a week for six months to meet criteria for BED. Also, to rule-out bulimia, binge-eating should not be accompanied by compensatory behaviors, such as purging, fasting, or compulsive exercising.

As the research base on BED expands, several researchers have called for changes in criteria for the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V; APA, 2013). BED was located in the appendix of the DSM-IV, but in DSM-V, BED is identified as a formal mental health diagnosis. Also, the threshold of binge frequency that meets criteria for BED was lowered to one binge per week for three months (for review of new criteria and background research, see Bravender et al., 2010; Striegel-Moore & Franko, 2008; Wonderlich et al., 2009). Some research indicates that changes to the criteria for children and adolescence may be warranted as well, although these changes are not represented in the DSM-V. Specifically, the low prevalence rate and validity concerns about child-report of binge episodes have led some researchers to suggest amending the criteria for children to include subjective binge-eating episodes characterized by experiencing loss of control while eating (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Research indicates that children who engage in binge-eating often struggle to remember or accurately report the amount of food that they consume during a binge (objective binge episode; Field, Taylor, Celio, & Colditz, 2004; Tanofsky-Kraff et al., 2003). Another issue that may undermine the usefulness of measuring objective binge episodes for children is that parent monitoring and expectations that parents set with children regarding food access and consumption may prevent a child from accessing a large enough quantity of food to meet the criteria of an objectively large quantity of food. Further, the experience of loss of control while eating appears to be a stronger predictor of BED for children four years later than objective binge episodes (Tanofsky-Kraff et al., 2011). Given these findings, the proposed study included measurement of BED symptoms that focus on affective

experiences while eating and thoughts about eating rather than the frequency and portions of food consumed during binge episodes.

Although, Bulimia Nervosa, which refers to binge-eating with compensatory behaviors to reduce weight gain and changes in shape from eating habits, appears conceptually similar to BED, important differences between the disorders exist. Primarily, the difference between BED and bulimia is that BED does not involve post-binge compensatory behaviors, whereas bulimia does. Bulimia criteria also include concern related to body shape and weight, whereas BED criteria do not. Current research provides inconsistent findings regarding associations between body dissatisfaction and BED symptoms (Goossens et al., 2011; but see Grilo, Hrabosky, White, Allison, & Stunkard, 2008; Masheb & Grilo, 2000), indicating that further research is necessary to clarify the nature of shape and weight concerns for BED. Other differences and similarities may exist between these disorders as well, yet the limited research on BED makes the correspondence between them unclear. Further research is necessary to identify the extent to which these disorders differ.

Prevalence of Binge-Eating Disorder. Most of the research on BED has occurred with samples of adult, European-American women. Although some research exists regarding the prevalence of BED in children and adolescents, research on differences in rates of BED based on gender and ethnicity has been insufficient. Further, questions remain about how BED develops and the frequency of binge-eating behavior for children and adolescents in general. Johnson, Rohan, and Kirk (2002) found that only 1% of a community sample (N = 822) of adolescents in sixth through twelfth grade met criteria for BED, which was replicated by Decalwue and Braet (2003). Results from a school-

based sample indicated that 3.1% of girls and .9% of boys met criteria for BED (Ackard et al., 2003), whereas about1% of adolescent girls (*N* = 849) met criteria for BED in a nationally representative sample (Stice, Marti, Shaw, & Jaconis, 2011).

Meeting criteria for BED appears to occur infrequently, but the presence of sub-threshold BED symptoms appear more frequently for children and adolescents than the rate of diagnosis. Prevalence of sub-threshold BED symptoms ranges widely depending on the characteristics of the population in the study and the definition used for sub-threshold BED symptoms. Stice and colleagues (2011) found that 4.6% of adolescent girls met sub-threshold BED criteria, which, in this study, referred to reducing the cut-off for the frequency of binge behavior from eight to two binge-eating episodes within a six month span of time. Similarly, Ackard and colleagues (2003) found that 7.9% of girls and 2.4% of boys displayed sub-threshold BED. One study with a large representative sample (*N* = 16,000) of children aged 9 – 14 years of age found that only 1.9% of girls and .8% of boys had engaged in binge-eating at least once a month (Field et al., 1999). Another study (*N* = 6,728) identified that about 4% of the adolescent males (grades 5 to 12) participated in binge-eating daily, compared to nearly 9% of the females (Neumark-Sztainer & Hannan, 2000).Other research indicates that up to 18.5% of adolescents have engaged in infrequent binge-eating (Johnson et al., 2002). Research on the prevalence of "loss of control" while eating indicates that 20% – 40% (treatment vs. non-treatment seeking) of children and adolescents experience this symptom while eating a large quantity of food (Tanofsky-Kraff et al., 2008).

Longitudinal Course of Binge-Eating Disorder. Only a handful of studies have assessed the longitudinal course of BED or binge-eating more generally and these studies

have all used analyses that consider the average level of symptoms within the sample rather than the initial level and shape of symptoms across multiple time periods. Currently, results conflict with some studies indicating that binge-eating decreases across adolescence, whereas other studies suggest that binge-eating increases as youth mature. In a ten year longitudinal study with a large representative sample (N = 2,287), the number of girls experiencing loss of control while eating increased from 9% at about 14 years of age to 16% at about 24 years, and increased similarly for boys from 3% to 6% during the same time frame (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). On the other hand, in another study, girls increased engagement in binge-eating behaviors from fifth to seventh grade (e.g., end of fifth grade: 6.8%; end of seventh grade: 7.5%), whereas binge-eating declined for boys (e.g., end of fifth grade: 9.0%, end of seventh grade: 3.2%; Pearson, Combs, Zapolski, & Smith, 2012). These results suggest binge-eating increases for girls, whereas changes in binge-eating for boys during adolescence are currently unclear. Given the limited longitudinal research on binge-eating, more research is necessary to clarify the developmental course of BED symptoms. However, although prevalence rates for BED and sub-threshold BED rates consistently indicate that these symptoms are lower in boys than girls, these longitudinal findings suggest that binge-eating symptoms are problematic for boys as well as girls, primarily during childhood rather than adolescence (Becker, Grinspoon, Klibanski, & Herzog, 1999; Johnston et al., 2002; for review of BED for adolescent males, see Muise, Stein, & Arbess, 2003). If the developmental course of BED differs by sex, the factors contributing to binge-eating symptoms may also differ between boys and girls. Thus, sex

differences in the associations among BED, ADHD, and depression symptoms were considered in the proposed study to address this gap in the literature.

Results with adults indicate that BED is a chronic, episodic disorder that remits and returns throughout the lifespan. Some research with adults indicates that BED remits at rates of 85% within five years (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000), whereas other studies indicate that BED frequently remits, but returns at other times throughout the lifespan (Pope et al., 2006).

Evaluation of the persistence of BED for children and adolescents varies widely across studies. Goosens and colleagues (2011) found little correspondence in binge-eating behavior between time points where participants were 10 to 17 years and 15 to 23 years at time points one and two, respectively. Unfortunately, a small sample (N = 56), the high 48% attrition rate at the second time point, and the wide age range for participants at both time points limit the usefulness of these findings. However, Tanofsky-Kraff and colleagues (2011) also found little correspondence in rates of BED in a four year follow-up when children were 10 to 17 years old (N = 118). About 52% of children who reported experiencing loss of control while eating continued to experience this symptom, whereas 30% of those who had never experienced loss of control while eating now reported the presence of this symptom at least once a month. Despite the apparent strong correspondence of loss of control while eating between time points, only five adolescents who reported loss of control during the first time point met criteria for BED at the second time, compared to none from the comparison group. Although the correspondence of BED across time points was not consistent, there was some correspondence of core symptoms of BED, such as loss of control while eating. These

results suggest that a significant number of children with binge-eating symptoms appear to consistently experience specific BED symptoms and that consistently experiencing specific BED symptoms in childhood may increase risk for meeting criteria for the disorder in adolescents.

However, some of the lack of correspondence of BED and BED symptoms across time may be explained by the methods currently used to evaluate the longitudinal nature of BED. A research design with only two time points may not capture the chronic episodic nature of this disorder or its progression, thus providing underestimates of the actual persistence of BED for children and adolescents. Because the proposed study did not include assessment of BED beyond one time point, the development of BED across time is beyond the scope of this study. However, a goal for future research is to evaluate the consistency of BED symptoms across childhood and adolescence to identify if these symptoms are as chronic for children and adolescents as they are adults.

Psychological and Physical Health Concerns Associated with Binge-Eating. The mental health implications of BED are significant. BED is highly cormorbid with other psychological disorders, such that nearly 78.9% of individuals with BED have a second psychological disorder (Hudson et al., 2007). Common comorbid disorders include depression, generalized anxiety, and panic disorders (Grucza, Przybeck, & Cloninger, 2007). Even sub-threshold BED symptoms are associated with additional mental health symptoms. Adolescent girls with sub-threshold BED symptoms were 3.5 times more likely to also have a depression diagnosis and 1.8 times more likely to have an anxiety disorder than girls with no mental disorders (Touchette et al., 2011).

BED is also associated with physical health risks as well. Longitudinal analysis of eating habits and obesity indicate that children who experience loss of control when overeating and who report binge-eating episodes show an increased risk for being overweight, obese, and experiencing obesity related health outcomes (Hasler et al., 2004; Hudson et al., 2010; Tanofsky-Kraff et al., 2006; Tanofsky-Kraff et al., 2009; Wonderlich et al., 2009), with boys who binge appearing more overweight than girls (Braun, Sunday, Huang, & Halmi, 1999; Striegel-Moore et al., 2009). One study indicated that children who demonstrate binge-eating behaviors were 5.33 times more likely to display metabolic syndrome five years later, with BMI only partially accounting for elevated risk (Tanofsky-Kraff et al., 2012). Regardless of obesity, BED is associated with chronic pain, irritable bowel syndrome, and fibromyalgia in adults and similar health risks may exist for children and adolescents as well (Bulik & Reichborn-Kjennerud, 2003; Javaras et al., 2008). Unfortunately, when binge-eating behaviors and symptoms lead to weight gain for children and adolescents, it appears more difficult to lose weight. BED has been shown to negatively impact response to weight-loss treatment for adults. For example, Pagota and colleagues (2007) found that obese and moderately obese adult participants of a behavioral weight loss program lost 1.73 times less weight compared to those without BED. Further, half as many BED participants as those without BED met their weight loss goals. These results are consistent with other studies as well (Gorin et al., 2008). BED may lead to similar problems with weight loss for children and adolescents who are overweight or obese. Further, boys with binge-eating symptoms appear less responsive to current weight loss interventions, demonstrating less weight loss after therapy compared to girls (Field et al., 2001). These results demonstrate

connections between obesity and binge-eating that raise concerns about the health about children and adolescents who engage in binge-eating behaviors.

Difference in Binge-Eating by Race/Ethnicity. Differences in rates of BED and binge-eating may exist between African-American and European-American youth. Research findings conflict regarding differences in binge-eating diagnoses between African-American and European-American youth. According to some studies, African-Americans display lower rates of binge-eating than European-American youth (23% vs. 30.6%, respectively; French et al., 1997). Other findings indicate that more African-American youth engage in binge-eating than European-American youth (20% vs. 18%; Johnson et al., 2002). Youth from both ethnicities share stress and peer acceptance as contributing factors to binge-eating, but weight dissatisfaction and perception of being overweight only predict binge-eating for European-American youth (French et al., 1997; Johnson et al., 2002). When considering differences in loss of control when overeating, research findings are also inconsistent regarding racial/ethnic differences. Some findings indicate that African-American youth experience these symptoms more (Swanson et al., 2011), less (Story, French, Resnick, & Blum, 1995), or equivalently to European-American youth (Cassidy et al., 2012).

Aspects of the environment that may differ for European-American and African-American youth may also account for some of the differences in rates of binge-eating symptoms and the factors that contribute to these symptoms. First, African-American youth experience racial/ethnic discrimination at rates that are higher than European-American youth. The stress of discrimination has been linked to both poor mental and physical health outcomes (Gaylord-Harden & Cunningham, 2009; Landrine & Klonoff,

1996; Sims et al., 2012). In fact, experience with discrimination has been cross-sectionally linked to higher fat content of physique for African-American adults compared to European-American adults (Hickson et al., 2012; Hunte & Williams, 2009). It is not clear what accounts for this link. These results may be due to the effects of stress on metabolic processes and factors influencing physical health and eating habits that may be related to cultural differences between African-American and European-American adults. However, these results suggest the possibility that the stress of discrimination may contribute to obesity with binge-eating behaviors as the mechanism of that link. Although little research has been conducted on the link between stress or discrimination and eating pathology for African-Americans (see Thompson, 1996), one study suggests that stress makes a unique contribution to eating disorders for African-American women (Harrington, Crowther, Henrickson, & Mickelson, 2006).

Second, African-American youth may have greater access to calorically dense, nutrient depleted foods and less access nutrient rich foods (see Haering & Syed, 2009; Horowitz, Colson, Hebert, & Lancaster, 2004). Epidemiological research indicates that predominantly minority communities are half as likely to have a supermarket and 2.4 times more likely to have a convenience store than predominantly European-American communities (Moore & Diez Roux, 2006). Comparison of stores selling food items between predominantly minority communities and predominantly European-American communities indicates that stores in minority communities are also less likely to sell fresh fruits and vegetables, offer fewer fresh items, place fresh items in the back rather than the front of the store, stock more calorically dense food items, and sell fewer non-fat food items than comparison stores (Sloane et al., 2003). These findings have led researchers to

suggest that these minority communities are “nutritionally disadvantaged” (Moore & Diez Roux, 2006). The greater access to energy dense, highly palatable foods that African-American youth have may also increase opportunity for experiencing loss of control while eating or engaging in binge-eating behavior.

Currently, no research has evaluated the role of ethnicity and food access considerations in BED symptoms. However, characteristics of the environment for African-American youth may impact the rate of BED symptoms and contributing factors to these symptoms for this population. Additional research is necessary to clarify characteristics of binge-eating that may be unique to African-American children and adolescents. The sample for the proposed study contribute to addressing this gap in the literature by assessing the contribution of ADHD and depression symptoms to binge-eating behaviors with participants most of whom identify as African-American.

Theories of the Development of Binge-Eating. Several theories exist that attempt to explain how binge-eating develops. A theory that implicates impulsivity suggests that individuals who tend to act rashly are more likely to experience loss of control when eating resulting in overeating and binge-eating than those who tend to display more restraint. Another aspect of impulsivity in particular, sensitivity to reward, may also contribute to vulnerability for binge-eating. Research indicates that those who engage in binge-eating prefer smaller, immediate rewards rather than larger, delayed rewards (for review see, Dawes & Loxton, 2004). Individuals who discount the benefits of delaying gratification may be more likely to value the hedonic pleasure of eating highly palatable foods and continue consuming these foods beyond the point at which their future reward oriented peers would stop. These facets of impulsivity, acting rashly, and reward

sensitivity, may also work together to initiate binge-eating behavior and then to maintain it. Some research support for this theory of binge-eating exists (Hartmann et al., 2010; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Nederkoorn, Jansen, Mulkins, & Jansen, 2007; Nasser, Gluck, & Geliebter, 2004), although these studies have primarily included adult women in samples.

Theories with more predominance in the field suggest that negative affect triggers binge-eating behavior. The affect regulation model proposes that individuals engage in binge-eating to distract from or reduce negative affect. Over time, binging-eating becomes a conditioned response to the distress of negative affect (Hawkins & Clement, 1984). Similarly, escape theory posits that an individual experiences intolerance for negative affect and seeks to reduce self-awareness in an effort to avoid awareness of negative affect. Binge-eating provides the opportunity to bring attention away from self, allowing the individual to escape their experience of distress (Baumeister, 1991). Based on learning theory, expectancy theory suggests that individuals who expect that eating behaviors will aid them in positively coping with negative affect are more likely to engage in binge-eating (Hohlstein, Smith, & Atlas, 1998). Finally, with restraint theory, negative affect disrupts the cognitive control necessary to maintain a calorically restricted diet. Negative affect triggers dietary disinhibition, resulting in binge-eating behavior (Herman & Polivy, 1980). Despite differences, all these theories hypothesize that negative affect in some way triggers binge-eating episodes, suggesting that negative affect precedes binge-eating. A recent meta-analysis of fourteen studies evaluating the presence of negative affect pre- and post-binge found a moderate cumulative effect size

(ES = .63) across studies, indicating that negative affect appears elevated prior to a binge-eating episode.

Dieting and dietary restraint may play an important role in the link between depression and binge-eating (Hawkins & Clement, 1984). Cultural pressure to maintain a thin physique may contribute to the negative affect that triggers binge-eating. Experiencing pressure to be thin may result in dissatisfaction with one's body for youth, particularly young women, which may then catalyze dietary restraint (Cattarin & Thompson, 1994; Field et al., 2001; Stice, 2001; Stice & Bearman, 2001; Stice, Mazotti, Weibel, & Agras, 1998; Stice & Whitenton, 2002; Wertheim, Koerner, & Paxton, 2001). However, reducing caloric intake can reduce serotonin levels (Attenburrow et al., 2003), which has both implications for physical and mental health that may subsequently affect food intake behavior. Given that release of serotonin in the hypothalamus triggers satiety and de-motivates food intake, lower levels of serotonin may leave an individual with weakened interoceptive cues to stop eating, thus contributing to binge-eating behavior (Chandler-Laney et al., 2007). Lowered serotonin levels may also account for physical fatigue, decreased positive affect, and increased negative affect that is associated with dieting (Stice & Bearman, 2001; Stice, Hayward, Cameron, Killen, & Taylor, 2000). As a result, dieting may set the stage for the link between negative affect and binge-eating (Stice, 2002).

Overall, these theories point to impulsivity and depression as major predictors of binge-eating. Impulsivity is a core symptom of ADHD and may play an important role in creating vulnerability for binge-eating. A hallmark of depression, on the other hand, is frequent experiences of negative affect, which may increase opportunities for binge-

eating. However, it is also possible that ADHD and depression interact to predict binge-eating behavior. Children and adolescents with ADHD often demonstrate deficits in regulating negative emotions, which have been linked to higher levels of depression for adolescents with ADHD than comparisons (Seymour et al., 2012). Furthermore, impulsivity may amplify the tendency to engage in maladaptive coping strategies such as binge-eating to reduce negative affect (Fischer & Smith, 2008). Thus, those with both ADHD and depression may experience greater risk for binge-eating than those with either symptom area alone.

ADHD Symptoms and Binge-Eating. Given the primary role that impulsivity plays in some theories explaining binge-eating and the prominence of impulsivity symptoms in ADHD diagnostic criteria, ADHD symptoms may increase risk for binge-eating. ADHD is a behavioral disorder with symptoms in three areas – inattention, hyperactivity, and impulsivity. It affects 7% - 10% of school-age children (APA, 2000; Froehlich et al., 2007) and represents over 50% of all child clinic cases (Waschbusch et al., 2002). One theory of binge-eating suggests that a propensity for immediate rather than delayed rewards contributes to vulnerability for binge-eating (Dawes & Loxton, 2004). ADHD symptoms are also associated with discounting delayed rewards and difficulties delaying gratification. Thus, ADHD symptoms and binge-eating may be related through a shared deficit in reward processing. Evidence from neuroimaging studies and those evaluating behavioral responses suggests that individuals with ADHD prefer smaller immediate rewards rather than larger rewards available at a later date (delay discounting; Anokhin et al., 2011; Bitsakou et al., 2009; Scheres et al., 2006; Solanto et al., 2001; Ströhle et al., 2008). As a result, ADHD symptoms may leave individuals prone to preference for

strong, immediate rewards rather than natural rewards that require time and effort. Consistent with this point, ADHD confers risk for use of substances such as tobacco, marijuana, and illicit stimulants (Lee, Humphreys, Flory, Liu, & Glass, 2011; Malone, Van Eck, Flory, & Lamis, 2010; Van Eck, Markle, Dattilo, & Flory, in press, Van Eck, Markle, & Flory, 2012). Some researchers have proposed that this pattern of reward sensitivity increases risk for substance use for those with ADHD symptoms and similarly increases risk for binge-eating (Cortese et al., 2008; Cortese, Isnard, Bernardino, & Mouren, 2007; Davis, Levitan, Smith, Tweed, & Curtis, 2006; Davis et al., 2010; 2011; for review, see Davis & Carter, 2009).

Despite the strong theoretical link between ADHD symptoms and binge-eating, no research has evaluated this association with a sample of children and adolescents, underscoring the importance of the proposed study. Indirectly supporting this link, several studies have indicated that children with ADHD demonstrate risk for developing obesity, suggesting that ADHD symptoms may be associated with difficulty resisting highly palatable food. Holtkamp and colleagues (2004) found in a clinical sample of boys with ADHD that nearly 20% had a BMI above the 90th percentile for their age, which is much higher than would be expected in the population. Agranat-Meged and colleagues (2005) identified that nearly 58% of a children receiving inpatient treatment for obesity met criteria for an ADHD subtype. The link between obesity and ADHD symptoms appears to occur despite research indicating that the primary treatment for ADHD, stimulant use, contributes to decreased height and weight growth compared to controls (Charach, Figueroa, Chen, Ickowicz, & Schachar, 2006; Swanson et al., 2011). It is possible that a link between binge-eating and ADHD symptoms may help to clarify the

mechanisms associated with heightened risk for obesity for children and adolescents with ADHD symptoms.

Some research also exists regarding the link between ADHD symptoms and Bulimia Nervosa. Although binge-eating is a primary component of Bulimia, BED and Bulimia are distinct disorders with some shared characteristics, such as body dissatisfaction and typical affect associated with binge episodes (Haedt-Matt & Keel, 2011). However, given the dearth of literature on this topic, associations between ADHD symptoms and Bulimia may provide important insight that can inform understanding of the association between ADHD and binge-eating.

Results from several longitudinal studies indicate the rate of Bulimia among young adults diagnosed in their childhood with ADHD generally appears higher than same age peers. Surman, Randall, and Biederman (2006) found weak support for a link between ADHD and Bulimia. Considering lifetime prevalence of Bulimia across three longitudinal studies collected by Biederman and his colleagues (2006), they found that a Bulimia diagnosis occurred among 1% - 12% of those with an ADHD diagnosis, which compared to 1% - 3% in control groups without an ADHD diagnosis. Young women with ADHD aged 11 to 23 years appeared particularly at risk as they were 5.6 times more likely to report sub-threshold or diagnostic symptom severity of Bulimia than the comparison group during a five-year follow-up study (Biederman et al., 2006). Unfortunately, several limitations reduce the validity of these findings. Samples included little to no representation of African-American individuals, leaving questions about the degree to which these results generalize to African-American children and adolescents. The latter study had an extremely wide age range and included both sub-threshold and

diagnostic level symptom severity in the category of Bulimia diagnosis. These limitations make unclear the correspondence between Bulimia and ADHD.

Additional research was conducted with research designs that improved upon these weaknesses. In a cross-sectional study, Mikami, Hinshaw, Patterson, and Lee (2008) compared rates of Bulimia for female adolescents aged 11 to 18 years with ADHD (n = 127) and without ADHD (n = 82). Results indicated that girls with only ADHD-Combined type demonstrated significantly higher Bulimia symptoms five years later. Longitudinal analyses supported these findings and extend them to apply to both males and females. Using the sample from the Multimodal Treatment of Attention Deficit Hyperactivity Disorder (MTA) study, Mikami and colleagues (2010) compared the prevalence of Bulimia in adolescents with ADHD (n = 432; 22% female) and without ADHD (n = 264; 20% female). Interestingly, parent report of impulsivity but not inattention symptoms predicted Bulimia six years later. Although this association was statistically significant for both genders, the degree of association was stronger for girls than boys.

These findings indicate that adolescents of both genders with ADHD experience greater risk for Bulimia compared to their peers. Further, impulsivity symptoms provide the strongest prediction of Bulimia for adolescents with ADHD, which supports the hypothesis that impulsivity increases risk for binge-eating for adolescents with ADHD. Given that binge-eating is an integral component of Bulimia, these findings may apply to binge-eating behavior as well as Bulimia. Although no research currently exists on the rate of binge-eating or BED for children and adolescents with ADHD, these results indicate that ADHD may increase risk for BED just as it does for Bulimia.

Research with adults with ADHD also provides some support for a link between binge-eating and ADHD. Davis and colleagues (2010) found that women in both the study's obesity (n = 60) and the obesity plus binge-eating (n = 60) groups demonstrated significantly more inattention and hyperactivity symptoms than the normal weight group (n = 61). These results suggest that ADHD symptoms may increase risk for both binge-eating and obesity.

Some but not all studies support a link between ADHD and binge-eating among adults seeking treatment for obesity. Both Pagoto and colleagues (2010) and de Zwaan and colleagues (2011) found that binge-eating moderated the link between ADHD diagnosis and BMI within a sample of adults receiving weight-loss treatment services for obesity. Among adults scheduled for bariatric surgery, Gruss, Mueller, Horbach, Martin, and de Zwaan (2012) identified that about 28% met criteria for an adult or childhood diagnosis of ADHD, which is 6.4 times higher than the 4.4% rate of ADHD in the general adult population (Kessler et al., 2006). Although these findings support the hypothesis that ADHD symptoms increase risk for BED and binge-eating behaviors, characteristics of these studies warrant further research on this topic. The cross-sectional design of these studies preclude inferences of causation and given the extreme scores individuals in these samples demonstrate on both BMI and ADHD symptoms, they may be outliers on other variables that influence both BMI and ADHD, accounting for their association. Thus, additional research is necessary to identify if ADHD increases risk for binge-eating and if this association exists for children and adolescents.

The limited research evaluating the link between ADHD symptoms and binge-eating provides support for this association. Findings with adults indicate that women

with ADHD symptoms engage in binge-eating more frequently and were more overweight than women without ADHD (Davis et al., 2010). Similarly, adults seeking weight loss treatment were significantly heavier when they engaged in binge-eating and had a diagnosis of ADHD (de Zwaan et al., 2011; Pagoto et al., 2010). Specific to children and adolescents, impulsivity may increase risk for binge-eating for children and adolescents with ADHD as it does for Bulimia (Mikami et al., 2010).

Impulsivity and Binge-Eating. Given the lack of research assessing the association between ADHD symptoms and binge-eating, it may be helpful to consider research linking impulsivity and binge-eating. Since impulsivity symptoms were the ADHD symptom area related to Bulimia (Mikami et al., 2008; 2010), research on impulsivity and binge-eating may help to elucidate the association between ADHD and binge-eating. Intuitively, characteristics of BED seem similar to impulsivity. Loss of control while eating indicates difficulty inhibiting behavior. Consuming food quickly and continuing to eat despite being uncomfortably full point to acting rashly with little forethought. Feelings of shame and depressed mood after a binge-eating episode suggest rash behavior that one regrets. Yet, surprisingly little research has evaluated the role of impulsivity in binge-eating or BED for children and adolescents. Some research on the link between impulsivity and binge-eating supports this link for adults with BED, and impulsivity appears to contribute to obesity and to binge-eating for adults who are obese. Studies that include impulsivity use a plethora of measures and constructs that represent different aspects of impulsivity, which include rash, thoughtless behavior, difficulty inhibiting behavior, and reward sensitivity (Hartmann et al., 2010; Nasser, et al., 2004). Evidence exists across these diverse measures of impulsivity that it contributes to obesity and

binge-eating for those who are overweight or obese (Bonato & Boland, 1983; Johnson, Parry, & Drabman, 1978; Nasser et al., 2004; Nederkoorn et al., 2006; 2007; Sigal & Adler, 1976).

Binge-eating may be linked to impulsivity through impulsive decision-making or reward sensitivity. Reward sensitivity refers to the degree to which reward rather than punishment drives behavior and is part of a neuropsychological theory of approach/avoidance learning (Gray, 1982; 1987), explaining behavioral decision-making including impulsive behavior (Whiteside & Lynam, 2001). The theory stipulates that behavior is motivated through a balance of reward and punishment. The Behavioral Activation System (BAS) governs recruitment of interpersonal resources to react and favors reward, whereas the Behavioral Inhibition System (BIS) impedes behavior responses and responds more strongly to punishment rather than reward (Carver & White, 1994). Individuals display a range of balance between BAS and BIS in their behavioral output. Those with a stronger BAS than BIS tend to demonstrate reward sensitivity in their behavior. Delay discounting indicates one aspect of reward sensitivity and serves as a fundamental component within several theories of self-regulation (Manuck, Flory, Muldoon, & Ferrell, 2003). It refers to the preference for immediate, small rewards compared to delayed, large rewards (Reynolds, 2006). Delay discounting correlates strongly with impulsivity within individuals (for reviews, see Green & Myerson, 2004; Reynolds, 2006), indicates one's level of sensitivity to reward (Reynolds, 2006), and represents an important deficit area associated with ADHD (Anokhin et al., 2008; for review, see Paloyelis, Mehta, Kuntsi, & Asherson, 2007). For example,

children who prefer smaller immediate rewards rather than larger delayed rewards may also prefer highly palatable foods and continue eating them when peers may stop.

Although this theory remains somewhat unevaluated related to binge-eating, evidence suggests that delay discounting may be related to food consumption affects. In several studies, children with obese weight status have preferred immediate rather than delayed rewards compared to healthy weight children (Bonato & Boland, 1983; Johnson et al., 1978; Sigal & Adler, 1976). Children who engage in binge-eating may also display greater sensitivity to reward compared to punishment (Nederkoorn et al., 2006). A reward based video game, which contrasted pursuit of reward to risk of receiving punishment, discriminated between obese and non-obese adolescents. Further, obese adolescents who reported binge-eating behaviors made significantly more choices to risk receiving punishment during the game than obese adolescents without binge-eating behaviors. These results suggest sensitivity to reward may result in children and adolescence with binge-eating symptoms being prone to experiencing highly palatable foods as more rewarding than those who do not display binge-eating symptoms.

Using questionnaire measures, some studies suggest that women with BED may be more likely to act without forethought and engage in excessive eating without thinking. These studies suggest that women with BED demonstrate higher rates of impulsivity compared to women without BED. Women in both the sub-threshold BED group (n = 11) and BED diagnosis group (n = 11) demonstrated significantly higher rates of motor impulsivity scores as measured by self-report on the Barratt Impulsivity Scale (Patton, Stanford, & Barratt, 1995) than women without BED (n = 11). Further, impulsivity was correlated with symptoms of BED, including “eating when not

physically hungry", "eating when alone", and "loss of control while eating" (Nasser, et al., 2004). Although this study had a very small sample size, these results indicate that impulsivity may contribute to binge-eating behavior. Impulsivity appears to predict loss of control while eating for children as well (Hartmann et al., 2010). Children (N = 128) aged 8 to 13 years from a community sample who had experienced loss of control over eating at least once in the past three months had higher impulsivity than children without this BED symptom. Although these findings support the association between impulsivity and binge-eating, the measure of disordered eating was quite weak as the inclusion criteria required that children experience only one episode where they experienced loss of control while eating in the past three months. Although additional research is necessary to clarify these links, the results of these studies suggest that impulsivity as measured with questionnaires may be associated with binge-eating.

Obese children appear to demonstrate less inhibitory control, which may impede response to weight loss treatment (Nederkoorn et al., 2006; 2007). However, it is unclear how inhibitory control capacity for children who binge-eat compares to healthy children or obese children. In one study, obese adolescents (n = 32) had significantly slower performance on a stop signal task than non-obese (n = 31) adolescents. Although obese binge-eaters (n = 15) had the slowest stop signal performance, results did not significantly differ from the adolescents who were only obese (Nederkoorn et al., 2006). Unfortunately, the very small sample size for the binge-eating comparison group limited the ability to adequately test this difference. Further, membership in the binge-eating group was based on frequency of binge episodes and no report of symptoms related to BED (Nederkoorn et al., 2006). Another study found that the slower obese children aged

8 to 12 years performed on a stop signal task, the less weight they lost in a 12 week weight loss treatment (Nederkoorn et al., 2007). This study did not assess treatment performance for children who engaged in binge-eating; thus, it is unclear if inhibitory control as measured on the stop signal task impacted response to treatment for children with binge-eating behavior. These results indicate that children with obesity demonstrate slower inhibitory control than those who are not obese, suggesting that inhibitory control bolsters a child's ability to resist consuming highly palatable foods, such as desserts, sweets, and snacks. Given these results, children with binge-eating problems may also demonstrate significantly lower inhibitory control than healthy children.

Together, these studies suggest that impulsivity is likely important in the development of BED and its symptomatology during childhood and adolescence. Given the link between impulsivity and binge-eating and the fact that impulsivity is a core symptom of ADHD, ADHD symptoms increase risk for binge-eating, as well. However, several methodological aspects of these studies necessitate additional research. First, most of these studies have been completed with children and adults who are also obese. Binge-eating is not unique to those who are obese. In fact, one study indicated that 54% of males and 42% of females from a representative sample of adolescents (N = 2,380) who reported binge-eating were obese, which accounted for only 5.5% of males and 16% of females who were obese (Neumark-Sztrainer et al., 2011). These findings suggest that evaluating binge-eating in a sample of obese children or adolescents accounts for only about half of those who engage in binge-eating. Thus, it is not clear the extent to which binge-eating is associated with impulsivity for those with and without obesity. Second, these studies are cross-sectional, precluding inference making regarding causality. It is

unclear if impulsivity increases engagement in binge-eating, if binge-eating leads to increased impulsivity, or if a third variable increases rates of both impulsivity and binge-eating. Thus, prospective designs are necessary to clarify the degree to which impulsivity contributes to the development of binge-eating and BED.

Link between Depression and BED. Depression symptoms may also increase risk for binge-eating. Depression refers to experiencing six of the following symptoms for at least two consecutive weeks: depressed mood, lack of interest in activities, disrupted appetite, disrupted sleep (i.e., hypo- or hyper-somnia), fatigue, feelings of worthlessness, lack of concentration or difficulty thinking, lethargy, and suicidality (APA, 2000). Most theories on binge-eating suggest that individuals binge eat to reduce the distress they experience from negative affect (for review, see Haedt-Matt & Keel, 2011). In fact, findings from several studies indicate that children and adolescents demonstrate depressed mood before binge-eating episodes. For example, one study found that feeling sad before a laboratory meal of highly palatable snacks was associated with increased snack consumption for overweight children (Goldschmidt, Tanofsky-Kraff, & Wilfley, 2011). In an international multi-site study, adolescents (N = 445) who ranged from overweight to morbidly obese reported having more negative feelings prior to binge-eating episodes and being more emotionally numb during episodes compared to normative eating (Tanofsky-Kraff et al., 2007). These results suggest that, indeed, binge-eating follows negative affect and depressed mood specifically, and that binge-eating may temporarily alleviate the distress of these feelings. These findings indicate that depressed mood and other depressive symptoms may increase risk for binge-eating.

Further, depression and BED are frequently comorbid, which also suggests that these two disorders influence each other. Comorbidity for depression and BED appears across a wide range of symptom severity for BED (Johnson et al., 2002; Touchette et al., 2011). Research findings indicate that up to 50% of those with BED also have a depressive disorder (Shisslak et al., 2006), 23% of individuals with sub-threshold BED have a co-occurring depressive disorder (Swanson et al., 2011), and those who binge just once a month are also significantly more likely to have a depression diagnosis (Rawana, Morgan, Nguyen, & Craig, 2010). Loss of control while eating is also associated with depression. In several studies, children who report experiencing loss of control while eating displayed significantly higher depression symptoms than those who had not (Morgan et al., 2002; Tankofsky-Kraff et al., 2004). Overweight children with even one experience of loss of control while eating appear to experience greater depression symptom severity than overweight children without this symptom (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Finally, frequency of experiencing loss of control while eating in the past three months for adolescents was related to depression symptoms (Glashofer et al., 2007). These results suggest that depression symptoms are associated with increases in binge-eating and BED symptoms.

However, the directional association between binge-eating and depression remains unclear with current longitudinal studies. Similarly, Skinner and colleagues (2012) found that depressive symptoms at age 14 years more than doubled the risk of developing binge-eating behaviors two years later for a representative sample of adolescent girls (N = 4,798). However, binge-eating doubled the risk of developing depression four years later as well. Similarly, Tanofsky-Kraff and colleagues (2011)

found that overweight children who reported loss of control while eating at 10 years of age had higher depression symptoms 4 years later than children who had acquired loss of control while eating symptoms by the second time point. However, depression symptoms from the first time point were not included in either study, leaving unclear the temporal order and directionality of the association between loss of control while eating and depression symptoms. Interestingly, depression significantly predicted binge-eating one year later for a sample of female college students (N = 127), but binge-eating did not predict depression with earlier depression symptoms included as a covariate (Spoor et al., 2006). On the other hand, Goosens and colleagues (2011) compared depression and loss of control over eating when adolescents were 10 to 17 years old (N = 56) in predicting future levels of loss of control over eating six years later. Results indicated that depression symptoms were the strongest predictor of loss of control over eating.

These longitudinal studies clearly support a link between depression and binge-eating. However, several methodological characteristics of these studies limit their findings. First, most of the prospective studies did not have designs that assessed depression symptoms prior to adolescence, which as a crucial design element in evaluating this association. Taken together, these findings suggest that it is possible that BED and depression symptoms share a bidirectional association. Once children and adolescents have developed BED symptoms, depression and binge-eating may exacerbate each other, generating a cycle that maintains symptom severity in both areas. This hypothesis deserves further empirical scrutiny. However, the methodological implication of this possible relation between BED and depression is that failing to include assessment of depression before BED symptoms have developed may undermine evaluation of the

contribution of depression symptoms to the development of BED. The proposed study clarifies the role that depression symptoms play in the development of BED symptoms as depression symptoms measured in childhood from first through third grade and adolescence from sixth through ninth grade were included in analyses. Given that depression was assessed much earlier than adolescence when both BED and depression symptoms appear to increase, the proposed study was able to shed light on the degree to which depression symptoms predict BED symptoms.

Unfortunately, most of these longitudinal studies included assessment of depression at only two time points (Goosens et al., 2011; Tanofsky-Kraff et al., 2012) and when more than two time points were available, researchers did not consider the growth and shape of growth in depression and binge-eating symptoms (Skinner et al., 2012). Given that depression appears to have a non-linear trajectory across childhood and adolescence, this limitation is problematic. Depression symptoms display a five-fold increase during early adolescence for young women and then symptoms level off during middle adolescence (Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin et al., 1998; Lewinsohn, Shankman, Gau, & Klein, 2004). Not allowing for this acceleration in symptom severity may misrepresent the contribution of depression symptoms to the development of binge-eating. This study addresses this gap in the literature by evaluating the link between depression symptoms and binge-eating with early measurement of depression symptoms and multiple time points throughout adolescence. This study also provides indication of which developmental period of depression symptoms contribute to binge-eating. For example, childhood symptoms may exert a stronger influence on the development of binge-eating. On the other hand, depression symptoms specific to

adolescence may be the strongest predictor of binge-eating. The transitional design of the model proposed for this study allowed for examination of how developmental periods impact the link depression symptoms share with binge-eating.

Interaction between ADHD and Depression Predicting Binge-Eating. Given that both depression symptoms and impulsivity, a primary symptom area of ADHD, increase risk for BED, both symptoms together may increase the risk for BED more than either symptom area alone. ADHD significantly increases risk for depression during childhood (Bagwell, Molina, Pelham, & Hoza, 2001; Reinke & Ostrander, 2008). Some research suggests that difficulty coping with negative emotions accounts for the link between ADHD and depression (Seymour et al., 2012). Given their struggle to cope with negative emotions, adolescents with ADHD may impulsively engage in maladaptive behaviors to relieve the distress of negative emotions, such as binge-eating, which is consistent with several theories regarding why people engage in binge-eating behaviors (See Haedt-Matt & Keel, 2011). The impulsivity associated with ADHD may magnify maladaptive coping related to negative affect, moderating the effect of depression on binge-eating symptoms.

Several studies indicate that ADHD increases risk for depression in adolescents and young adulthood (Bagwell et al., 2001; Biederman et al., 2006; Barkley, Fischer, Smallish, & Fletcher, 2002). Comorbidity between ADHD and depression is estimated to be 20–30%, with some researchers indicating that adolescents with ADHD experience 5.5 times greater risk of having a depressive disorder than adolescents without ADHD (Costello et al., 2003). Chronis-Tuscano et al. (2010) found that children diagnosed with ADHD during childhood were significantly more likely to receive a depression diagnosis,

report experiencing suicidal ideation, or have attempted suicide during adolescence. These studies demonstrate the close association between depression and ADHD during adolescence. Further, the fact that the rate of depression is higher in boys than girls with ADHD also indicates that adolescents with ADHD are not simply following the normal developmental pattern of depression symptoms, where girls demonstrate significantly higher rates than boys.

Several studies indicate that youth with ADHD struggle to cope with negative emotions. Children with ADHD persevere with frustration much less than their peers do. Milich and Okazaki (1991) found that boys with ADHD demonstrate greater frustration and negative affect than control boys, when asked to complete challenging puzzles, and also persisted in completing the puzzles for a much shorter period of time than controls. Likewise, O'Neill and Douglas (1996) also found that children with ADHD completed fewer puzzles than controls. Scime and Norvilitis (2006) found that children with ADHD not only reported experiencing more frustration while solving a difficult puzzle, quitting sooner than comparison children, they also reported making less effort to find ways to improve their mood. Melnick and Hinshaw (2000) found that not only were boys with ADHD more emotional reactive than controls when completing a difficult task with their parents, they also engaged in fewer effective patterns of coping. They gave more hostile responses, stopped communicating with their parents more often, engaged in problem solving much less, and were less accommodating than boys without ADHD. Coping ineffectively with negative affect can increase risk for depression (Aldoa, Nolen-Hoeksema, & Schweitzer, 2010), and one study has found that difficulty coping with and

managing negative emotions may account for the link between depression and ADHD (Seymour et al., 2012).

It is possible that the maladaptive coping strategies that children with ADHD demonstrate in response to frustration on difficult laboratory tasks extends to engaging in maladaptive coping, such as binge-eating, in response to negative affect. Some research indicates that impulsivity while negatively emotionally aroused in young adults with ADHD predicts impairment above and beyond inattention and hyperactivity/impulsivity symptoms (Barkley & Fischer, 2010). Thus, it is also possible that impulsive responding may increase in the presence of negative affect, increasing the likelihood that youth with ADHD are more vulnerable to engage in ineffective behaviors to escape the distress negative affect than peers. It is also possible that depression amplifies risk for binge-eating for youth with ADHD by increasing experience of negative affect.

Given the fact that both depression and impulsivity are associated with binge-eating, it is surprising that little to no literature exists regarding the degree to which these symptoms interact to predict binge-eating. Given that several theories of binge-eating behaviors point to engaging in binge-eating to relieve distress associated with negative affect (Haedt-Matt & Keel, 2011), an interaction between depression and impulsivity seems likely to provide better explanation of binge-eating behaviors than either symptom area alone. If negative affect increases the risk of impulsively binging on palatable food, it seems likely that depression symptoms would be associated with increased BED symptoms, but also that individuals with greater levels of impulsivity would experience a greater vulnerability to engage in BED behaviors when experiencing depression symptoms.

A burgeoning area of research related to urgency provides supports for the presence of an interaction between depression and impulsivity predicting binge-eating. Urgency refers to impulsively engaging in behavior when distressed to reduce negative affect (Whiteside & Lynam, 2001). Conceptually, it represents both impulsivity and negative affect, a characteristic of depression, potentially providing a rough indication of the effect that both impulsivity and depression symptoms together may have on binge-eating. Urgency has been found to predict binge-eating behavior. Fischer and Smith (2008) evaluated in a sample of college students (N = 249) the links between four constructs of impulsivity and alcohol use, gambling, and binge-eating. Only urgency predicted binge-eating and the association was only significant for women. Urgency also interacted with expectancies of eating problems, which referred to having positive expectations regarding the ability to manage negative affect with eating problems. Those with high urgency and high expectancies of eating problems engaged in high levels of binge-eating. Similarly, urgency measured in fifth grade indirectly predicted binge-eating in sixth grade through the mediating effect of expectancies of eating problems (Pearson et al., 2012). These studies suggest that the combined effect of impulsivity and depression, similar to urgency, may increase binge-eating.

Further support comes from latent class analysis and cluster analysis findings indicate that two subtypes of BED exist among adults. One subtype is associated with infrequent binge episodes typically following periods of dietary restriction. The second subtype is characterized by a myriad of psychopathology symptoms, including impulsivity and depression symptoms, as well as Axis II symptoms (Masheb & Grilo, 2008; Stice, Chase, Stormer, & Appel, 2001). Carrard, Crépin, Ceschi, Golay, and Van

der Linden (2012) replicated these two subtypes with cluster analyses, where urgency replaced impulsivity and depression symptoms. Given that both symptom areas were higher in the group with the highest BED symptom severity, it is possible that impulsivity and depression interact to contribute to higher BED symptoms than either symptom area alone. Thus, additional research is necessary with children and adolescents to identify if depression and impulsivity, a major symptom area of ADHD, work together to contribute to the development of binge-eating behavior and BED.

1.2 Predictor: ADHD Symptoms – Longitudinal Course of Symptoms

When considering how ADHD symptoms contribute to the development of binge-eating it is helpful to consider how ADHD symptoms develop. The research findings related to the developmental course of ADHD symptoms have shifted dramatically over the years. Although ADHD symptoms were associated uniquely with childhood and thought to remit by adolescence, longitudinal studies tracking children diagnosed with ADHD into adolescence demonstrate that ADHD symptoms persist into adolescence and adulthood. These studies indicate that 66% to 85% of children diagnosed with ADHD still demonstrate clinically significant symptoms and impairment in their early to mid 20's (Barkley, Fischer, Smallish, & Fletcher, 2002; Biederman, Petty, Clark, Lomendico, & Faraone, 2011; Hansen, Weiss, & Last, 1999).

Consideration of symptoms of ADHD dimensionally provides consistent results. Several studies evaluating the development of ADHD symptoms across childhood demonstrate that, although symptoms do decrease into adolescence, ADHD symptoms still remain clinically significant for most adolescents (Stepp, Burke, Hipwell, & Loeber, 2012). When considering separately the dimensions of ADHD symptoms, several studies

indicate that hyperactivity/impulsivity symptoms gradually decrease across childhood, whereas inattention symptoms tend to remain consistent (e.g., Biederman, Mick, & Faraone, 2000; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Larsson, Lichtenstein, & Larsson, 2006). For example, Biederman and colleagues (2000) found that, in those with a childhood ADHD diagnosis, hyperactivity and impulsivity symptoms reduced in severity by about 50% from 6 to 20 years of age, whereas inattention symptoms reduced in severity by only approximately 15%.

Importantly, the development of ADHD symptoms across childhood and adolescence appears to display heterogeneity. The fact that 68% - 85% of childhood diagnoses of ADHD persist into adolescence (Barkley, Fischer, Edelbrock, & Smallish, 1990) suggests that individual differences in trajectories of ADHD may occur. Studies using latent class growth analysis or growth mixture modeling to evaluate typical developmental patterns of ADHD symptoms across childhood support this notion. Most studies have identified four classes of symptom trajectories where the classes correspond to stable rates of symptom severity rather than changes in the growth symptoms across time (Côté, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002; Nagin & Tremblay, 1999; Pingault et al., 2011; Shaw, Lacourse, & Nagin, 2005; Van Lier, Van Der Ende, Koot, & Verhulst, 2007). Classes correspond to very high symptoms, moderately high symptoms, moderately low symptoms, and low symptoms. All four classes appear to slightly decrease with age, although the moderately high symptoms class appears to increase until middle childhood and then decrease. As an exception, Côté and colleagues (2002) found that four classes fit growth in ADHD symptoms for male participants, but three classes fit symptom growth for female participants. The extra class for the male participants

represented symptoms that started moderately low, decreased to age nine, but increased into middle school. Nagin and Tremblay (1999) also differed from other studies in that all four classes demonstrated decreasing trajectories. Although most studies have identified four classes, other studies find support for three trajectory classes (Malone et al., 2010; Robbers et al., 2011) or even two trajectory classes of ADHD symptoms (Jester al., 2005; Larsson, Dilshad, Lichtenstein, & Barker, 2011).

Together, these studies suggest some interesting characteristics and differences of ADHD symptom growth. First, all studies identified a low symptom class, which represents children who display few ADHD symptoms across the measurement period, and a high symptom class, which corresponds to persistent high levels of ADHD symptoms that display a slight decrease across time. Second, the age of 10 years may be important with several classes changing direction in symptoms at this age. Some children may maintain moderate levels of ADHD symptoms until age 10 and then decrease (Côté et al., 2002; Pingault et al., 2011; Malone et al., 2010). This change in direction for symptom severity may correspond to the development of self-regulatory skills that improve a child's impulse control and ability to manage attention. Specifically, working memory abilities, attention shifting, and inhibitory control appear to improve and impulsivity decreases during middle childhood (Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999; Raffaelli, Crockett, & Shen, 2005). Some research suggests that children with ADHD demonstrate a delay in brain development (Kinsbourne, 1973; Shaw et al., 2007, 2012; Vaughn et al., 2011). As cognitive and self-regulation capacity improves with development, children with ADHD may display reductions in symptom severity (Faraone et al., 2000). Third, a similar point may occur at age 12 years, where some

children may display a resurgence in ADHD symptom severity, which is consistent with research demonstrating increases in ADHD symptoms during the transition to middle school (e.g., Langberg et al., 2008). The heightened demands on planning, organization, time management, and self-regulation associated with the transition to middle school present a significant challenge for youth with ADHD, who specifically struggle in these areas. Middle school typically requires management of four different class periods, teachers, homework activities, and testing schedules. Although the skill required to succeed in middle school may quickly emerge for youth without ADHD, adolescents with ADHD often display an increase in ADHD symptoms, decreased grades, and increased disruptive behavior as they adjust to the demands of middle school (Langberg et al., 2011).

Although many of these studies present strong, methodological approaches, several factors necessitate further research on the developmental trajectories of ADHD symptoms. Most of the studies on this topic use international samples that may or may not apply to an American population. Although understanding how ADHD symptoms develop in these environments is important, ethnic and cultural differences may lead to different trajectories of ADHD symptoms in the African-American population. Further, the studies conducted in the United States had very low representation of African-American children and adolescents, which makes unclear the extent to which the trajectory classes from these studies apply to African-American youth. Only two studies contained children who identified as African-American (Malone et al., 2010; Shaw et al., 2005); only Malone and colleagues (2010) included race/ethnicity as a covariate and reported its influence on class membership. These results indicated that African-

American children were slightly more likely to have class membership in the trajectory with the greatest symptom severity. These results suggest that there may be differences in trajectories of ADHD symptoms for African-American children and identifying these differences may hold important treatment and outcome implications for African-American children.

1.3 Predictor: Depression Symptoms – Longitudinal Course of Symptoms

Depression symptoms may also contribute to the development of binge-eating. Depression symptoms affect 2% - 5% of children (Reynolds & Johnston, 1994). However, as children transition into adolescence, depression symptoms substantially increase. According to self-report, about 20% - 40% of adolescents experience depressed mood (Petersen, Compas, Brooks-Gunn, Stemmler, Ey, & Grant, 1993), and clinical levels of depression increase to 15% to 20% (Lewinsohn et al., 2004). Some research suggests that the prevalence rate of clinical depression demonstrates a six-fold increase from age 15 years to age 18 years, with many individuals encountering their first depressive episode between 15 and 18 years of age (Hankin et al., 1998). Until adolescence, rates of depression display gender equivalence. At age 13 years, however, a gender difference begins to emerge with girls experiencing more depression than boys. By age 15 years, about twice as many girls as boys meet criteria for clinical depression (Hankin et al., 1998; Wade, Cairney, & Pevalin, 2002). Studies assessing change across time in depression symptoms as a continuous dimension provide similar results (Ge, et al., 1994; Holsen, Kraft, & Vitterso, 2000), although the trajectories of both studies possessed significant heterogeneity, which was especially characteristic of trajectories for boys.

Important gender differences regarding the developmental course of depression have emerged from longitudinal assessments of growth in depression symptoms rather than change in diagnostic category. Assessing depression every six months rather than every year, Cole and colleagues (2002) found that depression symptoms began to increase for girls as early as fifth grade rather than at age thirteen. Although depression symptoms for girls decreased until tenth grade in another study, depression increased thereafter, whereas boys demonstrated a continued declension in symptoms (Burstein, Ginsburg, Petras, & Ialongo, 2010). These findings are inconsistent with previous reports and raise questions regarding the trajectory that best fits growth in depression symptoms.

Considering the significant heterogeneity in trajectories present in the above studies, it is possible that more than one typical developmental trajectory of depression best fits changes in symptoms during adolescence. Approximately four studies exist that use latent trajectory analysis to identify typical trajectory classes of depression symptoms. Given that these studies represent different periods of time during childhood and adolescence, together these studies provide a unique window into the development of depression symptoms across childhood and adolescence.

During second to eighth grade, one study indicated that five trajectories best fit the development of depression (Mazza, Fleming, Abbott, Haggerty, & Catalano, 2010). These trajectories corresponded to symptoms that were consistently low, low and increasing, consistently moderate, moderate and decreasing, or moderate and increasing. Another study found that four classes of trajectories represented the depression symptoms of children 11 to 14 years of age, where symptoms could be described as consistently high, increasing, consistently moderate, and consistently low (Brendgen, Wanner, Morin,

& Vitaro, 2005). These results suggest that some adolescents remain unaffected by depression symptoms, whereas other children display moderate and high levels of depression symptoms during childhood, which they maintain into adolescence.

Growth of depression during middle adolescence appears to fit with three trajectory classes of growth that correspond to low, medium, and high symptom severity (Wiesner & Kim, 2006). Although the trajectories remain relatively stable across middle adolescence, the high symptoms trajectory showed a slightly concave shape, whereas the moderate symptoms trajectory displayed a slightly convex shape (Rodriguez, Moss, & Audrain-McGovern, 2005). Also, four classes may provide better fit of depression symptoms for boys, where moderate symptoms are increasing for some and decreasing for other (Wiesner & Kim, 2006), which is consistent with other research identifying trajectory classes of depression for young men aged 14 to 24 years of age (Stoolmiller, Kim, & Capaldi, 2005).

The dramatic increase in risk for depression symptoms in adolescence is consistent across studies evaluating the development of depression symptoms as a continuous or a categorical construct. However, a subset of children, as identified in latent class growth analyses, demonstrate elevated depression symptoms before adolescence. For these children, instead of experiencing a worsening of symptoms during adolescence, many children with high depression symptoms display a decrease in symptoms during adolescence. Also, although girls were more likely to be in the classes with higher symptom severity of every latent trajectory class study, boys also appear to experience clinically significant depression symptoms during adolescence. In several studies, boys demonstrated increases in depression symptoms that may not have been as

dramatic as symptom increases for girls, but may be quite meaningful (Ge et al., 1994; Stoolmiller et al., 2005; Wiesner & Kim, 2006).

These studies reviewed above on the development of depression provide limited representation of African-American youth in the samples, leaving questions about the extent to which these studies extend to African-American youth. Four studies included no African-American or black children in the sample, whereas for another three studies, less than 5% of the sample was African-American. For Cole and colleagues (2002), 36% of the sample was African-American, but differences in the initial level and growth in depression symptoms based on ethnicity were not evaluated and race/ethnicity was not included as a covariate in analyses. The only study to date to evaluate growth in depression symptoms during childhood or adolescence that has produced results that can clearly extend to African-American youth is Burstein and colleagues (2010), given that their samples was 88.7% African-American. Their results were not consistent with the shape of growth in depression symptoms found in most other studies. For girls, depression symptoms displayed concave rather than convex growth towards the end of adolescence. These results suggest that differences may exist in the development of depression symptoms between European-American and African-American youth. Further research is necessary to clarify the shape of growth in depression symptoms for African-American youth.

1.4 Methodological Issues

Most of the research conducted on depression, ADHD, and binge-eating has included homogeneous samples with low representation of African-Americans. However, growth in ADHD and depression symptoms appears to differ between European-

Americans and African-Americans and differences in symptom presentation or rate of diagnosis for BED between ethnicities is unclear. Thus, a crucial methodological characteristic of the proposed study is that it identifies growth in ADHD and depression symptoms and evaluates their contribution to binge-eating symptoms in a sample that includes strong African-American representation and analyses considered the effect of race on model parameters.

Most research related to eating disorders has also focused exclusively on women and girls. However, the very limited research that has included both sexes in samples has indicated that men and boys also display eating problems. In particular, although females are more likely to engage in binge-eating, it is a behavior that clearly also affects males. Binge-eating for males is associated with consuming more food during a binge (Striegel-Moore et al., 2009), becoming more obese (Braun et al., 1999; Striegel-Moore et al., 2009), less frequently seeking treatment (Bramon-Bosch, Troop, & Treasure, 2000; Carlat & Carmargo, 1991), and responding less to treatment (Field et al., 2001). Given these clearly deleterious outcomes, it is crucial that mechanisms related to binge-eating be evaluated for males as well as females. This study addressed this gap in the literature by including both sexes in the sample used for the proposed study.

An important methodological characteristic of this study is that the sample was randomized to a control condition and one of two interventions during the first year of measurement. One intervention was a classroom-based program (i.e., the Good Behavior Game; Barrish, Saunders, & Wolf, 1969), where positive behavioral supports were used to encourage self-regulation skills to increase prosocial, compliant, and on-task classroom behavior (Werthamer-Larsson, Kellam, & Wheeler, 1991). The second intervention

targeted school to home communication to support teachers and staff in engaging parents in their children's academic achievement (based on Canter & Canter, 1992; Webster-Stratton, 1984; for details, see lalongo, Werthamer, Kellani, Brown, Wang, & Lin, 1999). These interventions may influence the shape and initial level of growth in the development in ADHD and depression symptoms in both childhood and adolescence, as well as adolescent binge-eating behavior.

Although some studies demonstrate that universal classroom behavioral interventions can produce modest improvements in attention and reductions in hyperactivity (DuPaul, Ervin, Hook, & McGoey, 1998; Plummer & Stoner, 2005; Van Lier, Muthén, Van Der Sar, & Crijnen, 2004), declension in symptoms appears to occur only for children with low to moderate ADHD symptom severity rather (Van Lier et al., 2004). Likewise, improving communication about academics and behavior between the home and school context has also contributed to improvements in ADHD symptoms (Pfiffner et al., 2007). Positive behavioral management in the classroom as well as universal interventions targeting self-regulation skill development has also been associated with prevention of depression symptoms (Kellam, Rebok, Mayer, Ialongo, & Kalodner, 1994). As a result, it is possible that intervention status would influence the shape and rate of growth in ADHD and depression symptoms as well as membership in symptom severity classes. No research has assessed the effect of early interventions targeting self-regulation and inhibitory control capacity on binge-eating in adolescence has been evaluated. Thus, considering the effect of intervention status on binge-eating provides a unique contribution to the literature.

Another methodological consideration is the conceptualization of mental health diagnosis. In this study, mental health diagnoses were considered with continuous symptom dimensions. Researchers have argued on both a conceptual and statistical basis that ADHD, depression, and binge-eating are better classified as continuous symptom dimensions than categorical diagnosis. Klein argues that using a categorical approach to conceptualize depression results in numerous combinations of symptoms and cut-points that correspond to different new "disorders" of depression. Taking a continuous symptoms approach can avoid the "balkanization of chronic depression into numerous categories and specifiers" (Klein, 2008). Using a continuous symptom dimension to represent ADHD has been found to consistently provide better reliability and validity than discrete models of ADHD diagnosis (Frazier, Youngstrom, & Naugle, 2007; Haslam et al., 2006; Hudziak et al., 1998; Marcus & Barry, 2010). Given that mental health symptoms occur on a continuum, using diagnostic categories corresponds to splitting up a continuous variable and would have carried the corresponding limitations, such as reduced variability and statistical power (Cohen, Cohen, Aiken, & West, 2003, Keselman et al., 1998; MacCallum, Zhang, Preacher, & Rucker, 2002). Thus, use of continuous dimensions increases the possibility of detecting significant effects if they are present.

The combination of person-centered and variable-centered analysis was selected to evaluate hypotheses for a variety of reasons. Variable-centered analyses refer to statistical tests that aggregate data based on a variable level of analyses. These analyses characterize what is typical in a sample, usually assume cases are drawn from one population, and that together case scores on the variable of interest display a normal distribution. Examples of variable-centered analyses include regression, analysis of

variance (ANOVA), path analyses, multiple group comparisons, and confirmatory factor analyses. Unfortunately, variable-centered approaches assume that associations apply to all individuals in analysis, conceptualizing individual differences as error. Thus, the utility of variable-centered approaches breaks down when more than one population or subtype of a variable exists, which can obscure important associations among constructs (Bauer & Curran, 2004). Person-centered analyses refer to statistical tests that identify the number of groups of individuals within the sample that display similar response patterns. Person-centered approaches assume that identified groups represent different populations from which cases were drawn (Bauer & Curran. 2004; Muthén & Muthén, 2000). Examples of person-centered analyses include latent class analysis, cluster analyses, and mixture models.

Given the literature reviewed on ADHD and depression symptoms, these symptoms appear to display different developmental patterns across childhood and adolescence. If the trajectory of symptom severity differs among children, it stands to reason that different patterns of development in these symptoms during childhood may also make different contributions to the prediction of binge-eating behaviors in adolescents. Further, the degree to which ADHD and depression symptoms independently or jointly affect binge-eating would also be likely to differ. Accounting for these differences in analyses may help to accurately identify which developmental patterns of ADHD and depression symptoms contribute to binge-eating behaviors (Bauer & Curran, 2004; Muthén & Muthén, 2000).

Chapter 2: Current Study

Given the literature regarding the contribution that ADHD and depression symptoms may make to binge-eating symptoms, I proposed a set of five primary research questions and one secondary question. The primary questions pertained to identifying typical developmental patterns of ADHD and depression symptoms during childhood (i.e., fall of first grade, spring of first grade, second grade, and third grade) and adolescence (i.e., sixth through ninth grade), the degree to which youth display similar symptom severity across developmental periods of childhood and adolescence, and the degree to which these child and adolescent patterns of symptoms predict binge-eating symptoms in adolescence (i.e., tenth grade). The secondary questions are related to differences in outcomes based on contextual predictors of gender, intervention status, race, and lunch status. These contextual predictors may influence the development and subtypes of ADHD and depression symptoms, the transition among subtypes of symptom severity across two developmental periods, and the association these developmental patterns may have with binge-eating symptoms.

Data from the Johns Hopkins Field Trial was used for these analyses. The sample completed annual assessments from first through third grade and sixth through tenth grade. During first grade, whole classrooms were enrolled in the study and randomized to one of two interventions or a control condition. The interventions continued only throughout the first grade year. An extra assessment was included at the end of first grade and is included in these analyses as well. Classrooms from urban public schools in the

Baltimore city school district participated in the study. Most children in the study were African-American and received free or reduced lunch. Thus, this sample provides a unique opportunity to evaluate the effects of race and economic status on the development of ADHD and depression symptoms in childhood and adolescence.

2.1 Foundational Research Questions

Before evaluating the role of ADHD and depression symptoms in increasing binge-eating behaviors in adolescence, it was necessary to explicate the role of ADHD and depression symptoms in the transition model. Thus, the first four research questions related to the developmental trajectory, the description of the latent growth classes, and associations among latent classes of ADHD and depression symptoms within and between developmental periods. Once these aspects of the model were established, the link between childhood and adolescence symptom classes and binge-eating was evaluated, which is described in Primary Research Question I.

1. **Foundational Research Question I:**
    a. **What are the developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

For this step, I completed independent growth models of ADHD symptoms and depression symptoms in childhood (i.e., fall of first grade, spring of first grade, second grade, and third grades) and adolescence (i.e., sixth through ninth grades). The results produced four summative trajectories for each type of symptom in each developmental period. Based on previous research, I expected to find that the trajectory of ADHD

symptoms in childhood would increase, the trajectory of depression symptoms in childhood would stay stable, the trajectory of ADHD symptoms in adolescence would decrease slightly, and the trajectory of depression symptoms in adolescence would increase.

1. **Foundational Research Question I:**
    b. **What are the typical subgroups of the developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

In a second step, I completed a latent class analysis with each of these four trajectories. Based on the developmental course of depression and ADHD symptoms found in previous research, I expected to find three classes of ADHD symptoms in childhood and two classes of ADHD symptoms in adolescence. In childhood, when ADHD symptoms are more prevalent than in adolescence, I expected that one class would have high ADHD symptoms, the second would have moderate symptom severity, and the third class would have very low ADHD symptoms. In adolescence, I expected that one class would have moderate to high ADHD symptoms and the second would have very low ADHD symptoms. I expected there to be two classes of childhood depression symptoms and three classes of adolescent depression symptoms. In childhood, I expected there to be a low symptom severity class and a moderate symptom severity in class. In adolescence, I expected there to be a low symptom severity class, a moderate symptom severity class, and a high symptom severity class, given that depression symptoms are more prevalent in adolescence than in childhood.

2. **Foundational Research Question II: Do children remain in the same symptom severity class of ADHD and/or depression symptoms or change to classes with higher or lower ADHD and/or depression symptom severity?**

Analyses used to consider this question involved latent transition analyses with the latent classes of the growth models of symptom severity described in the first research question. Latent transition analysis provided an estimate of the likelihood of membership in latent trajectory classes in adolescence based on membership in latent trajectory classes in childhood. Analyses also produced transition posterior probabilities of membership in adolescent symptom classes given membership in childhood symptom classes, which were evaluated along with the log odds estimates representing the prediction of the adolescent symptom class membership from childhood symptom class membership. Several transition models were assessed. First, models with each symptom type were independently evaluated to identify the transition probability from childhood to adolescence. Second, models with both symptom types during both developmental periods were considered within the same model. I expected to find that students in the high ADHD symptom severity class in childhood would transition to the high symptom severity class in adolescence, but that the low and moderate symptom classes would transition to the low ADHD symptom severity class in adolescence. I expected that the students in the high depression symptom severity class in childhood would transition to the high symptom severity class in adolescence. For the low depression symptoms class, I expected students to transition to both the low, moderate, and high symptom severity class in adolescence, given the significant increase in depression symptoms during adolescence.

3. **Foundational Research Question III: Are individuals in the high ADHD symptom severity class in childhood or adolescence also more likely to concurrently be in moderate or high depression symptom classes?**

To evaluate the likelihood of membership in high ADHD symptom severity classes increasing membership in moderate and high depression symptom severity classes, the concurrent posterior probabilities between symptom type at the same developmental period and the log odds estimates representing the association of the depression symptoms class membership from ADHD symptom class membership were considered for childhood and adolescent symptoms. I expected to find that the high ADHD symptom severity class would be more related to the high depression symptom severity class than the low ADHD symptom severity class, but that the probability of being in both high symptom severity classes would be stronger in adolescence than childhood.

4. **Foundational Research Question IV: Are individuals in the high ADHD symptom severity class in childhood more likely to be in moderate or high depression symptom severity classes in adolescence?**

To identify if membership in the high ADHD symptom severity class increases risk for membership in the moderate and/or high depression symptom severity class, the transition posterior probabilities, and the log odds estimates representing the prediction of the adolescent class membership from childhood membership were evaluated from the latent transition models. I expected to find that likelihood of membership in the high ADHD symptoms severity class in childhood would increase the probability of membership in the high depression symptom severity class in adolescence.

2.2 Primary Research Question

1. **Primary Research Question I: How do latent classes of ADHD and depression symptoms in childhood and adolescence predict engagement in binge-eating behavior during tenth grade?**

To evaluate this question, I evaluated the degree to which ADHD and depression symptom classes from childhood and adolescence predict binge-eating behaviors. I used the most likely class membership for symptom classes and identified the degree to which class membership in moderate and high symptom severity classes shared stronger associations with binge-eating behaviors than low symptom severity classes for each symptom area and developmental period. This process identified the extent to which growth in depression and ADHD symptoms in childhood and adolescence contributed to binge-eating symptoms in adolescence.

Given the previously reviewed literature, I expected to find that classes with high ADHD symptoms and high depression symptoms would predict higher binge-eating symptoms than low ADHD symptoms or depression symptoms. I also expected to find that classes including elevations of both ADHD and depression symptoms would demonstrate the strongest prediction of binge-eating symptoms.

2.3 Secondary Research Questions

1. **Secondary Research Question I: How do contextual predictors (i.e., gender, intervention status, race, and lunch status) affect the latent trajectories and class memberships for ADHD and depression symptoms in childhood and adolescence as well as their prediction of binge-eating behavior?**

Given the differences present across gender, race, lunch status, and the effects of intervention status on the development of depression and ADHD symptoms, these factors may influence the initial level and shape of growth in symptom trajectories. These factors may also influence the quantity and quality of latent classes of ADHD and depression symptoms and the association latent growth classes share with binge-eating behaviors. These possibilities were evaluated by estimating conditional models at each stage of analysis that include contextual predictors. For latent growth models, contextual predictors were included in conditional models as predictors of the fixed effects of the intercepts and slopes. In latent class growth models, contextual predictors were included as predictors of class membership. In the latent transition models, contextual predictors were also included as predictors of class membership. Contextual predictors were also included as predictors of binge-eating behavior when assessing the association between binge-eating and class membership.

I expect to find that the gender effects would indicate that boys have higher ADHD symptoms during both developmental periods than girls, and that girls have higher depression symptoms during adolescence than boys. I expect that the gender distribution of depression symptoms during childhood would be equivalent. In accord with these predictions, I also expect that boys would be more likely than girls to have membership in the high ADHD symptoms classes in childhood and adolescence and that girls would be more likely than boys to belong to the high depression symptoms class in adolescence. Girls may also be more likely than boys to engage in binge-eating behavior and membership in the high depression class may be more closely associated with binge-eating for girls than boys.

Regarding other contextual predictors, race and lunch status are not expected to produce significant differences in model parameters. However, regarding intervention status, I expect that children who received either the Good Behavior Game or the Family-Centered Intervention would show lower ADHD and depression symptoms during childhood and adolescence than children who did not receive either intervention. I also expect that children in the treatment condition would be more likely than children in the control condition to be in the low ADHD and depression symptom severity classes in childhood and adolescence. It is not expected that binge-eating behavior would be significantly different based on treatment condition.

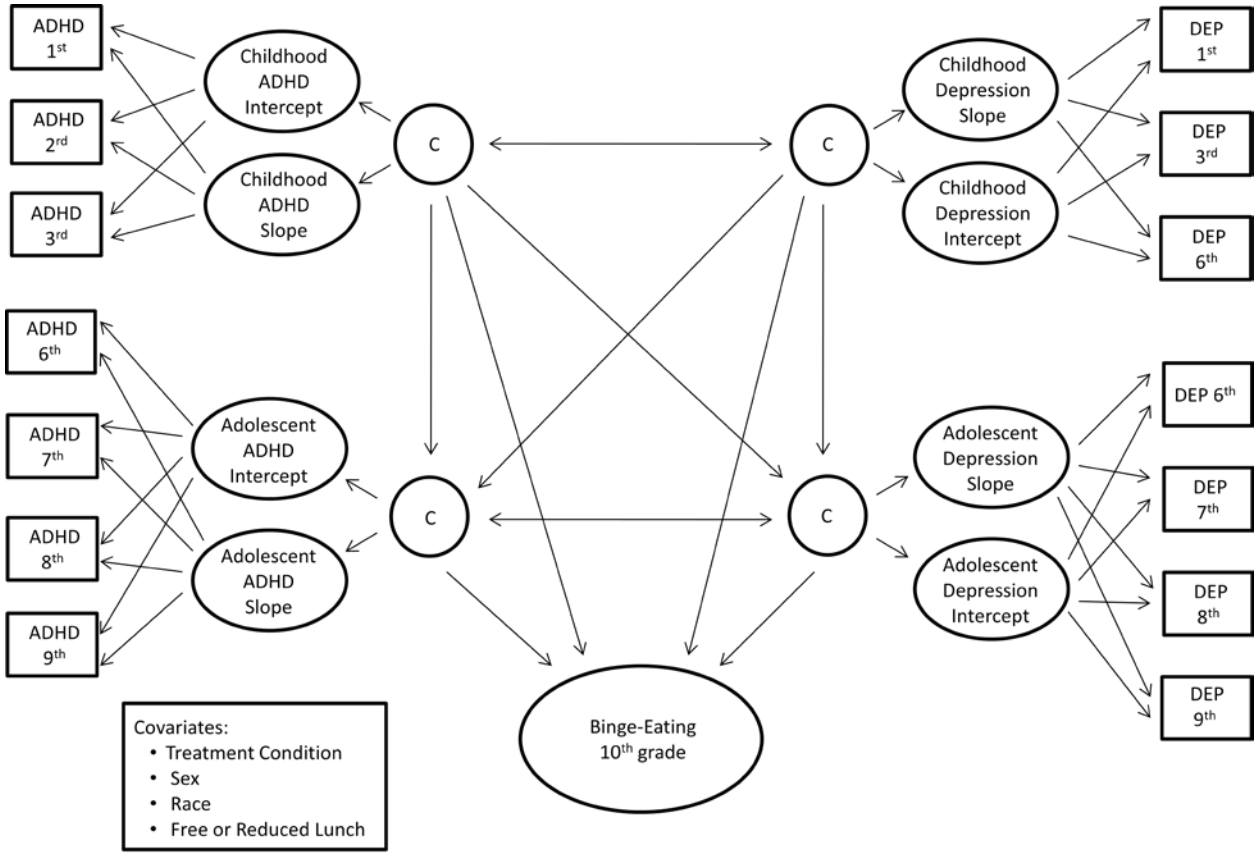


Figure 2.1 *Latent Class Analysis of a Parallel Process Model of ADHD and Depression Symptoms Predicting Binge-Eating*

Chapter 3: Methods

Annual assessments from the Johns Hopkins Field Trial were used to evaluate the research questions for this study. The primary questions of this study was to identify the developmental patterns of ADHD and depression symptoms during childhood (i.e., fall of first grade, spring of first grade, second grade, and third grade) and adolescence (i.e., sixth through ninth grade), the degree of correspondence of symptom severity between the developmental periods of childhood and adolescence, and the degree to which these child and adolescent patterns of symptoms predict binge-eating symptoms in adolescence (i.e., tenth grade). The secondary questions referred to the degree to which contextual predictors of gender, intervention status, race, and lunch status influenced the development and subtypes of ADHD and depression symptoms, the transition among subtypes of symptom severity across two developmental periods, and the association these developmental patterns may have with binge-eating symptoms.

3.1 Participants

Beginning in 1993, children (N = 678) from 27 first grade classrooms located in 9 urban elementary schools in Baltimore, Maryland participated in the Johns Hopkins Field Trial. At the start of first grade, children were 5.3 to 7.7 years old (M = 6.2, SD = .34) with nearly 46.8% being female and 86.8% African-American. Approximately 63.4% of children received free or reduced lunch. Parents provided written consent for their child's participation for 97% of children. Information about psychiatric medication use was available only at the eighth through tenth grade time points. According to teacher-report,

21 students took medication for Conduct Disorder (CD), 25 students took ADHD medication, and 6 students took medication for depression symptoms.

3.2 Procedures

Children were recruited for participation after their school administration agreed to participate in the study. Schools were randomly assigned to one of three conditions, a classroom-centered intervention, a family-school partnership intervention, or a treatment as usual condition. The classroom-centered intervention involved use of evidence-based curriculum, the Good Behavior Game (GBG; Barrish, Saunders, & Wolf, 1969) for classroom management, and enhanced behavioral management for children with additional behavioral concerns. The interventions only occurred during first grade and the beneficial effects of these interventions are described in more detail in several treatment outcome papers (Kellam et al., 2008; Kellam, Reid, & Balster, 2008; Werthamer-Larsson et al., 1991). Procedures used to analyze the effect of treatment condition on this study's research questions are described below.

The objective of the GBG was to reduce disruptive behavior by helping children to build the skills necessary to display positive behaviors in the classroom. The GBG required teachers to create three diverse groups of students that provided equal representation of student characteristics. These three groups competed with each other throughout the day, week, and month with the goal of demonstrating positive classroom behaviors and keeping disruptive behaviors below a specified frequency. All disruptive behaviors are reworded to positive behaviors, to support skill building by providing replacement behaviors. Rewards consisted of tangible items like stickers, pins, or treats or enrichment activities, like art, music, and games.

Rather than focusing solely on the classroom, the family-school partnership intervention oriented school staff towards encouraging parent involvement in their child's academic achievement. Research staff trained school staff and teachers to use methods shown to increase communication between parents and school employees (Canter & Canter, 1992). Parents and teachers were also required to engage in regular home-learning activities intended to develop partnerships between parents and teachers. School mental health professionals also teamed with first grade teachers to present nine weekly parenting groups on effective strategies for communicating with teachers and for supporting a child's behavioral and academic development. The behavioral management strategies that teachers and school staff promoted in parenting groups were based on parenting strategies developed by Webster-Stratton (1984).

Children, their parents, and their teachers participated in annual assessments regarding psychopathology symptoms, impairment in areas of daily functioning, and use of mental health services from first through third grade and sixth through twelfth grade and several years into young adulthood. Assessments were completed during the spring either at school or at the Baltimore Prevention and Research Center. During first grade, both the Family-Engagement intervention and the GBG were administered. Thus, children completed an assessment in the fall and the spring to provide a baseline and end of treatment evaluation. Ratings from both of these assessments are included in the model. During elementary school, children completed self-report scales at school, where a three-person team administered the questionnaires to a full classroom of students. One person read the questions to students, while two individuals monitored children throughout the classroom, assisting children who need additional explanation or support.

Assessments of ADHD and depression symptoms from the fall and spring of first grade and fall of second and third grade made up the four time points of the childhood trajectories, whereas assessments of symptoms from the fall of sixth, seventh, eighth, and ninth grade made up the adolescent trajectories.

3.3 Measures

Baltimore How I Feel-Young Child, Child Report (BHIF; Ialongo, Kellam, & Poduska, 1999; see Appendix A). The BHIF is a forty-five-item, self-report measure for elementary school aged children to indicate the frequency of their depression and anxiety symptoms during the past two weeks, using a three-point scale (0 = "Never", 1 = "Sometimes", 2 = "Almost Always"). Items for this measure form two subscales that correspond to depression and anxiety symptom severity. Only the depression subscale was used for the proposed analyses. Items were drawn from a wide range of typically used depression and anxiety measures for children. The measure was designed to assess depression and anxiety symptoms, according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R; American Psychiatric Association, 1987). Internal consistency for all items on the BHIF was acceptable, although a few values were below the preferred level of .80 (α's .77, .78, and .82 in first – third grades, respectively). Regarding concurrent validity, first grade self-report of depression was associated with increased likelihood that parents and teachers reported the child as requiring services for depressed mood.

During sixth through ninth grade, adolescents completed the same questionnaire, although they read the items themselves rather than having the items read to them as was done during first through third grade. Internal consistency was strong for assessments

during adolescence (range of α's: .81 – .85). Test-retest reliability for a two week interval was acceptable (r = .83). Self-report of depression symptoms on the BHIF was significantly associated with a diagnosis of depression on the Major Depressive Disorder on the Diagnostic Interview Schedule for Children-IV (DISC-IV, Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), demonstrating strong concurrent validity for the BHIF.

Eating Disorders Inventory (EDI; Garner, Olmsted, & Polivy, 1983; see Appendix A). The EDI is a self-report questionnaire of eating related attitudes and behaviors with sixty-four items and eight subscales including: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears. Participants used a 6 point response scale (1 = "Never", 2 = "Rarely", 3 = "Sometimes", 4 = "Frequently", 5 = "Usually", 6 = "Always") to answer items. This measure has been shown to possess adequate reliability and validity (Garner et al., 1983). Test-retest reliability on the EDI is high (Wear & Pratz, 1987), and the construct validity for the measure is quite strong (Espelage et al., 2003). This measure is one of the premier measures used to diagnose Anorexia Nervosa, Bulimia Nervosa, Binge-Eating Disorder, and Eating Disorders Not Otherwise Specified in clinical practice and for the purposes of research. During the tenth grade assessment, only the seven items of the bulimia scale were administered in the Field Study. For the proposed analyses, one item pertaining to compensatory behavior ("I have the thought of vomiting to lose weight") was removed, so that only items related to binge-eating were included. Thus, six items were used to create a subscale of binge-eating symptoms.

Given the restructuring of the bulimia subscale to create the measure of binge-eating behavior, psychometric evaluation of the items used was completed. Using

Confirmatory Factor Analysis (CFA) with Maximum Likelihood estimation (MLR), the items for the bulimia scale were evaluated (see Table 1 for inter-item correlations). Although the chi-square test indicated that the subscale did not have exact fit (χ^2 (20) = 31.63, p = .047; see Table 2 for fit indices and factors loadings for all models), some indices of model fit were satisfactory (Root Mean Square Error of Approximation (RMSEA) = .034; Standardized Root Mean Square Residual (SRMR) = .048), whereas others were not (CFI = .945). Further, item 8 had a factor loading well below .5 ("In your opinion, what is your current body weight?"). A contributing factor may be the use of a different response scale for this item. Instead of a 6 point response scale ranging from "Never" (1) to "Always" (6), this item has a 5 point response scale (1 = "Very Underweight", 2 = "Underweight", 3 = "Average", 4 = Overweight, 5 = "Very Overweight"). When item 8 was removed, the chi-square test displayed modest improvement (χ^2 (14) = 24.194, p = .043), although only subjective comparison between models was possible as models are not nested. Other model fit indices remained static (CFI = .947; RMSEA = .038; SRMR = .049). When the item referring to thoughts about purging was removed, the model demonstrated subjective improvements in model fit. Although the model did not achieve exact fit (χ^2 (9) = 17.357, p = .043), all other fit indices exceeded cut-points, displaying satisfactory fit (CFI = .956; RMSEA = .043; SRMR = .043). The standardized factor loadings were above .5 for all items except for item 1 ("I eat when upset") and item 7 ("I eat/drink in secrecy"), and the reliability for the latent binge-eating factor was slightly lower than preferred (α = .775). Thus, the binge-eating behavior factor demonstrated satisfactory psychometric properties and viability as a latent factor.

Household Structure and Demographics. Family socio- and demographic characteristics were assessed from this measure. Items pertaining to household income and parental education were used in analyses for this study.

Service Assessment for Children and Adolescent-Parent Report (SACA-P; Horwitz et al., 2001). Staff administered a structured interview with parents regarding their child's engagement in mental health treatments, including medication and psychotherapy. Only questions related to medications taken for management of ADHD and depression symptoms were used for this study, which were reported in the participant subsection of the methods section.

Teacher Observation of Classroom Adaptation-Revised (TOCA-R; Werthamer-Larsson et al., 1991; see Appendix A). From first through third grade, teachers engaged in a structured interview with trained assessment staff to identify each child's behavior along a wide variety of dimensions necessary for successful performance in the classroom. These areas included accepting authority (aggressive behavior), social participation (shy or withdrawn behavior), self-regulation (impulsivity), motor control (hyperactivity), concentration (inattention), and peer likeability (rejection). With the exception of withdrawn behavior and social rejection, questions for all subscales were drawn from criteria for corresponding disorders in the DSM-III-R. For the purposes of the proposed study, only self-regulation (impulsivity), motor control (hyperactivity), concentration (inattention) subscales were used. Concentration was reverse scored to reflect problems with attention.

During sixth through ninth grade, teachers responded to a shortened version of the same questions on a paper-and-pencil checklist version of the measure rather than a

structured interview. This decision was made to reduce the burden of time and effort for both research and school staff. Both the Language Arts and Mathematics teachers completed the checklists. Items for inattention, hyperactivity, and impulsivity are listed under Measure 3 in Appendix B.

Internal consistency for these subscales was acceptable (α's .79 to .97) as were the four-month test-retest correlations (r > .60). The selected subscales of concentration problems, hyperactivity, and impulsivity demonstrated concurrent validity, given that report of these behaviors correlated with teacher report of needing medication for learning and behavioral concerns at all time points.

3.4 Analytic Technique

Primary Research Questions. To identify the degree to which ADHD symptoms and depression symptoms independently and jointly affected binge-eating, a four step analytic approach was undertaken in M*Plus* v.7.1 (Muthén & Muthén, 2013). First, four sets of independent latent growth models were estimated to represent the initial level and growth in ADHD and depression symptoms during childhood (i.e., fall of first grade, spring of first grade, second grade, and third grade) and adolescence (i.e., sixth through ninth grade). Second, four latent class growth analyses were conducted with each of the four independent growth models to identify subgroups of distinct growth in symptoms within the sample. Third, the four latent class growth models were joined within a latent transition model to evaluate the transitions in ADHD and depression symptoms between childhood and adolescence. Transition models with each symptom type considered independently were first considered and then transitions among both symptom types across both developmental periods were considered in the same model. Fourth and

finally, the degree to which the latent symptom classes from the four latent class growth models predicted binge-eating behavior was assessed.

To represent the construct of ADHD, impulsivity, concentration, and hyperactivity symptoms were added together at each time point to form summary variables of ADHD symptoms at each time point for the childhood and adolescent trajectories. It was not possible to allow concentration/attention problems, hyperactivity, and impulsivity problems to remain separate in analyses. The initial longitudinal latent class models for childhood and adolescent ADHD symptoms did not reach convergence when ADHD symptoms were entered into the model as independent constructs. This result may be due to the collinearity among these constructs at each time point. Concurrent correlations among constructs ranged from .363 to .802. Thus, attention problems, hyperactivity, and impulsivity were combined at each time point and entered into the model as a single construct of ADHD symptoms.

Latent Growth Models. The first step with analyses involved estimating four separate growth models for ADHD and depression symptoms during childhood (i.e., fall of first grade, spring of first grade, second grade, and third grade) and adolescence (i.e., sixth through ninth grade). A series of nested models were evaluated by comparing model fit and considering fixed and random effects to identify the shape of growth in symptoms (see Raudenbush & Bryk, 2002; Singer and Willett, 2003). Unconditional means models were evaluated to identify the intercept and its variability for each symptom area in each developmental period. To identify the shape of growth, unconditional growth models were estimated, which considered the fixed and random effects of the slope in each model. Once the shape of growth was established, a third set of models, conditional

growth models, were estimated to identify the effect of contextual predictors (i.e., gender, intervention status, race, and lunch status) on the fixed effects of intercepts and slopes. All models used maximum likelihood with robust standard errors (MLR) to minimize standard errors. Models were evaluated for fit, statistical significance of fixed and random effects, and interpretability of growth models through graphical representation.

Fit indices included the chi-square test, Standardized Root Mean Square Residual (SRMR), Root Mean Square Error of Approximation (RMSEA), and Cumulative Fit Index (CFI; see Hu and Bentler, 1999; Marsh et al., 2004). The chi-square test displays the exact fit of the model. It identifies how closely the predicted covariance matrix replicates the actual covariance matrix based on the range allowed by the model's available degrees of freedom. Several criticisms of the chi-square test exist. Increases in sample size inflate the chi-square value. Samples may not have an underlying chi-square distribution for the covariance structure, making the test irrelevant. Further, the chi-square test holds models to a stringent standard that few can meet (Brown, 2006). Thus, considering fit indices in addition to the chi-square test is advised. Also an index of absolute fit, the SRMR is less stringent. It represents the discrepancy between the estimated and actual correlation matrix. Values range from 1.0 to 0 with smaller values indicating better fit. Values of .07 and below indicate acceptable fit.

The value of the RMSEA is that it includes adjustment for parsimony. The RMSEA (Hu & Bentler, 1999) incorporates representation of model complexity by including the discrepancy in fit for each degree of freedom. Referred to as a population-based index, the RMSEA also incorporates a noncentrality parameter that adjusts the test of fit for distributions that display non-normality (MacCallum, Browne, & Cai, 2006).

Values range from 0 to 1.0, with fit improving as values decrease. Values below .05 indicate adequate fit.

Unlike the chi-square test, the CFI is not affected by sample size and demonstrates incremental model fit. It functions by comparing the estimated model to a model where all latent factors are uncorrelated. As the CFI value increases, the estimated model demonstrates less similarity to the null, uncorrelated base model. Values for the CFI range from 0 to 1.0; appropriate model fit falls above .95. Another index of incremental fit, the chi-square difference test can be used to compare fit between two models. The difference in chi-square values between two nested models can be calculated and compared to the chi-square value expected based on the difference between degrees of freedom for the two models. If above the critical value, the additional model complexity improves model fit (Brown, 2006).

In addition to evaluating model fit, the interpretability of the fixed and random effects of the model was considered. The intercepts and slopes of models were evaluated for statistical significance, direction for the slope parameters, and effect size. A graphical representation of the models also aided interpretation of models.

Latent Class Growth Models. Once growth in ADHD and depression symptoms has been established, four independent unconditional latent class analyses were conducted with the slope and intercept terms of each type of symptom for each developmental period. Latent class analyses were conducted with growth models to evaluate the variability cases displayed around the mean slope and intercept of trajectories. This variability suggested that subgroups of cases may exist that possess distinct slopes and intercepts. These distinct trajectories may hold meaningful

information; allowing distinct expression of these subgroup trajectories may contribute to interpretation of ADHD and depression symptoms. Nested models of *k* classes were evaluated and compared to models with *k*-1 classes to identify the best fitting number of classes for each analysis. Once the appropriate number of classes was identified, conditional models were conducted that included the effect of contextual predictors (i.e., gender, intervention status, race, and lunch status) on class membership.

Latent class analyses differed from the growth models in that the variances for the intercepts and slopes were constrained to zero (Nagin, 1999). That is, the intercept and slope were assumed to be homogeneous within class and that the number of classes extracted from data fully accounts for the heterogeneity in trajectory slope and intercept. Models were constrained in this way to minimize complexity and maximize parsimony.

Maximum likelihood estimation was used and occurred in two steps. The predicted model is iteratively replicated a specified number of times (i.e., starting values). Optimization champions the predicted model with the highest log likelihood value. As model complexity and the number of classes extracted increases, the risk of the solution representing local rather than global optima increases. Local optima refer to solutions that provide the biggest log likelihood value possible within the range of the starting values, even though a better solution exists outside that range. Thus, as model complexity and number of classes estimated increases, the number of starting values should also increase to expand the range of possible solutions. Standard practice for latent class analyses specifies the use of 1000 random starting values and 100 sets of optimization for model fit (Nylund, Asparouhov, & Muthén, 2007), which was used here with the latent class analyses.

Model fit and class viability were identified by considering model fit and several class characteristics. Both the Aikake Information Criteria (AIC) and the Bayesian Information Criteria (BIC) are indicators of relative fit. According to the AIC and the BIC, the model with the smallest value for these indices indicates the model with the best fit. The Lo-Mendell-Rubin Likelihood-Ratio Test (LMR-LRT; Lo, Mendell, & Rubin, 2001) compares the log likelihood of a model with *k* classes to that of a model with *k*-1 classes. The model with the most classes that still produces a significant LMR-LRT displays the best fit. Bootstrapped Likelihood Ratio Test (BLRT; McLachlan & Peel, 2000) is another test that compares the log likelihood value of *k* classes to *k*-1 classes to identify if the increase in model complexity is commensurate with the corresponding improvements in model fit.

It is also important to consider the degree to which classes represent distinct and non-overlapping subgroups within the data. Entropy refers to the degree to which classes accurately represent subgroups present in the data and cases accurately fall within those classes. Entropy values of .8 and above indicate good classification of the proposed classes. Although entropy should not be used to decide on the best number of classes, it can be useful for delineating between two models with similar model fit. The posterior probabilities of class membership indicate both the homogeneity of class membership and class separation. Homogeneity of class membership (diagonal values) indicate that the likelihood a case has of belonging to the class to which it has been assigned. These values should be high. The class separation probabilities (off diagonal values) represent the probability of a case belonging to a class to which it has not been assigned. Class separation probabilities should be low and at least below .10. Further, the intercept and

slope of each class should be evaluated for interpretability and meaningfulness. Finally, the size of the smallest class should be considered to ensure that each class has adequate case representation and to avoid over-fitting the data. These aspects of the models were evaluated to identify the model with the best fit and number of classes for each set of symptoms and developmental periods.

Latent Transition Models. Latent transition analysis extends latent class analysis to a longitudinal context to identify the degree to which individuals remain in a similar class or transition to a different class across time. The latent transition analysis identified the likelihood of transitioning to an adolescent symptom class given each childhood symptom class. This phase clarified the extent to which classes of symptom trajectories are specific to each developmental period or continue from childhood into adolescence. Several transition models were assessed. First, models with latent class growth models from different developmental periods but the same symptom type were evaluated to identify the transition probability from childhood to adolescence (i.e., one model for ADHD symptoms and a second model for depression symptoms). Second, all four latent class growth models were linked together within the same transition model.

Latent transition analysis uses similar fit indices and model evaluation approaches to latent class growth models. Thus, the AIC and BIC were evaluated for relative model fit. Instead of the LMR-LRT and BLRT, the log likelihood difference test was used. In the log likelihood difference test, the difference in log likelihood values from two models (i.e., less restrictive model vs. more restrictive, nested, comparison model) were compared to the chi-square value that corresponds to the difference in degrees of freedom between models. If the log likelihood difference exceeds the critical value, the increase in

model complexity appears to contribute significant explanatory value to the model, supporting the superiority of the model's complexity. The entropy was also assessed as well as the posterior probabilities for class separation and homogeneity of class. Sample size of the smallest class and sample size of the smallest combination of classes were assessed to identify adequate case coverage.

Maximum likelihood estimation was used. Given the complexity of the transition models, the specified number of random starting values was increased to 10,000 with 1,000 model fit optimizations. The increase in random starting values was implemented to avoid local optima.

To identify likelihood of transitioning in and out of symptom classes of similar severity levels for the same type of symptom (i.e., high ADHD symptom severity class in childhood to high ADHD symptom severity class in adolescence), concurrent probabilities were evaluated. To identify the likelihood of transitioning into high symptom classes given membership in opposite symptoms classes (i.e., likelihood of transitioning into high depression symptom severity class in adolescence given membership in ADHD symptom severity class in childhood), transition probabilities and log odds estimates representing the prediction of the adolescent class membership from childhood membership were also evaluated.

To calculate the concurrent and transition probabilities, the equations identified in Table 2.3 were used. These equations together identify the log odds of transitioning from childhood to adolescent symptom classes compared to the reference group. In the equations, the threshold for the reference group (a_1) serves as the intercept and the log odds of belonging to another group compared to the reference group (b_{11} and b_{12}) is

added to the intercept for each corresponding group. These equations produce a log odds value that can be transformed to a probability value, representing the transition probability among classes (see Muthén & Asparouhov, 2011; Nylund, 2007). The log odds of membership in each group compared to other groups is accompanied by a significance test and was also reported.

Predicting Binge-Eating Behavior. Finally, the initial level of binge-eating behavior in tenth grade was identified for each combination of trajectory classes and linked to binge-eating behaviors assessed in tenth grade. When the transition model was estimated, class membership given the posterior probabilities of each class was saved in a dataset created during estimation. The most likely class membership from model estimation was then entered as predictors in a series of ANCOVAs that included contextual predictors (i.e., gender, intervention status, race, and lunch status). Each class membership for each symptom and developmental period were entered in separate models to identify their independent prediction of binge-eating. A separate model included childhood and adolescent ADHD symptom classes as well as their interaction to identify the effect of ADHD symptoms in the development of binge-eating behavior. A third model with childhood and adolescent depression symptom classes in addition to their interaction was also evaluated. Finally, an ANCOVA with childhood ADHD, adolescent depression symptom classes, and their interaction was also evaluated, while controlling for childhood depression symptoms.

Secondary Analyses. The effect of contextual predictors was evaluated through a variety of means. First, the basic associations that contextual predictors shared with variables of interest in the model were explored with simple statistics. T-tests were

conducted to identify significant differences that dichotomous contextual predictors (i.e., treatment condition, gender, and race/ethnicity) have with ADHD, depression, and binge-eating symptoms at all time points. Second, contextual predictors were entered as predictors of the initial level and growth in ADHD and depression symptoms during childhood and adolescence during explication of the latent growth models. Third, contextual predictors were entered as predictors of class membership of ADHD and depression symptoms during childhood and adolescence in the latent class growth models and in the latent transition models. Finally, contextual predictors were added as predictors of binge-eating behaviors when evaluating the association class membership for ADHD and depression symptoms shared with binge-eating behaviors.

Contextual Predictors. Contextual predictors included in the model were treatment (0 = "control condition", 1 = "classroom-centered intervention condition" or "family-school partnership intervention condition"), gender (0 = "male", 1 = "female"), race/ethnicity (0 = "European-American", 1 = "African-American"), and free or reduced lunch status (0 = "full priced lunch", 1 = "free or reduced lunch"). Although inclusion of psychotropic medication use was considered as a covariate, inclusion in the model was not possible, given the small number of students who were taking medication for psychological symptoms. Rates of psychiatric medication use among the sample for ADHD and depression symptoms is reported above in the participant subsection of the methods section.

Missing Data. A total of 60.6% of participants were missing data at one or more time points on one or more variable across the nine time periods included in these analyses. The rate of missing data at each time point was lower than the overall rate (i.e.,

first grade: 13%; second grade: 21.4%; third grade: 33.5%; sixth grade: 25.1%; seventh grade: 24.5%; eighth grade: 29.8%; ninth grade: 28.3%; tenth grade: 26.1%) and 60.6% of participants had data available for six or more time points. At the most, analyses had 96 different patterns of missingness. The covariance coverage for all variables included in models ranged from 65% to 100%, indicating acceptable coverage for the proposed analyses.

To evaluate the degree to which missingness was related to variables in these analyses, chi-square and t-test analyses were used. All the ADHD variables demonstrated a pattern where individuals with missing data were more likely to have higher ADHD ratings than those with complete data. Those with missing data also reported lower depression symptoms in second grade. Participants with missing data were more likely to be male and European American. Although certain characteristics of individuals were associated with missingness, no data were missing on demographic characteristics, making all missing data conditional on variables included in analyses (Graham, 2009). Missing data was managed in M*plus* v.7.1 (Muthén & Muthén, 2013) with full information maximum likelihood (FIML) estimation, which produces parameter estimates that are less biased than other missing data strategies when MAR cannot be assumed (Shafer & Graham, 2002).

Power Analyses. To evaluate the degree of statistical power possible with the sample to evaluate the proposed hypotheses, assessment of statistical power was conducted when analyses were complete. Several Monte Carlo simulations were conducted in M*Plus* using a Markov chain and the coefficients identified in the final analyses. A simulation to assess power was conducted for the full transition model as

well as each independent symptoms transition model for a total of three Monte Carlo simulation analyses with 100 replications. Parameters of interest in the power analyses were the log odds of the distinction between concurrent classes, the log odds of transitions among class memberships between developmental periods, and the log odds of the effect of covariates on class membership.

Several characteristics of the simulations were assessed to identify if analyses had adequate power to evaluate hypotheses (see Muthén & Muthén, 2002). The parameter values and the standard error bias for all parameters of interest in power analyses should not exceed 10%, and coverage must not fall below .91. Coverage refers to the proportion of replications of the simulation in which the true or specified parameter value was identified. Sample size should then be identified so that power remains above or equal to .80.

The results of the Monte Carlo simulation indicated the statistical power was available with this sample size to identify the reported coefficients (Muthén & Muthén, 2002). The ADHD symptoms independent transition model demonstrated very low bias with standard errors and parameter values, which all fell below 10%. Also, 100% of the replications contained the specified values for all log odds. The power analysis for the depression symptoms independent transition model did not meet the criteria necessary to establish sufficient sample size for power to identify the significant effects that were found. Large biases were present for both standard errors and parameter values. Between 18.6% and 63.9% of the replications contained the specified values for all log odds, which fell well below the cut point of 80% that serves as the standard for acceptable statistical power. The full transition model demonstrated moderate levels of bias with

standard errors and parameter values. Bias fell below 10% for the ADHD classes from childhood and adolescence, but rose above 10% for the depression classes. Similarly, 100% of the replications for the ADHD classes containing the specified values for log odds, but for the depression classes fewer than 80% of simulations replicated these values. Thus, the sample size for this study was sufficient to identify the statistical significance demonstrated in the results for transition models containing ADHD symptoms classes, but was lower than desirable for the depression classes.

Table 3.1. *Correlations of Bulimia Items from the Eating Disorders Inventory*

Items	1	2	3	4	5	6	7	8
1. I eat when I am upset	1.00							
2. I stuff myself with food	.361*	1.00						
3. I have gone on eating binges where I felt that I could not stop	.307*	.570*	1.00					
4. I have the thought of trying to vomit to lose weight	.166*	.199*	.272*	1.00				
5. I think about bingeing or overeating	.146*	.410*	.544*	.421*	1.00			
6. I eat moderately in front of others and stuff myself when they are gone	.172*	.436*	.459*	.281*	.471*	1.00		
7. I eat or drink in secrecy	.247*	.267*	.314*	.160*	.226*	.346*	1.00	
8. In your own opinion, what is your current body weight?	.051	-.042	-.057	.015	-.071	.059	.060	1.00

*$p < .05$.

Table 3.2. *Binge-eating factor: Standardized factor loadings and model fit indices*

	Model 1: Full	Model 2: Trimmed	Model 3: Binge-Eating
Items	*B* (SE)	*B* (SE)	*B* (SE)
1. I eat when I am upset	.377 (.073)	.377 (.073)	.382 (.073)
2. I stuff myself with food	.685 (.049)	.685 (.049)	.702 (.049)
3. I have gone on eating binges where I felt that I could not stop	.782 (.059)	.781 (.059)	.796 (.061)
4. I have the thought of trying to vomit to lose weight	.410 (.104)	.410 (.104)	--
5. I think about bingeing or overeating	.677 (.077)	.677 (.077)	.648 (.081)
6. I eat moderately in front of others and stuff myself when they are gone	.634 (.085)	.635 (.083)	.624 (.087)
7. I eat or drink in secrecy	.417 (.103)	.418 (.103)	.418 (.104)
8. In your own opinion, what is your current body weight?	-.029 (.079)	--	--
Fit Statistics			
χ^2	31.634 (df = 20)	24.194 (df = 14)	17.363 (df = 9)
CFI	.945	.947	.956
RMSEA	.034	.038	.043
SRMR	.048	.049	.043
A	.780	.780	.775

Notes. SE = Standard Error. Standardized regression coefficients are reported for each item. All factor loadings were significant except for item 8 in Model 1. All chi-square values were statistically significant. Cut-offs for the Confirmatory Factor Index (CFI) is ≥ .95, for the Root Mean Square Error of Approximation (RMSEA) ≤ .05, and for the Standardized Root Mean Residual (SRMR) ≤ .05.

Table 3.3 *Equations for Calculating Transition Probabilities*

	Adolescent Symptom Classes		
Childhood Symptoms Classes		1	2
	1	$a_1 + b_{11}$	$1 - p_{11}$
	2	$a_1 + b_{12}$	$1 - p_{12}$
	3	a_1	$1 - p_{13}$

Note. To calculate the transition probabilities, the above equations were used that produce the log odds of transition from childhood to adolescent symptom classes, where the third childhood symptoms class serves as the reference group. In the equations, a_1 = the threshold or intercept for the first adolescent symptoms class, b_{11} = slope representing the difference in association the first adolescent class has of belonging to the first childhood symptom class compared to the third childhood symptom class, b_{12} = slope representing the difference in association the first adolescent class has of belonging to the second childhood symptom class compared to the third childhood symptom class. These equations produce a log odds value that can be transformed to a probability value, representing the transition probability among classes (see Muthén & Asparouhov, 2011; Nylund, 2007).

Chapter 4: Results

4.1 Preliminary Analyses

Testing Statistical Assumptions. Assumptions of regression require that continuous variables demonstrate values that do not exceed |1| for skew and |3| for kurtosis (Cohen et al., 2003). Variables for ADHD and depression symptoms at all time points demonstrated skew and kurtosis within acceptable bounds (see Table 3.1). The binge-eating factor, however, had extreme skew and kurtosis, which may be due to many individuals reporting very low binge-eating symptoms. Analyses were conducted with the binge-eating factor log transformed to evaluate differences in outcomes due to a skewed distribution. The log transformed version of binge-eating had a skew of 2.244 and kurtosis of 5.464, which are still outside acceptable bounds. No differences were identified in the association binge-eating shared with class membership between transformed and non-transformed versions of binge-eating. Thus, the untransformed version of binge-eating was used. Although a few univariate and multivariate outliers were identified, these cases did not influence effect size or statistical significance and remained in analyses.

Descriptive Statistics. All descriptive statistics (see Table 3.1) and correlations (see Table 3.2) were assessed for continuous model variables. ADHD symptoms were moderately to strongly associated with each other at all time points, where the closer in time that time points were to each other the stronger the correlations. Depression symptoms ranged from time points not being at all related to strongly related. Depression

symptoms during first through third grade demonstrated small to moderate correlations, whereas symptoms in sixth through ninth grade were moderately to strongly related. First grade depression symptoms did not appear to be related to depression symptoms in adolescence, although symptoms in second and third grade shared small to moderate association with symptoms in adolescence. Depression and ADHD symptoms shared concurrent associations during only first and sixth grade. At other time points, depression and ADHD symptoms were either not significantly related or had a small association. Binge-eating symptoms were not related to ADHD symptoms at any time point, but did share a small association with depression symptoms at most time points.

Differences Variables of Interest across Contextual Predictors. Model variables were significantly different across the contextual predictors that were included in the model. Results of t-tests indicated that boys displayed significantly higher ADHD symptoms at all time points. Girls had significantly higher depression symptoms at all time points except for 3rd and 6th grade. The one-way ANOVA comparing the family-school partnership intervention condition, classroom-centered intervention condition, and the control group indicated that the family and classroom conditions displayed significantly higher ADHD symptoms than the control group during the fall of 1st grade (F (2, 674) = 8.46, $p < .001$; Family: $M_{diff} = .359$, $p = .001$; Classroom: $M_{diff} = .339$, $p = .002$), although the family and classroom condition were not different from each other ($M_{diff} = .020$, $p = .977$). However, only the family condition continued to display significantly higher ADHD symptoms at the spring 1st grade assessment (F (2, 629) = 5.315, $p = .005$, $M_{diff} = .311$, $p = .004$). African-American students displayed significantly higher depression symptoms compared to other race/ethnicities at the fall ($t = 3.123$, $p =$

.002) and spring of 1st grade (t = 3.701, p < .001), as well as 2nd grade (t = 2.011, p = .045). Also, students who received free or reduced lunch displayed significantly higher depression and ADHD symptoms, but only during 2nd grade (t = 2.180, p = .030).

4.2 Foundational Research Questions

Although the primary objective of this study was the degree to which ADHD and depression symptoms from childhood and adolescence predicted binge-eating behaviors in adolescence, it was necessary to explicate the role of ADHD and depression symptoms in the transition model prior to evaluating the full transition model. Thus, the first four research questions are related to the developmental trajectories of ADHD and depression symptoms, the description of the latent growth classes, and associations among latent classes of ADHD and depression symptoms within and between developmental periods. Once these aspects of the model had been established, the link between childhood and adolescent symptom classes and binge-eating was evaluated and is described below in the primary research question section.

4.3 Foundational Research Questions IA

1. **Foundational Research Question I:**
    a. **What are the developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

Model building began by identifying the shape of growth in ADHD and depression symptoms with conventional growth models. Unconditional means models with fixed and random effects for only the intercept explored the initial level of

symptoms across childhood and then adolescent time points. Unconditional growth models with fixed and random effects for the intercept and slope identified the initial level of symptoms and shape of growth. Model fit and fixed and random effects were evaluated to identify the best representation of the initial level and growth in symptoms (for model fit results, see Table 3.3; for fixed and random effects, see Table 3.4). A third set of models, conditional growth models, showed the effect of contextual predictors on the intercepts and slopes (see Table 3.5). Effects of contextual predictors on the fixed effects of the intercept and slope are reported in the secondary analysis section.

The need for a quadratic factor to account for nonlinear growth in symptoms was also evaluated. Given that quadratic factors did not account for a significant amount of variance above and beyond the linear slope and the fixed quadratic factors were not statistically significant in any model, quadratic factors were not included.

4.4 Foundational Research Questions IA: Growth Models of ADHD Symptoms

ADHD Symptoms in Childhood. The first model estimated only the fixed and random effects of the intercept, and demonstrated poor model fit with all indices outside of the critical value (see Table 3.3). The fixed and random effects for the intercept were both significant (intercept: $B = 2.407$, SE = .035, $p < .001$; σ: $B = .665$, SE = .043, $p < .001$; see Table 3.4). The addition of the slope and slope variance significantly improved the fit of the model according to the chi-square difference test ($\Delta\chi^2$ (3) = 165.619, $p < .001$), and most fit indices demonstrated adequate model fit. The intercept remained significant and the slope was also positive and significant (slope: $B = .033$, SE = .014, $p = .021$). The significant variability in the slope and intercept ($\sigma_{intercept}$: $B = .867$, SE = .061, $p < .001$; σ_{slope}: $B = .055$, SE = .011, $p < .001$) suggested that substantial individual

differences in the initial level and growth in ADHD symptoms were present, which was supported by a line graph including a random selection of 50 cases (Figure 3.1). These individual differences may represent a variety of subtypes that could be captured in latent class growth analyses.

ADHD Symptoms in Adolescence. The intercept only model demonstrated strong model fit according to all indices (see Table 3.3). The fixed and random effects of the intercept were both significant (intercept: $B = 2.468$, SE = .033, $p < .001$; σ: $B = .498$, SE = .033, $p < .001$; see Table 3.4). When both the slope and its variance were added, the model did not reach convergence owing to a non-positive definite matrix from the limited variability in the slope. Thus, the slope variance was set to zero, which allowed the model to converge and fit the data well with all fit indices within the acceptable range. The chi-square difference test indicated that adding the fixed slope parameter significantly improved model fit compared to the intercept only model ($\Delta\chi^2$ (1) = 25.758, $p < .001$). In this model, the intercept remained significant, but the slope was negative and non-significant (slope: $B = -.013$, SE = .012, $p = .304$). The significant variability in the intercept ($B = .498$, SE = .033, $p < .001$) suggested the presence of substantial individual differences in the initial level of ADHD symptoms, as can be seen in a line graph of a random selection of 50 cases (Figure 3.2). This variability indicates that distinct subtypes of the initial level and growth in symptoms may be present, supporting the use of latent class growth analyses with these data.

4.5 Foundational Research Questions IA: Growth Models of Depression Symptoms

Depression Symptoms in Childhood. The model with fixed and random effects of the intercept demonstrated poor model fit with all indices outside of the critical value (see

Table 3.3). Both the intercept and its variance were significant (intercept: B = .759, SE = .009, p < .001; σ: B = .027, SE = .003, p < .001; see Table 3.4). The addition of the slope and slope variance significantly improved the fit of the model according to the chi-square difference test ($\Delta\chi^2$ (3) = 81.044, p < .001), and all fit indices. The intercept remained significant and the slope was negative and significant (slope: B = -.047, SE = .006, p < .001). The significant variability in the slope and intercept ($\sigma_{intercept}$: B = .044, SE = .007, p < .001; σ_{slope}: B = .006, SE = .002, p = .001) suggested the presence of substantial individual differences in the initial level and growth in depression symptoms during childhood, as can be seen in a line graph of a random selection of 50 cases (Figure 3.3). The variability indicates that distinct subtypes of the initial level and growth in symptoms may be present, supporting the use of latent class growth analyses with these data.

Depression Symptoms in Adolescence. The fixed and random intercept effects model demonstrated poor model fit with all indices outside of the critical value (see Table 3.3). Both the fixed and random effects of the intercept were positive and significant (intercept: B = .641, SE = .017, p < .001; σ: B = .127, SE = .011, p < .001; see Table 3.4). The addition of the slope and its variance significantly improved the fit of the model according to the chi-square difference test ($\Delta\chi^2$ (3) = 370.691, p < .001), and most fit indices. The intercept remained significant and the slope was negative and significant (slope: B = -.042, SE = .007, p < .001). The significant variability in the slope and intercept ($\sigma_{intercept}$: B = .144, SE = .016, p < .001; σ_{slope}: B = .01, SE = .003, p < .001) suggested the presence of substantial individual differences in the initial level and growth in depression symptoms, as can be seen in a line graph of a random selection of 50 cases (Figure 3.4). The variability indicates that distinct subtypes of the initial level and growth

in symptoms may be present, supporting the use of latent class growth analyses with these data.

4.6 Foundational Research Questions IB

1. **Foundational Research Question I:**

    b. **What are the typical subgroups of the developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

Once the conventional growth models were identified, four separate latent class growth analyses were conducted to identify common developmental trajectories for ADHD and depression symptoms in childhood and in adolescence. A series of nested unconditional latent class models were explored first to identify the number of classes that provided the best fit for the data based on a balance of parsimony and model fit. Model fit characteristics are reported in Table 3.6 and probabilities of class membership are reported in Table 3.7. Conditional latent class growth models were then estimated with contextual predictors that included gender, treatment status, race, and lunch status (see Table 3.8 and 3.9). Contextual predictors were added as predictors of class membership in conditional models. Effects of contextual predictors on class membership are reported in the secondary analysis section.

4.7 Foundational Research Questions IB: ADHD Latent Class Growth Models

Childhood ADHD Symptoms. A latent class analysis of the growth model of childhood ADHD symptoms was estimated to identify the quantity and quality of typical developmental patterns of ADHD symptoms during childhood. Results from comparing

unconditional models indicated that 3 classes provided the best fit for the data (see Table 3.6). The AIC and the BIC continued to decrease as complexity increased, although improvements in relative fit appeared to slow as the number of classes increased. The LMR-LRT and BLRT remained significant as the number of classes increased as well. Thus, the fit indices provided little direction regarding the best fitting model. The probabilities for class separation and homogeneity of classes were the most compelling for the model with three classes (See Table 3.7). Class separation (non-diagonal probabilities) ranged from 0 to .104, whereas homogeneity of class (diagonal probabilities) ranged from .865 to .924. At .788, entropy indicated an adequate amount of classification occurred with the three class solution. The smallest class size was 93 (13.7%), which was reasonably large.

The three classes corresponded to interpretable trajectories. The first class was best identified as the "increasing low" symptoms class. The intercept was the lowest of the classes (B = 1.643, SE = .942, $p < .001$), and the slope was positive but very small (B = .063, SE = .018, $p < .001$). The second class was labeled as the "stable, moderate" symptoms class. The intercept was significant (B = 2.676, SE = .098, $p < .001$) and the slope was positive but non-significant (B = .067, SE = .038, p = .077). The third class fit the description of "decreasing high" symptoms. The intercept was higher than the other classes (B = 4.175, SE = .114, $p < .001$), and the slope was negative and significant (B = -.147, SE = .063, p = .018).

A conditional model was then estimated where contextual predictors (i.e., gender, intervention status, race, and lunch status) were entered as predictors of class membership. Model fit remained strong. Although model fit between non-nested models

cannot be compared, the AIC and the BIC were much lower in the conditional three class model than the unconditional three class model. The LMR-LRT and BLRT were both statistically significant as well. The probabilities for class separation ranged from .881 to .928, and the probabilities for homogeneity of classes ranged from 0 to .082, demonstrating strong classification of cases. Entropy at .805 also supported the classification provided in the model.

The three classes corresponded to nearly identical trajectories as the unconditional model (see Table 3.8 and Figure 3.5). The first class remained the "increasing low symptoms" class. The intercept was the lowest of the classes (B = 1.63, SE = .044, p < .001), and the slope was positive (B = .071, SE = .021, p < .001). The second class also remained the "stable moderate symptoms" class. The intercept was significant (B = 2.681, SE = .104, p < .001) with a non-significant slope (B = .052, SE = .042, p = .127). The third class changed from the "decreasing high symptoms" class to the "stable high symptoms" class. The intercept was the highest of the three classes (B = 4.098, SE = .114, p < .001), and the slope was non-significant (B = -.096, SE = .063, p = .219).

Adolescent ADHD Symptoms. A latent class analysis of the growth model of adolescent ADHD symptoms was estimated to identify the quantity and quality of typical developmental patterns of ADHD symptoms during adolescence. Results from comparing unconditional models indicated that two classes provided the best fit for the data (see Table 3.6). The AIC and the BIC continued to decrease as complexity increased, but the LMR-LRT and BLRT were not significant when three rather than two classes were estimated. As a result, the two class model was the highest level of complexity where the LMR-LRT and BLRT were still significant. Class separation (non-diagonal probabilities)

ranged from 0 to .063 (see Table 3.7), whereas homogeneity of class (diagonal probabilities) ranged from .937 to .964. Entropy was satisfactory at .843, indicating that an acceptable amount of classification occurred with the two class solution. The smallest class size was 191 (36.2%), which was relatively large.

Both classes displayed interpretable trajectories. The first class was best identified as the "stable low" symptoms class. The intercept was low (B = 1.983, SE = .04, p < .001), and the slope was non-significant (B = .003, SE = .014, p = .821). The second class was labeled as the "stable high" symptoms class. The intercept appeared higher than the "stable low" symptoms class (B = 3.355, SE = .066, p < .001), and the slope was negative, but non-significant (B = -.042, SE = .028, p = .133).

A conditional model was then estimated where contextual predictors (i.e., gender, intervention status, race, and lunch status) were entered as predictors of class membership. Model fit remained strong (see Table 3.6). Although model fit between non-nested models cannot be directly compared, the AIC and the BIC were somewhat lower in the conditional two class model than the unconditional two class model. The LMR-LRT and BLRT were both statistically significant, as well. The probabilities for class separation ranged from .037 to .049, and the probabilities for homogeneity of classes ranged from .936 to .951, demonstrating strong classification of cases (see Table 3.7). Entropy at .858 also supported the classification provided in the model.

The two classes corresponded to nearly identical trajectories as the unconditional model (see Table 3.8). The first class remained the "stable low" symptoms class. The intercept remained quite low (B = 1.993, SE = .041, p < .001; see Figure 3.6), and the slope was still non-significant (B = .002, SE = .014, p = .897). The second class also

remained the "stable high" symptoms class. The intercept was significant (B = 3.349, SE = .067, p < .001) with a non-significant slope (B = -.035, SE = .029, p = .228).

4.8 Foundational Research Questions IB: Depression Latent Class Growth Models

Childhood Depression Symptoms. A latent class analysis of the growth model was estimated to identify the quantity and quality of typical developmental patterns of depression symptoms during childhood. Results from comparing unconditional models indicated that two classes provided the best fit for the data (see Table 3.6). The AIC and the BIC continued to decrease as complexity increased throughout all estimated models, although improvements in relative fit appeared to slow as the number of classes increased. The LMR-LRT and BLRT demonstrated that the two class model had better fit than the one class model, but also that the three class model had better fit than the two class model. Consideration of the posterior probabilities (see Table 3.7) guided selection of the optimal number of classes to extract from these data. Although the addition of a third class provided better distinction between class 1 and class 2, class 3 demonstrated significant overlap with both classes 1 and 2 with class separation values for class 3 ranging from .132 to .222. Thus, the two class model was selected as the best representation of the data, despite weaknesses in classification. Class separation (non-diagonal probabilities) ranged from .147 to .188, which was quite high. The homogeneity of class (diagonal probabilities) ranged from .812 to .853. Entropy was also low at .477, indicating poor classification. The smallest class size was 220 (39.2%), which was reasonably large.

Both classes displayed interpretable trajectories. The first class was the "decreasing low" symptoms class. The intercept was low (B = .637, SE = .035, p < .001),

and the slope was negative and significant (B = -.044, SE = .017, p = .009). The second class was the "decreasing high" symptoms class. The intercept was significant (B = .966, SE = .026, p < .001) with a small, negative slope (B = -.051, SE = .012, p < .001).

A conditional model was then estimated where contextual predictors (i.e., gender, intervention status, race, and lunch status) were entered as predictors of class membership. Although model fit between non-nested models cannot be compared, the AIC and the BIC were lower in the conditional two class model than the unconditional two class model, indicating improved fit (see Table 3.6). The LMR-LRT and BLRT were both statistically significant when comparing the two class conditional model to the one class conditional model, as well. Comparison of the conditional three class model to the conditional two class model did not produce significant LMR-LRT and or BLRT values, supporting the stance that the two class model better represented the data than a three class model. The probabilities for class separation ranged from .131 to .172 (see Table 3.7), and the probabilities for homogeneity of classes ranged from .828 to .869, demonstrating adequate classification of cases. Entropy remained quite low at .525, suggesting that cases within these classes displayed a notable amount of variability with regard to the initial level and shape of depression symptoms displayed during childhood.

The two classes corresponded to nearly identical trajectories as the unconditional model (see Table 3.8). The first class remained the "decreasing low" symptoms class. The intercept remained quite low (B = .642, SE = .04, p < .001; see Figure 3.7), and the slope was negative and significant (B = -.048, SE = .017, p < .001). The second class also remained the "decreasing high" symptoms class. The intercept was significant (B = .954, SE = .022, p < .001) with a small, negative slope (B = -.050, SE = .011, p < .001).

Adolescent Depression Symptoms. A latent class analysis of the growth model was estimated to identify the quantity and quality of typical developmental patterns of depression symptoms during adolescence. Results from comparing unconditional models indicated that three classes provided the best fit for the data (see Table 3.6). The AIC and the BIC continued to decrease as complexity increased throughout all estimated models, although improvements in relative fit appeared to slow as the number of classes increased. The LMR-LRT demonstrated that the three class model had better fit than the two class model, but the BLRT did not reach significance when comparing the three class model to the two class model. Consideration of the posterior probabilities (see Table 3.7) also guided selection of the optimal number of classes to extract from these data. Class separation for the three class model was strong with non-diagonal probabilities ranging from 0 to .096. Homogeneity of class (diagonal probabilities) ranged from .883 to .935. Entropy was also high at .821, indicating good classification, but the smallest class size was 23 (4.3%), which was small.

All three classes corresponded to interpretable trajectories. The first model was best identified as the "decreasing low" symptoms class. The intercept was the lowest of the classes (B = .487, SE = .041, p < .001), and the slope was negative and very small (B = -.061, SE = .011, p < .001). The second class was labeled as the "stable moderate" symptoms class. The intercept was significant (B = .966, SE = .046, p < .001) and the slope was negative but non-significant (B = -.018, SE = .029, p = .533). The third class was labeled as the "stable high" symptoms class. The intercept was significant (B = 1.57, SE = .166, p < .001) and the slope was positive but non-significant (B = .049, SE = .049, p = .315).

A conditional model was then estimated where contextual predictors (i.e., gender, intervention status, race, and lunch status) were entered as predictors of class membership. Model fit remained strong (see Table 3.6). Although model fit between non-nested models cannot be directly compared, the AIC and the BIC were much lower in the conditional three class model than the unconditional three class model. The LMR-LRT and BLRT were both statistically significant in the conditional model as well. The probabilities for class separation ranged from 0 to .054, and the probabilities for homogeneity of classes ranged from .906 to .958, demonstrating strong classification of cases (see Table 3.7). A high entropy value of .85 also supported the classification provided in the model.

The three classes corresponded to nearly identical trajectories to the unconditional model (see Table 3.8). The first class remained the "decreasing low" symptoms class. The intercept was the lowest of the classes (B = .502, SE = .028, p < .001; see Figure 3.8), and the slope was negative (B = -.064, SE = .009, p < .001). The second class also remained the "stable moderate" symptoms class. The intercept was significant (B = .977, SE = .04, p < .001) with a non-significant slope (B = -.012, SE = .022, p = .600). The third class stayed the "stable high" symptoms class. The intercept was the highest of the three classes (B = 1.588, SE = .125, p < .001), and the slope was non-significant (B = -.061, SE = .046, p = .179).

4.9 Foundational Research Question II

2. **Foundational Research Question II: Do children remain in the same symptom severity class of ADHD and/or depression symptoms or change to classes with higher or lower ADHD and/or depression symptom severity?**

Transition models were used to evaluate this question and were assessed in several stages. First, transitions in symptom classes between childhood and adolescence were considered independently for ADHD and depression symptoms through separate transition models with each symptom type. Second, all four ADHD and depression latent class growth models were combined in a latent growth transition model to identify the degree to which membership in childhood symptom classes predicted membership in the same symptom severity classes or transitions to other classes. The full transition model also demonstrated the degree which childhood ADHD symptom classes contributed to membership in adolescent depression symptom classes and the degree to which childhood depression classes contributed to adolescent ADHD symptom classes.

Several versions of the model were tested based on the individual symptom latent class growth models from each developmental period to identify the model with the best combination of parsimony and model fit. Regarding ADHD symptoms, two unconditional transition models were tested based on results from the latent class growth models during childhood and adolescence. The first unconditional model included two classes for childhood ADHD symptoms and two classes for adolescence ADHD symptoms (i.e., ADHD1 (2) ADHD2 (2)). The second unconditional model expanded to three childhood symptom classes and two adolescent symptom classes (i.e., ADHD1 (3) ADHD2 (2)). Two unconditional transition models for depression symptoms were also tested, the first with two classes for childhood depression symptoms and two adolescent depression symptom classes (i.e., DEP1 (2) DEP2 (2)) and the second model with two childhood depression classes and three adolescent classes (i.e., DEP1 (2) DEP2 (3)).

These transition models were analyzed a second time with contextual predictors. Contextual predictors included gender, intervention status, race, and lunch status and were added to evaluate the effect of contextual predictors on model fit and differentiation between classes. Effects of contextual predictors on the fixed effects of the intercept and slope are reported in the secondary analysis section.

To identify the best full transition model, four unconditional transition models with all symptom types and developmental periods were evaluated. The first unconditional model had two classes for all symptom types and developmental periods (i.e., childhood depression model with two classes, childhood ADHD model with two classes, adolescent depression model with two classes, adolescent ADHD model with two classes: DEP1 (2) ADHD1 (2) DEP2 (2) ADHD2 (2)). The second unconditional model had two classes for all symptoms types and developmental periods except for childhood ADHD symptoms, where three classes were proposed (i.e., DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2)). The third unconditional model maintained two classes for all symptom types and developmental periods except adolescent depression symptoms, which had three classes (i.e., DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2)). Finally, the fourth unconditional model had two classes for childhood depression and adolescent ADHD symptoms and three classes for childhood ADHD and adolescent depression symptoms (i.e., DEP1 (2) ADHD1 (3) DEP2 (3) ADHD2 (2)).

Once fit for the unconditional models were established, conditional models were evaluated with contextual predictors added to viable models. Contextual predictors included gender, intervention status, race, and lunch status and were added to evaluate the effect of contextual predictors on model fit and differentiation between classes. Effects of

contextual predictors on the fixed effects of the intercept and slope are reported in the secondary analysis section.

To evaluate transitions among symptom classes and developmental periods, transition probabilities and log odds point estimates representing the prediction of the adolescent class membership from childhood membership was evaluated. These values indicated the likelihood of transitioning in and out of symptom classes of similar severity levels for the same type of symptom (i.e., high ADHD symptom severity class in childhood to high ADHD symptom severity class in adolescence). Values also indicated the likelihood of transitioning into high symptom classes given membership in opposite symptoms classes (i.e., likelihood of transitioning into high depression symptom severity class in adolescence given membership in ADHD symptom severity class in childhood). It was also possible to evaluate the probability of belong to concurrent symptom classes of different symptom types (i.e., high ADHD symptom severity class in childhood as well as the high depression symptom severity class in childhood).

4.10 Foundational Question II: ADHD Transition Models

Model fit statistics for latent transition models are reported in Table 3.10 with concurrent and transition probabilities reported in Table 3.11. To review fixed effects of childhood class membership on adolescent membership, see Table 3.12. The intercepts and slopes of each class combination are reported in Table 3.13. The effects of contextual predictors on class membership are reported in Table 3.15. Results of contextual predictors on class membership are discussed in the secondary analyses section.

Model Fit for ADHD Transition Models. Of the unconditional models, the ADHD transition model that demonstrated the strongest fit characteristics was the second model,

ADHD1 (3) ADHD2 (2) (see Table 3.10). The log likelihood and AIC values continued to decrease as model complexity increased, although the BIC increased as complexity increased. The log likelihood difference test was significant when the second model was compared to the first model, ADHD1 (2) ADHD2 (2). Entropy decreased as model complexity increased, although entropy was slightly lower than is preferred for appropriate classification for both models (second model: .776). Consideration of the posterior probabilities also guided selection of the optimal number of classes to extract from these data. Class separation for the second transition model was acceptable, but not ideal with non-diagonal probabilities ranging from 0 to .112. Homogeneity of class (diagonal probabilities) was also appropriate although low, ranging from .801 to .868. The smallest class size for the second unconditional model, ADHD1 (3) ADHD2 (2), was 91 (13.4%), and the smallest sample size for the class combinations was 34 (8.5%), which were an appropriate size.

Contextual predictors of gender, intervention status, race, and free and reduced lunch status were then added to the models. Although model fit between non-nested models cannot be directly compared, the model fit showed slight improvements with the addition of the contextual predictors (see Table 3.10). The AIC and the BIC were much lower in the second conditional compared to the second unconditional model (i.e., ADHD1 (3) ADHD2 (2)). Similar relationships among other fit indices were also evident between conditional and unconditional models, as well. The second conditional model, ADHD1 (3) ADHD2 (2), still fit significantly better than the first conditional model, ADHD1 (2) ADHD2 (2), according to the log likelihood difference test. The second conditional model also had a high entropy value of .821, which supported the

classification provided in the model. Class separation for the third transition model was strong with non-diagonal probabilities ranging from 0 to .08. Homogeneity of class (diagonal probabilities) was acceptable, ranging from .848 to .915. The smallest class size was 109 (19.1%) and the smallest class combination size was 32 (5.7%), both of which were an acceptable size.

Transitions from Childhood to Adolescent ADHD Symptoms. The transition probabilities indicated that individuals in all the childhood ADHD symptom classes were most likely to transition to the "stable low" ADHD symptoms class in adolescence (see Table 3.11). Of the childhood classes, the "high" childhood symptoms class was the most likely to transition to the "stable high" adolescent symptom class (transition probability = .297), but this was only .06 times higher than the probability of the "decreasing moderate" and "increasing low" symptom classes transitioning to the "stable high" adolescent class. Not surprisingly, neither the "increasing low" nor the "decreasing moderate" classes significantly differed from the "high" childhood symptoms class in predicting membership to the adolescent ADHD symptom classes ("increasing low" classes: B = -.079, SE = .626, t = -.127, p = .899; "decreasing moderate" class: B = -.288, SE = .542, t = -.531, p = .595; see Table 3.12).

The most common transition was to maintain low ADHD symptoms from childhood to adolescence, represented by "increasing low" childhood ADHD symptoms transitioning to "stable low" adolescent ADHD symptoms (n = 208; transition probability = .722). Children in the "increasing low" symptoms class demonstrated a low intercept relative to the other childhood ADHD symptoms classes (B = 1.615, SE = .05, t = 32.295, p < .001; see Table 3.13 and Figure 3.11) and a small increase in symptoms across

childhood (B = .045, SE = .021, t = 2.106, p = .035). During adolescence, symptoms demonstrated similar characteristics (intercept: B = 1.735, SE = .047, t = 36.583, p < .001; slope: (B = .035, SE = .016, t = 2.145, p = .032).

Many children also transitioned from the "decreasing moderate" symptoms class to the "stable low" symptoms class in adolescence (n = 125; transition probability = .721), demonstrating a common trend for mild childhood ADHD symptoms to decline in adolescence. Children in the "decreasing moderate" symptoms class had the highest initial level of ADHD symptoms (intercept: B = 2.871, SE = .139, t = 20.639, p < .001) when compared with other childhood symptom classes that transitioned to the "stable low" adolescent symptoms class. However, symptoms had a decreasing trajectory across childhood that remained stable in adolescence (childhood slope: B = -.110, SE = .052, t = -2.13, p = .003; adolescence slope: B = -.041, SE = .027, t = -1.490, p = .136) and demonstrated one of the lowest levels of symptoms in adolescence (B = 2.291, SE = .093, t = 24.715, p < .001).

Approximately the same number of children transitioned from both the "decreasing moderate" and "increasing low" symptoms classes to the "stable high" symptoms class in adolescence (n's = 80; transition probabilities = .279 and .278, respectively). These childhood symptoms classes displayed opposite trajectories during childhood but transitioning to very similar adolescent trajectories of ADHD symptoms. Children in the "increasing low" symptoms class started with the lowest symptom level of the childhood classes (intercept: B = 2.401, SE = .01, t = 23.96, p < .001) that transitioned to the "stable high" adolescent symptoms class. They maintained stable adolescent ADHD symptoms (slope: B = -.014, SE = .052, t = -.266, p = .791) that were

higher than those of the "decreasing moderate" symptoms class ("increasing low" adolescent intercept: B = 3.362, SE = .116, t = 29.018, p < .001; "decreasing moderate" adolescent intercept: B = 3.011, SE = .251, t = 12.0, p < .001). In contrast, the "decreasing moderate" class had a high initial level of childhood ADHD symptoms (intercept: B = -.156, SE = .076, t = -2.056, p = .040) that decreased in adolescence (slope: B = -.041, SE = .097, t = -.423, p = .672), where symptoms stayed relatively low.

Fewer children transitioned from the "high" symptoms class to the "stable low" symptoms class in adolescence (n = 76; transition probability = .703). Although children in the "high" symptoms class had a low initial level of ADHD symptoms when compared with other childhood symptom classes (intercept: B = 1.664, SE = .079, t = 21.175, p < .001), symptoms continued to increase across childhood (slope: B = .165, SE = .04, t = 4.175, p < .001) and represented the highest level of symptoms during adolescence of childhood classes transitioning to the "stable low" adolescent symptom class (intercept B = 2.887, SE = .142, t = 20.368, p < .001).

The least common trajectory was for children in the "high" symptoms class transitioning to the "stable high" adolescent class (n = 15; probability = .297). Childhood symptoms displayed notable variability across measurement periods, but little consistent change over time (slope: B = .03, SE = .105, t = .286, p = .775). Those in the "high" childhood symptoms class maintained the highest symptoms during adolescence (B = 4.06, SE = .238, t = 17.057, p < .001), although symptoms slightly declined into 9th grade (B = -.155, SE = .110, t = -1.046, p = .295).

These results demonstrated that ADHD symptoms generally decreased from childhood to adolescence. Further, symptoms that began decreasing in childhood

appeared to predict ADHD class membership nearly equivalently to childhood symptoms that were initially low. Finally, the chronicity and difficulty associated with developmental transitions associated with ADHD was present among the "high" childhood symptom class that transitioned to the "stable high" adolescent class. Although a small portion of the sample (15/596; 2.5%), this proportion falls within the prevalence range of ADHD within the population.

4.11 Foundational Question II: Depression Transition Models

Model fit statistics for latent transition models are reported in Table 3.10 with concurrent and transition probabilities reported in Table 3.11. To review fixed effects of class membership of childhood on membership in adolescence, see Table 3.12. The intercepts and slopes of each class combination are reported in Table 3.14. The effects of contextual predictors on class membership are reported in Table 3.15 and are discussed in the second on secondary analyses.

Model Fit for Depression Transition Models. Of the unconditional models, the depression transition model that demonstrated the strongest fit characteristics was the second model, DEP1 (3) DEP2 (2) (see Table 3.10). The log likelihood and AIC values continued to decrease as model complexity increased, although the BIC increased as complexity increased. The log likelihood difference test was significant when the second model was compared to the first model, DEP1 (2) DEP2 (2). Entropy decreased as model complexity increased, although entropy was slightly lower than is preferred for appropriate classification for both models (second model: .636). Consideration of the posterior probabilities (see Table 3.11) also guided model selection. Class separation for the second transition model was acceptable, but not ideal with non-diagonal probabilities

ranging from 0 to .141. Homogeneity of class (diagonal probabilities) was appropriate although low, ranging from .688 to .942. The smallest class size for the second unconditional model, DEP1 (2) DEP2 (3), was 84 (12.5%), and the smallest sample size for the class combinations was 17 (2.6%), both of which were an appropriate size.

Contextual predictors of gender, intervention status, race, and free and reduced lunch status were then added to the models. Although model fit between non-nested models cannot be directly compared, the model fit showed slight improvements with the addition of the contextual predictors (see Table 3.10). The AIC and the BIC were much lower in the second conditional compared to the second unconditional model (i.e., DEP1 (3) DEP2 (2)). Similar relationships among other fit indices were also evident between conditional and unconditional models. The second conditional model, DEP1 (3) DEP2 (2), still fit significantly better than the first conditional model, DEP1 (2) DEP2 (2). The second conditional model also had an acceptable entropy value of .701, which supported the classification provided in the model. Class separation for the second depression transition model was adequate with non-diagonal probabilities ranging from 0 to .130. Homogeneity of class (diagonal probabilities) was acceptable, ranging from .750 to .986. The smallest class size was 103 (18.2%), which was an acceptable size, whereas the smallest class combination size was quite small at 15 (2.66%).

Transitions from Childhood to Adolescent Depression Symptoms. The transition probabilities indicated that individuals in both the “stable high” and “stable low” depression symptoms class in childhood were more likely to transition into the “decreasing moderate” symptoms class in adolescence than other symptom classes (“stable high” class: probability = .567; “stable low” class: probability = .467, see Table

3.11). Both classes were also least likely to be in the "increasing high" symptoms class in adolescence compared to other adolescent classes ("stable high" class: probability = .086; "stable low" classes: probability = .225). These significance tests of class membership comparisons also supported these results (see Table 3.12). Children in the "stable low" depression symptoms class where less likely than the "increasing high" depression class to be members of the "stable high" depression symptoms class (B = 1.622, SE = .459, t = -3.533, $p < .001$). Similarly, children in the "stable low" depression symptoms class were also less likely than the "increasing high" depression class to be in the "decreasing moderate" symptoms class in adolescence (B = 1.406, SE = .609, t = -2.307, p = .021).

The most common transition was to maintain low depression symptoms from childhood to adolescence, represented by the "stable low" childhood class transitioning to the "stable low" adolescent class (n = 183; transition probability = .467). Children in the "stable low" symptoms class demonstrated a moderate intercept relative to the other childhood depression symptoms classes (B = .838, SE = .06, t = 13.945, $p < .001$; see Table 3.14 and Figure 3.12) and a small decrease in symptoms across childhood (B = -.048, SE = .02, t = -2.323, p = .02). During adolescence, symptoms remained at about the same level and continued to decrease (intercept: B = .732, SE = .039, t = 18.579, $p < .001$; slope: B = -.062, SE = .03, t = -2.083, p = .037).

The second most frequent class combination was transitioning from the "stable low" symptoms class to the "decreasing moderate" symptoms class in adolescence (n = 121; transition probability = .308). Children in the "decreasing moderate" symptoms class had some of the lowest initial levels of depression symptoms in both childhood and adolescence (childhood intercept: B = .622, SE = .042, t = 14.80, $p < .001$; adolescent

intercept: *B* = .396, SE = .052, *t* = 7.68, *p* < .001). Symptoms maintained a decreasing trajectory during both childhood and adolescence (childhood slope: *B* = -.065, SE = .018, *t* = -3.527, *p* < .001; adolescence slope: *B* = -.047, SE = .015, *t* = -3.178, *p* = .001).

Another combination included transitioning from the "stable high" class in childhood to the "stable low" class adolescence (*n* = 99; transition probabilities = .347). Childhood symptoms demonstrated a high initial level, but slightly declined (intercept: *B* = .978, SE = .04, *t* = 24.68, *p* < .001; slope: *B* = -.084, SE = .02, *t* = -4.233, *p* < .001). In adolescence, symptoms were quite low and continued to decline (intercept: *B* = .354, SE = .097, *t* = 3.659, *p* < .001; slope: *B* = -.048, SE = .023, *t* = -2.03, *p* = .042).

Transitioning from the "stable low" childhood class to the "increasing high" adolescent class demonstrated a unique pattern of depression symptoms (n = 88; transition probability = .225) Childhood symptoms demonstrated a moderate initial level relative to the other childhood depression classes, but slightly declined (intercept: *B* = .835, SE = .055, *t* = 15.106, *p* < .001; slope: *B* = -.025, SE = .023, *t* = -1.078, *p* = .281). In adolescence, symptoms also began at a moderate level and increased significantly across adolescence (intercept: *B* = .87, SE = .099, *t* = 8.742, *p* < .001; slope: *B* = .135, SE = .035, *t* = 3.812, *p* < .001).

Transitioning from the "stable high" class in childhood to the "decreasing moderate" class in adolescence had the strongest transition probability (*n* = 61; transition probabilities = .567). In this class combination, childhood symptoms demonstrated a high and stable initial level (intercept: *B* = .965, SE = .093, *t* = 10.337, *p* < .001; slope: *B* = -.023, SE = .025, *t* = -.886, *p* = .376). In adolescence, depression symptom initially began

quite high but declined into 9th grade (intercept: B = 1.40, SE = .075, t = 18.734, p < .001; slope: B = -.241, SE = .048, t = -5.028, p < .001).

The least common trajectory was for children in the "stable high" symptoms class to transition to the "increasing high" adolescent class (n = 15; probability = .086). Childhood symptoms displayed the highest initial level of the childhood classes and increased across childhood (intercept: B = .914, SE = .056, t = 16.387, p < .001; slope: B = .068, SE = .031, t = 2.213, p = .027). Those in the "high" adolescent symptoms class demonstrated an initial level of depression symptoms much greater than other adolescent classes and continued to increase into 9th grade (intercept: B = 1.738, SE = .137, t = 12.692, p < .001; slope: B = .075, SE = .06, t = 1.258, p = .208).

Although the transition probabilities suggest that depression symptoms in childhood provide limited prediction of adolescent depression symptoms, consideration of the shape and initial level of classes in childhood and adolescence indicated interesting and potentially meaningful growth in depression symptoms. Depression symptoms displayed limited variability in childhood, but adolescent symptoms varied significantly. Even small elevations in depression in childhood appear to contribute to increased depression symptoms during the transition to adolescence. Given that low childhood symptoms also predicted membership in depression classes with high and increasing symptoms, it is likely that factors other than childhood depression symptoms have a strong influence on the development of depression symptoms in adolescence.

4.12 Foundational Question II: Full Transition Models

Model fit statistics for latent transition models are reported in Table 3.16, descriptive labels for class combinations can be found in Table 3.17, and concurrent and

transition probabilities are reported in Table 3.18. To review the effects of class membership on membership in other symptom type classes or classes of other developmental periods, see Table 3.19. The effects of contextual predictors on class membership are reported in Table 3.20. Results of contextual predictors on class membership are discussed in the secondary analyses section.

Of the unconditional models, the transition model that demonstrated the strongest fit characteristics was the second model, DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2) (see Table 3.16). The log likelihood and AIC values continued to decrease as model complexity increased, although the BIC increased as complexity increased. This difference in direction with the BIC may reflect the level of complexity present within the model. The log likelihood difference test was significant for all models when compared to the first model, DEP1 (2) ADHD1 (2) DEP2 (2) ADHD2 (2). However, the fourth unconditional model, DEP1 (2) ADHD1 (3) DEP2 (3) ADHD2 (2), did not have a significant difference in log likelihood value when compared to the second, DEP (2) ADHD1 (3) DEP2 (2) ADHD2 (2), and third unconditional models, DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2). Entropy continued to increase as model complexity increased. For the second unconditional model, entropy was acceptable at .737, indicating appropriate classification. Consideration of the posterior probabilities also guided selection of the optimal number of classes to extract from these data. Class separation for the second transition model was strong with non-diagonal probabilities ranging from 0 to .106. Although some values for the homogeneity of class (diagonal probabilities) were low, most values were acceptable, ranging from .638 to .958. The smallest class size for the second unconditional model, DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2), was 221

(32.66%), which was large, although the smallest sample size for the class combinations was 7 (1.09%), which is very small.

Contextual predictors of gender, intervention status, race, and free and reduced lunch status were then added to both the second model, DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2), and the third model, DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2). The third model, DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2), did not converge on an acceptable solution, resulting in a non-positive definite matrix. The second model, DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2), did converge on a solution and demonstrated significantly better model fit than its unconditional version, although model fit between non-nested models cannot be directly compared (see Table 3.16). The AIC, BIC, and log likelihood values were smaller than the unconditional version of the model. The conditional model also had a high entropy value of .833, which supported the classification provided in the model. Class separation for the second transition model was strong with non-diagonal probabilities, ranging from 0 to .096. Homogeneity of class (diagonal probabilities) was acceptable, ranging from .701 to .989. The smallest class size was 163 (28.6%) and the smallest class combination size was 5 (.88%). Although the smallest class combination size was quite small, the smallest class size was adequate.

Transitions from Childhood to Adolescent ADHD Symptoms. The transition probabilities were calculated according to the equations in Table 2.3 and can be used in conjunction with the significance tests of the log odds of class membership comparisons to consider the likelihood for transitioning to adolescent symptoms classes given childhood symptoms class membership (see Table 3.19). Transition probabilities indicated that individuals in the “high” and “increasing moderate” ADHD symptoms

class in childhood had a slightly greater probability of transitioning to the "high" rather than "low" symptoms class in adolescence (probability = .531; probability = .553, respectively; see Table 3.18). However, only children in the "low" ADHD symptoms class were much less likely than children in the "high" ADHD symptoms class to be members of the "high" ADHD symptoms class during adolescence (B = -1.897, SE = .15, t = -3.572, p < .001). Children in the "increasing moderate" ADHD symptoms class and the "high" symptoms class did not differ significantly from the "high" childhood symptoms class in predicting membership in the "high" adolescence ADHD symptoms class (B = -.97, SE = .379, t = -1.702, p = .089). Children in the "low" ADHD symptoms class were most likely to stay in the "low" ADHD symptoms class in adolescence (probability = .712).

These results demonstrated that the ADHD symptoms classes from childhood and adolescence are quite consistent. Individuals with high symptoms in childhood relative to their peers appear to maintain their position as having similarly high symptoms relative to their peers in adolescence. Children with "increasing moderate" ADHD symptoms in childhood appear to display symptoms in adolescence that are more consistent with "high" ADHD symptoms than "low" ADHD symptoms.

Transitions from Childhood to Adolescent Depression Symptoms. The depression symptoms classes demonstrated similar consistency across developmental periods. Children in the "low" depression class were most likely to also be in the "decreasing low" adolescent depression symptoms class (probability = .647). Children in the "high" depression symptoms class were also slightly more likely to be in the "high" symptoms class in adolescence (probability = .508). These results were consistent with the capacity

for childhood class membership to predict adolescent class membership. The "decreasing low" symptoms class in childhood was significantly less likely than the "high" symptoms class to be members of the "high" adolescent depression symptoms class (probability = .353; *B* = -1.051, SE = .361, *t* = -2.907, *p* = .004; see Table 3.19). These results indicated that depression symptoms demonstrated relatively strong correspondence in symptom severity between childhood and adolescence.

4.13 Foundational Research Question III

3. **Foundational Research Question III: Are individuals in the high ADHD symptom severity class in childhood or adolescence more likely to also concurrently be in moderate or high depression symptoms classes?**

Concurrent Symptoms during Childhood. For the most part, membership in high ADHD symptoms classes in childhood corresponded slightly with membership in high childhood depression symptom classes (see Tables 3.18 and 3.19). Children in the "low" ADHD symptoms class were 1.57 times more likely to belong to the "low" depression class (probability = .415) than the "high" depression class (probability = .265). Similarly, "high" childhood ADHD symptom class membership increased odds by 1.52 times of belonging to the "high" childhood depression class (probability = .404) compared to the "low" depression class (probability = .332). Children in the "increasing moderate" ADHD symptoms class were also 1.31 times more likely to also belong to the "high" childhood depression symptom class (probability = .331) compared to the "low" depression class (probability = .253).

Concurrent Symptoms during Adolescence. Adolescents in both ADHD symptoms classes were about equivalently likely to belong to either adolescent

depression symptom class. The "high" adolescent ADHD class was 1.04 times more likely to correspond with the "high" rather than "low" ADHD symptoms class, whereas the "low" ADHD symptom class was .96 times more likely to be in the "high" compared to "decreasing low" depression symptoms class.

4.14 Foundational Research Question IV

4. **Foundational Research Question IV: Are individuals in the high ADHD symptom severity class in childhood more likely to be in moderate or high depression symptom severity classes in adolescence?**

The most likely class combination was for children in the "increasing moderate" ADHD symptoms class to transition to the "high" depression symptom class in adolescence (transition probability = .632), which was 1.72 times more likely than being in the "low" depression symptoms class in adolescence (probability = .368). However, the "increasing moderate" ADHD symptoms class in childhood was not significantly less likely than the "high" class (probability = .540) to belong to the "high" depression symptoms class (B = -.298, SE = .698, t = -.427, p = .669). The "low" symptoms ADHD class in childhood (probability = .461) was also not significantly less likely than the "high" symptoms class (probability = .540) to belong to the "high" depression symptoms class in adolescence (B = -.777, SE = .468, t = -1.661, p = .097).

4.15 Primary Research Question

1. **Primary Research Question I: How do latent classes of ADHD and depression symptoms in childhood and adolescence predict engagement in binge-eating behavior during tenth grade?**

To identify the effect of childhood and adolescent ADHD and depression symptom classes on binge-eating behaviors, cases were assigned class membership based on posterior probabilities from the full transition model. Class membership was then used to predict 10th grade binge-eating behaviors in a series of several ANCOVAs. Analyses included contextual predictors of gender, intervention status, race, and lunch status. Effects of the contextual predictors on the link between ADHD and depression symptom class membership and binge-eating behaviors are reviewed in the secondary research question section. ANCOVAs assessed the independent effect each set of symptom classes from each developmental period had on binge-eating behavior, which required four models (see Table 3.21). The childhood and adolescent classes for each symptom type as well as their interaction were also evaluated in separate models, which generated two models (see Table 3.22). Finally, the effect of childhood ADHD symptoms classes and adolescent depression symptoms classes as well as their interaction were tested to identify the synergistic, developmental effect of ADHD and depression symptoms on binge-eating behaviors (Table 3.23). This model included childhood depression symptom classes as a covariate.

Independent Effects of Each Symptom Class from Each Developmental Period. Childhood ADHD symptom classes had a significant effect on binge-eating behavior in adolescence (F (6, 450) = 5.695, p = .004, η^2= .025; see Table 3.21). The "high" ADHD symptoms class displayed the highest binge-eating behavior. The Tukey's post-hoc test indicated that the "high" ADHD symptoms class had significantly higher binge-eating symptoms than the "low" class (M_{diff} = -.148, SE = .051, p = .011; see Figure 3.19). The adolescent ADHD symptoms classes also showed significantly different binge-eating

behavior (F (4, 451) = 15.14, $p < .001$, $\eta^2 = .025$). The "high" ADHD symptom class in adolescence had higher binge-eating behaviors than the "low" symptoms class ("high" class: $M = 1.334$, SE = .04; "low" class: $M = 1.161$, SE = .039; see Figure 3. 20).

Depression classes from neither childhood nor adolescence predicted differences in binge-eating behaviors (childhood classes: F (4, 451) = 5.847, $p = .453$, $\eta^2 = .001$; adolescent classes: F (4, 452) = .053, $p = .819$; $\eta^2 < .001$).

Interaction of Childhood and Adolescent Symptoms Classes. Childhood ADHD symptoms classes interacted with the adolescent ADHD symptoms classes to predict binge-eating behaviors (F (7, 446) = 3.376, $p = .035$, $\eta^2 = .015$; see Table 3.22). Results showed that binge-eating was highest for the class combination of "high" childhood ADHD symptoms and "high" adolescent ADHD symptoms (see Figure 3. 21). The main effects of both childhood ADHD symptoms classes and adolescent ADHD symptoms classes were significant (childhood classes: F (7, 446) = 3.363, $p = .036$, $\eta^2 = .015$; adolescent classes: F (7, 446) = 14.426, $p < .001$, $\eta^2 = .031$). The interaction of childhood and adolescent depression classes was not significant (F (7, 449) = .649, $p = .421$, $\eta^2 = .001$; see Table 3.21), and the main effects of both childhood and adolescent depression classes remained non-significant as well (childhood classes: F (7, 449) = .714, $p = .398$, $\eta^2 = .002$; adolescent classes: F (7, 449) = .317, $p = .574$, $\eta^2 = .001$).

Interaction of Childhood ADHD and Adolescent Depression Symptom Classes. None of the childhood ADHD symptoms classes interacted with adolescent depression symptoms classes to predict binge-eating behaviors (see Table 3.23; see Figure 3.22), but the main effect of childhood ADHD symptoms classes remained statistically significant in the model.

4.16 Secondary Research Question

1. **Secondary Research Question I: How do contextual predictors (i.e., gender, intervention status, race, and lunch status) affect the latent trajectories and class membership of ADHD and depression symptoms in childhood and adolescence as well as their prediction of binge-eating behavior?**

To evaluate the effect that contextual predictors (i.e., gender, intervention status, race, and lunch status) may have on the development of ADHD and depression symptoms across childhood and adolescence, contextual predictors were entered in the conditional growth models to predict fixed effects of the intercept and slope (see Table 3.5). Contextual predictors were included in conditional latent class growth models as predictors of class membership (see Table 3.9). In transition analyses, contextual predictors were also added as predictors of class membership in both independent transition models as well as the full transition model (see Table 3.13 and 3.17, respectively). Finally, in binge-eating analyses, contextual predictors were added when identifying differences in binge-eating behaviors across class combinations. The direct effect of contextual predictors on binge-eating behaviors was evaluated in a separate ANCOVA (see Table 3.24).

4.17 Secondary Research Questions: Gender

Childhood ADHD Symptoms. Results of the conditional growth model indicated that the initial level of childhood ADHD symptoms was significantly different between male and female participants (B = -.462, SE = .08, $p < .001$). Boys demonstrated an initial level of ADHD symptoms that was .462 units higher than girls.

Gender was also significantly different across classes in the latent class growth model. Compared to the "increasing low" symptoms class, boys were more likely than girls to be members of both the "stable moderate" symptoms class (B = -.720, SE = .235, p = .002) and the "stable high" symptoms class (B = -2.254, SE = .430, p < .001). Membership in the "stable high" symptoms class was also more likely for boys than girls (B = -1.534, SE = .459, p = .001).

Results were consistent across the independent and full transition models and mirrored the individual latent class growth model in that gender significantly affected class membership. In the independent transition model, boys were similarly less likely to be members of the "increasing low" ADHD symptoms class compared to the "high" ADHD symptoms class (B = -.945, OR = .389, SE = .345, p = .006). Although the trend was similar, gender did not differ significantly between the "high" and "decreasing moderate" symptoms classes (B = -.613, OR = .542, SE = .435, p = .159). Compared to the "high" symptoms class in the full transition model, girls were much more likely than boys to belong to the "low" symptoms class (B = -2.036, OR = .131, SE = .501, p < .001). Girls were also more likely to be members of the "increasing moderate" than "high" symptoms class compared to boys (B = -1.029, OR = .357, SE = .406, p = .026).

Adolescent ADHD Symptoms. The conditional growth model indicated that the initial level of adolescent ADHD symptoms was significantly different between male and female participants (B = -.628, SE = .073, p < .001). Boys had an initial level of ADHD symptoms that were .628 units higher than girls. Gender did not significantly predict the slope. In the latent class growth model, the two classes demonstrated statistically significant differences by gender. Compared to the "stable low" symptoms class, boys

were more likely than girls to be members of the "stable high" symptoms class (*B* = 1.945, SE = .245, *p* < .001). In the independent transition model with only ADHD symptoms in childhood and adolescence, girls were significantly more likely to belong to the "stable low" class compared to the "stable high" class (*B* = -1.987, OR = .137, SE = .274, *p* < .001). Similarly, in the full transition model, girls were more likely to belong to the "low" symptoms class than the "high" symptoms class (*B* = -2.237, OR = .107, SE = .357, *p* < .001).

Childhood Depression Symptoms. A conditional growth model with contextual predictors was evaluated. Results indicated that the initial level of childhood depression symptoms was significantly different between male and female participants (*B* = .066, SE = .027, *p* = .013). Girls displayed initial levels of depression symptoms that were .066 units higher than boys. In the latent class growth model, the two classes demonstrated statistically significant differences by gender. Compared to the "decreasing low symptoms" class, girls were more likely than boys to be members of both the "decreasing high symptoms" class (*B* = -1.423, SE = .292, *p* < .001). Similarly, in the independent transition analyses, boys were more likely than girls to belong to the "stable low" depression symptoms class compared to the "decreasing high" symptoms class (*B* = .916, OR = 2.50, SE = .413, *p* = .004). In the full transition model, girls were less likely to belong to the "decreasing low" symptoms class than the "high" symptoms class (*B* = .681, OR = 1.976, SE = .305, *p* = .026).

Adolescent Depression Symptoms. Results from the conditional growth model indicated that the initial level of adolescent depression symptoms was significantly different between male and female participants (*B* = .111, SE = .041, *p* = .007). Girls

demonstrated an initial level of depression symptoms that were .111 units higher than boys. In the latent class growth model, the distribution of gender among the three classes demonstrated statistically significant differences. Compared to the "decreasing low" symptoms class, girls were more likely than boys to be members of the "stable high" symptoms class (B = -1.698, SE = .565, p = .003). Girls rather than boys were more likely to have membership in the "stable high" symptoms class when compared to the "stable moderate" class as well (B = -.923, SE = .23, p < .001).

In the independent transition model, girls were more likely to be members of the "high" symptoms class when compared to both the "decreasing moderate" and "stable low" symptoms classes ("decreasing moderate" class: B = 1.314, OR = 3.823, SE = .360, p < .001; "stable low" class: B = 1.865, OR = 6.456, SE = .413, p < .001). The full transition model demonstrated similar effects. Girls were also more likely to members of the "high" depression symptoms class compared to the "decreasing low" or "low" class in childhood and adolescence (childhood: B = .681, OR = 1.976, SE = .305, p = .026; adolescence: B = 1.639, OR = 5.149, SE = .357, p > .001).

Binge-Eating Behaviors. Regarding binge-eating analyses, gender had a significant effect (F (4, 452) = 5.847, p = .016, η^2= .013). Girls reported engaging in significantly more binge-eating behaviors than boys (girls: M = 1.293, SE = .037; boys: M = 1.193, SE = .041). Gender did not interact with either depression or ADHD symptoms during childhood or adolescence to predict binge-eating behaviors.

Summary of Gender Effects. To summarize the gender effects, boys consistently demonstrated higher ADHD symptoms during both developmental periods and were more likely to belong to higher rather than lower ADHD symptom severity classes.

Similarly, girls had higher depression symptoms during both developmental periods and were more likely to belong to higher rather than lower depression symptoms classes during both developmental periods. Girls also reported engaging in more binge-eating behaviors than boys did (F (4, 452) = 5.847, p = .016, η^2= .013), although this effect was independent of ADHD and depression symptoms. Neither symptoms from either developmental period interacted with gender to predict binge-eating.

4.18 Secondary Research Questions: Intervention Status

Childhood and Adolescent ADHD Symptoms. Intervention status demonstrated some influence on childhood ADHD symptoms. In the conditional growth model, the initial level of symptoms and the rate of change in symptoms were significantly different based on intervention status (intercept: B = -.363, SE = .088, p < .001; slope: B = .14, SE = .031, p < .001). Those in either the GBG or Family-Centered intervention showed an initial level of ADHD symptoms that was .363 units higher and decreased .14 units faster than those in the control group. Intervention also affected class membership in latent class growth models. Those who received either the GBG or the Family-Centered intervention were more likely to have membership in the "increasing low" symptoms class than the "stable moderate" symptoms class (B = -.838, SE = .275, p = .002).

In the independent transition analyses, children who receiving either the GBG or Family-Centered intervention in first grade were more likely to belong to the "increasing low" or the "decreasing moderate" symptoms classes than the "high" symptoms class ("increasing low" class: B = -.648, OR = .523, SE = .318, p < .001; "decreasing moderate" class: B = -1.688, OR = .185, SE = .383, p < .001). Similarly, those who received either treatment were also more likely to be members of the "high" symptoms

class than other classes in the full transition model (B = -1.245, OR = .288, SE = .544, p < .001). There were no significant differences in class membership according to intervention status between the "increasing moderate" class and the "high" class (B = .173, OR = 1.189, SE = .382, p = .65).

Intervention status did not affect the intercept or slope of the conditional growth model with adolescent ADHD symptoms and was not a significant predictor of membership for adolescent symptom classes of ADHD.

Childhood and Adolescent Depression Symptoms. No significant effects of intervention status were present in the conditional growth model for adolescent ADHD symptoms or childhood and adolescent depression symptoms. Intervention status also did not significantly affect class membership for these symptom areas in the latent class growth model. No significant effects of intervention status were found in the independent transition analyses, and intervention status did not affect membership in childhood or adolescent depression symptom classes in the full transition model, either.

Binge-Eating Behaviors. Intervention status was not a significant predictor of binge-eating behaviors (F (4, 452) = .563, p = .453, η^2= .001). Further, intervention status also did not interact with either ADHD or depression symptoms classes during childhood or adolescence to predict binge-eating behaviors.

Summary of Intervention Status Effects. In summary, children in the GBG and Family-Centered intervention conditions had a higher initial level of ADHD symptoms, but also had ADHD symptoms that declined faster than children in the control condition. No differences in intervention status in ADHD symptoms were significant during adolescence, suggesting that the interventions may have contributed to equalizing ADHD

symptom severity levels between intervention conditions and that this effect was maintained into adolescence. Adolescent ADHD symptom classes differed across intervention status, where receiving either the GBG or Family-Centered intervention increased the likelihood of belonging to the "high" ADHD symptoms class. A similar pattern was found for adolescent depression symptoms classes. Receiving the GBG or Family-Centered intervention in childhood predicted membership in the "high" rather than "low" depression symptoms class in adolescence.

4.19 Secondary Research Questions: Race

Childhood and Adolescent ADHD Symptoms. No significant effects of race were present in the conditional growth model for childhood or adolescent ADHD symptoms. Race was not significantly different among classes for childhood or adolescent ADHD symptoms in latent class growth models, independent transition analyses, or the full transition model.

Childhood Depression Symptoms. In the conditional growth model, the initial level of symptoms was significantly different based on race (B = -.135, SE = .044, p = .002). Those with African-American race or ethnicity had an initial level of depression symptoms that was .135 units higher than those with European-American race or ethnicity. In the latent class growth model, race was also significantly different between the two classes. African-American children were more likely to be members of the "decreasing high" symptoms class rather than the "decreasing low" symptoms class than European-American children (B = -1.328, SE = .437, p = .002). Race was not a significant predictor of childhood depression class membership in the independent or full transition models.

Adolescent Depression Symptoms. Although no significant effects of race were found in the conditional growth model, the three classes demonstrated statistically significant differences. African-American adolescents were more likely to have membership in the "stable high" symptoms class compared to the "decreasing low" symptoms class than European-American adolescents (B = 1.423, SE = .667, p = .033).

Binge-Eating Behaviors. Binge-eating analyses did not demonstrate any significant affects of race (F (4, 452) = .081, p = .775). Also, race did not interact with any of the symptom classes to predict binge-eating. Race was not a significant predictor of class membership in the independent or full transition analyses or in binge-eating analyses.

Summary of Race/Ethnicity Effects. In summary, race did not affect the initial level, shape, or class membership for ADHD symptoms during either developmental period. However, race did influence depression symptoms during childhood and adolescence. African-American children had slightly higher depression symptoms during childhood and were more likely to belong to the "decreasing high" symptoms class when compared to European-American children. On the other hand, European-American adolescents were also more likely to belong to the "high" depression symptoms class than the African-American adolescents.

4.20 Secondary Research Questions: Lunch Status

ADHD and Depression Symptoms from Childhood and Adolescence. No significant effects of lunch status were present in the conditional growth model. Likewise, lunch status was not significantly different among classes in latent class growth models for childhood and adolescent ADHD symptoms as well as childhood and

adolescent depression symptoms. Lunch status did not affect membership in any of the independent transition models.

However, in the full transition model, lunch status affected membership in childhood ADHD and adolescent depression classes. Children in the "increasing moderate" ADHD class were much more likely than children in the "high" ADHD symptoms class to have received free or reduced lunch (B = -.828, SE = .356, p = .02). Adolescents in the "high" depression class were much more likely than the "low" depression class to receive free or reduced lunch at school (B = .603, SE = .287, p = .036).

Binge-Eating Behaviors. Lunch status was not a significant predictor of binge-eating behaviors (F (4, 452) = .081, p = .776), and none of the symptom types from either developmental period interacted with lunch status to predict binge-eating behaviors.

Summary of Lunch Status Effects. Lunch status had very little effect on the initial level, shape of growth, or class membership of either symptom type in either developmental period. However, small effects were found in the full transition model, where children in the "increasing moderate" ADHD class and adolescents in the "high" depression class were more likely than other classes to receive free or reduced lunch. These results may suggest that other contextual characteristics have a bigger effect on the development of ADHD and depression symptoms in childhood and adolescence than lunch status.

Table 4.1. *Descriptive Statistics of Continuous Model Variables*

	Mean	Standard Deviation	Skew	Kurtosis	Minimum	Maximum
ADHD 1	2.415	1.045	.804	.236	1.00	5.81
ADHD 1a	2.312	1.001	.905	.641	1.00	5.89
ADHD 2	2.435	1.101	.762	-.060	1.00	6.00
ADHD 3	2.494	1.014	.652	-.012	1.00	6.00
ADHD 6	2.509	.941	.582	-.199	1.00	5.43
ADHD 7	2.433	.886	.677	.070	1.00	5.60
ADHD 8	2.438	.899	.599	-.180	1.00	5.33
ADHD 9	2.435	.883	.874	.488	1.00	5.44
Depression 1	.810	.355	.128	-.158	1.00	2.00
Depression 1a	.794	.344	.395	.392	1.00	2.00
Depression 2	.741	.315	.347	.158	1.00	1.86
Depression 3	.673	.324	.310	-.071	1.00	1.79
Depression 6	.744	.506	.778	.514	1.00	2.86
Depression 7	.637	.456	.944	1.025	1.00	2.71
Depression 8	.593	.466	.910	.508	1.00	2.50
Depression 9	.610	.501	1.155	1.339	1.00	2.79
Binge-Eating	1.225	.430	3.456	14.958	1.00	4.39

Notes. "1a" refers to spring time assessments. All other assessment were conducted in the fall; Binge-Eating was assessed in 10th grade.

Table 4.2. *Correlations among Continuous Model Variables.*

	ADHD1	ADHD1a	ADHD2	ADHD3	ADHD6	ADHD7	ADHD8	ADHD9	DEP1	DEP1a	DEP2	DEP3	DEP6	DEP7	DEP8	DEP9
ADHD1a	.710*															
ADHD2	.528*	.591*														
ADHD3	.459*	.506*	.546*													
ADHD6	.391*	.437*	.504*	.539*												
ADHD7	.382*	.393*	.453*	.453*	.628*											
ADHD8	.346*	.396*	.438*	.493*	.604*	.646*										
ADHD9	.342*	.350*	.423*	.423*	.542*	.528*	.543*									
DEP 1	.123*	.049	.082	.082	.002	.034	-.067	-.013								
DEP 1a	.057	.040	.060	.003	-.041	-.007	-.083	.011	.306*							
DEP 2	.016	.012	.024	.026	.002	.004	-.022	.070	.156*	.303*						
DEP 3	.133*	.100*	.089	.064	.076	.085	.113*	.096	.154*	.208*	.339*					
DEP 6	.065	.051	.046	.080	.114*	.030	.121*	.071	.088*	.163*	.163*	.309*				
DEP 7	-.007	.037	-.036	-.033	.008	-.032	.047	.042	.023	.128*	.144*	.269*	.591*			
DEP 8	.021	.051	-.001	.007	-.001	-.025	.052	-.025	.039	.176*	.214*	.285*	.467*	.545*		
DEP 9	-.030	.009	-.007	-.003	.003	-.029	.009	.035	.010	.112*	.171*	.237*	.444*	.545*	.609*	
Binge	-.061	-.047	.015	-.006	-.006	.033	-.017	.021	.053	.195*	.082	.151*	.192*	.203*	.221*	.293*

Notes. "ADHD1a" and "DEP 1a" refers to symptoms assessed during the spring. All other assessment were conducted in the fall; DEP = Depression symptoms; Binge = Binge-eating latent factor.

* $p < 0.05$.

Table 4.3. *Model Fit Statistics for Conventional Growth Curve Models*

Model	Log Likelihood	χ^2 (df)	*p*	$\Delta\chi^2$ (df)	*p*	CFI	RMSEA	SRMR
Childhood ADHD Symptoms (Grades 1-3)								
Unconditional Means Model	-3026.87	68.756 (8)	< .0001	-	-	.912	.106	.123
Unconditional Growth Model	-3001.75	21.188 (5)	.0007	165.619 (3)	< .0001	.976	.069	.037
Conditional Growth Model	-2572.55	36.273 (13)	.0005	-	-	.973	.056	.029
Childhood Depression Symptoms (Grades 1-3)								
Unconditional Means Model	-700.516	87.362 (8)	< .0001	-	-	.459	.123	.091
Unconditional Growth Model	-660.136	12.576 (5)	.0277	559.988 (3)	< .0001	.948	.048	.029
Conditional Growth Model	-555.712	20.47 (15)	.0841	-	-	.956	.032	.024
Adolescent ADHD Symptoms (Grades 6-9)								
Unconditional Means Model	-2293.957	16.889 (8)	.0313	-	-	.984	.044	.067
Unconditional Growth Model *	-2293.44	15.675 (7)	.0283	25.758 (1)	< . 0001	.984	.047	.072
Conditional Growth Model	-2143.212	25.382 (13)	.045	-	-	.986	.036	.039
Adolescent Depression Symptoms (Grades 6-9)								

Unconditional Means Model	-1148.2	73.35 (8)	< .0001	-	-	.874	.119	.085
Unconditional Growth Model	-1118.73	24.23 (5)	.0002	370.691 (3)	< .0001	.963	.082	.034
Conditional Growth Model	-1058.12	38.64 (13)	.0002	-	-	.96	.061	.025

Notes. CFI = Cumulative Fit Index; critical value: ≥ .96. RMSEA = Root Meant Square Error of Approximation; critical value: ≤ .05. SRMR= Standardized Root Mean Square Residual; critical value: ≤ .07. Conditional Growth Models included the following contextual predictors as predictors of the intercept and slope: gender, intervention status, race, and lunch status.

* The slope variance in the Unconditional Growth Model for Adolescent ADHD Symptoms was constrained to zero to achieve convergence.

Table 4.4. *Fixed and Random Effects for Convention Growth Models*

	Fixed Effect				*Random Effect*			
Models	*B*	SE	*t*	*P*	*B*	SE	*t*	*p*
Childhood ADHD Symptoms (Grades 1-3)								
Unconditional Means Model: Intercept	2.407	.035	69.138	< .0001	.665	.043	15.328	< .0001
Unconditional Growth Model: Intercept	2.379	.04	59.708	< .0001	.867	.061	14.128	< .0001
Slope	.033	.014	2.317	.021	.055	.011	5.113	< .0001
Conditional Growth Model: Intercept	2.344	.178	13.17	< .0001	.859	.064	13.422	< .0001
Slope	-.083	.059	-1.398	.162	.053	.01	5.30	< .0001
Childhood Depression Symptoms (Grades 1-3)								
Unconditional Means Model: Intercept	.759	.009	82.287	< .0001	.027	.003	9.169	< .0001
Unconditional Growth Model: Intercept	.825	.013	64.734	< .0001	.044	.007	6.093	< .0001
Slope	-.047	.006	-7.615	< .0001	.006	.002	3.368	.001
Conditional Growth Model: Intercept	.832	.014	18.489	< .0001	.039	.008	4.875	.0003
Slope	-.05	.007	-2.931	.003	.005	.002	2.50	.027
Adolescent ADHD Symptoms (Grades 6-9)								
Unconditional Means Model: Intercept	2.468	.033	75.896	< .0001	.498	.033	14.975	< .0001
Unconditional Growth Model: Intercept	2.487	.039	63.93	< .0001	.498	.033	14.962	< .0001
Slope*	-.013	.012	-1.027	.304	-	-	-	-
Conditional Growth Model: Intercept	2.485	.04	14.012	< .0001	.502	.036	13.940	< .0001
Slope*	-.012	.012	-.076	.939	-	-	-	-

Adolescent Depression Symptoms (Grades 6-9)								
Unconditional Means Model: Intercept	.641	.017	37.809	< .0001	.127	.011	11.265	< .0001
Unconditional Growth Model: Intercept	.709	.02	35.627	< .0001	.144	.016	9.189	< .0001
Slope	-.042	.007	-5.741	< .0001	.01	.003	3.554	< .0001
Conditional Growth Model: Intercept	.715	.02	8.563	< .0001	.145	.016	9.063	< .0001
Slope	-.044	.007	-.175	.861	.01	.003	3.323	.005

Notes. SE = Standard Error. Conditional Growth Model including the following contextual predictors as predictors of the intercept and slope: sex, intervention status, race, and lunch status.

*To achieve convergence, the random effect for the slope for the Adolescent ADHD Symptoms model was held constant.

Table 4.5. *Fixed Effects of Contextual Predictors in Convention Growth Models*

	Fixed Intercept Effects			*Fixed Slope Effects*		
Models	*B*	OR	SE	*B*	OR	SE
Childhood ADHD Symptoms (Grades 1-3)						
Gender	**-.462****	**1.587**	**.080**	-.044	1.045	.029
Intervention Status	**-.363****	**.696**	**.088**	**.140****	**1.150**	**.031**
Race	-.188	.829	.147	.052	1.053	.043
Lunch Status	.152	1.164	.085	-.005	.995	.031
Childhood Depression Symptoms (Grades 1-3)						
Gender	**.066***	**.936**	**.027**	.02	.980	.013
Intervention Status	-.007	.993	.029	.017	1.017	.014
Race	**-.135***	**.874**	**.044**	.028	1.028	.02
Lunch Status	.019	1.019	.028	.003	1.003	.014
Adolescent ADHD Symptoms (Grades 6-9)						
Gender	**-.628****	**1.874**	**.073**	.029	.971	.025
Intervention Status	.073	1.076	.08	.001	1.001	.028
Race	-.127	.881	.116	.005	1.005	.042
Lunch Status	.112	1.119	.081	.004	1.004	.028
Adolescent Depression Symptoms (Grades 6-9)						
Gender	**.111***	**.895**	**.041**	.027	.973	.016
Intervention Status	-.026	.974	.045	.023	1.023	.017
Race	.057	1.059	.063	-.015	.985	.026
Lunch Status	.033	1.034	.042	-.021	.979	.017

Notes. B = log odds estimates; OR = Odds Ratios; SE = Standard Error. Conditional Growth Model including the following contextual predictors as predictors of the intercept and slope: sex, intervention status, race, and lunch status. Significant contextual predictors are notated in bold text. *$p < .05$. **$p < .001$.

Table 4.6. *Model Fit Statistics for Fitting Latent Class Growth Models*

							LMR-LRT			BLRT			
Models	df	log likelihood	AIC	BIC	Δ2x log	Δdf	Mean	SD	*p*-value	BLRT	*p*-value	Entropy	Smallest Class *n* (%)
Childhood ADHD Symptoms (Grades 1-3)													
Unconditional Models													
1 class	6	-3458.302	6928.605	6955.72	-	-	-	-	-	-	-		-
2 classes	9	-3080.298	6178.596	6219.269	756.009	3	16.138	26.221	< .0001	719.233	< .0001	.815	211 (31.1%)
3 classes	12	-2966.766	5957.532	6011.761	227.065	3	0.179	30.43	< .0001	216.019	< .0001	.788	93 (13.7%)
4 classes	15	-2939.661	5909.322	5977.11	54.209	3	4.82	20.657	.0199	51.572	.0232	.751	83 (12.2%)
Conditional Models with Contextual Predictors													
3 classes	20	-2555.412	5150.823	5237.701	216.49	7	0.987	35.608	< .0001	211.722	< .0001	.806	79 (13.9%)
Childhood Depression Symptoms (Grades 1-3)													
Unconditional Models													
1 class	6	-744.295	1500.59	1527.55	-	-	-	-	-	-	-	-	-
2 classes	9	-671.101	1360.202	1400.646	146.387	3	6.587	6.586	< .0001	139.24	< .0001	.477	220 (39.2%)
3 classes	12	-660.864	1345.728	1399.653	20.474	3	2.831	6.441	.0131	19.474	.016	.572	51 (9.2%)
4 classes	15	-653.964	1337.927	1405.334	13.801	3	6.174	9.238	.147	13.127	.159	.464	45 (6.8%)
Conditional Models with Contextual Predictors													
2 classes	13	-568.644	1163.288	1219.552	160.98	7	11.454	8.175	< .0001	157.426	< .0001	.525	191 (36.2%)

3 classes	20	-552.794	1145.588	1232.147	31.70	7	14.793	15.272	.114	31	.119	.601	51 (9.2%)
Adolescent ADHD Symptoms (Grades 6-9)													
Unconditional Models													
1 class	6	-2682.72	5377.43	5403.47	-	-	-	-	-	-	-	-	-
2 classes	9	-2324.78	4667.563	4706.626	715.867	3	5.565	14.8	< .0001	680.111	< .0001	.843	191 (36.2%)
3 classes	12	-2278.97	4581.943	4634.027	91.62	3	232.171	236.699	.534	87.044	.544	.773	118 (22.2%)
Conditional Model with Contextual Predictors													
2 classes	13	-2180.52	4387.036	4442.559	776.79	7	9.98	16.239	< .0001	759.488	< .0001	.858	191 (36.2%)
Adolescent Depression Symptoms (Grades 6-9)													
Unconditional Model													
1 class	6	-1477.67	2967.33	2993.45	-	-	-	-	-	-	-	-	-
2 classes	9	-1194.72	2407.442	2446.615	565.892	3	19.415	30.64	< .0001	537.679	< .0001	.815	153 (28.9%)
3 classes	12	-1118.23	2260.461	2312.693	152.98	3	29.342	65.577	.047	145.353	.0532	.821	23 (4.3%)
4 classes	15	-1082.03	2194.067	2259.356	72.395	3	-0.392	69.061	.0974	68.785	.106	.73	14 (2.5%)
Conditional Model with Contextual Predictors													
2 classes	13	-1130.71	2287.412	2343.008	576.877	7	21.37	29.115	< .0001	564.039	< .0001	.831	154 (28.9%)
3 classes	20	-1053.27	2146.548	2232.081	154.86	7	6.098	37.291	.0009	151.418	.0011	.85	23 (4.3%)

Notes. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; df = degrees of freedom; LMR-LRT = Lo-Mendell-Rubin Likelihood-Ratio Test; BLRT = Bootstrapped Likelihood-Ratio Test; SD = Standard Deviation. Models in bold indicated best fitting models for each symptoms set. Conditional Models with contextual predictors included gender, intervention status, lunch status, and race.

Table 4.7. *Probabilities for Class Membership for Latent Class Growth Models*

Childhood ADHD Symptoms (Grades 1-3)												
Model with 2 Class Solution			Model with 3 Class Solution			Model with 4 Class Solution				Conditional Models		
	Class 1	Class 2	Class 1	Class 2	Class 3	Class 1	Class 2	Class 3	Class 4	Class 1	Class 2	Class 3
Class 1	.913	.087	.924	.076	0	.758	.057	.128	.057	.928	0	.072
Class 2	.038	.962	.101	.865	.035	.029	.92	.051	0	0	.922	.078
Class 3	-	-	0	.104	.896	.154	.1	.735	.011	.082	.037	.881
Class 4	-	-	-	-	-	.056	0	.028	.916	-	-	-
Childhood Depression Symptoms (Grades 1-3)												
Class 1	.812	.188	.797	.001	.202	-	-	-	-	.869	.131	-
Class 2	.147	.853	0	.777	.222	-	-	-	-	.172	.828	-
Class 3	-	-	.132	.067	.801	-	-	-	-	-	-	-
Adolescent ADHD Symptoms (Grades 6-9)												
Class 1	.937	.063	.837	.078	.084	-	-	-	-	.936	.037	-
Class 2	.036	.964	.06	.939	0	-	-	-	-	.049	.951	-
Class 3	-	-	.168	0	.832	-	-	-	-	-	-	-
Adolescent Depression Symptoms (Grades 6-9)												
Class 1	.917	.083	.935	.065	0	.787	0	.128	.086	.906	.084	.01
Class 2	.038	.962	.096	.883	.021	0	.943	0	.057	.054	.946	0
Class 3	-	-	0	.067	.933	.137	0	.861	.002	.042	0	.958
Class 4	-	-	-	-	-	.106	.026	.001	.867	-	-	-

Table 4.8. *Intercepts and Slopes for Best Fitting Conditional Latent Class Growth Models*

	Intercept				Slope			
Models	*B*	SE	*T*	*p*	*B*	SE	*t*	*p*
Childhood ADHD Symptoms (3 Classes)								
Increasing Low Symptoms	**1.63**	**0.044**	**36.812**	**< 0.001**	**.071**	**.021**	**3.461**	**.001**
Stable Moderate Symptoms	**2.681**	**0.104**	**25.892**	**< 0.001**	.052	.042	1.229	.127
Stable High Symptoms	**4.098**	**0.115**	**35.605**	**< 0.001**	-.096	.063	-1.526	.219
Childhood Depression Symptoms (2 Classes)								
Decreasing Low Symptoms	**0.642**	**0.04**	**16.087**	**< 0.001**	**-.048**	**.017**	**-2.791**	**< .001**
Decreasing High Symptoms	**0.954**	**0.022**	**42.43**	**< 0.001**	**-.05**	**.011**	**-4.649**	**< .001**
Adolescent ADHD Symptoms (2 Classes)								
Stable Low Symptoms	**1.993**	**0.041**	**48.521**	**< 0.001**	.002	.014	.129	.897
Stable High Symptoms	**3.349**	**0.067**	**50.35**	**< 0.001**	-.035	.029	-1.206	.228
Adolescent Depression Symptoms (3 Classes)								
Decreasing Low Symptoms	**0.502**	**0.028**	**18.16**	**< 0.001**	**-.064**	**.009**	**-6.989**	**< .001**
Stable Moderate Symptoms	**0.977**	**0.04**	**24.458**	**< 0.001**	-.012	.022	-.525	.600
Stable High Symptoms	**1.588**	**0.125**	**12.684**	**< 0.001**	.061	.046	1.344	.179

Notes. SE = Standard Error. Significant parameters are denoted in bold text. Parameters come from models that included contextual predictors (i.e., gender, intervention status, lunch status, and race).

Table 4.9. Fixed Effects of Contextual Predictors on Class Membership in Latent Class Growth Models

	Gender			Intervention Status			Race			Lunch Status		
Models	B	OR	SE	B	OR	SE	B	OR	SE	B	OR	SE
Childhood ADHD Symptoms (Grades 1-3)												
Compared to Increasing Low Class												
Stable Moderate Class	**.720****	**2.054**	**.235**	**-.838****	**.433**	**.275**	-.290	1.481	.393	.399	1.300	.262
Stable High Symptoms	**2.254****	**9.526**	**.430**	-.514	1.384	.325	-.148	1.581	.458	.426	1.531	.316
Compared to Stable Moderate Class												
Stable High Symptoms	**1.534****	**4.637**	**.459**	.324	1.448	.370	.142	1.632	.490	.027	1.027	.357
Childhood Depression Symptoms (Grades 1-3)												
Compared to Decreasing Low Class												
Decreasing High Class	**-1.423****	**.241**	**.292**	.266	1.339	.302	**-1.328****	**.265**	**.437**	.218	1.244	.297
Adolescent ADHD Symptoms (Grades 6-9)												
Compared to Stable Low Class												
Stable High Class	**1.945****	**6.994**	**.245**	-.278	1.278	.230	.135	1.259	.321	-.224	.799	.233
Adolescent Depression Symptoms (Grades 6-9)												
Compared to Decreasing Low Class												
Stable Moderate Class	**-.774****	**.461**	**.579**	.363	1.438	.515	**1.423***	**4.150**	**.667**	-.850	.427	.632
Stable High Class	**-1.698****	**.183**	**.565**	.412	1.510	.489	1.174	3.235	.585	.569	1.767	.609
Compared to Decreasing Moderate Class												

Stable High Class	**-.923****	**.397**	**.23**	.049	1.050	.242	-.249	.780	.341	.281	1.325	.230

Notes. B = log odds estimates; OR = Odds Ratios; SE = Standard Error. Conditional Latent Class Growth Models included the following contextual predictors as class membership for each symptom type at each developmental period: gender, intervention status, race, and lunch status.
**p* < .05. ***p* < .01.

Table 4.10. *Model Fit Statistics for Independent Symptoms Latent Transition Growth Models*

	df	Log Likelihood	AIC	BIC	Δ2x log	Δdf	*p*	Entropy	Smallest Class *n* (%)	Smallest Class Combination *n* (%)
ADHD Transition Models: Unconditional Models										
ADHD1 (2) ADHD2 (2)	27	-5191.19	10436.37	10558.39	--	--	--	.801	237 (35%)	90 (13.3%)
ADHD1 (3) ADHD2 (2)	37	-5111.34	10296.48	10463.69	79.85	10	<.0001	.776	91 (13.4%)	34 (8.5%)
ADHD Transition Models: Conditional Models										
ADHD1 (2) ADHD2 (2)	35	-4658.23	9386.46	9538.50	--	--	--	.845	192 (33.8%)	75 (13.1%)
ADHD1 (3) ADHD2 (2)	49	-4574.28	9246.56	9459.41	83.95	14	< .0001	.821	109 (19.1%)	32 (5.67%)
Depression Transition Models: Unconditional Models										
DEP1 (2) DEP2 (2)	27	-1769.35	3592.71	3714.44	--	--	--	.676	255 (38%)	18 (2.66%)
DEP1 (2) DEP2 (3)	37	-1703.54	3481.08	3657.91	55.81	10	<.0001	.636	84 (12.5%)	17 (2.6%)
Depression Transition Models: Conditional Models										
DEP1 (2) DEP2 (2)	35	-1617.39	3298.79	3450.7	--	--	--	.716	193 (34.1%)	16 (2.8%)
DEP1 (2) DEP2 (3)	49	-1542.7	3183.39	3396.07	74.69	14	<.0001	.701	103 (18.21%)	15 (2.66%)

Notes. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; df = degrees of freedom; DEP = Depression. Conditional Models included contextual predictors (i.e., gender, intervention status, race, and lunch status).

Table 4.11. *Transition Probabilities for Independent Symptom Latent Transition Models.*

ADHD Transition Probabilities		
	Adolescent ADHD	
	Stable Low	Stable High
Childhood ADHD		
Increasing Low	.722	.278
Decreasing Moderate	.721	.279
High	.703	.297

Depression Transition Probabilities			
	Adolescent Depression		
	Stable Low	Decreasing Moderate	Increasing High
Childhood Depression			
Stable Low	.308	.467	.225
Stable High	.347	.567	.086

Table 4.12. *Fixed Effect of Childhood Class Membership on Adolescent Class Membership for Independent Symptom Transition Models*

ADHD Transition Model	Adolescent ADHD Symptoms				
	Stable High Symptoms Class				
	B	OR	SE	*t*-value	*p*
Childhood ADHD Symptoms					
Compared to High Class					
Increasing Low Class	-.079	.924	.626	-.127	.899
Decreasing Moderate Symptoms	-.288	.750	.542	-.531	.595

Depression Transition Model	Adolescent Depression Symptoms									
	Stable High Symptoms Class					Decreasing Moderate Class				
	B	OR	SE	*t*-value	*p*	B	OR	SE	*t*-value	*p*
Childhood Depression Symptoms										
Compared to Increasing High Class										
Stable Low Class	**-1.406**	**.245**	**.609**	**-2.307**	**.021**	**-1.622**	**.198**	**.459**	**-3.533**	**< .001**

Notes. B = log odds estimates; OR = Odds Ratio; SE = Standard Error. Conditional Latent Class Growth Models included the following contextual predictors as predictors of class membership for each symptom type at each developmental period: gender, intervention status, race, and lunch status. Bold parameter estimates denote statistically significant effects.

Table 4.13. *Intercepts and Slopes for Best Fitting Independent Conditional ADHD Transition Model*

	Intercept				*Slope*			
Models	*B*	SE	*t*	*p*	*B*	SE	*t*	*p*
Increasing Low Symptoms + Stable Low Symptoms (21: *n* = 208)								
Increasing Low Symptoms	**1.615**	**.05**	**32.295**	**< .0001**	**.045**	**.021**	**2.106**	**.035**
Stable Low Symptoms	**1.735**	**.047**	**36.583**	**< .0001**	**.035**	**.016**	**2.145**	**.032**
Increasing Low Symptoms + Stable High Symptoms (22: *n* = 80)								
Increasing Low Symptoms	**2.401**	**.010**	**23.96**	**< .0001**	**.280**	**.059**	**4.745**	**< .0001**
Stable High Symptoms	**3.362**	**.116**	**29.018**	**< .0001**	-.014	.052	-.266	.791
Decreasing Moderate Symptoms + Stable Low Symptoms (11: *n* = 125)								
Decreasing Moderate Symptoms	**2.871**	**.139**	**20.639**	**< .0001**	**-.110**	**.052**	**-2.130**	**.003**
Stable Low Symptoms	**2.291**	**.093**	**24.715**	**< .0001**	-.041	.027	-1.490	.136
Decreasing Moderate Symptoms + Stable High Symptoms (12: *n* = 80)								
Decreasing Moderate Symptoms	**4.094**	**.170**	**24.049**	**< .001**	**-.156**	**.076**	**-2.056**	**.040**
Stable High Symptoms	**3.011**	**.251**	**12.000**	**< .0001**	-.041	.097	-.423	.672
High Symptoms + Stable Low Symptoms (31: *n* = 76)								
High Symptoms	**1.664**	**.079**	**21.175**	**< .0001**	**.165**	**.040**	**4.175**	**< .0001**
Stable Low Symptoms	**2.887**	**.142**	**20.368**	**< .0001**	-.031	.051	-.615	.538
High Symptoms + Stable High Symptoms (32: *n* = 32)								
High Symptoms	**4.012**	**.211**	**19.012**	**< .0001**	.03	.105	.286	.775
Stable High Symptoms	**4.060**	**.238**	**17.057**	**< .0001**	-.115	.110	-1.046	.295

Notes. SE = Standard Error. Classes from childhood ADHD symptoms are listed first and then adolescent ADHD symptoms. Significant parameters are denoted in bold text. Parameters come from models that included contextual predictors (i.e., gender, intervention status, lunch status, and race. Significant parameters denoted in bold text.

Table 4.14. *Intercepts and Slopes for Best Fitting Independent Conditional Depression Transition Model*

	Intercept				*Slope*			
Models	*B*	SE	*t*	*p*	*B*	SE	*t*	*p*
Stable Low Symptoms + Stable Low Symptoms (11: *n* = 183)								
Stable Low Symptoms	**.838**	**.060**	**13.945**	**< .0001**	**-.048**	**.020**	**-2.323**	**.020**
Stable Low Symptoms	**.732**	**.039**	**18.579**	**< .0001**	**-.062**	**.030**	**-2.083**	**.037**
Stable Low Symptoms + Decreasing Moderate Symptoms (12: *n* = 121)								
Stable Low Symptoms	**.622**	**.042**	**14.800**	**< .0001**	**-.065**	**.018**	**-3.527**	**< .0001**
Decreasing Moderate Symptoms	**.396**	**.052**	**7.680**	**< .0001**	**-.047**	**.015**	**-3.178**	**.001**
Stable Low Symptoms + Increasing High Symptoms (13: *n* = 88)								
Stable Low Symptoms	**.835**	**.055**	**15.106**	**< .0001**	-.025	.023	-1.078	.281
Increasing High Symptoms	**.870**	**.099**	**8.742**	**< .0001**	**.135**	**.035**	**3.812**	**< .0001**
Stable High Symptoms + Stable Low Symptoms (21: *n* = 99)								
Stable High Symptoms	**.978**	**.040**	**24.680**	**< .0001**	**-.084**	**.020**	**-4.233**	**< .0001**
Stable Low Symptoms	**.354**	**.097**	**3.659**	**< .0001**	**-.048**	**.023**	**-2.030**	**.042**
Stable High Symptoms + Decreasing Moderate Symptoms (22: *n* = 61)								
Stable High Symptoms	**.965**	**.093**	**10.337**	**< .0001**	-.023	.025	-.886	.376
Decreasing Moderate Symptoms	**1.400**	**.075**	**18.734**	**< .0001**	**-.241**	**.048**	**-5.028**	**< .0001**
Stable High Symptoms + Increasing High Symptoms (23: *n* = 15)								
Stable High Symptoms	**.914**	**.056**	**16.387**	**< .0001**	**.068**	**.031**	**2.213**	**.027**
Increasing High Symptoms	**1.738**	**.137**	**12.692**	**< .0001**	.075	.060	1.258	.208

Notes. SE = Standard Error. Classes from childhood depression symptoms are listed first and then adolescent ADHD symptoms. Significant parameters are denoted in bold text. Parameters come from models that included contextual predictors (i.e., gender, intervention status, race, and lunch status). Significant parameters denoted in bold text.

Table 4.15. *Effects of Contextual Predictors on Class Membership in Independent Symptoms Transition Models.*

	Gender			Intervention Status			Race			Lunch Status		
Models	B	OR	SE	B	OR	SE	B	OR	SE	B	OR	SE
ADHD Transition Model: Childhood ADHD Symptoms												
Compared to High (3) Class												
Increasing Low Class (2)	**-.945****	**.389**	**.345**	**-.648***	**.523**	**.318**	1.038	2.824	.764	-.183	.833	.342
Decreasing Moderate Class (1)	-.613	.542	.435	**-1.688****	**.185**	**.383**	.563	1.756	.930	.164	1.178	.371
ADHD Transition Model: Adolescent ADHD Symptoms												
Compared to Stable High Class (2)												
Stable Low Class (1)	**-1.987****	**.137**	**.274**	.158	1.171	.322	-.108	.898	.352	-.305	.737	.243
Depression Transition Model: Childhood Depression Symptoms												
Compared to Stable High Class (2)												
Stable Low Class (1)	**.916****	**2.50**	**.318**	.133	1.142	.294	.732	2.079	.518	-.468	.626	.347
Depression Transition Model: Adolescent Depression Symptoms												
Compared to Increasing High Class (3)												
Stable Low Class (2)	**1.865****	**6.456**	**.413**	-.791	.453	.411	.649	1.914	.560	.028	1.028	.371
Decreasing Moderate Class (1)	**1.341****	**3.823**	**.360**	-.283	.754	.362	-.965	.381	.646	.304	1.355	.358

Notes. B = log odds; OR = Odds Ratios; SE = Standard Error. Conditional Latent Class Growth Models included the following contextual predictors as predictors of class membership: gender, intervention status, race, and lunch status. Statistically significant parameters are denoted in bold text. Significant parameters denoted in bold text.

*$p < .05$. **$p < .01$.

Table 4.16. *Model Fit Statistics for Full Latent Transition Growth Models*

Transition Model	df	Log Likelihood	AIC	BIC	Δ2x log	Δdf	*p*	Entropy	Smallest Class *n* (%)	Smallest Class Combination *n* (%)
Unconditional Models										
1. DEP1 (2) ADHD1 (2) DEP2 (2) ADHD2 (2)	123	-6932.79	14111.59	14667.44	--	--	--	.733	196.57 (29%)	8 (1.22%)
2. DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2)	174	-6838.36	14024.71	14811.04	94.43	51	<.001	.746	121 (17.84%)	6 (.92%)
3. DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2)	174	-6839.55	14027.10	14813.43	93.24	51	<.001	.737	221 (32.66%)	7 (1.09%)
4. DEP1 (2) ADHD1 (3) DEP2 (3) ADHD2 (2)	250	-6751.52	14003.05	15132.83	181.27	127	.001	.743	178 (26.26%)	3 (.47%)
Conditional Models with Contextual Predictors										
DEP1 (2) ADHD1 (2) DEP2 (3) ADHD2 (2)*	238	-5971.75	12419.51	13453.35	--	--	--	.854	122 (21.4%)	3 (.56%)
DEP1 (2) ADHD1 (3) DEP2 (2) ADHD2 (2)	238	-5959.90	12365.78	13429.62	--	--	--	.833	163 (28.6%)	5 (.88%)

Notes. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; df = degrees of freedom; DEP = Depression. All unconditional models were compared to the first unconditional model (i.e., DEP1 (2) ADHD1 (2) DEP2 (2) ADHD2 (2)). When the fourth unconditional model (i.e., DEP1 (2) ADHD1 (3) DEP2 (3) ADHD2 (2)) was compared to the second and third unconditional model, the log likelihood difference test was not significant. Conditional Models included contextual predictors (i.e., gender, intervention status, race, and lunch status). Conditional Models with Outcome included contextual predictors and binge-eating behaviors.

*The model did not converge; model non-identification due to a non-positive definite matrix made parameters and standard errors unreliable.

Table 4.17. *Class Combination Labels for Full Latent Transition Model*

Class Combination Label				
	ADHD		Depression	
	Childhood Symptoms	Adolescent Symptoms	Childhood Symptoms	Adolescent Symptoms
1111	increasing moderate	high	high	decreasing low
1112	increasing moderate	high	high	high
1121	increasing moderate	high	low	decreasing low
1122	increasing moderate	high	low	high
1211	increasing moderate	low	high	decreasing low
1212	increasing moderate	low	high	high
1221	increasing moderate	low	low	decreasing low
1222	increasing moderate	low	low	high
2111	low	high	high	decreasing low
2112	low	high	high	high
2121	low	high	low	decreasing low
2122	low	high	low	high
2211	low	low	high	decreasing low
2212	low	low	high	high
2221	low	low	low	decreasing low
2222	low	low	low	high
3111	high	high	high	decreasing low
3112	high	high	high	high
3121	high	high	low	decreasing low
3122	high	high	low	high
3211	high	low	high	decreasing low
3212	high	low	high	high
3221	high	low	low	decreasing low
3222	high	low	low	high

Table 4.18. *Concurrent and Transition Probabilities for the Full Transition Model.*

	Childhood ADHD			Adolescent ADHD	
	Low	Increasing Moderate	High	Low	High
Childhood Depression					
Low	.415	.253	.332	.565	.435
High	.265	.331	.404	.633	.367
Adolescent Depression					
Decreasing Low	.539	.368	.460	.534	.466
High	.461	.632	.540	.514	.486

	Adolescent Depression	
Childhood Depression	Decreasing Low	Stable High
Low	.647	.353
High	.492	.508

	Adolescent ADHD	
Childhood ADHD	Low	High
Low	.712	.288
Increasing Moderate	.447	.553
High	.469	.531

Notes. Between developmental period/within symptom transition probabilities calculated using the equations described in Table 3.11 (see Muthén & Asparouhov, 2011).

Table 4.19. *Fixed Effect of Class Membership on Membership in other Classes*

	Adolescent ADHD Symptoms (Grades 6-9)					Adolescent Depression Symptoms (Grades 6-9)				
	High Symptoms Class					High Symptoms Class				
Models	B	OR	SE	*t*-value	*p*	B	OR	SE	*t*-value	*p*
Childhood ADHD Symptoms (Grades 1-3)										
Compared to High Symptoms Class										
Low Symptoms Class	**-1.897****	**.150**	**.531**	**-3.572**	**< .001**	-.777	.460	.468	-1.661	.097
Increasing Moderate Symptoms	-.970	.379	.570	-1.702	.089	-.298	.742	.698	-.427	.669
Childhood Depression Symptoms (Grades 1-3)										
Compared to High Symptoms Class										
Decreasing Low Symptoms Class	--	--	--	--	--	**-1.051**	**.350**	**.361**	**-2.907**	**.004**

Notes. B = log odds estimates; OR = Odds Ratio; SE = Standard Error. Conditional Latent Class Growth Models included the following contextual predictors as predictors of class membership for each symptom type at each developmental period: gender, intervention status, race, and lunch status. Significant parameters are denoted in bold text.

*$p < .05$. **$p < .01$.

Table 4.20. *Effects of Contextual Predictors on Class Membership in the Full Latent Transition Model.*

	Gender			Intervention Status			Race			Lunch Status		
Models	B	OR	SE	B	OR	SE	B	OR	SE	B	OR	SE
Childhood ADHD Symptoms (Grades 1-3)												
Compared to High Class												
Low Class	**-2.036****	**.131**	**.501**	**-1.245***	**.288**	**.544**	-1.042	.353	.565	-.252	.778	.460
Increasing Moderate Symptoms	**-1.029***	**.357**	**.406**	.173	1.189	.382	-.354	.702	.495	**-.828***	**.437**	**.356**
Childhood Depression Symptoms (Grades 1-3)												
Compared to High Class												
Decreasing Low Symptoms Class	**.681***	**1.976**	**.305**	.222	1.249	.371	.077	1.08	.441	-.083	.92	.319
Adolescent ADHD Symptoms (Grades 6-9)												
Compared to High Class												
Low Class	**-2.237****	**.107**	**.469**	.286	1.331	.390	-.338	.713	.487	.190	1.209	.324
Adolescent Depression Symptoms (Grades 6-9)												
Compared to High Class												
Low Class	**1.639****	**5.149**	**.357**	-.405	.667	.334	-.801	.449	.424	**.603***	**1.828**	**.287**

Notes. B = log odds; OR = Odds Ratios; SE = Standard Error. Conditional Latent Class Growth Models included the following contextual predictors as predictors of class membership for each symptom type at each developmental period: gender, intervention status, race, and lunch status. Significant parameters are denoted in bold text.

*$p < .05$. **$p < .01$.

Table 4.21. *ANCOVA for ADHD and Depression Symptoms Classes Predicting 10th Grade Binge-Eating*

	df	F	η^2	*p*	Binge-Eating: Mean Difference (SE)	
ADHD1 (3 Classes)	6, 450	**5.695**	**.025**	**.004**		
Class 1 vs. Class 2					.069 (.051)	
Class 2 vs. Class 3					**-.148 (.051)***	
Class 1 vs. Class 3					-.079 (.048)	
					Class 1: Mean (SE)	Class 2: Mean (SE)
ADHD2 (2 Classes)	5, 451	**15.14**	**.032**	**< .001**	**1.334 (.04)**	**1.161 (.039)**
DEP1 (2 Classes)	5, 451	.564	.001	.453	1.262 (.041)	1.226 (.041)
DEP2 (2 Classes)	5, 451	.053	.000	.819	1.238 (.039)	1.248 (.039)
DEP1 x DEP2	7, 449					
DEP1		.714	.002	.398	1.259 (.042)	1.217 (.042)
DEP2		.317	.001	.574	1.226 (.041)	1.196 (.039)
DEP1 x DEP2		.649	.001	.421		
DEP2 (Class 1)					1.264 (.045)	1.187 (.059)
DEP2 (Class 2)					1.254 (.056)	1.247 (.047)

Notes. ADHD1 = Childhood ADHD classes; ADHD2 = Adolescent ADHD classes; DEP1 = Childhood depression classes; DEP2 = Adolescent depression classes. ANCOVAs with ADHD and depression classes were conducted that included contextual predictors of gender, intervention status, race, and lunch status. Significant parameters are denoted in bold text. For ADHD1, Class 1 = "Increasing Moderate" Symptoms; Class 2 = "Low" Symptoms; Class 3 = "High" Symptoms. For ADHD2, Class 1 = "High" Symptoms; Class 2 = "Low" Symptoms. For DEP1, Class 1 = "High" Symptoms; Class 2 = "Low" Symptoms. For DEP2, Class 1 = "Low" Symptoms; Class 2 = "High" Symptoms.

*$p < .05$.

Table 4.22. *ANCOVA for Interactions between Childhood ADHD and Adolescent Symptom Classes Predicting 10th Grade Binge-Eating*

					Binge-Eating			
	df	F	η^2	*p*	Mean Difference (SE)	Class 1: Mean (SE)	Class 2: Mean (SE)	Class 3: Mean (SE)
ADHD1	7, 446	**3.363**	**.015**	**.036**		1.468 (.06)	1.336 (.07)*	1.523 (.067)
Class 1 vs. Class 2					.157 (.072)			
Class 2 vs. Class 3					**-.188 (.071)***			
Class 1 vs. Class 3					-.043 (.068)			
ADHD2		**14.426**	**.031**	**< .001**		1.57 (.057)	1.315 (.059)	--
ADHD1 x ADHD2		**3.376**	**.015**	**.035**				
ADHD2 ("High" Symptoms, Class 1)						1.663 (.088)	1.354 (.088)	1.693 (.077)
ADHD2 ("Low" Symptoms, Class 2)						1.272 (.069)	1.318 (.092)	1.354 (.09)
ADHD1	7, 446	**3.749**	**.017**	**.007**		1.424 (.060)	1.36 (.071)*	1.56 (.068)
Class 1 vs. Class 2					.146 (.073)			
Class 2 vs. Class 3					**-.188 (.073)***			
Class 1 vs. Class 3					-.043 (.069)			
DEP2		.899	.002	.343		1.418 (.058)	1.478 (.057)	--
ADHD1 x DEP2		1.558	.007	.212				
DEP2 ("Low" Symptoms, Class 1)						1.323 (.073)	1.348 (.094)	1.583 (.081)
DEP2 ("High" Symptoms, Class 2)						1.524 (.088)	1.372 (.086)	1.537 (.087)

Notes. ADHD1 = Childhood ADHD classes; ADHD2 = Adolescent ADHD classes; DEP1 = Childhood depression classes; DEP2 = Adolescent depression classes. ANCOVAs were conducted that included contextual predictors of gender, intervention status, race, and lunch status. Significant parameters denoted in bold text. For ADHD1, Class 1 = "Increasing Moderate" Symptoms; Class 2 = "Low" Symptoms; Class 3 = "High" Symptoms. For ADHD2, Class 1 = "High"

Symptoms; Class 2 = “Low” Symptoms. For DEP1, Class 1 = “High” Symptoms; Class 2 = “Low” Symptoms. For DEP2, Class 1 = “High” Symptoms; Class 2 = “Low” Symptoms.

*$p < .05$.

Table 4.23. *ANCOVA for Effect of Contextual Predictors on 10th Grade Binge-Eating*

	F	η^2	*p*	Mean (SE)	Mean (SE)
				Males	Females
Gender	**5.847**	**.013**	**.016**	**1.193 (.041)**	**1.293 (.037)**
				Control	Treatment
Intervention Status	.563	.001	.453	1.227 (.035)	1.260 (.044)
				European-American	African-American
Race	.081	.000	.775	1.234 (.025)	1.252 (.060)
				Paid for Lunch	Free or Reduced Lunch
Lunch Status	.081	.000	.776	1.237 (.042)	1.25 (.037)

Notes. df = 4, 452. Significant parameters denoted in bold text.

Observed Trajectories of ADHD Symptoms in Childhood

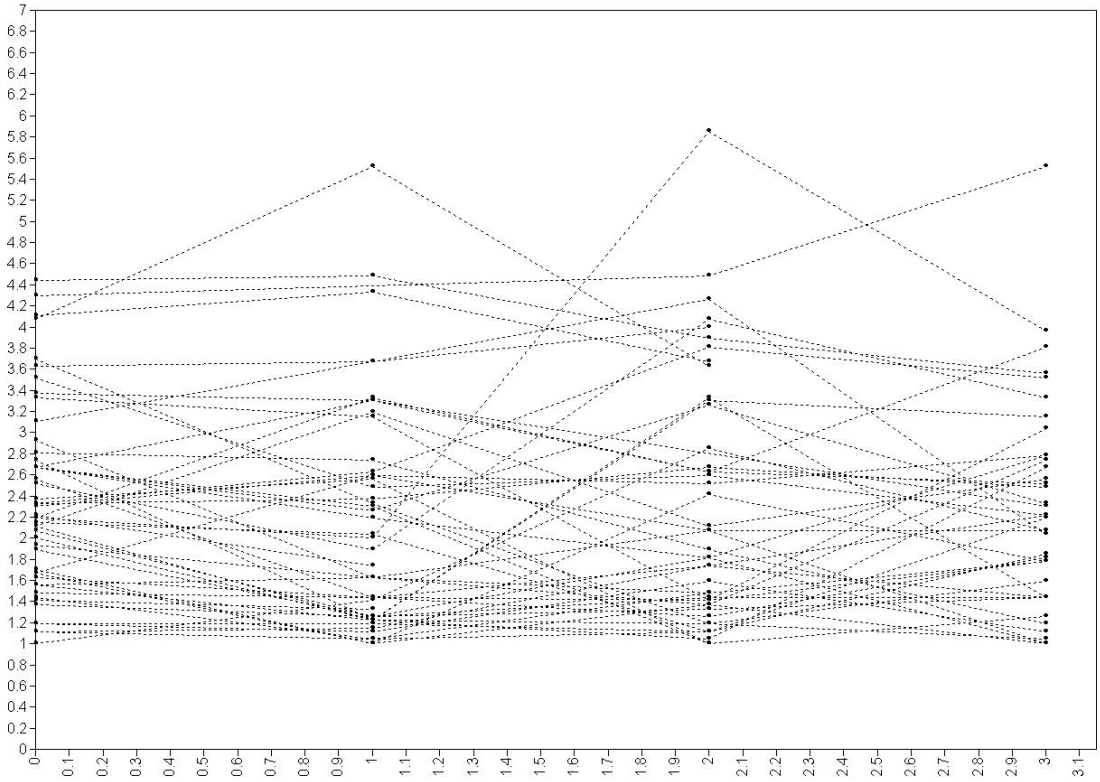


Figure 4.1.

Random Selection of 50 Observed Trajectories of ADHD Symptoms in Childhood.

Observed Trajectories of ADHD Symptoms in Adolescence

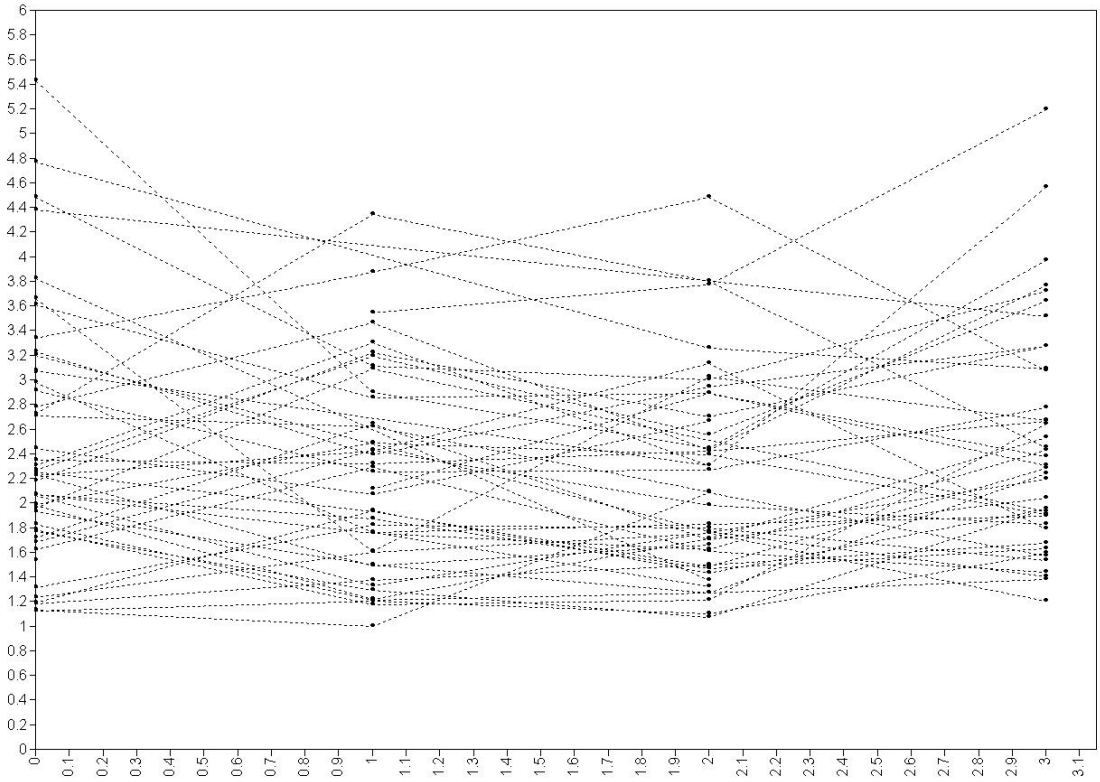


Figure 4.2.

Random Selection of 50 Observed Trajectories of ADHD Symptoms in Adolescence.

Observed Trajectories of Depression Symptoms in Childhood

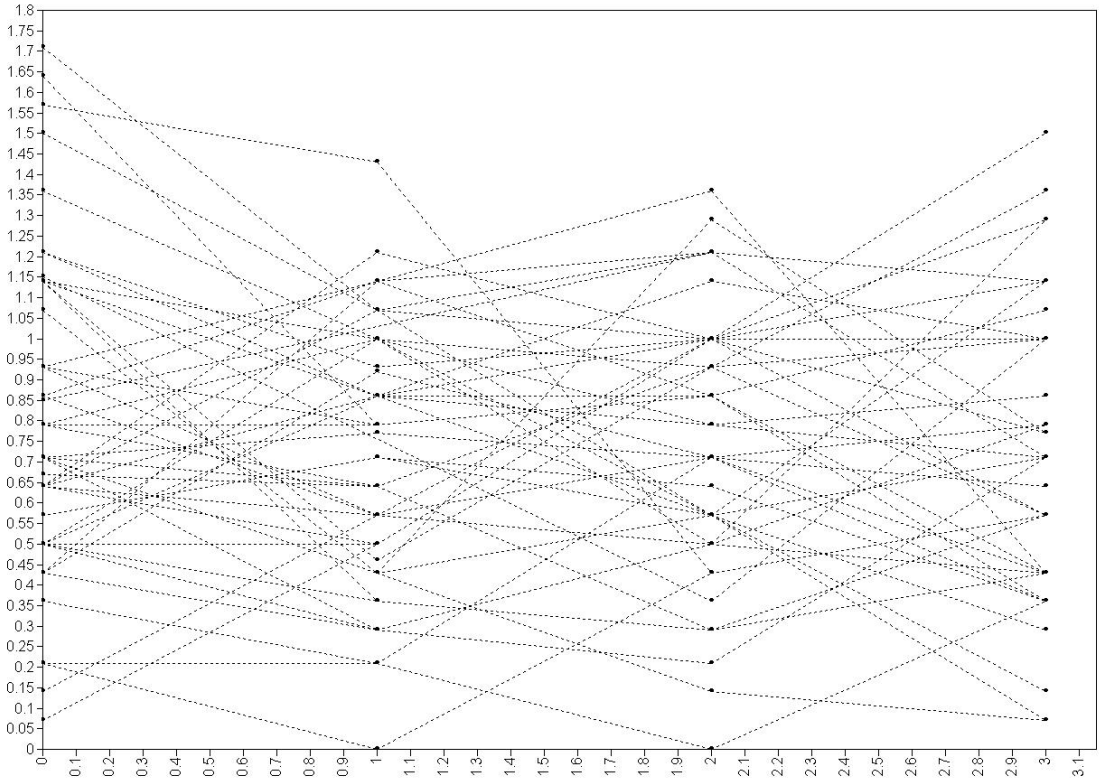


Figure 4.3.

Random Selection of 50 Observed Trajectories of Depression Symptoms in Childhood.

Observed Trajectories of Depression Symptoms in Adolescence

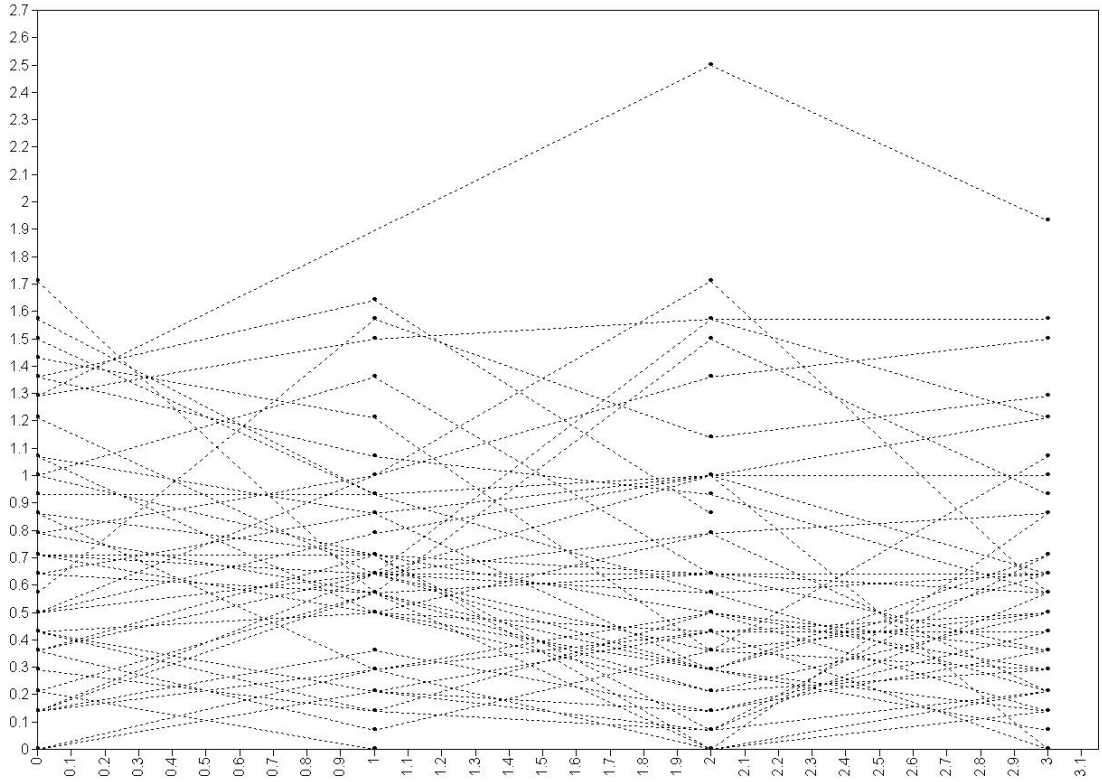


Figure 4.4.

Random Selection of 50 Observed Trajectories of Depression Symptoms in Adolescence

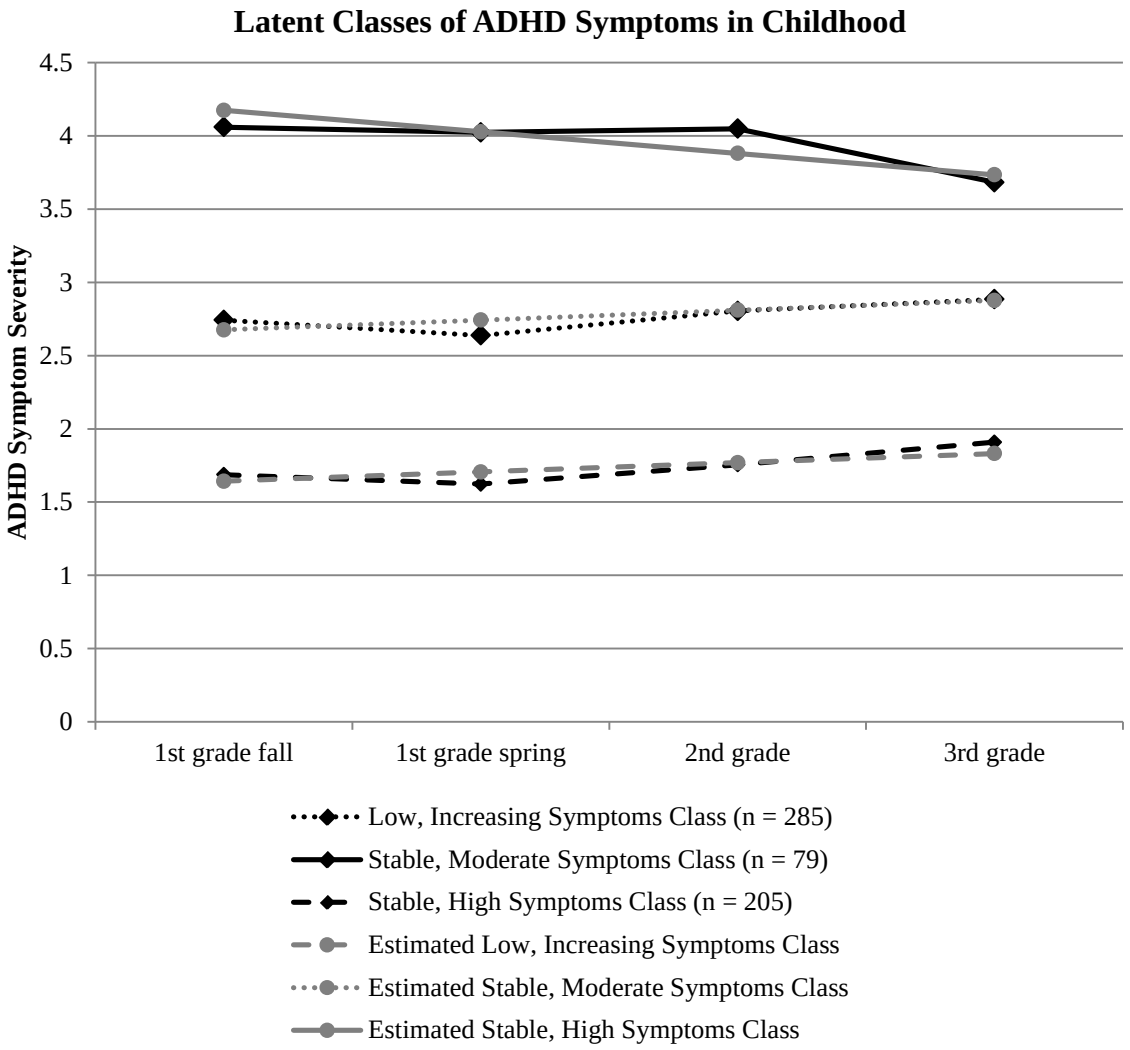


Figure 4.5.

Three Class Solution of the Latent Growth Model of ADHD Symptoms in Childhood

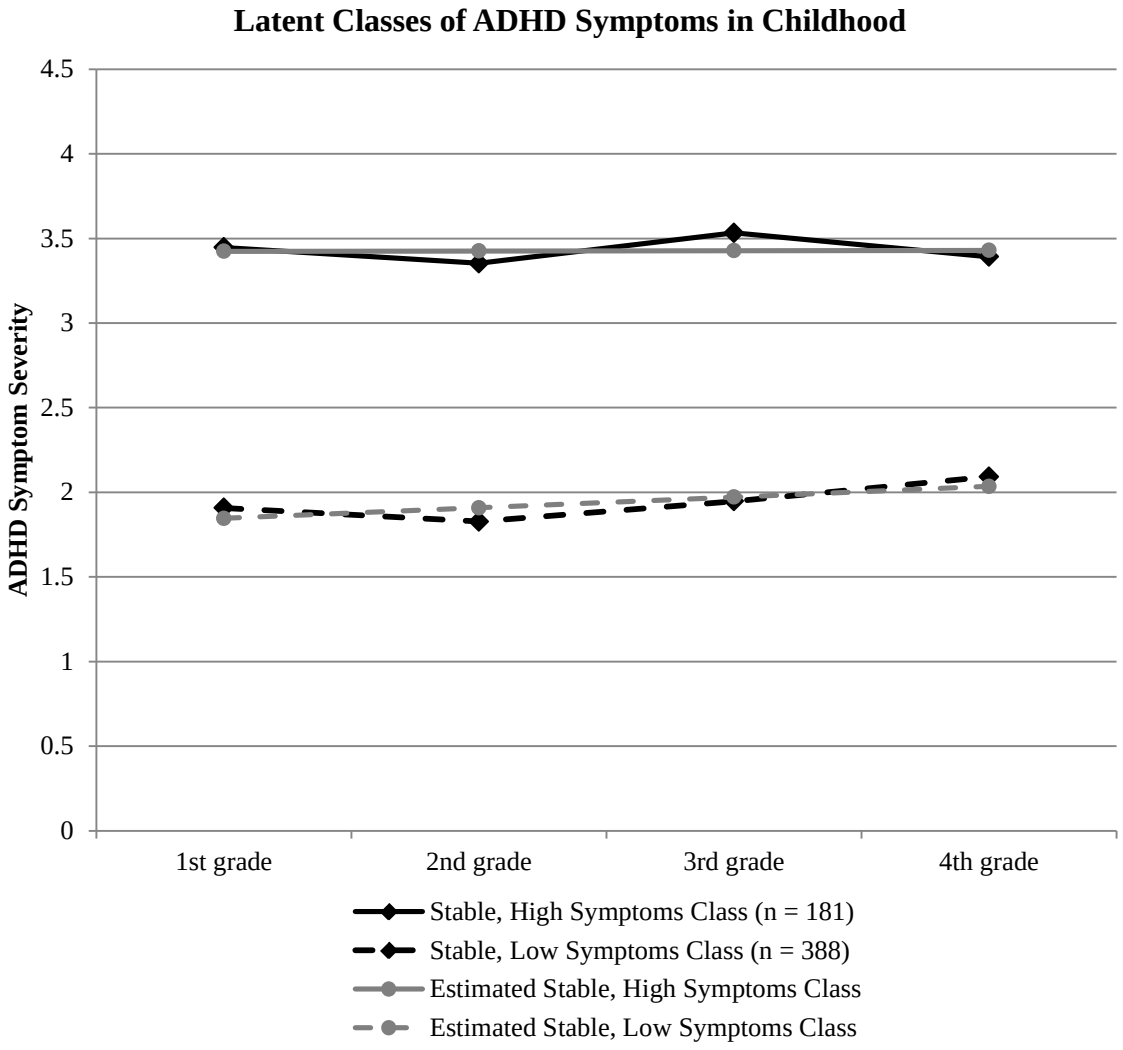


Figure 4.6.

Two Class Solution for the Latent Growth Model of ADHD Symptoms in Childhood

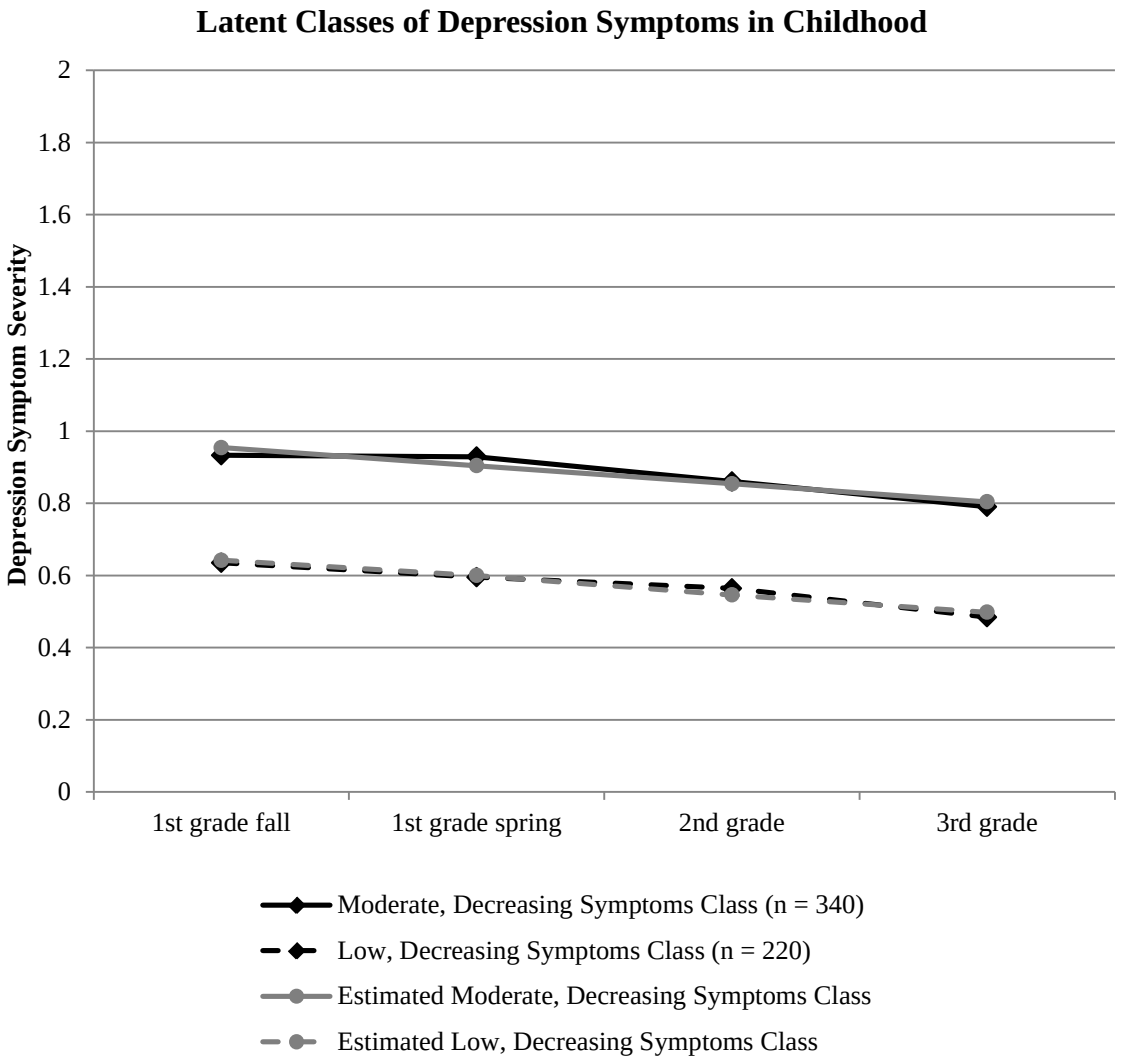


Figure 4.7.

Latent Class Growth Trajectories for Depression Symptoms in Childhood

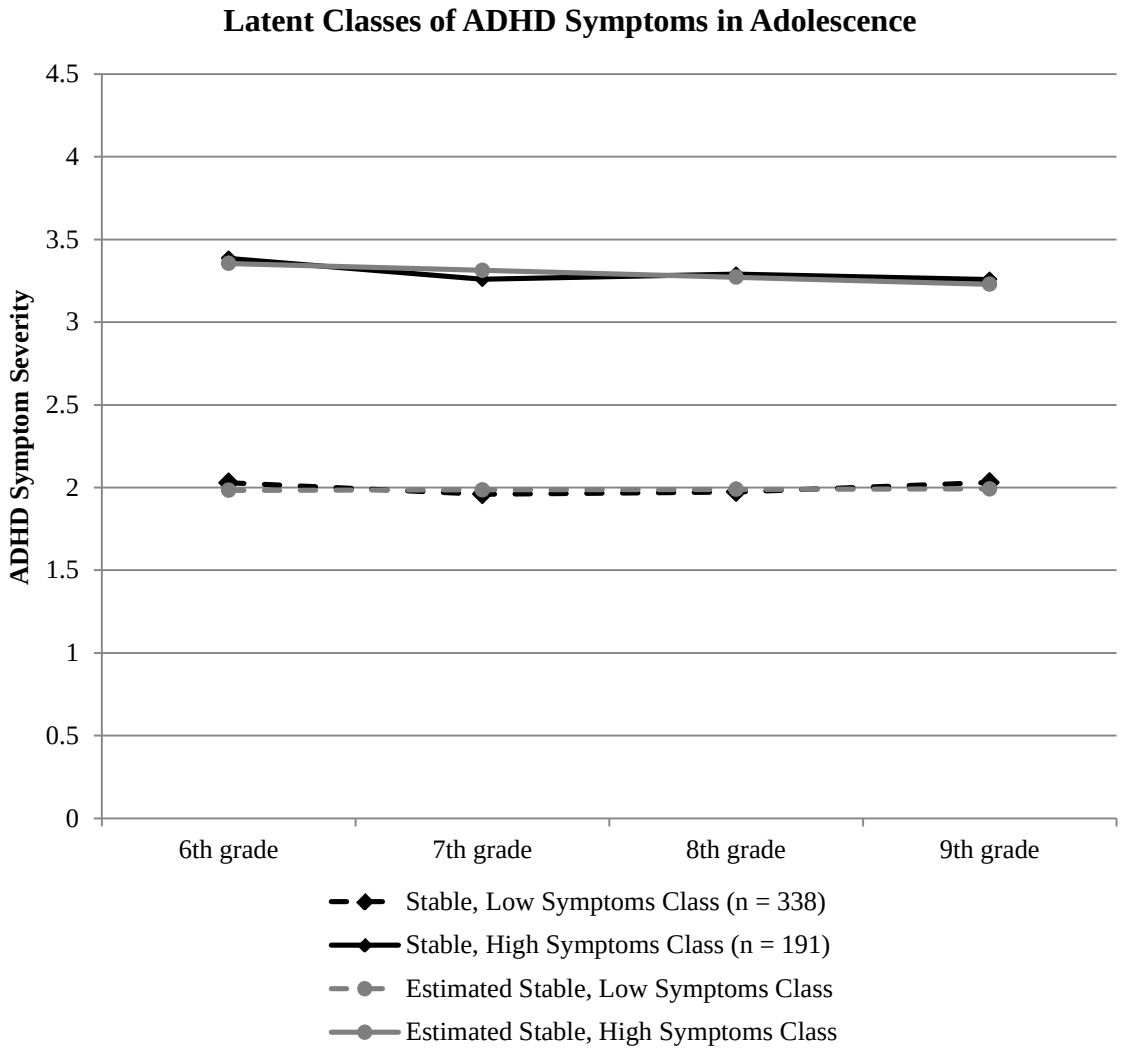


Figure 4.8.

Latent Class Growth Trajectories for Adolescent ADHD Symptoms

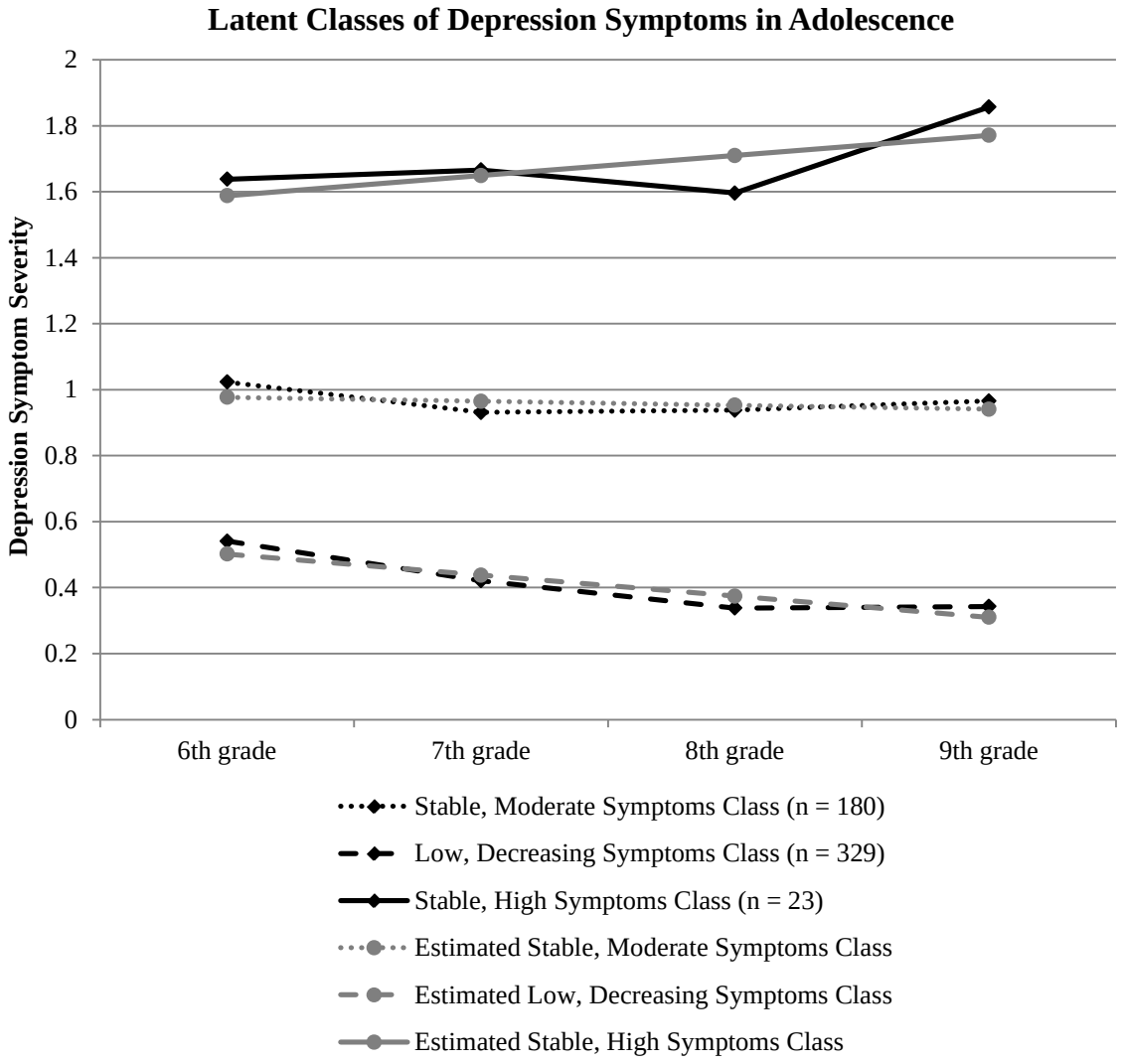


Figure 4.9.

Three Class Solution for the Latent Growth Model of Adolescent Depression Symptoms

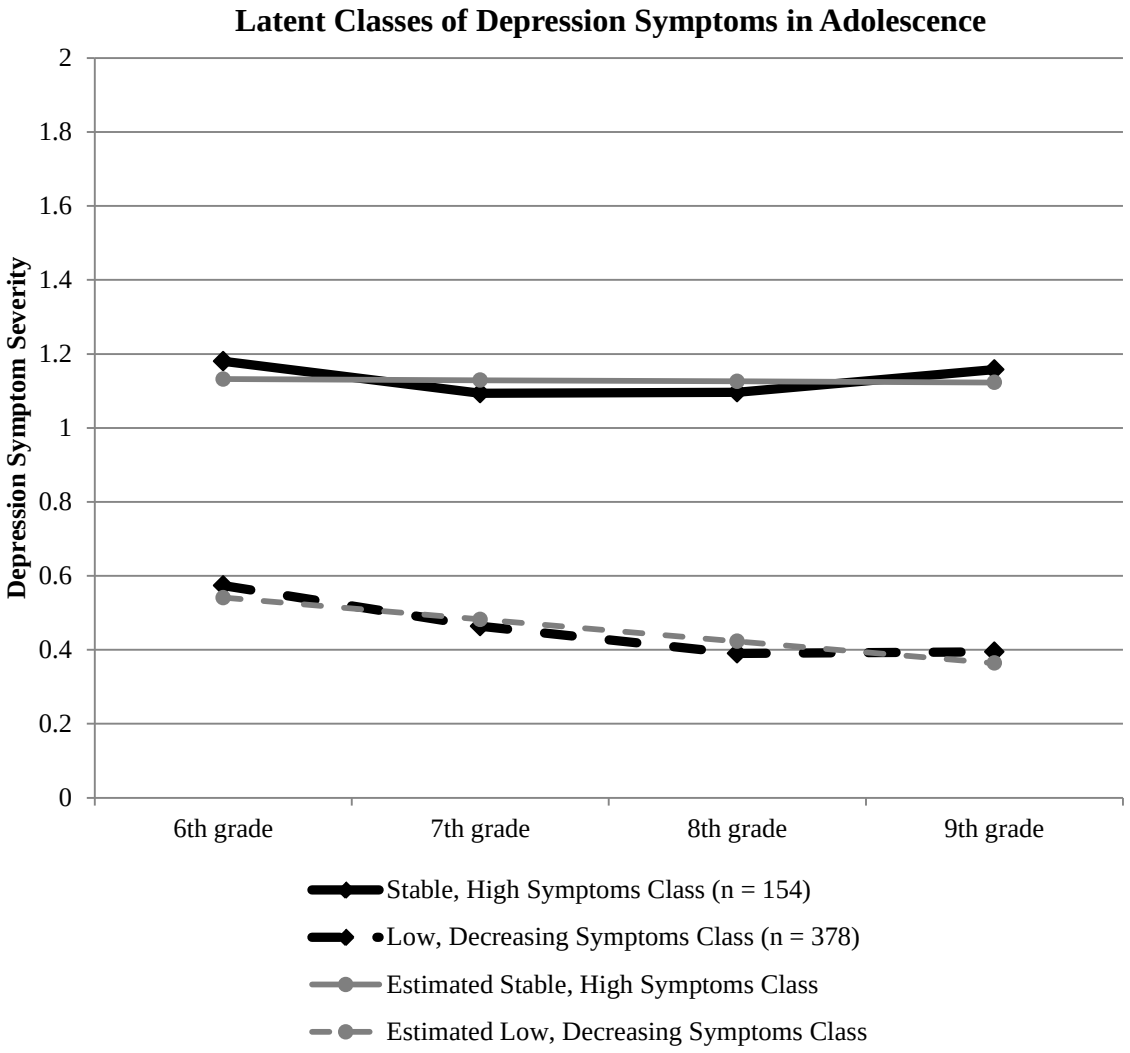


Figure 4.10.

Two Class Solution for the Latent Growth Model of Adolescent Depression Symptoms

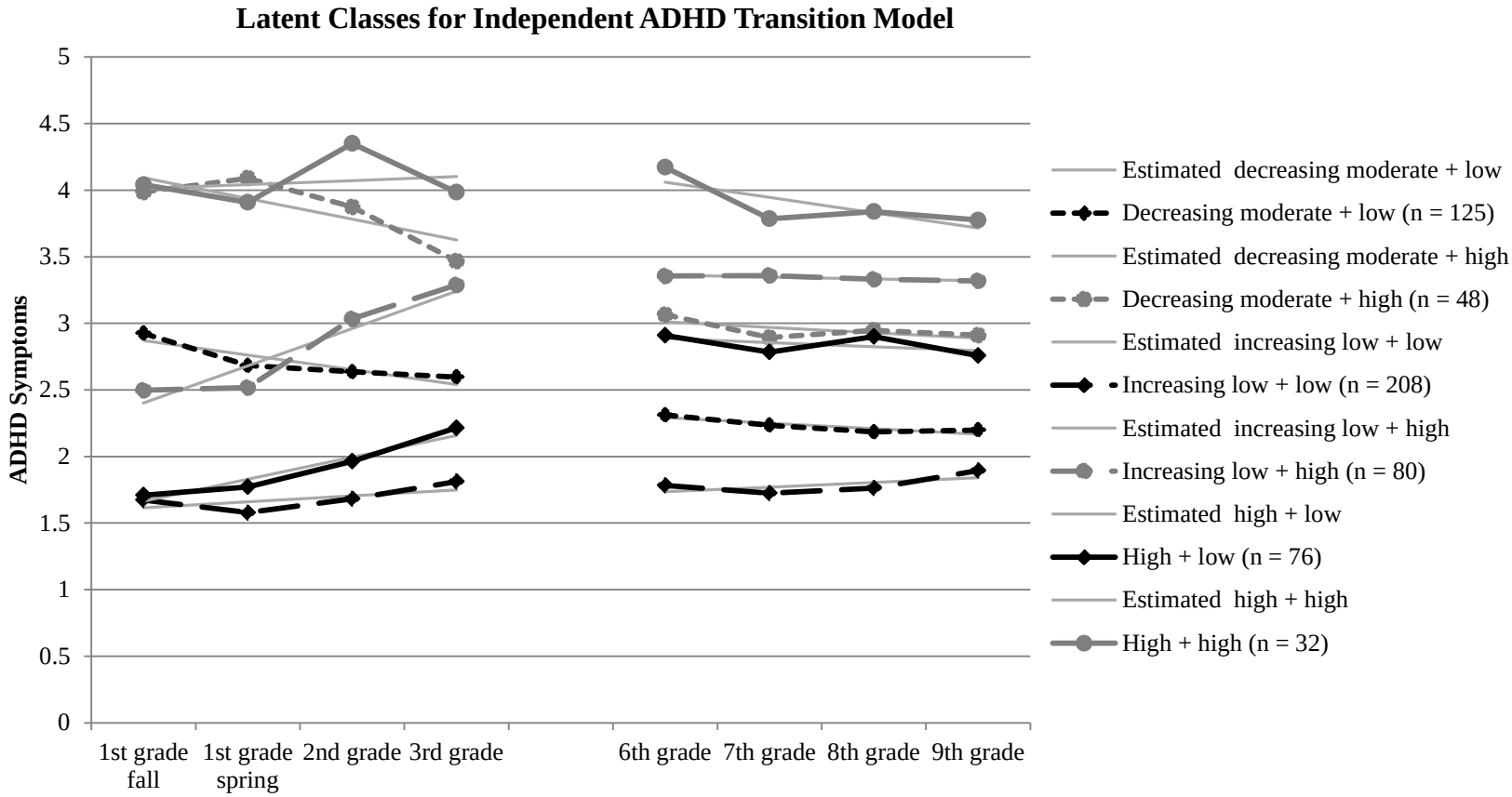


Figure 4.11.

Latent Classes of Childhood and Adolescent ADHD Symptoms in the Independent ADHD Latent Transition Model

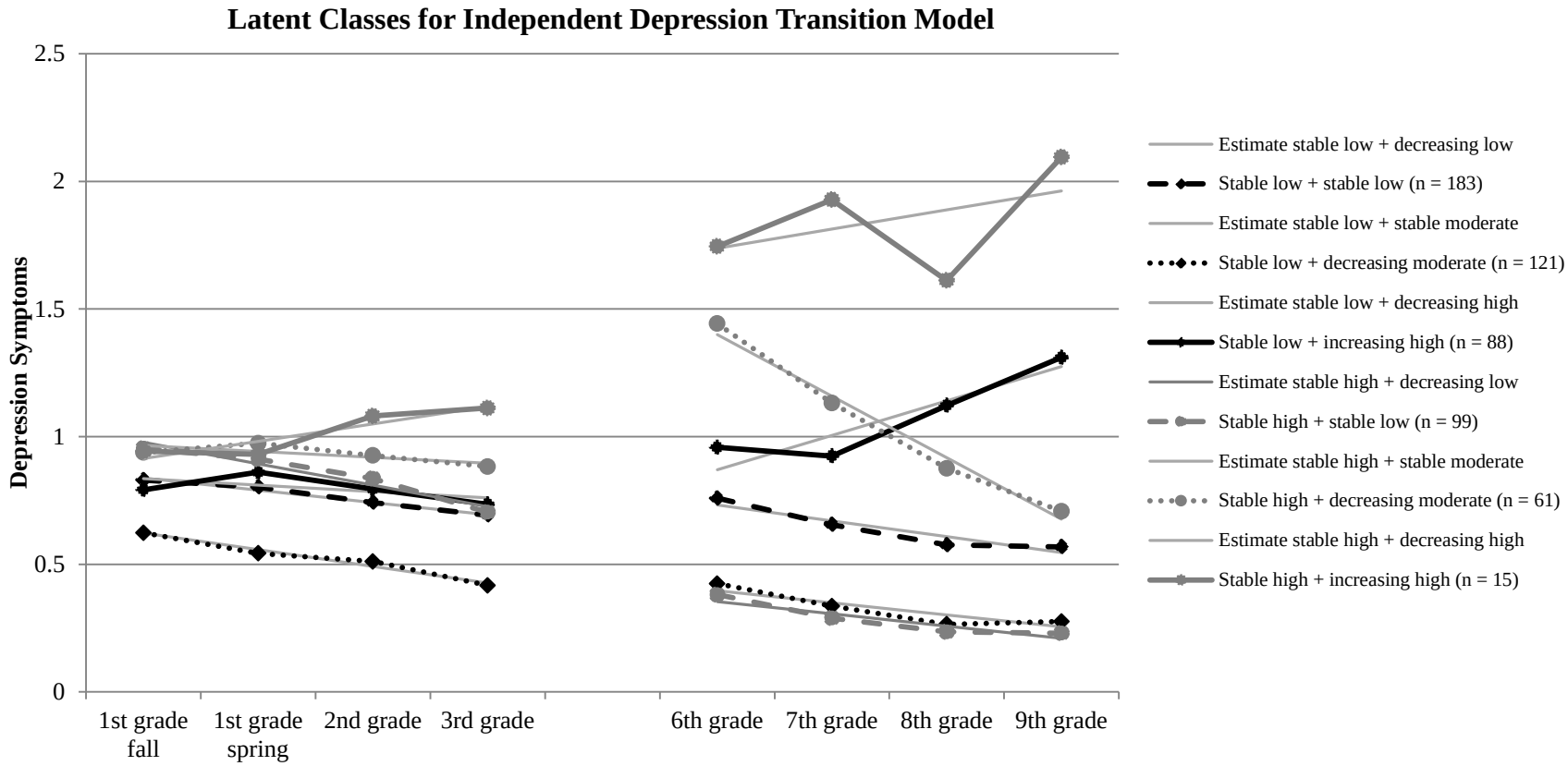


Figure 4.12.

Latent Classes of Childhood and Adolescent Depression Symptoms in the Independent Depression Transition Model

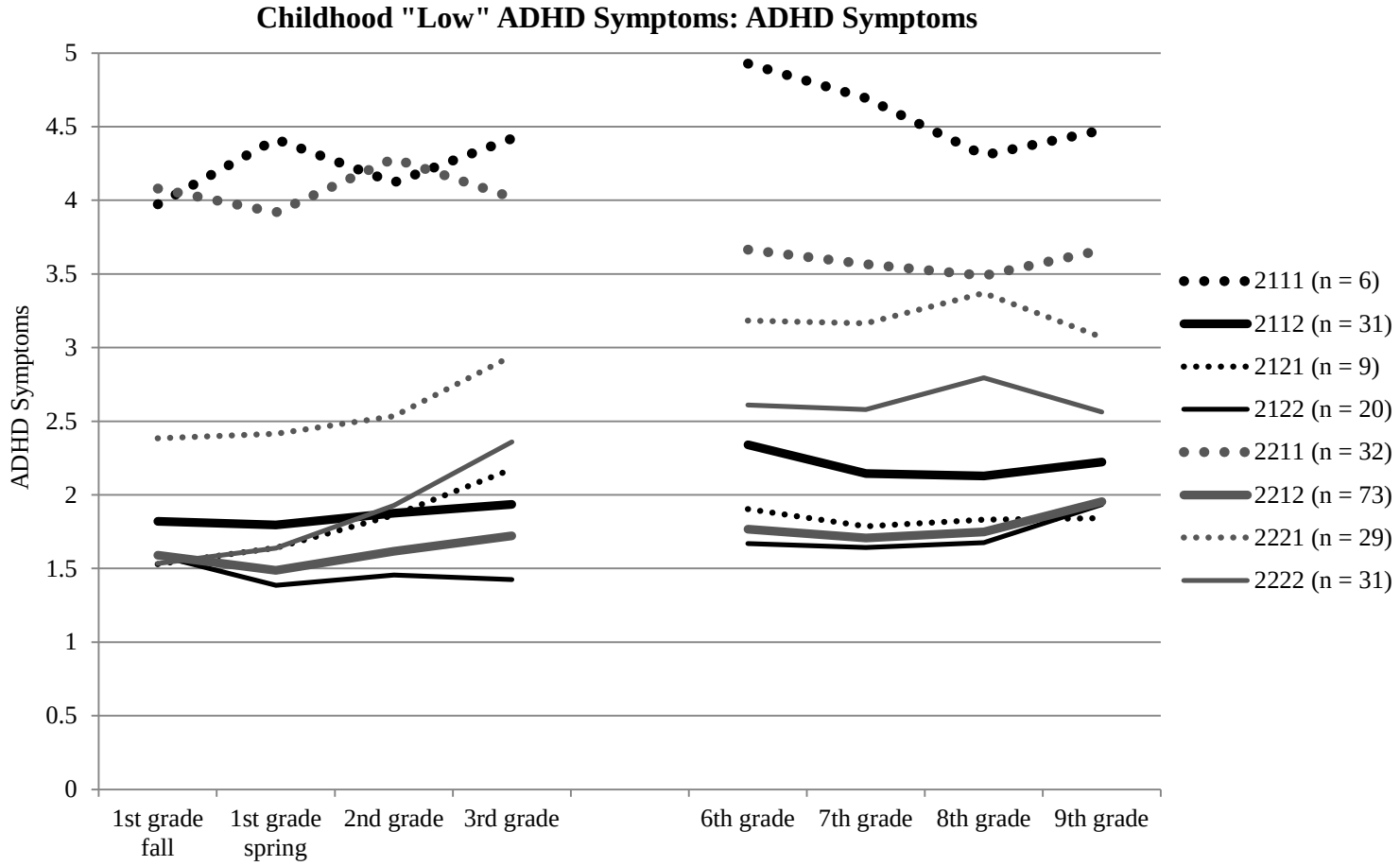


Figure 4.13.

Latent Classes from the Full Transition Model: Childhood "Low" ADHD Symptoms (Class 1) – ADHD Symptoms

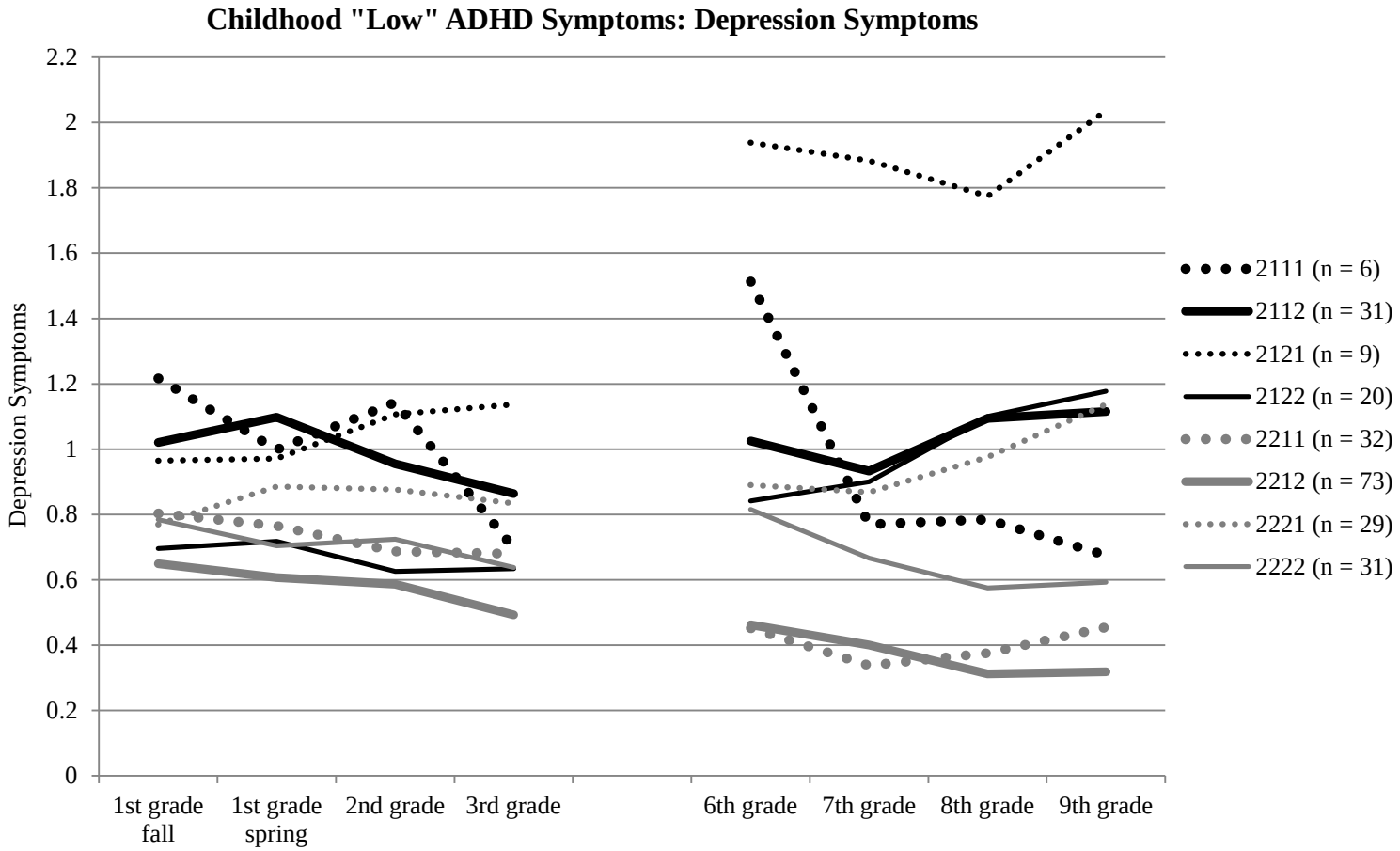


Figure 4.14.

Latent Classes from the Full Transition Model: Childhood "Low" ADHD Symptoms (Class 1) – Depression Symptoms

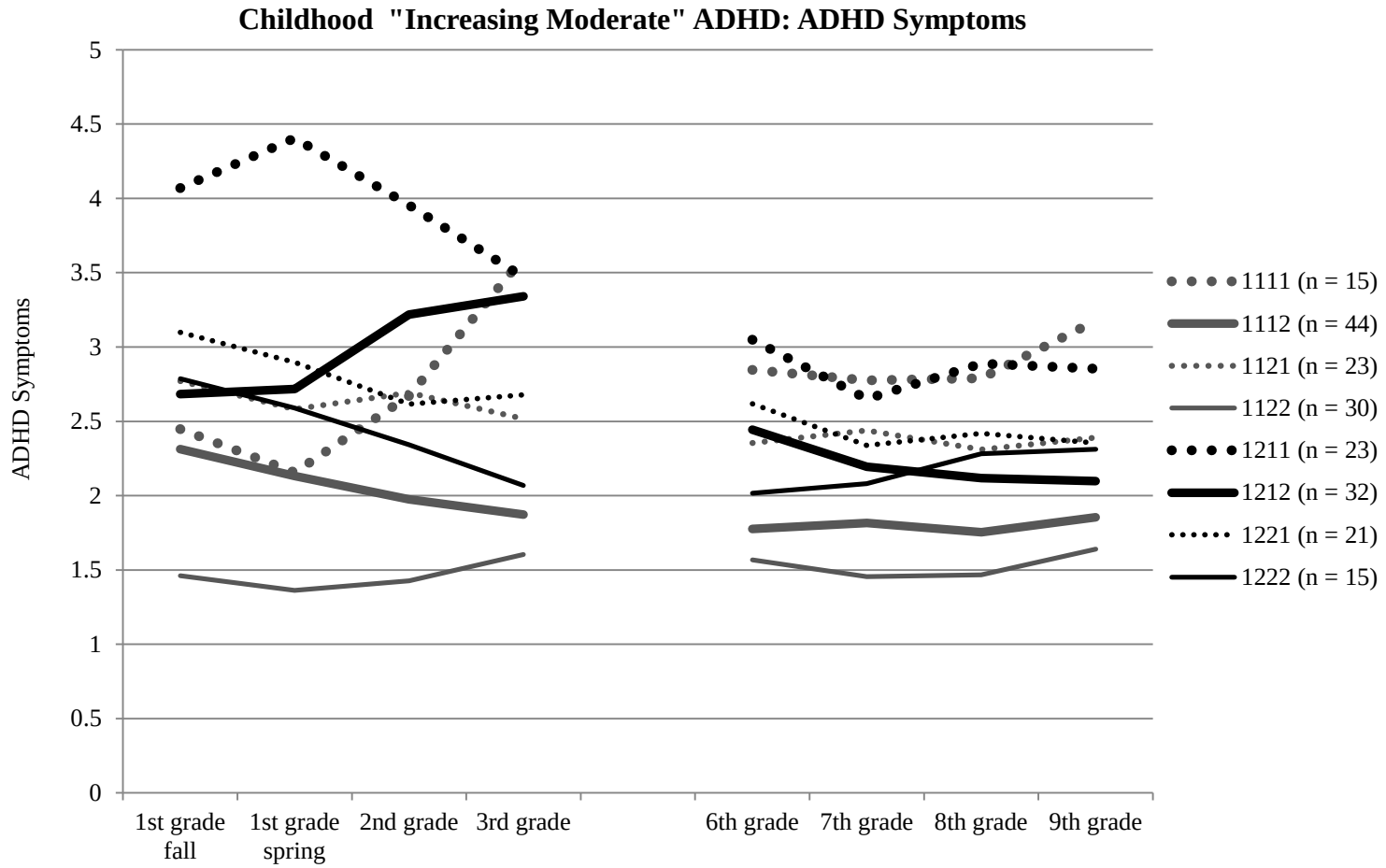


Figure 4.15.

Latent Classes from the Full Transition Model: Childhood "Increasing Moderate" ADHD Symptoms (Class 2) – ADHD Symptoms

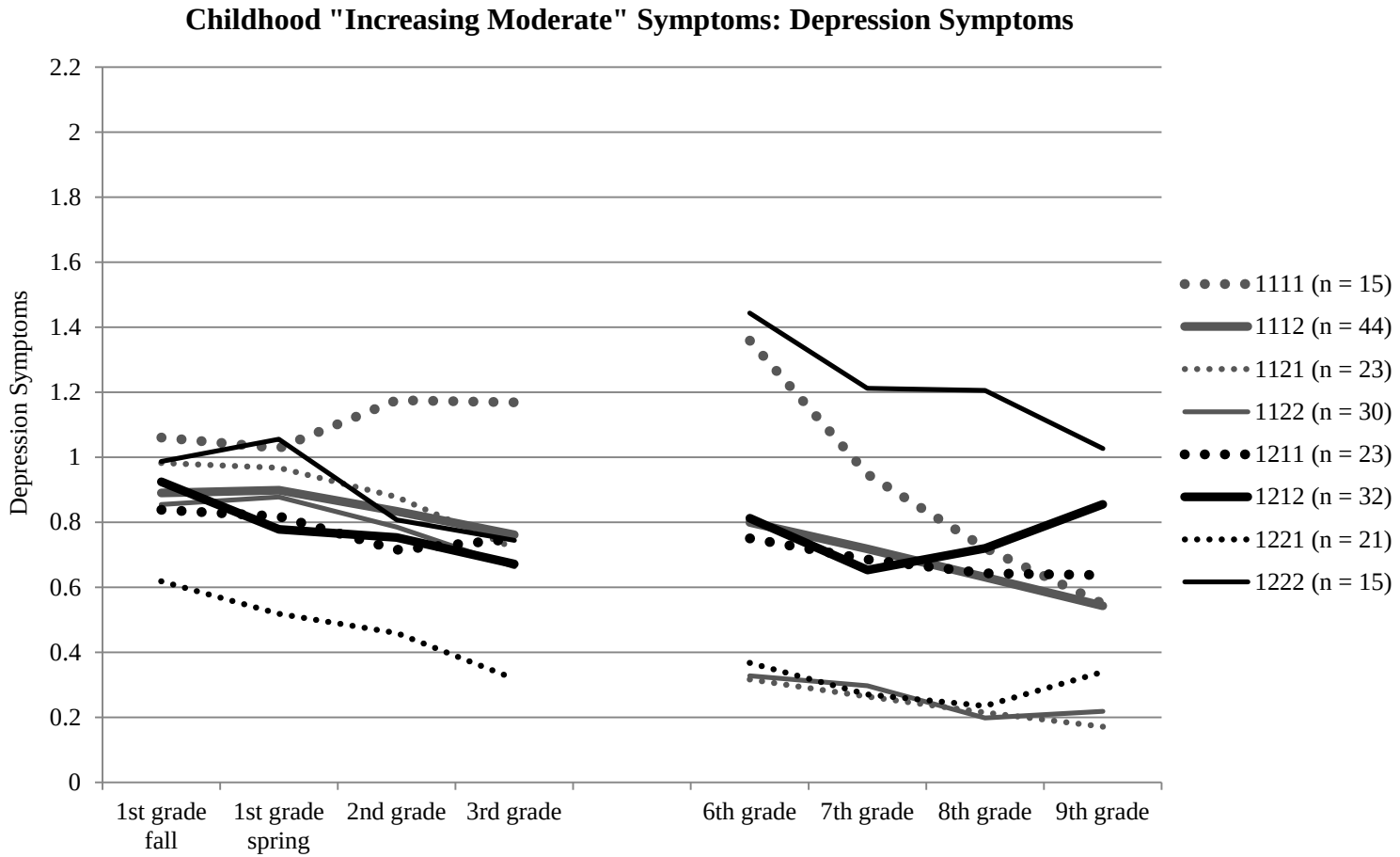


Figure 4.16.

Latent Classes from the Full Transition Model: Childhood "Increasing Moderate" ADHD Symptoms (Class 2) – Depression Symptoms

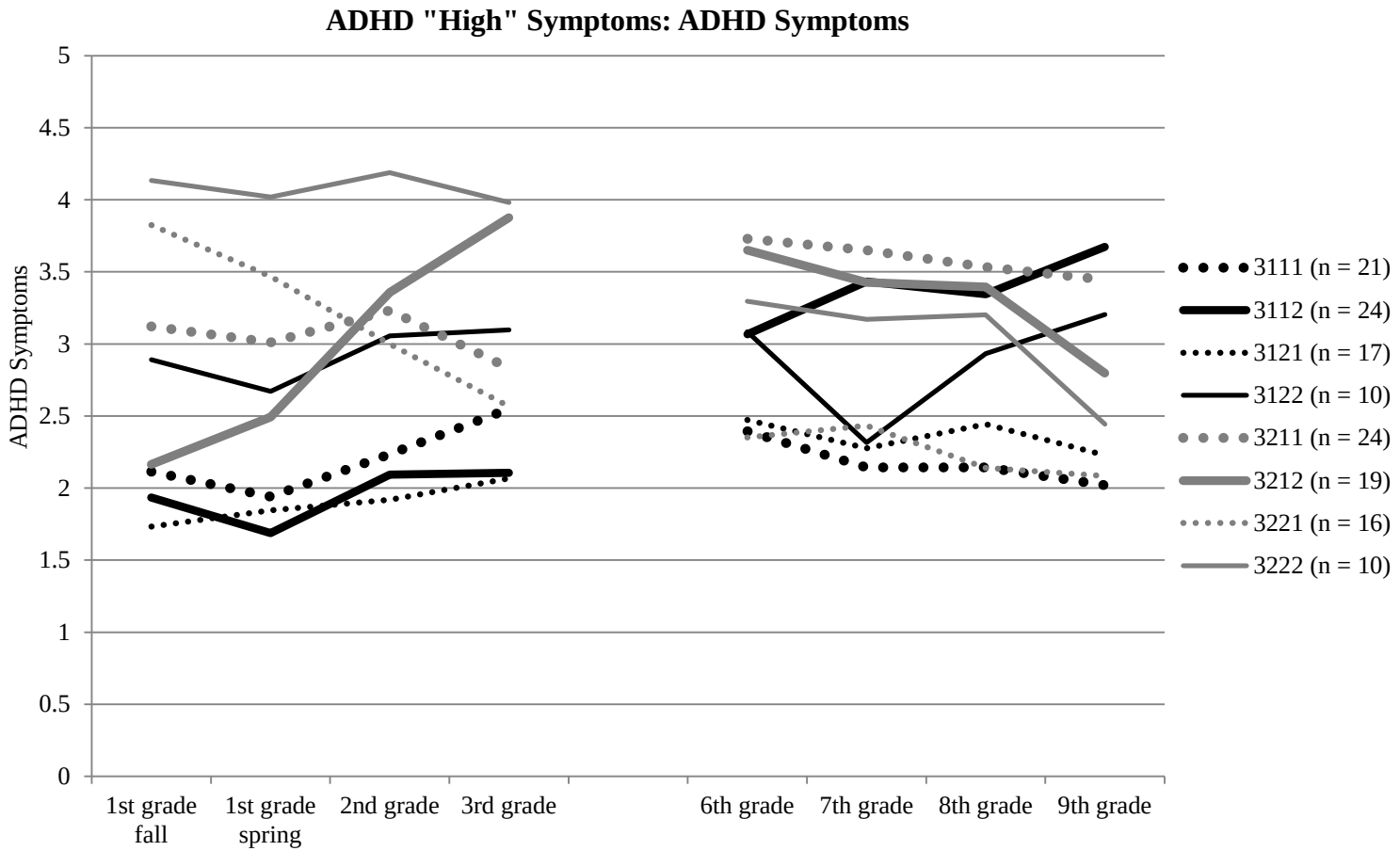


Figure 4.17.

Latent Classes from the Full Transition Model: Childhood "High" ADHD Symptoms (Class 3) – ADHD Symptoms

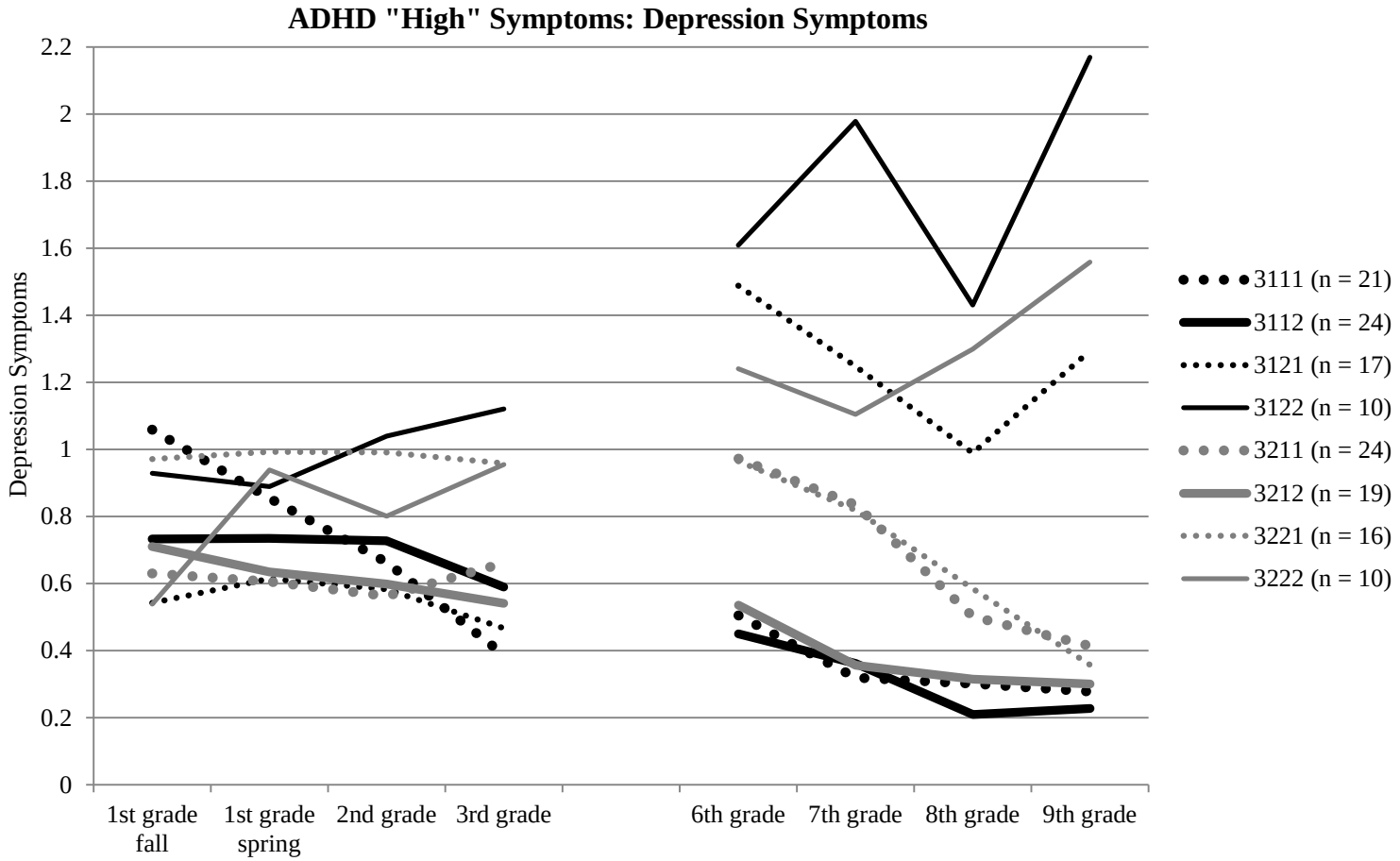
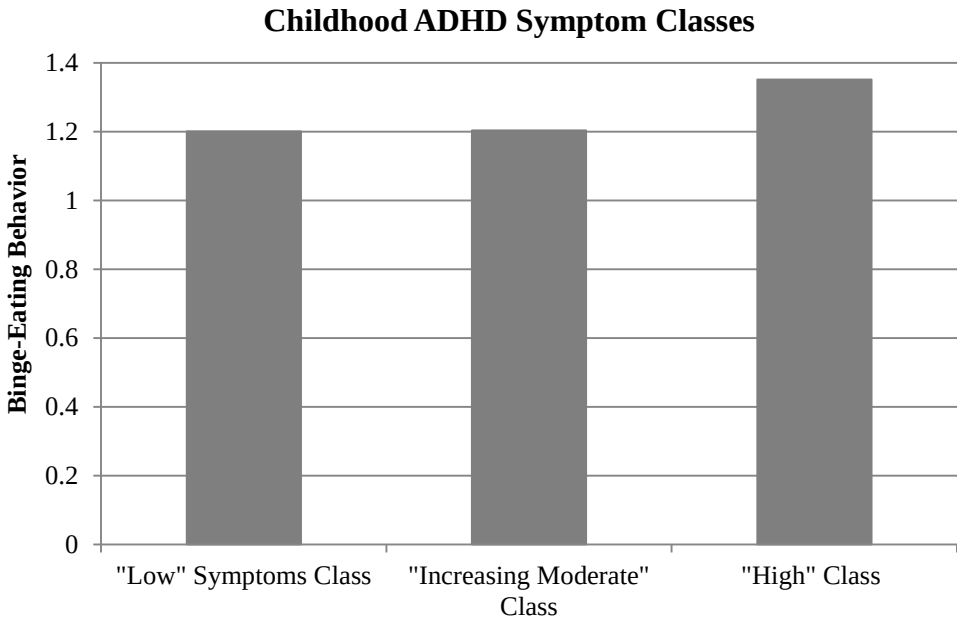


Figure 4.18.

Latent Classes from the Full Transition Model: Childhood "High" ADHD Symptoms (Class 3) – Depression Symptoms



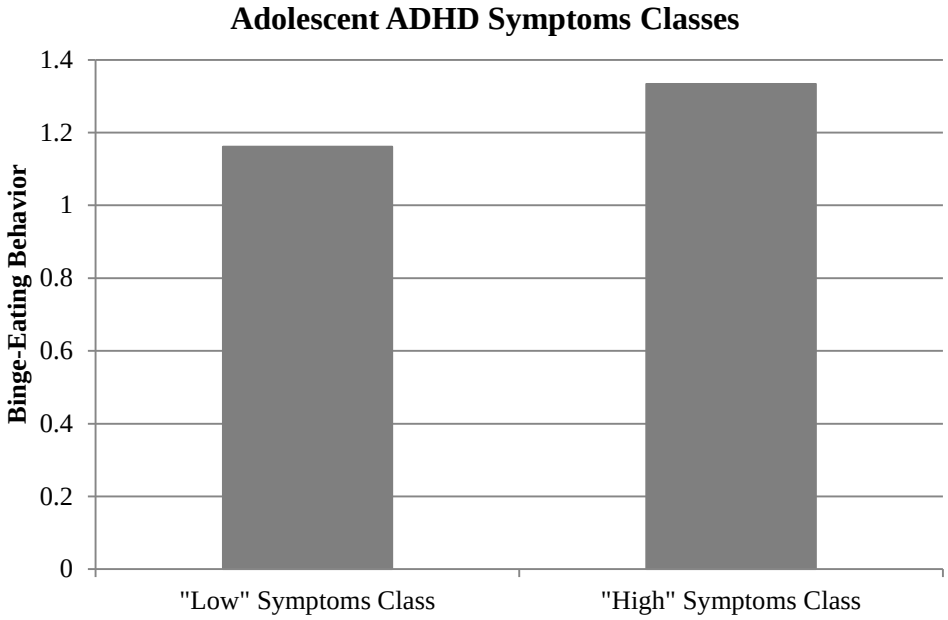


Figure 4.19.

Main Effects of Childhood and Adolescent Symptoms Classes on Binge-Eating Behaviors

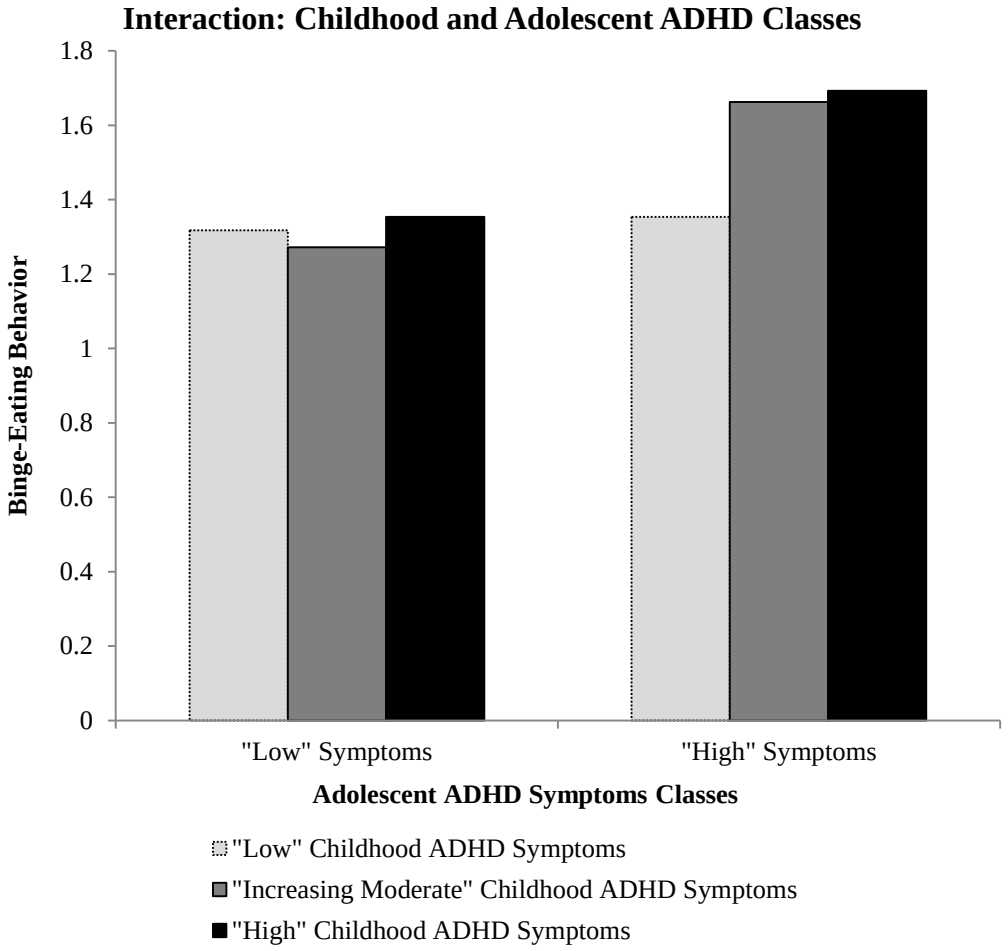


Figure 4.20.

Childhood ADHD Classes Interacting with Adolescent ADHD Classes to Predict Binge-Eating Behaviors

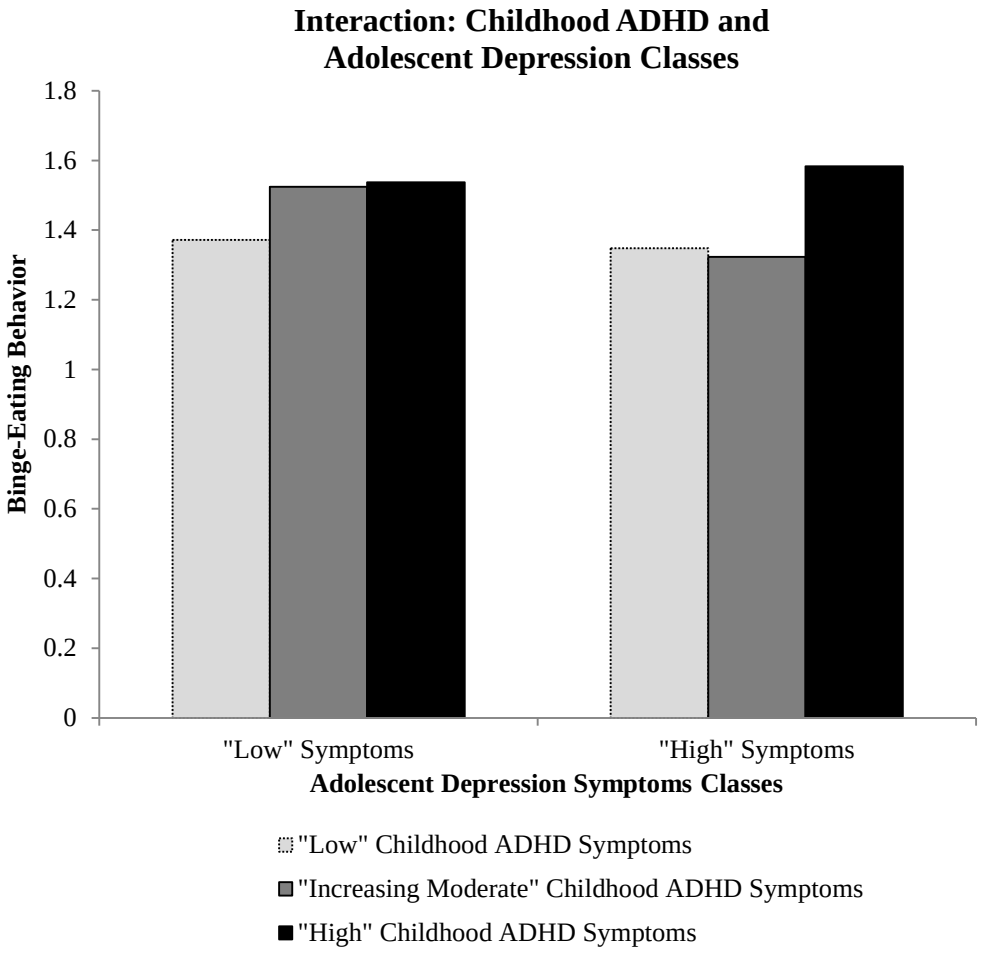


Figure 4.21.

Childhood ADHD Classes Interacting with Adolescent Depression Classes to Predict Binge-Eating Behaviors

Chapter 5: Discussion

In this dissertation, a latent transition model of four latent class growth models representing classes of growth in ADHD symptoms and depression symptoms in childhood and adolescence were evaluated along with their prediction of binge-eating behavior in adolescence. This model was proposed to identify the developmental patterns of both ADHD and depression symptoms in childhood and adolescence and the consistency in symptom severity between developmental periods. This model also allowed for evaluation of the concurrent and longitudinal association between symptom classes of ADHD and depression symptoms to identify the degree to which ADHD and depression symptom severity are related during the same development period and the degree to which childhood ADHD symptoms predict depression symptoms in adolescence. Finally, this study evaluated the degree to which ADHD and depression symptoms independently or jointly predicted binge-eating symptoms and the degree to which ADHD and depression symptoms differentially predicted binge-eating during 10^{th} grade depending on the developmental period.

Several aspects of the latent class transition model required explication prior to the evaluation of the effect of ADHD and depression symptom classes from childhood and adolescence on binge-eating. When considering latent class analyses for each symptom type in each developmental period, it was identified that three classes best represented the individual differences in growth patterns for both ADHD symptoms in childhood and depression symptoms in adolescence. Two classes provided the best fit for

symptom growth in ADHD symptoms during adolescence and depression symptoms in childhood. When these classes were entered into independent transition models for each symptom type, childhood ADHD symptom classes did not display significantly different probabilities of transitioning to adolescent ADHD symptom classes with all classes about 2.6 times more likely to transition to the "low" than "high" adolescent symptoms classes. However, the depression classes did not demonstrate significant difference in transition probabilities between childhood and adolescence. Children in the "stable low" childhood depression symptoms class were less likely than those in the "increasing high" symptoms class to be members of either the "stable high" or "decreasing" moderate" depression symptoms class in adolescence. These results demonstrated strong correspondence between childhood and adolescent symptoms of depression. Also, childhood ADHD symptom classes did not predict membership in depression symptom classes in adolescence.

Finally, results regarding binge-eating suggested that ADHD symptoms classes from both childhood and adolescence predicted binge-eating behavior in adolescence. Further, childhood and adolescent ADHD symptom classes interacted to predict binge-eating where youth with high symptoms in both childhood and adolescence displayed the highest binge-eating behaviors. None of the depression symptoms classes from childhood or adolescence predicted binge-eating behavior. Childhood and adolescent depression symptoms classes did not interact to predict binge-eating behaviors and childhood ADHD symptoms did not interact with adolescent depression symptoms to predict binge-eating behaviors either. Results and their implications are reviewed in greater depth below.

5.1 Primary Research Question

1. **Primary Research Question I: How do latent classes of ADHD and depression symptoms in childhood and adolescence predict engagement in binge-eating behavior during tenth grade?**

Binge-eating is characterized by experiencing a loss of control while eating high quantities of food in a short period of time. A serious public health concern, binge-eating is highly comorbid with other psychological disorders and increases risk for obesity and other health concerns (Hudson et al., 2007; Wilfley et al., 2000), such as metabolic disorder and diabetes (Tanofsky-Kraff et al., 2006; Tanofsky-Kraff et al., 2009; Wonderlich et al., 2009). Little is known about what mental health symptoms contribute to the development of binge-eating for children and adolescents. Research with adults indicates that two strong predictors of binge-eating behavior include impulsivity and depression (Goosens et al,, 2011; Hartmann et al., 2010; Tanofsky-Kraff et al., 2012), and these symptom areas may contribute to BED for youth, as well.

Effect of ADHD Symptoms. Findings of this study indicate that ADHD symptoms, which include impulsivity, predicted binge-eating, whereas depression symptoms did not. Individuals with both a childhood history of ADHD symptoms and current ADHD symptoms in adolescence displayed the highest rates of binge-eating behaviors. These results are consistent with emerging research, demonstrating that impulsivity is a strong risk factor for binge-eating behavior (Dawes & Loxton, 2004; Davis et al., 2006; 2010; de Zwaan et al., 2011; Nasser et al., 2004; Nederkoorn et al., 2006; 2007; Pagoto et al., 2010).

Several theories of binge-eating behaviors incorporate impulsivity as a major component. These theories may explain why ADHD symptoms increase risk in binge-eating behavior. For example, impulsivity associated with ADHD may predispose youth to experience loss of control while eating, a distinguishing characteristic of binging. Sensitivity to reward, specifically preference for immediate rather than delayed rewards, may also contribute to increased risk for binge-eating. Difficulty with delayed gratification is associated with both ADHD (Anokhin et al., 2011; Solanto et al., 2001) and binge-eating (see Dawes & Loxton, 2004; Davis et al., 2006; 2010) and may be a mechanism for engaging in binges for youth with ADHD. Very little research on binge-eating in children let alone binge-eating in youth with ADHD has been conducted. Future research should evaluate which components of impulsivity contribute to binge-eating for youth with ADHD.

Other theories of binge-eating do not explicitly point to ADHD symptoms but ADHD symptoms may exacerbate aspects of the theory that predict engagement in binge-eating. For example, some research suggests that individuals engage in pleasurable activities to regulate negative affect by distracting themselves from the discomfort of distress (Hawkins & Clement, 1984). In some theories, the function of distraction may serve to reduce self-awareness to also avoid awareness of negative affect (Baumeister, 1991). Youth with ADHD may use binge-eating in ways consistent with these theories as a maladaptive coping strategy to avoid distress. The propensity to engage in activities that provide short term coping benefits but negative long-term consequences may be linked to difficulty planning for the future, challenges with delaying gratification, or simply acting rashly, all of which are associated with ADHD. Future research should explore which

theories best apply to binge-eating behavior associated with ADHD. Understanding the mechanisms that account for the link between binge-eating and ADHD will aid in the effective development of prevention and treatment efforts.

Another theory of binge-eating involves dietary restraint, which stipulates that cultural pressure to meet the expectations of a thin ideal lead to dieting and dietary restraint (Stice, 2001; Stice & Bearman, 2001). However, as youth maintain dietary restraint, serotonin levels in the body deplete and reduce, leading to greater levels of negative affect and disinhibition, which set the stage for binge-eating episodes (Stice & Bearman, 2001; Stice et al., 2000). It is unclear the degree to which this theory may apply to youth with ADHD.

Self-perceptions of physical appearance may be a crucial component in understand how dietary restraint may contribute to binge-eating for youth with ADHD symptoms. However, very little is known about the impact of ADHD symptoms on self-perceptions of physical appearance. Self-perceptions in other areas, such as behavior, academic, and social functioning, demonstrate that youth display overestimations of their performance and competence (Hoza, Murray-Close, Arnold, Hinshaw, & MTA Cooperative Group, 2010). This positive bias has been linked to a lack of awareness and insufficient self-monitoring of their behavior (McQuade, Tomb, Hoza, Waschbusch, Hurt, & Vaughn, 2011). Thus, this tendency to underestimate the impact of their negative behaviors and overestimate the success of their positive behavior may extend to perceptions of their physical appearance and eating habits. For example, youth with ADHD may display a similar positive illusory bias regarding their physical appearance, their food intake, or the impact of their diet on their appearance. No research has

explored how ADHD symptoms impact self-perceptions of physical appearance, body image, or food intake behaviors. Thus, future research should consider the degree to which dietary restraint theory displays concordance with perceptions of self and eating behaviors for youth with ADHD.

Effect of Depression Symptoms. Interestingly, most of the theories of binge-eating involve negative affect, yet depression was not a significant predictor of binge-eating and also did not interact with ADHD symptoms to predict binge-eating. Thus, ADHD symptoms appeared to impact binge-eating independent of depression symptoms and potentially negative affect. It is unclear why depression symptoms appeared unrelated to binge-eating behaviors in this study.

Methodological issues associated with depression in analyses may have contributed to the lack of association. For example, initial latent class analyses for childhood depression demonstrated low entropy and limited variability in initial level of symptoms and shape of growth across time, which may have compromised the validity of the childhood depression classes and artificially reduced the association between depression and binge-eating. Further, only two adolescent depression classes were feasible to extract in the full latent transition model, whereas the latent class analyses with adolescent depression and the independent depression transition model demonstrated that three classes provided the best fit for adolescent depression symptoms. The lack of the third adolescent depression class may have also led to a lack of association between depression classes and binge-eating. Further, the independent depression symptoms transition model also had low statistical power to support results. These limitations may have obscured the link between depression and binge-eating.

Another explanation may be that binge-eaters experience elevated depression symptoms but are less aware of their symptoms than others with depression symptoms. This interpretation of results is consistent with theories that posit binge-eating functions as an avoidance strategy for negative affect (Baumeister, 1991; Hawkins & Clement, 1984). The link between negative affect and binge-eating is hypothesized to become conditioned over many repetitions (Haedt-Matt & Keel, 2011). Over time, youth who engage in binge-eating may have low awareness of their negative affect and engage in a cycle of negative affect and binge-eating that is so engrained that they may be less aware of their mood concerns than others. This interpretation of results would leave the link between depression and binge-eating undetectable with the current measurement approach in this study.

However, it is also possible that depression symptoms manifest differently among African-American youth compared to European-American youth who predominantly comprised the samples with which symptoms and diagnostic criteria for depression among children and adolescents were developed and refined (Kessler et al, 2008; Merikangas, Avenevoli, Costello, Koretz, & Kessler, 2009; Merikangas et al., 2010; Moffitt et al., 2007). Some research demonstrates that African-Americans are more likely that European-Americans to express depression through irritability, anger, somatic symptoms, and physical expressions of symptoms (Myers et al., 2002; Pickering, 2000) rather feelings of sadness, tearfulness, feeling overwhelmed, or reporting a sense of helplessness. Although the depression items included a broad range of symptom presentation, the culturally focused presentation of symptoms that may have been present in these data may have limited the variability of depression as a construct and reduced its

association with binge-eating. Future research should explore how the association between binge-eating and depression changes depending on the presentation of depression symptoms generally and for African-American youth specifically.

On the other hand, the aspects of depression symptoms that predict binge-eating may also be characteristic of ADHD symptoms. Because regression approaches evaluate the independent contribution of a predictor to an outcome, this shared variance between ADHD and depression would have been eliminated, reducing the capacity for depression symptoms to account for variability in binge-eating above and beyond the influence of ADHD symptoms. For example, low tolerance for negative affect, avoidance of distress, and low cognitive control are critical symptoms of depression and major components of binge-eating theories, but are also symptoms that are characteristic of ADHD.

Finally, the causal order between depression and binge-eating may not be that depression symptoms cause binge-eating, but that binge-eating behaviors cause depression. Factors other than depression symptoms may account for the initial engagement in the behavior, which then contribute to the development of depression. Following a binge-eating episode, many individuals experience shame, guilt, and a lack of control over their behavior. After many binges repeated over time, these feelings may contribute to a sense of hopelessness and helplessness about one's capacity to display behavioral control. Over time, depression and binge-eating behaviors may become conjoined in a cyclical behavioral pattern where each reinforces the other. Although this behavioral pattern has not been research with regard to binge-eating, evidence exists to suggest that depression and bulimia symptoms display reciprocal causal associations

(Stice, 2001; Stice & Bearman, 2001). Future research should evaluate the extent to which these findings apply to link between depression and binge-eating as well.

5.2 Effect of Contextual Predictors on Binge-Eating Outcome

Of the contextual predictors, only gender had a significant effect on binge-eating behaviors. No significant differences in binge-eating behaviors among treatment status, race, or lunch status emerged. The gender effect indicated that girls reported engaging in more binge-eating behaviors than boys did. Neither symptom type from either developmental period interacted with gender to predict binge-eating. Thus, girls in this sample engaged in binge-eating behaviors more than boys did independent of having higher depression or ADHD symptoms. These results are consistent with previous research indicating that two boys for every three girls engage in binge-eating (Johnson et al., 2002; Ricciardelli, Williams, & Kiernan, 1999).

Results indicated that ADHD symptoms confer risk for binge-eating equivalently for boys and girls. However, binge-eating may serve a different function or result from different mechanisms for boys and girls. Boys may engage in disinhibited eating that meets the criteria of binge-eating but may be developmentally appropriate, given a typically increased growth rate during adolescence for boys. Binge-eating in this situation may be driven primarily by the need to increase dense caloric intake to achieve growth demands and expectations of daily activities (Tanofsky-Kraff et al., 2008). Disinhibited eating for boys that follows this pattern may not function as a maladaptive coping strategy for boys, whereas binge-eating for girls may be more likely to be linked to dietary restraint. Girls may experience greater susceptibility for binge-eating behaviors as they may be more likely to engage in dieting and dietary restraint that disregulate both

mood and inhibitory control related to food intake (Stice, 2001; Stice & Bearman, 2001). Cultural norms and expectations related to maintaining a thin physique apply primarily to girls rather than boys and may contribute to different outcomes associated with binge-eating. Girls may experience distress after a binge-eating episode related to concerns about their behavior working against their ideal body image, whereas boys may not experience the same distress. Future research should evaluate further how gender impacts binge-eating behaviors as these gender differences may hold important implications for differences in intervention needs for binge-eating between boys and girls.

5.3 Foundational Research Questions

The foundational research questions existed to develop the model in which ADHD and depression classes were used to predict binge-eating behaviors. However, the research questions contain interesting results regarding the developmental course of ADHD and depression, transitions in symptoms between childhood and adolescence, and the degree of correspondence between ADHD and depression during and across developmental periods.

5.4 Foundation Research Question IA

1. **Foundational Research Question I:**
    a. **What are developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

Conditional Growth Models: ADHD Symptoms. Results of the conditional growth models indicated that childhood ADHD symptoms displayed a trajectory with a slight

increase from 1st through 3rd grade. Research findings display little consistency across studies regarding the typical developmental trajectory of inattention, impulsivity, and hyperactivity symptoms. Whereas these results are at odds with some research suggesting that children decrease these behaviors during childhood (Biederman et al., 2000; Côté et al., 2002), other research shows either slight increases or stable rates of these behaviors (Jester et al., 2005; Pingault et al., 2011). One factor contributing to the maintenance and increase in ADHD symptoms may be the adversity exposure characteristics of youth in this study. This study produced results that appear remarkably consistent with research on developmental trends in disruptive behavior among youth living with adversity or ongoing stressors within their home or community (Jester et al., 2005). Youth in this study experienced community violence exposure, domestic violence, un-enriched environments at home and at school, and parental substance use, all of which predict the maintenance of impulsivity, inattention, and low executive functioning skills (Halperin & Healey, 2011; Jester et al., 2005; 2008; Nikolas, Friderici, Waldman, Jernigan, & Nigg, 2010), and may account for the increase in ADHD symptoms across childhood.

ADHD symptoms from 6th through 9th grade demonstrated a flat trajectory of symptom growth, which demonstrated no change in symptoms across middle school. These results are consistent with past research demonstrating that ADHD symptoms in adolescence remain relatively equivalent across time (Biederman et al., 2000 Côté et al., 2000; Jester et al., 2005; Pingault et al., 2011).

Conditional Growth Models: Depression Symptoms. On average across participants, depression symptoms in childhood demonstrated a slight decrease into 3rd grade. These findings are consistent with one previous study demonstrating that

depression symptoms generally decrease for children in middle childhood (Dekker et al., 2007), but it conflicts with other research suggesting that depression symptoms increase across middle childhood (Mazza et al., 2010). Additional research is necessary to identify the typical developmental trajectory for depression symptoms in childhood.

The typical trajectory for depression symptoms during early adolescence is also not clear. During adolescence, depression symptoms on average continued to slightly decrease. These findings are consistent with some research indicating that depression symptoms on average decrease (Burstein et al., 2010; Dekker et al., 2010). However, other studies demonstrate moderate to quite dramatic increases in depression during this age (Brendgen et al., 2005; Cole et al., 2002; Ge et al., 1994). This lack of clarity may be attributable to the wide discrepancy in depression symptom severity across adolescents. Early adolescence is when depression symptoms begin to emerge and escalate for some youth, whereas other youth do not experience significant risk for depression symptoms. Thus, considering the latent classes of depression symptoms may serve to clarify typical subgroups regarding growth in depression symptoms during adolescence.

5.5 Foundational Research Questions IB

1. **Foundational Research Question I:**
    b. **What are the typical subgroups of the developmental trajectories in childhood (i.e., first through third grades) and adolescence (i.e., sixth through ninth grades) of teacher-reported ADHD symptoms and child self-reported depression symptoms?**

Latent Class Models: ADHD Symptoms. The latent class analyses for each symptoms type during each developmental period produced interesting findings

regarding the quantity and quality of subgroups present. For the childhood ADHD symptoms model, three classes resulted that corresponded to “increasing low” symptoms, “stable moderate” symptoms, and “stable high” symptoms, although all classes had very minimal change over time. Several studies evaluating latent classes of ADHD symptoms have results with similar aspects to these findings. In general, most other studies demonstrated more change in symptoms across time periods than was displayed in this study. However, all studies identified a class with very low symptoms that typically decrease in symptom severity across the measurement periods (Côté et al., 2002; Jester et al., 2005; Nagin & Tremblay, 1999; Robbers et al., 2011). Also, several studies included a class with moderate symptom severity, although this class is either increasing or decreasing, but not stable across time (Nagin & Tremblay, 1999; Côté et al., 2002). Finally, the high symptoms class appears consistent with other studies as well (Côté et al., 2002; Jester et al., 2005; Nagin & Tremblay, 1999; Robbers et al., 2011).

Results from the latent class growth model for adolescent ADHD symptoms indicated that two classes provided the best fit for the data - “stable low” symptoms class and “stable high” symptoms class. These results replicated findings from several other studies. Both Jester and colleagues (2005) and Larson and colleagues (2011) identified two classes of symptoms that extended across the same developmental period. Both studies identified high and low classes of ADHD symptoms that demonstrated stable levels of symptom severity across time. Although Nagin and Tremblay (1999) identified four classes, the two classes identified in this study corresponded with two of the classes among the four found in the Nagin and Tremblay (1999) study.

Latent Class Models: Depression Symptoms. Childhood depression symptoms resulted in two classes that corresponded to a "decreasing low" symptoms class and a "decreasing high" symptoms class. Previous research on childhood depression symptoms using latent class research has rarely included children as young as first through third grade. As a result, few studies are available for comparison. Dekker and colleagues (2010) identified six classes of depression symptoms from symptoms assessed annually from 4 to 18 years of age. These classes primarily fell into two categories for low stable symptoms and high decreasing symptoms, which show similarities to the symptoms classes identified in this study. Mazza and colleagues (2010) found five classes of depression symptoms when symptoms were assessed yearly from 2nd to 8th grade. Between the 2nd and 3rd grade assessment, these classes demonstrated a variety of shapes and initial levels that were quite different from the classes in this study.

Adolescent depression symptoms were best represented with three classes, which corresponded to a "decreasing low" symptoms class, a "stable moderate" symptoms class, and a "stable high" symptoms class. These three classes demonstrated some similarities with previous research. Although Brendgen and colleagues (2005) identified four rather than three classes, the three classes identified in this study replicated these four classes with the exception of the "increasing moderate" class. Similarly, Dekker and colleagues (2007) identified six classes, which shared many similarities with the three classes identified in this study, although a class with increasing depression was missing from the current study. Mazza and colleagues (2010) identified five classes, none of which demonstrated symptoms that significantly increasing slope. It is not clear why a class of increasing depression symptoms did not emerge at this level of analysis.

The variability in the number of classes, shape of growth, and initial level of symptoms across studies may have resulted from several methodological characteristics, such as the number of measurement points across time, the sample size, and reporter of symptoms. Increasing time points and sample size increases variability, which can sustain a greater number of classes and facilitate a wider range of slopes and intercept levels represented among the classes.

The individual reporting the symptoms may also influence the number of classes as well as their shape and initial level. For example, children may lack full awareness or insight regarding their cognitions and internal, emotional states, limiting their ability to represent the full range of their depression symptoms. Thus, the use of self-report for depression symptoms may have contributed to low variability and the emergence of fewer classes than previous research for the childhood depression symptoms model.

On the other hand, teachers served as reporters of ADHD symptoms in childhood and adolescence, which may have also been a limitation. Although teachers may be excellent reporters of ADHD symptoms in childhood, during adolescence, their report of student ADHD symptoms may be limited. The shift from displaying hyperactivity through physical business (e.g., running, climbing on things, frequently leaving one's seat) to restlessness and fidgeting may make ADHD symptoms less obvious and more difficult to detect for teachers of adolescents. Further, middle and high school teachers see students for one class period rather than the whole day. With less access to students, accurately identifying ADHD symptoms may be challenging. Thus, use of teacher report of adolescent ADHD symptoms may have contributed to identification of fewer symptom classes than other studies.

5.6 Foundation Research Question II

2. **Foundational Research Question II: Do children remain in the same symptom severity class of ADHD and/or depression symptoms or change to classes with higher or lower ADHD and/or depression symptom severity?**

ADHD Symptoms in Transition Model. In the full transition model, ADHD symptoms classes demonstrated significant correspondence from childhood to adolescence. The "high" childhood ADHD symptoms class was significantly more likely than the "low" class to transition to the "high" adolescent ADHD symptoms class. These results are consistent with previous research on the course of ADHD symptoms. ADHD is largely a chronic disorder where individuals continue to display symptoms throughout the lifespan. Research suggests that 65% - 85% of children with ADHD display diagnostic levels of the disorder in adolescence (Barkley et al., 1990) and nearly 75% of children with ADHD continue to display symptoms that impair functioning in early adulthood (Biederman et al., 2011).

However, the "high" childhood class was not significantly more likely than the "increasing moderate" class to transition to the "high" adolescent ADHD symptoms class. These results demonstrated that the "increasing moderate" class demonstrated a relatively similar developmental outcome with regard to symptom severity and maintenance as the "high" childhood symptoms class. It is possible that over time, teachers may perceive youth with even a few ADHD symptoms as being similar to classmates with far more severe ADHD symptoms. Teachers may draw this conclusion due to academic and behavioral impairment associated with subthreshold ADHD symptoms. For example, research demonstrates that even three prevalent ADHD

symptoms can lead to academic and social impairment for youth (Scahill et al., 1999). Likewise, other studies show that youth with subthreshold ADHD symptoms display levels of impairment and severity of comorbidities equivalent with youth who have quite severe levels of ADHD symptom (Hong et al., in press).

The correspondence in symptoms differed from the independent transition model which included only ADHD symptoms rather than both ADHD and depression symptoms. In the independent transition model, ADHD symptoms did not display correspondence between childhood and adolescent developmental periods. Instead, all childhood classes demonstrated about a 2.6 times greater likelihood of belonging to the "low" ADHD symptoms class in adolescence. These differences in transition probabilities between the full and independent symptoms transition models suggest the effect that the development of depression symptoms may have on the development of ADHD symptoms in childhood and adolescence. For example, the presence of depression symptoms during childhood may increase the likelihood that youth with ADHD symptoms maintain significant ADHD symptoms in adolescence.

Depression Symptoms in Transition Model. In the full transition model, the "high" depression symptoms class in childhood was much more likely than the "decreasing low" symptoms class to be members of the "high" adolescent depression symptoms class. These results were consistent across the independent depression symptoms transition model and the full transition model. In the independent model, children in the "decreasing high" symptoms class were more likely than the "stable low" symptoms class to belong to both the "stable high" and the "decreasing moderate" symptoms classes in adolescence. Similarly, children in the "decreasing high" symptoms class were more

likely than the “stable low” symptoms class to transition to the “stable high” class in adolescence. These results indicated that depression symptoms demonstrated relatively strong correspondence in symptom severity between childhood and adolescence.

Although the transition probabilities suggest that depression symptoms in childhood provided limited prediction of adolescent depression symptoms, consideration of the shape and initial level of classes in childhood and adolescence indicated interesting and potentially meaningful growth in depression symptoms. Depression symptoms displayed limited variability in childhood, whereas adolescent symptoms displayed greater variance in slope and initial level. Although the mean level of depression symptoms was quite low and variability appeared small in childhood, childhood symptoms strongly distinguished high from low adolescent depression classes. Thus, even small elevations in depression in childhood appeared to contribute to increased depression symptoms during the transition to adolescence.

These results are important as they indicate the impact of subthreshold depression symptoms on the burgeoning expression of depression during adolescence. Subthreshold depression symptoms may engender vulnerability for the development of depression through a variety of mechanisms. Early signs of depression may correspond to a propensity to develop a negative inferential style towards one’s self and environment. Hopelessness theory of depression indicates that responding to adversity by attributing events to stable causes, catastrophizing, and deducing negative meaning about one’s self from negative events increases depression symptoms (Abela & Hankin, 2008; Abramson, Metalsky, & Hankin, 1989). As self-awareness and ability to have insight about internal

experience develops, this negative inferential style may grow into beliefs that become engrained perceptions of one's world view, particularly as youth face negative life events.

On the other hand, subthreshold depression symptoms in childhood may correspond to characteristics of temperament that predispose one to the development of depression. Childhood depression symptoms in this study may correspond to negative affect or negative emotionality. Children who experience greater levels of negative emotionality also experience more stress and expend more attentional control focusing on negative events than other children, which may over time predispose them to depression symptoms (Compas, Connor-Smith, & Jaser, 2004; Wetter & Hankin, 2009).

As youth transition into adolescence, exposure to negative life events significantly increases (Ge, Lorenz, Conger, Elder, & Simons, 1994). Academic expectations increase; peer dynamics vacillate more in adolescence than any other time across the life span; bullying and relational aggression reach their zenith during adolescence (Pelligrini & Long, 2002); and youth experience dramatic physical changes due to puberty that impact peer dynamics and an already shifting sense of identify (Steinberg & Morris, 2001). Subthreshold childhood depression symptoms may suggest maladaptive coping skills for negative events that lead to the emergence of high levels of depression symptoms in adolescence when youth are faced with the totality of these stressors typical during this time period.

These results, then, highlight the crucial need for prevention services for youth during childhood that bolster coping skills to replace negative cognitive styles. Targeting coping skills development during childhood may be an important strategy for preventing the development of depression symptoms during adolescence. Importantly, although the

"high" childhood depression symptoms class was more likely than the "low" class to transition to the "high" adolescent depression symptoms class, the probability of children in the "low" depression symptoms class transitioning to the "high" class in adolescence was very low. These results suggest that, although youth with high depression symptoms in childhood may experience the greatest vulnerability for developing depression in adolescence, a fair number of children with low depression also experience risk for developing depression. These results suggest that reducing depression symptoms during adolescence may be helped by the use of universal prevention approaches to building coping skills and reducing a negative cognitive style.

5.7 Foundational Research Question III

3. **Foundational Research Question III: Are individuals in the high ADHD symptom severity class in childhood or adolescence also more likely to concurrently be in moderate or high depression symptom classes?**

Results of the concurrent class memberships of symptom types demonstrated that children with "moderate increasing" ADHD symptoms were most likely to concurrently belong to the "high" depression symptoms class. On one hand, this finding is consistent with the symptoms of depression. Concentration problems, distractibility, difficulty maintaining cognitive engagement on tasks, and flagging motivation that impairs following through on initiated tasks are all symptoms that overlap between depression and ADHD. On the other hand, this correspondence may suggest vulnerability for depression associated with ADHD. In support of this stance, children in the "high" depression class were 1.52 times more likely to be in the "high" ADHD symptoms class than the "low" ADHD symptoms class.

Although childhood ADHD and depression symptoms displayed slight correspondence in symptom severity class membership, the adolescent symptoms classes clearly did not. There was little to no similarity in symptom severity class membership between ADHD and depression symptoms. These results suggest that ADHD symptoms in adolescence may appear quite distinct from depression symptoms, despite the overlap in symptoms. It is also possible that teachers, who provided report of ADHD symptoms, may not observe impairment or behavioral challenges for youth in this study who also experienced depression symptoms.

5.8 Foundation Research Question IV

4. **Foundational Research Question IV: Are individuals in the high ADHD symptom severity class in childhood more likely to be in moderate or high depression symptom severity classes in adolescence?**

Childhood ADHD classes did not significantly distinguish adolescent depression class membership. The "low" symptoms ADHD class in childhood was not significantly less likely than the "high" symptoms class to belong to the "high" depression symptoms class in adolescence. These results suggest that childhood ADHD symptoms did not provide significant risk for depression symptoms in adolescence. However, the rate of symptom severity for both the "high" symptoms classes for childhood ADHD and adolescent depression were quite low. These findings should be evaluated in a sample with higher elevations of symptom severity to further explore this research question.

5.9 Secondary Research Questions

1. **Secondary Research Question I: How do contextual predictors (i.e., gender, intervention status, race, and lunch status) affect the latent trajectories and**

class memberships for ADHD and depression symptoms in childhood and adolescence and their association with binge-eating behavior?

5.10 Effect of Contextual Predictors: Gender

The gender effects within this study are consistent with previous findings. Boys consistently demonstrated higher ADHD symptoms during both developmental periods and were more likely to belong to higher rather than lower ADHD symptom severity classes. These results are consistent with research on gender differences associated with ADHD symptoms. Research consistently demonstrates that about three boys to every one girl demonstrated clinically significant levels of ADHD symptoms (Froehlich et al., 2007; Gaub & Carolson, 1997). Although this study did not include diagnosis, identifying higher symptoms in boys than girls parallels findings on gender effects related to ADHD.

On the other hand, girls had higher depression symptoms during both developmental periods and were more likely to belong to higher rather than lower depression symptoms classes during both developmental periods. Research suggests that depression displays gender equivalence in childhood (Hankin & Abramson, 2001; Merikangas & Avenevoli, 2002), a finding with which this study is inconsistent. However, these results are in line with previous research, which consistently demonstrates that adolescent girls are about twice as likely as adolescent boys to develop depression (Ge, et al., 1994; Holsen et al., 2000; Wade et al., 2002).

5.11 Effect of Contextual Predictors: Intervention Status

Children in the GBG and Family-Centered intervention conditions had a higher initial level of ADHD symptoms, but also displayed a decline in ADHD symptoms that was faster than children in the control condition. The effect of intervention status

appeared also in the full transition model, where children in the "high" ADHD symptoms class were more likely than the "low" class to receive the GBG or Family-Centered intervention. However, the effect of intervention was not apparent during adolescence, suggesting that the effect of the interventions may have contributed to equalizing ADHD symptom severity levels between intervention and control conditions and that this effect was maintained into adolescence. No differences in class membership were present between intervention conditions in depression symptoms for either developmental period, suggesting that the difference in depression symptom severity for intervention status may have been small.

5.12 Effect of Contextual Predictors: Race & Lunch Status

Race did not affect the initial level, shape, or class membership for ADHD symptoms during either developmental period. However, race did influence depression symptoms during childhood and adolescence. African-American children had slightly higher depression symptoms during childhood and were more likely to belong to the "decreasing high" symptoms class compared to European-American children. These results were the opposite during adolescence. European-American adolescents were more likely to belong to the "high" depression symptoms class than the African-American adolescents.

Lunch status had very little effect on the initial level, shape of growth, or class membership of either symptom type in either developmental period. However, small effects were found in the full transition model, where children in the "increasing moderate" ADHD class and adolescents in the "high" depression class were more likely than other classes to receive free or reduced lunch. Given that lunch status is a proxy for

socio-economic status, these results may suggest, that while socio-economic status appears to have small effects of ADHD symptoms, other contextual characteristics have a bigger effect on the development of ADHD and depression symptoms in childhood and adolescence than economic adversity.

5.13 Strengths and Limitations

A strength of the current study is the novel research question. The present study represents the first examination of the effects that ADHD and depression symptoms have within the same analysis on binge-eating during adolescence. However, the fact that the sample over-represented African-American children compared to the population could be viewed as a limitation of this study. But, although the sample may not be representative of the population, most samples used for study of these constructs are also not representative of the population. Typical samples include primarily European-American individuals with low representation of ethnic/racial minority populations, such as African-Americans. These sample characteristics reduce the degree to which results generalize to African-American youth. The sample demographics for the present study address this gap in the literature.

Another limitation of this study is that depression symptoms were measured through self-report rather parent or teacher report. Accurate self-report of mental health symptoms requires that individuals possess the cognitive capacity to be aware of their emotional states, to accurately identify the emotion they experience, and to be willing to accurately represent these symptoms during assessment (Nisbett & Wilson, 1977; Perry & Carroll, 2008). Research indicates that some youth may lack these internal skills, which appears to reduce the validity of their self-report of internalizing symptoms

(Achenbach, McConaughy, & Howell, 1987; Measelle, John, Ablow, Cowan, & Cowan, 2005). On the other hand, other research indicates that self-report of depression symptoms provides equivalent to more accurate ratings of depression compared to teacher and parent report (Breland-Noble & Weller, 2012), and teachers and parents often lack awareness of internalizing symptoms and under-report symptom severity for identified youth (Kolko & Kazdin, 1993). Given these findings, it appears that self-report of depression is a valid and viable way to represent depression symptoms in this study.

The fact that not all diagnostic symptom criteria of ADHD were assessed with the measurement strategy used in this study represents another limitation. Also, binge-eating symptoms were assessed only through measurement of attitudes and feelings related to eating rather than specific behavior, such frequency of binges and quantity consumed during a binge episode. Assessing attitudes and emotions towards eating may have greater validity with measurement of binge-eating for children, given that children appear to significantly under-report their consumption of food (Field et al., 2004; Tanofsky-Kraff et al., 2003). Binge-eating symptoms also appear to display a variable developmental course, with some children consistently experiencing symptoms across childhood and other children experiencing remission of symptoms. Unfortunately, these analyses include assessment of binge-eating symptoms at only one time period.

Data used for these analyses came from a study where youth in two out of the three conditions received treatment for emotional and behavior difficulties, which may have decreased the symptom severity measured in the study. Rigorous procedures were undertaken to evaluate the contribution of treatment to these results, such as comparing

results for all models with and without contextual predictors that included intervention status and reporting the effects on intervention status on all parameters of interest.

The internal consistency of several constructs included in analyses was somewhat lower than would be preferred. Reliability was slightly lower than desirable for depression in childhood and binge-eating factor. The binge-eating factor and the depression symptoms construct for two childhood time points had internal consistency values below .80 but above .75. Although these values are below the cut point for acceptable reliability, evaluation of this study's research questions was still feasible and reported results are not significantly affected.

Finally, results from the power analyses indicated low power to assess this study's research questions for the independent transition model of depression symptoms. These problems impacted the power analyses of the full transition model as well. Full classification of participants into symptom classes was used to evaluate the link between binge-eating and symptom classes. This was also a limitation in that the probabilities for membership and transitions associated with classes were far less than 100% for all participants. As a result, some misclassification in membership associated with symptoms may have impacted the results of the binge-eating analyses.

5.14 Summary of Findings

Several important findings emerged from this study regarding the effect of ADHD and depression symptoms on binge-eating as well as the development of ADHD and depression symptoms across the childhood and adolescent developmental periods. Childhood and adolescent ADHD symptom classes but not depression symptom classes predicted 10th grade binge-eating behaviors. The "high" ADHD symptoms class from

childhood and adolescence had higher binge-eating symptoms than other ADHD symptoms classes. Further, childhood and adolescent ADHD symptom classes interacted where binge-eating behaviors were highest for those who were members of both the "high" childhood and adolescent ADHD symptoms classes.

Consideration of the development of ADHD and depression symptoms across childhood and adolescence revealed several interesting results. Latent growth modeling with ADHD and depression symptoms in childhood and adolescence indicated that three classes best fit childhood ADHD symptoms and adolescent depression symptoms, whereas two classes best fit childhood depression symptoms and adolescent ADHD symptoms. The full transition model resulted in two classes for childhood and adolescent depression and adolescent ADHD symptoms, whereas three classes best fit childhood ADHD symptoms. Both ADHD and depression symptoms displayed strong correspondence from childhood and adolescence, although ADHD and depression symptom classes did not predict each other across development periods.

References

Abela, J. R. Z., & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents: A developmental psychopathology perspective. In: Abela, J. R. Z., Hankin, B. L., editors. *Handbook of child and adolescent depression*. New York: The Guilford Press. pp. 35–78.

Abramson, L., Metalsky, G., & Alloy, L. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96,* 358–372.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin, 101,* 213-232.

Ackard, D. M., Neumark-Sztainer, D., Story, M., & Perry, C. (2003). Overeating among adolescents: Prevalence and association with weight-related characteristics and psychological health. *Pediatrics, 111,* 67–74.

Agranat-Meged, A. N., Deitcher, C., Goldzweig, G., Leibenson, L., Stein, M., & Galili-Weisstub, E. (2005).Childhood obesity and attention deficit/hyperactivity disorder: a newly described comorbidity in obese hospitalized children. *International Journal of Eating Disorders, 37,* 357-9.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review 30,* 217–237.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th Edition). Washington DC: American Psychiatric Association.American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th Edition), Text Revision. Washington DC: American Psychiatric Association.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (third Edition – Revised). Washington, DC: American Psychiatric Association.

Anokhin, A. P., Golosheykin, S., Grant, J. D., & Heath, A. C. (2011). Heritability of delay discounting in adolescence: A longitudinal twin study. *Behavioral Genetics, 41,* 175–183.

Attenburrow, M. J., Williams, C., Odontiadis, J., Powell, J., van de Ouderaa, F.,… Cowen, P. J. (2003). The effect of a nutritional source of tryptophan on dieting-induced changes in brain 5-HT function. *Psychological Medicine, 33,* 1381–1386.

Bagwell, C. L., Molina, B. S., Pelham, W. E., & Hoza, B. (2001). Attention-deficit hyperactivity disorder and problems in peer relations: Predictions from childhood to adolescence. *Journal of American Academy of Child and Adolescent Psychiatry, 40,* 1285-92.

Barkley, R. A., & Fischer, M. (2010). The unique contribution of emotional impulsiveness to impairment in major life activities in hyperactive children as adults. *Journal American of Academy Child and Adolescent Psychiatry, 49,* 503-13.

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2002). The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal Abnormal Psychology, 111,* 279-89.

Barkley, R. A., Fischer, M., Edelbrock, C. S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. *Journal American Academy of Child and Adolescent Psychiatry, 29,* 546-57.

Barrish, H. H., Saunders, M., & Wolf, M. M. (1969). Good behavior game: Effects of individual contingencies for group consequences on disruptive behavior in a classroom. *Journal of Applied Behavior Analysis, 2,* 119-24.

Baumeister, R. F. (1990). *Self-inference processes: The Ontario symposium.* Vol. 6. Hillsdale, NJ & England: Lawrence Erlbaum Associates, Inc.

Bauer, D. J., & Curran, P. J. (2004). The integration of continuous and discrete latent latent variable models: Potential problems and promising opportunities. *Psychological Methods, 9,* 3-29.

Becker, A. E., Grinspoon, S. K., Klibanski, A., & Herzog, D. (1999). Eating disorders. *New England Journal of Medicine, 24,* 1092–8.

Biederman, J., Monuteaux, M., Mick, E., Spencer, T. J., Wilens, T. E., Klein, K. L.,… Faraone, S. V. (2006). Psychopathology in females with attention deficit/ hyperactivity disorder: A controlled, five-year prospective study. *Biological Psychiatry, 60,* 1098–1105.

Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *American Journal of Psychiatry, 157,* 816–818.

Biederman, J., Petty, C. R., Clarke, A., Lomedico, A., & Faraone, S. V. (2011). Predictors of persistent ADHD: An 11-year follow-up study. *Journal of Psychiatric Research, 45,*150-155.

Bitsakou, P., Psychogiou, L., Thompson, M., & Sonuga-Barke, E. J. (2009). Delay aversion in attention-deficit/hyperactivity disorder: An empirical investigation of the broader phenotype. *Neuropsychologia, 47,* 446-56.

Bonato, D. P, & Boland, F. J. (1983). Delay of gratification in obese children. *Addictive Behavior, 8,* 71-74.

Bramon-Bosch, E., Troop, N. A., & Treasure, J. L. (2000). Eating disorders in males: A comparison with female patients. *European Eating Disorders, 8,* 321–328.

Brendgen, M., Wanner, B., Morin, A. J., & Vitaro, F. (2005). Relations with parents and with peers, temperament, and trajectories of depressed mood during early adolescence. *Journal of Abnormal Child Psychology, 33,* 579-594.

Braun, D. L., Sunday, S. R., Huang, A., & Halmi, K. A. (1999). More males seek treatment for eating disorders. *International Journal of Eating Disorders, 25,* 415–424.

Bravender, T., Bryant-Waugh, R., Herzog, D., Katzman, D., Kriepe, R. D., Lask, B., & Le Grange, D. (2010). Classification of eating disturbance in children and adolescents: Proposed changes for the DSM-V. *European Eating Disorders Review, 18,* 79–89.

Breland-Nobel, A. M., & Weller, B. (2012). Examining African-American adolescent depression in a community sample: The impact of parent/child agreement. *Journal of Child and Family Studies, 21,* 869-876.

Brown, T. A. (2006). *Confirmatory factor analysis for applied research: Methodology in the social sciences.* New York: The Guilford Press.

Bulik, C. M., & Reichborn-Kjennerud, T. (2003). Medical morbidity in binge-eating disorder. *International Journal of Eating Disorders, 34,* S39-S46.

Burstein, M., Ginsburg, G. S., Petras, H., & Ialongo, N. (2010). Parent psychopathology and youth internalizing symptoms in an urban community: A latent growth model analysis. *Child Psychiatry and Human Development, 41,* 61-87.

Canter, L., & Canter, M. (1992). *Assertive discipline: Positive behavior management for today's classroom*. Santa Monica, CA: Canter and Associates.

Carlat, D. J., & Camargo, C. A. (1991). Review of bulimia nervosa in males. *American Journal of Psychiatry, 148,* 831–43.

Carrard, I., Crépin, C., Ceschi, G., Golay, A., & Van der Linden, M. (2012). Relations between pure dietary and dietary-negative affect subtypes and impulsivity and reinforcement sensitivity in binge-eating individuals. *Eating Behaviors, 13,* 13-9.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319-333.

Cassidy, O. L., Matheson, B., Osborn, R., Vannucci, A., Kozlosky, M., Shomaker, L. B.,…Tanofsky-Kraff, M. (2012). Loss of control eating in African-American and Caucasian youth. *Eating Behaviors, 13,* 174–178.

Cattarin, J. A., & Thompson, J. K. (1994). A three-year longitudinal study of body image, eating disturbance, and general psychological functioning in adolescent females. *Eating Disorders: The Journal of Treatment and Prevention, 2,* 114-125.

Chandler-Laney, P. C., Castaneda, E., Viana, J. B., Oswald, K. D., Maldonado, C. R., & Boggiano, M. M. (2007). A history of caloric restriction induces neurochemical and behavioral changes in rats consistent with models of depression. *Pharmacology Biochemistry and Behavior, 87,* 104-114.

Charach, A., Figueroa, M., Chen, S., Ickowicz, A., & Schachar, R. (2006). Stimulant treatment over 5 years: effects on growth. *Journal of American Academy of Child and Adolescent Psychiatry, 45,* 415-21.

Chronis-Tuscano A., Molina, B. S., Pelham, W. E., Applegate, B., Dahlke, A., Overmyer, M., & Lahey, B. B. (2010). Very early predictors of adolescent depression and suicide attempts in children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 67,* 1044-51.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (third ed.). NJ: Erlbaum.

Cole, D. A., Tram, J. M., Martin, J. M., Hoffman, K. B., Ruiz, M. D., Jacquez, F. M., & Maschman, T. L. (2002). Individual differences in the emergence of depressive symptoms in children and adolescents: a longitudinal investigation of parent and child reports. *Journal of Abnormal Psychology, 111,* 156-65.

Compas, B. E., Connor-Smith, J., & Jaser, S. S. (2004). Temperament, stress reactivity, and coping: Implications for depression in childhood and adolescence. *Journal of Clinical Child and Adolescent Psychology, 33,* 21–31.

Costello, J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60,* 837-844.

Côté, S., Tremblay, R. E., Nagin, D., Zoccolillo, M., & Vitaro, F. (2002). The development of impulsivity, fearfulness, and helpfulness during childhood: Patterns of consistency and change in the trajectories of boys and girls. *Journal of Child Psychology and Psychiatry, 43,* 609-18.

Cortese, S., Angriman, M., Maffeis, C., Isnard, P., Konofal, E., Lecendreux, M.,… Mouren, M. C. (2008): Attention-deficit/hyperactivity disorder (ADHD) and obesity: A systematic review of the literature. *Critical Reviews in Food Science and Nutrition, 48,* 524-537.

Cortese, S., Isnard, P., Bernardino, B. D., & Mouren, M. C. (2007). Attention-deficit / hyperactivity disorder (ADHD) and binge-eating, *Nutrition Reviews, 65,* 404-417.

Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder: a review of theory and evidence. *Appetite, 53,* 1–8.

Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that food addiction' is a valid phenotype of obesity. *Appetite, 57,* 711-7.

Davis, C., Levitan, R.D., Smith, M., Tweed, S., & Curtis, C. (2006). Associations among overeating, overweight, and attention deficit/hyperactivity disorder: A structural equation modeling approach. *Eating Behaviors, 7,* 266–74.

Davis, C., Patte, C., Levitan, R. D., Carter, J., Kaplan, A. S., & Zai, C. (2010). A psycho-genetic study of associations between the symptoms of binge-eating disorder and those of attention deficit/hyperactivity disorder. *Journal of Psychiatric Research, 43,* 687–696.

Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience & Biobehavioral Reviews 28,* 343–351.

Decaluwé, V., & Braet, C. (2003). Prevalence of binge-eating disorder in obese children and adolescents seeking weight-loss treatment. *International Journal of Obesity and Related Metabolic Disorders, 27,* 404-409.

Dekker, M. C., Ferdinand, R. F., van Lang, N. D. J., Bongers, I. L., van der Ende, J., & Verhulst, F. C., (2007). Developmental trajectories of depressive symptoms from early childhood to late adolescence: Gender differences and adult outcome. *Journal of Child Psychology and Psychiatry, 48,* 657–666.

de Zwaan, M., Gruss, B., Müller, A., Philipsen, A., Graap, H., Martin, A.,…Hilbert, A. (2011). Association between obesity and adult attention-deficit/hyperactivity disorder in a German community-based sample. *Obesity Facts, 4,* 4-11.

DuPaul, G. J., Ervin, R. A., Hook, C. L., & McGoey, K. E. (1998). Peer tutoring for children with attention deficit hyperactivity disorder: effects on classroom

behavior and academic performance. *Journal of Applied Behavioral Analysis, 31,* 579–592.

Espelage, D. L., Mazzeo, S. E., Aggen, S. H., Quittner, A. L., Sherman, R., & Thompson, R. (2003). Examining the construct validity of the Eating Disorder Inventory. *Psychological Assessment, 15,* 71-80.

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. (2000). The natural course of bulimia nervosa and binge-eating disorder in young women. *Archives of General Psychiatry, 57,* 659–665.

Faraone S. V., Biederman J., Spencer T., Wilens T., Seidman L. J., Mick E., & Doyle, A. E. (2000). Attention-deficit/hyperactivity disorder in adults: An overview. *Biological Psychiatry, 48,* 9–20.

Field, A. E., Camargo, C. A., Taylor, C. B., Berkey, C. S., Frazier, A. L., Gillman, M. W., & Colditz, G. A. (1999). Overweight, weight concerns, and bulimic behaviors among girls and boys. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 754–760.

Field, A . E., Camargo, C. A., Taylor, C. B., Berkey, C. S., Roberts, S. B., & Colditz, G. A. (2001). Peer, parent and media influences on the development of weight concerns and frequent dieting among preadolescent girls and boys. *Pediatrics, 107,* 54–60.

Field, A. E., Taylor, C. B., Celio, A., & Colditz, G. A. (2004). Comparison of self-report to interview assessment of bulimic behaviors among preadolescent and adolescent girls and boys. *International Journal of Eating Disorders, 35,* 86–92.

Fischer, S., & Smith, G. T. (2008). Binge-eating, problem drinking, and pathological gambling: Linking behavior to shared traits and social learning. *Personality and Individual Differences, 44,* 789–800.

Frazier, T. W., Youngstrom, E. A., & Naugle, R. I. (2007). The latent structure of ADHD in a clinic referred sample. *Neuropsychology, 21,* 45-64.

French, S. A., Story, M., Neumark-Sztainer, D., Downes, B., Resnick, M., & Blum, R. (1997). Ethnic differences in psychosocial and health behavior correlates of dieting, purging, and binge-eating in s population-based sample of adolescent females. *International Journal of Eating Disorders, 22,* 315-322.

Froehlich, T. E., Lanphear, B. P., Epstein, J. N., Barbaresi, W. J., Katusic, S. K., & Kahn, R. S. (2007). Prevalence, recognition, and treatment of attention-deficit/hyperactivity disorder in a national sample of U. S. children. *Archives of Pediatric and Adolescent Medicine, 161,* 857-64.

Garner, D. M., Olmstead, M., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2,* 15–34.

Gaub, M., & Carolson, C. L. (1997). Gender differences in ADHD: a meta-analysis and critical review. *Journal of American Academy Child and Adolescent Psychiatry, 36,* 1036-45.

Gaylord-Harden, N. K., & Cunningham, J. A. (2009). The impact of racial discrimination and coping strategies on internalizing symptoms in African American youth. *Journal of Youth and Adolescence, 38,* 532-43.

Ge, X., Lorenz, F. O., Conger, R. D., Elder, G. H., & Simons, R. L. (1994). Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental Psychology, 30,* 467-483.

Glashofer, D. R., Tanofsky-Kraff, M., Eddy, K. T., Yanovski, S. Z., Theim, K. R., Mirch, M. C., …Yanovski, J. A. (2007). Binge-eating in overweight treatment-seeking adolescents. *Journal of Pediatric Psychology, 32,* 95–105.

Goldschmidt, A. B., Tanofsky-Kraff, M., & Wilfley, D. E. (2011). A laboratory-based study of mood and binge-eating behavior in overweight children. *Eating Behaviors, 12,* 37-43.

Goosens, L., Braet, C., Verbeken, S., Decaluwe´, V. & Bosmans, G. (2011). Long-term outcome of pediatric eating pathology and predictors for the onset of loss of control over eating following weight-loss treatment. *International Journal of Eating Disorders, 44,* 397–405.

Gorin, A. A., Hogan, P., DiLillo, V. G., Wadden, T. A., Williamson, D., Niemeier, H. M.,… Action for Health in Diabetes (Look AHEAD) Research Group. (2008). Binge-eating and weight loss outcomes in overweight and obese individuals with type 2 diabetes: Results from the LOOK AHEAD trial. *Archives of General Psychology, 65,* 1447–1455.

Graham, J. W., (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology, 60,* 549–576.

Gray, J. A. (1987). *The psychology of fear and stress.* NY: Cambridge University Press.

Gray, J. A. (1982). *The neuropsychology of anxiety: An inquiry of the septo-hippocampal system.* Oxford: Oxford University Press.

Green, L., & Myerson, J. (2004). A discounting framework for choice with delayed and probabilistic rewards. *Psychological Bulletin, 130,* 769 –792.

Grilo, C. M., Hrabosky, J. I., White, M. A., Allison, K. C., & Stunkard, A. J. (2008). Overvaluation of shape and weight in binge-eating disorder and overweight controls: Refinement of BED as a diagnostic construct. *Journal of Abnormal Psychology, 117,* 414–419.

Grucza, R. A., Przybeck, T. R., & Cloninger, C. R. (2007). Prevalence and correlates of binge-eating disorders in a community sample. *Comprehensive Psychiatry, 48,* 124–131.

Gruss, B., Mueller, A., Horbach, T., Martin, A., & de Zwaan, M. (2012). Attention-deficit/hyperactivity disorder in a prebariatric surgery sample. *European Eating Disorder Review, 20,* e103-107.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge-eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137,* 660–681.

Haering, S. A., & Syed, S. B. (2009). Community Food Security in United States Cities: A Survey of the Relevant Scientific Literature. Baltimore, MD: Future Center for a Livable Future, Johns Hopkins Bloomberg School of Public Health.

Halperin, J. M., & Healey, D. M. (2011). The influences of environmental enrichment, cognitive enhancement, and physical exercise on brain development: can we alter the developmental trajectory of ADHD? *Neuroscience and Biobehavioral Review, 35,* 621-623.

Hankin B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: an elaborated cognitive vulnerability-transactional stress theory. *Psychology Bulletin, 127,* 773-796.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107,* 128-140.

Hansen, C., Weiss, D., & Last, C. G. (1999). ADHD boys in young adulthood: Psychosocial adjustment. *Journal of American Academy of Child and Adolescent Psychiatry, 38,* 165-171.

Harrington, E. F., Crowther, J. H., Henrickson, H. C. P., & Mickelson, K. D. (2006). The relationships among trauma, stress, ethnicity, and binge eating. *Cultural Diversity and Ethnic Minority Psychology, 12,* 212–229.

Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. *Journal of Abnormal Child Psychology, 23,* 729–749.

Hartmann, A. S., Czaja, J., Rief, W., & Hilbert, A. (2010). Personality and psychopathology in children with and without loss of control over eating. *Comprehensive Psychiatry, 51,* 572–578.

Haslam, N., Williams, B., Prior, M., Haslam, R., Graetz, B., & Sawyer, M. (2006). The latent structure of attention-deficit/hyperactivity disorder: A taxometric analysis. *Australian and New Zealand Journal of Psychiatry, 40,* 639-647.

Hasler, G., Pine, D. S., Gamma, A., Milos, G., Ajdacic, V., Eich, D.,…Angst, J. (2004). The associations between psychopathology and being overweight: A 20-year prospective study. *Psychological Medicine, 34,* 1047–1057.

Hawkins, R. C., & Clement, P. F. (1984). *Binge-eating: Measurement problems and a conceptual model.* In: Hawkins, R. C., Fremouw, W. J., & Clement, P. F., editors. The binge purge syndrome: Diagnosis, treatment, and research. New York: Springer, p. 229-251.

Herman, C. P., & Polivy, J. (1980). *Restrained eating.* In: Stunkard, J. A., editor. Obesity. Philadelphia, PA: W. B. Saunders Company, p. 208-225.

Hickson, D. M. A., Lewis, T. T., Liu, J. Mount, D. L., Younge, S. N., Jenkins, W. C., …Williams, D. R. (2012). The Associations of Multiple Dimensions of Discrimination and Abdominal Fat in African American Adults: The Jackson Heart Study. *Annals of Behavioral Medicine, 43,* 4–14.

Hohlstein, L. A., Smith, G. T., & Atlas, J. G. (1998). An application of expectancy theory to eating disorders: Development and validation of measures of eating and dieting expectancies. *Psychological Assessment. 10,* 49–58.

Holtkamp, K., Hebebrand, J., & Herpertz-Dahlmann, B. (2004). The contribution of anxiety and food restriction on physical activity levels in acute anorexia nervosa. *International Journal of Eating Disorders, 36,* 163-71.

Holsen, I., Kraft, P., & Vittersø, J. (2000). Stability in depressed mood in adolescence: Results from a 6-year longitudinal panel study. *Journal of Youth and Adolescence, 29,* 61-78.

Hong, S. B., Dwyer, D., Kim, J. W., Park, E. J., Shin, M. S., …Cho, S. C. (in press). Subthreshold attention-deficit/hyperactivity disorder is associated with functional impairments across domains: a comprehensive analysis in a large-scale community study. *European Child and Adolescent Psychiatry.*

Horowitz, C. A., Colson, K. A., Hebert, P. L., & Lancaster, K. (2004). Barriers to buying healthy foods for people with diabetes: evidence of environmental disparities. *American Journal of Public Health, 94,* 1549–1554.

Horwitz, S. M., Hoagwood, K., Stiffman, A. R., Summerfeld, T., Weisz, J. R., Costello, E. J., Rost, K. B.,…Norquist, G. (2001). Reliability of the services assessment for children and adolescents. *Psychiatric Service, 52,* 1088–1094.

Hoza, B., Murray-Close, D., Arnold, L. E., Hinshaw, S. P. & MTA Cooperative Group. (2010). Time-dependent changes in positive illusory self-perceptions of children with ADHD: A developmental psychopathology perspective. *Development and Psychopathology, 22,* 375-390.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6,* 1-55.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychology, 61,* 348–58.

Hudson, J. I., Lalonde, J. K., Coit, C. E., Tsuang, M. T., McElroy, S. L., Crow, S. J.,…Pope, H. G. (2010). Longitudinal study of the diagnosis of components of the

metabolic syndrome in individuals with binge-eating disorder. *American Journal of Clinical Nutrition, 91,* 1568-1573.

Hudziak, J. J., Heath, A. C., Madden, P. F., Reich, W., Bucholz, K. K., Slutske, W.,… Todd, R. D. (1998). Latent class and factor analysis of DSM-IV ADHD: A twin study of female adolescents. *Journal of American Academy of Child and Adolescent Psychiatry, 37,* 848-857.

Hunte, H. E., & Williams, D. R. (2009). The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. *American Journal of Public Health, 99,* 1285–1292.

Ialongo, N., Kellam, S. & Poduska, J. (1999). A developmental epidemiologic framework for clinical and pediatric psychology research. In D. Drotar (ed.), *Handbook on Pediatric and Clinical Psychology* (pp. 1-25). New York: Kluwer Academic/Plenum Press.

Javaras, K. N., Pope, H. G., Lalonde, J. K., Roberts, J. L., Nillni, Y. I., Laird, N. M.,…Hudson, J. I. (2008). Co-occurrence of binge-eating disorder with psychiatric and medical disorders. *Journal of Clinical Psychiatry, 69,* 266-273.

Jester, J. M., Nigg, J. T., Adams, K., Fitzgerald, H. E., Puttler, L. I., Wong, M. M., & Zucker, R. A. (2005). Inattention/hyperactivity and aggression from early childhood to adolescence: Heterogeneity of trajectories and differential influence of family environment characteristics. *Developmental Psychopathology, 17,* 99-125.

Jester, J. M., Nigg, J. T., Buu, A. Puttler, L. I., Glass, J. M., Fitzgerald, H. E., & Zucker, R. A. (2008). Trajectories of childhood aggression and inattention/hyperactivity:

Differential effects on substance abuse in adolescence. *Journal of American Academy of Child and Adolescent Psychiatry, 47,* 1158–1165.

Johnson, W. G., Parry, W., & Drabman, R. S. (1978). The performance of obese and normal size children on a delay of gratification task. *Addictive Behavior, 3,* 205-208.

Johnson, W. J., Rohan, K. J., & Kirk, A. A. (2002). Prevalence and correlates of binge-eating in white and African American adolescents. *Eating Behaviors 3,* 179–189.

Kellam, S. G., Brown, C. H., Poduska, J. M., Ialongo, N. S., Wang, W., Toyinbo, P.,… Wilcox, H. C. (2008). Effects of a universal classroom behavior management program in first and second grades on young adult behavioral, psychiatric, and social outcomes. *Drug and Alcohol Dependence, 95, Supplement 1,* S5-S28.

Kellam, S. G., Rebok, G. W., Mayer, L. S., Ialongo, N., & Kalodner, C. R. (1994). Depressive symptoms over first grade and their responsiveness to a preventive trial aimed at improving achievement. *Development and Psychopathology, 6,* 463-491.

Kellam, S. G., Reid, J., & Balster, R. L. (2008). Effects of a universal classroom behavior program in first and second grades on young adult problem outcomes. *Drug and Alcohol Dependence, 95, Supplement 1,* S1-4.

Keselman, H. J., Huberty, C. J., Lix, L. M., Olejnik, S., Cribbie, R., Donahue, B.,… Levin, J. R. (1998). Statistical practices of educational researchers: An analysis of their ANOVA, MANOVA, and ANCOVA analyses. *Review of Educational Research, 68,* 350– 386.

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O.,… Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry, 163*, 716-723.

Kessler, R. C., Gruber, M., Hettema, J. M., Hwang, I., Sampson, N., & Yonkers, K. A. (2008). Co-morbid major depression and generalized anxiety disorders in the National Comorbidity Survey follow-up. *Psychological Medicine*, 38, 365-374.

Kinsbourne, M. (1973). Minimal brain dysfunction as a neurodevelopmental lag. *Annals of the New York Academy of Sciences, 205,* 268–273.

Klein, D. N. (2008). Classification of depressive disorders in the DSM-V: Proposal for a two-dimension system. *Journal of Abnormal Psychology, 117*, 552-560.

Kolko, D. J., & Kazdin, A. E. (1993). Emotional/behavioral problems in clinic and nonclinic children: Correspondence among child, parent and teacher reports. *Journal of Child Psychology and Psychiatry, 34*, 991-1006.

Landrine, H., & Klonoff, E. A. (1996). The schedule of racist events: A measure of racial discrimination and a study of its negative physical and mental health consequences. *Journal of Black Psychology*, 22, 144-168.

Langberg, J. M., Epstein, J. N., Altaye, M., Molina, B. S., Arnold, L. E., & Vitiello, B. (2008). The transition to middle school is associated with changes in the developmental trajectory of ADHD symptomatology in young adolescents with ADHD. *Journal of Clinical Child and Adolescent Psychology, 37*, 651-663.

Langberg, J. M., Molina, B. S. G., Arnold, L. E., Epstein, J. N., Altaye, M., Hinshaw, S. P.,…Hechtman, L. (2011). Patterns and predictors of adolescent academic

achievement and performance in a sample of children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Clinical Child and Adolescent Psychology, 40*, 519-531.

Larsson, H., Dilshad, R., Lichtenstein, P., & Barker, E. D. (2011). Developmental trajectories of DSM-IV symptoms of attention-deficit/hyperactivity disorder: Genetic effects, family risk and associated psychopathology. *Journal of Child Psychology and Psychiatry, 52*, 954-963.

Larsson, H., Lichtenstein, P., & Larsson, J. O. (2006). Genetic contributions to the development of ADHD subtypes from childhood to adolescence. *Journal of American Academy of Child and Adolescent Psychiatry, 45*, 973-981.

Lee, S. S., Humphreys, K. L., Flory, K., Liu, R., & Glass, K. (2011). Prospective association of childhood attention-deficit/hyperactivity disorder (ADHD) and substance use and abuse/dependence: A meta-analytic review. *Clinical Psychology Review, 31*, 328-341.

Lewinsohn, P. M., Shankman, S. A., Gau, J. M., & Klein, D. N. (2004). The prevalence and co-morbidity of sub-threshold psychiatric conditions. *Psychological Medicine, 34*, 613-622.

Lo, Y., Mendell, N., & Rubin, D. (2001). Testing the number of components in a normal mixture. *Biometrika, 88*, 767–778.

MacCallum, R. C., Browne, M. W., & Cai, L. (2006). Testing differences between nested covariance structure models: Power analysis and null hypotheses. *Psychological Methods*, *11*, 19-35.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods, 7,* 19-40.

Malone, P. S., Van Eck, K., Flory, K., & Lamis, D. (2010). A mixture-model approach to linking ADHD to adolescent onset of illicit drug use. *Developmental Psychology, 46,* 1543–1555.

Manuck, S. B., Flory, J. D., Muldoon, M. F., & Ferrell, R. E. (2003). A neurobiology of intertemporal choice. In: *Time and decision: Economic and psychological perspectives on intertemporal choice* (Loewenstein, G., Read, D., & Baumeister, R. F., eds), 139–172. New York: Sage.

Marcus, D. K., & Barry, T. D. (2010). Does ADHD have a dimensional latent structure? A taxometric analysis. *Journal of Abnormal Psychology, 120,* 427–442.

Marcus, M. D., & Kalarchian, M. A. (2003). Binge eating in children and adolescents. *International Journal of Eating Disorders, 34,* S47-57.

Marsh, H. W., Hau, K. T., & Wen, Z. (2004). In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) findings. *Structural Equation Modeling, 11,* 320-341.

Masheb, R. M., & Grilo, C. M. (2000). Binge-eating disorder: A need for additional diagnostic criteria. *Comprehensive Psychiatry, 41,* 159–162.

Masheb, R. M., & Grilo, C. M. (2008). Examination of predictors and moderators for self-help treatments of binge-eating disorder. *Journal of Consulting and Clinical Psychology, 76,* 900-904.

Mazza, J. J., Fleming, C. B., Abbott, R. D., Haggerty, K. P., & Catalano, R. F. (2010). Identifying trajectories of adolescents' depressive phenomena: An examination of early risk factors. *Youth Adolescence, 39,* 579–593.

McLachlan, G., & Peel, D. (2000). *Finite Mixture Models.* New York: John Wiley and Sons.

McQuade, J. D., Tomb, M., Hoza, B., Waschbusch, D. A., Hurt, E. A., & Vaughn, A. J. (2011). Cognitive deficits and positively biased self-perceptions in children with ADHD. *Journal of Abnormal Child Psychology, 39,* 307-319.

Measelle, J. R., John, O. P., Ablow, J. C., Cowan, P. A., & Cowan, C. (*2005).* Can young children provide coherent, stable, and valid self-reports on the Big Five dimension? A longitudinal study from ages 5 to 7. *Journal of Personality and Social Psychology, 89,* 90-106.

Melnick, S. M., & Hinshaw, S. P. (2000). Emotion regulation and parenting in AD/HD and comparison boys: Linkages with social behaviors and peer preference. *Journal of Abnormal Child Psychology, 28,* 73-86.Merikangas, K. R., & Avenevoli, S. (2002). Epidemiology of mood and anxiety disorders in children and adolescents. in:M.T. Tsuang, M. Tohen (Eds.) *Textbook in psychiatric epidemiology. 2nd ed.* New York: John Wiley and Sons, 657–704.Merikangas, K. R., Avenevoli, S., Costello, J., Koretz, D., & Kessler, R. C. (2009). National comorbidity survey replication adolescent supplement (NCS-A): I. Background and measures. *Journal of American Academy of Child and Adolescent Psychiatry, 48,* 367–369.Merikangas, K. R., He, J., Burstein, M., Swanson, S. A., Avenevoli, S., …Swendsen, J. (2010). Lifetime Prevalence of Mental Disorders in

US Adolescents: Results from the National Comorbidity Study-Adolescent Supplement (NCS-A). *Journal of American Academy of Child and Adolescent Psychiatry, 49,* 980-989.

Mikami, A. Y., Hinshaw, S. P., Arnold, L. E., Hoza, B. Hechtman, L., Newcorn, J. H., & Abikoff, H. B. (2010). Bulimia nervosa symptoms in the multimodal treatment study of children with ADHD. *International Journal Eating Disorders, 43,* 248–259.

Mikami, A. Y., Hinshaw, S. P., Patterson, K. A., & Lee, J. C. (2008). Eating pathology among adolescent girls with attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology, 117,* 225–235.

Milich, R., & Okazaki, M. (1991). An examination of learned helplessness among attention-deficit/hyperactivity disordered boys. *Journal of Abnormal Child Psychology, 19,* 607-623.

Moffitt, T. E., Caspi, A., Harrington, H. L., Milne, B. J., Melchior, M., Goldberg, D., & Poulton, R. (2007). Generalized anxiety disorder and depression: Childhood risk factors in a birth cohort followed to age 32. *Psychological Medicine*, 37, 441-452.

Moore, L., & Diez Roux, A. (2006). Association of neighborhood characteristics with the location and type of food stores. *American Journal of Public Health, 96,* 325-31.

Morgan, C., Yanovski, S., Nguyen, T., McDuffie, J., Sebring, N., Jorge, M.,…Yanovski, J. A. (2002). Loss of control over eating, adiposity, and psychopathology in overweight children. *International Journal of Eating Disorders, 31,* 430–441.

Muise, A. M., Stein, D. G., & Arbess, G. (2003). Eating disorders in adolescent boys: a review of the adolescent and young adult literature. *Journal of Adolescent Health, 33,* 427-35.

Murphy, B. C., Shepard, S., Eisenberg, N., Fabes, R. A., & Guthrie, I. K. (1999). Contemporaneous and longitudinal relations of young adolescents' dispositional sympathy to their emotionality, regulation, and social functioning. *Journal of Early Adolescence, 29,* 66–97.

Muthén, B. and Asparouhov, T. (2011). *LTA in Mplus: Transition probabilities influenced by covariates.* Mplus Web Notes, 13, July 27, 2011. Dowloaded March 15, 2014 from http://www.statmodel.com/examples/webnote.shtml.

Muthén, B., & Muthén, L. K. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcohol Clinical and Experimental Research, 24,* 882-891.

Muthén, L., & Muthén, B. (2002). How to use a Monte Carlo study to decide on sample size and determine power. *Structural Equation Modeling, 4,* 599–620.

Muthén, L. K., & Muthén, B. O. (2013). *Mplus user's guide. Seventh Edition.* Los Angeles, CA: Muthén & Muthén.

Myers, J. E. B., Berliner, L., Briere, J., Hendrix, C. T., Jenny, C., & Reid, T. A. (2002). *APSAC handbook on child maltreatment* (2nd ed.). Thousand Oaks, CA: Sage Publications.

Nagin, D.S. 1999. "Analyzing Developmental Trajectories: A Semi-parametric, Group-based Approach." *Psychological Methods, 4,* 139-177.

Nagin, D., & Tremblay, R. E. (1999). Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Development, 70,* 1181-1196.

Nasser, J. A., Gluck, M. E., & Geliebter, A. (2004). Impulsivity and test meal intake in obese binge-eating women. *Appetite, 43,* 303-7.

Nederkoorn, C., Braet, C., Van Eijs, Y., Tanghe, A., & Jansen, A. (2006). Why obese children cannot resist food: The role of impulsivity. *Eating Behaviors, 7,* 315–322.

Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2007). Impulsivity predicts treatment outcome in obese children. *Behavior Research Therapy, 45,* 1071-1075.

Neumark-Sztainer, D., & Hannan, P. J. (2000). Weight-related behaviors among adolescent girls and boys. *Archives of Pediatric and Adolescent Medicine, 154,* 569–77.

Neumark-Sztainer, D., Wall, M., Larson, N. I., Eisenberg, M. E., & Loth, M. (2011). Dieting and disordered eating behaviors from adolescence to young adulthood: Findings from a 10-year longitudinal study. *Journal of American Dietetic Association, 111,* 1004-1011.

Nikolas, M., Friderici, K., Waldman, I., Jernigan, K., & Nigg, J. T. (2010). Gene x environment interactions for ADHD: Synergistic effect of 5HTTLPR genotype and youth appraisals of inter-parental conflict. *Behavior and Brain Function, 16,* 23.

Nisbett, R. E., & Wilson, T. D. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review, 84,* 231-259.

Nylund, K. (2007). Latent transition analysis: Modeling extensions and an application to peer victimization. Doctoral dissertation, University of California, Los Angeles.

Nylund, K. L., Asparouhov, T., & Muthén, B. O. (2007). Deciding on the number of classes in latent class analysis and growth mixture modeling: A monte carlo simulation study. *Structural Equation Modeling, 14,* 535–569.

O'Neill, M. E., & Douglas, V. I. (1996). Rehearsal strategies and recall performance with boys with and without attention deficit/hyperactivity disorder. *Journal of Pediatric Psychology, 21,* 73-88.

Pagoto, S. L., Curtin, C., Bandini, L. G., Anderson, S. E., Schneider, K. L., Bodenlos, J. S., & Ma, Y. (2010). Weight loss following a clinic-based weight loss program among adults with attention deficit/hyperactivity disorder symptoms. *Eating and Weight Disorders, 15,* e166-72.

Paloyelis, Y., Mehta, M. A., Kuntsi, J., & Asherson, P. (2007). Functional MRI in ADHD: A systematic literature review. *Expert Review of Neurotherapeutics, 7,* 1337–1356.

Patton, J. M., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of Clinical Psychology, 51,* 768-774.

Pearson, C. M., Combs, J. M., Zapolski, T. C. B, & Smith, G. T. (2012). A longitudinal transactional risk model for early eating disorder onset. *Journal of Abnormal Psychology, 121,* 707–718.

Pellegrini, A. D., & Long, J. D. (2002). A longitudinal study of bullying, dominance, and victimization during the transition from primary school through secondary school. *British Journal of Developmental Psychology, 20,* 259–280.

Perry, J. L., & Carroll, M. E. (2008). The role of impulsive behavior in drug abuse. *Psychopharmacology, 200, 1-26.*

Petersen, A. C., Compas, B. E., Brooks-Gunn, J., Stemmler, M., Ey, S., & Grant, K. E. (1993). Depression in adolescence. *American Psychology, 48,* 155-68.

Pfiffner, L. J., Mikami, A., Huang-Pollock, C. L., Easterlin, B., Zalecki, C., & McBurnett, K. (2007). A randomized controlled trial of integrated home-school behavioral treatment for ADHD, Predominantly Inattentive Type. *Journal of the American Academy of Child and Adolescent Psychiatry, 46,* 1041-1050.

Pickering, T. (2000). Depression, race, hypertension, and the heart. *Journal of Clinical Hypertension, 2,* 410–412.

Pingault, J. B., Tremblay, R. E., Vitaro, F., Carbonneau, R., Genolini, C., Falissard, B., & Côté, S. M. (2011).Childhood trajectories of inattention and hyperactivity and prediction of educational attainment in early adulthood: A 16-year longitudinal population-based study. *American Journal of Psychiatry, 168,* 1164-70.

Plummer, P. J., & Stoner, G. (2005). The relative effects of classwide peer tutoring and peer coaching on the positive social behaviors of children with ADHD. *Journal of Attention Disorders, 9,* 290–300.

Pope, H. G., Lalonde, J. K., Pindyck, L. J., Walsh, T., Bulik, C. M., Crow, S. J.,… Hudson, J. I. (2006). Binge-eating disorder: A stable syndrome. *American Journal of Psychiatry, 163,* 2181–2183.

Raffaelli, M., Crockett, L. J., & Shen, Y. (2005). Developmental stability and change in self-regulation from childhood to adolescence. *Journal of Genetic Psychology, 166,* 54–75.

Raudenbush, S. W., & Bryk, A. S. (2002). Hierarchical Linear Models (Second Edition). Thousand Oaks, CA: Sage Publications.

Rawana, J. S., Morgan, A. S., Nguyen, H., & Craig, S. G. (2010). The relation between eating- and weight-related disturbances and depression in adolescence: A review. *Clinical Child and Family Psychology Review, 13,* 213–230.

Reinke W. M., & Ostrander, R. (2008). Heterotyic and homotypic continuity: The moderating effects of age and gender. *Journal of Abnormal Child Psychology, 36,* 1109-1121.

Reynolds, B. (2006). A review of delay-discounting research with humans: Relations to drug use and gambling. *Behavioral Pharmacology, 17,* 651–667.

Reynolds, W. M., & Johnston, H. F. (1994). The nature and study of depression in children and adolescents. In W. M. Reynolds & H. F. Johnston (Eds.), *Handbook of depression in children and adolescents,* (pp. 3-17). New York: Plenum.

Robbers, S. C., van Oort, F. V., Polderman, T. J., Bartels, M., Boomsma, D. I., Verhulst, F. C.,…Huizink, A. C. (2011). Trajectories of CBCL attention problems in childhood. *European Child and Adolescent Psychiatry, 20,* 419-27.

Rodriguez, D., Moss, H. B., & Audrain-McGovern, J. (2005). Developmental heterogeneity in adolescent depressive symptoms: Associations with smoking behavior. *Psychosomatic Medicine, 67,* 200-10.

Scahill, L., Schwab-Stone, M., Merikangas, K. R., Leckman, J. F., Zhang, H., & Kasl, S. (1999). Psychosocial and clinical correlates of ADHD in a community sample of school-age children. *Journal of American Academy of Child and Adolescent Psychiatry, 38,* 976-84.

Scheres, A., Dijkstra, M., Ainslie, E., Balkan, J., Reynolds, B., Sonuga-Barke, E., & Castellanos, F. X. (2006). Temporal and probabilistic discounting of rewards in children and adolescents: effects of age and ADHD symptoms. *Neuropsychologia, 44,* 2092–2103.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods, 7,* 147-177.

Scime, M., & Norvilitis, J. M. (2006). Task performance and response to frustration in children with attention deficit hyperactivity disorder. *Psychology in the Schools, 43,* 377-386.

Seymour, K. E., & Chronis-Tuscano, A., Halldorsdottir, T., Stupica, B., Owens, K., & Sacks, K. (2012). Emotion regulation mediates the relationship between ADHD and depressive symptoms in youth. *Abnormal Child Psychology, 40,* 595–606.

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D.,…Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences, U.S.A., 104,* 19649–19654.

Shaw, D. S., Lacourse, E., & Nagin, D. S. (2005). Developmental trajectories of conduct problems and hyperactivity from ages 2 to 10. *Journal of Child Psychology and Psychiatry, 46,* 931-42.

Shaw, P., Malek, M., Watson, B., Sharp, W., Evans, A., & Greenstein D. (2012). Development of cortical surface area and gyrification in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 72,* 191–197.

Shisslak, C. M., Mays, M. Z., Crago, M., Jirsak, J. K., Taitano K., & Cagno, C. (2006). Eating and weight control behaviors among middle school girls in relationship to body weight and ethnicity. *Journal of Adolescent Health, 38,* 631-633.

Sigal, J. J., & Adler, L. (1976). Motivational effects of hunger on time estimation and delay of gratification in obese and non-obese boys. *Journal of Genetic Psychology, 128,* 7-16.

Sims, M., Diez-Roux, A. V., Dudley, A., Gebreab, S., Wyatt, S. B.,…Herman A. Taylor, H. A. (2012). Perceived Discrimination and Hypertension among African Americans in the Jackson Heart Study. *American Journal of Public Health, 102,* S258-S265.

Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence.* New York: Oxford University Press.

Skinner, H. H., Haines, J., Austin, S. B., & Field, A. E. (2012). A prospective study of overeating, binge-eating, and depressive symptoms among adolescent and young adult women. *Journal of Adolescent Health, 50,* 478–483.

Sloane, D. C., Diamant, A. L., Lewis, L. B., Yancey, A. K., Flynn, G.,… Cousineau, M. R. (2003). Improving the nutritional resource environment for healthy living through community-based participatory research. *Journal of General Internal Medicine, 18,* 568–575.

Solanto, M. V., Abikoff, H., Sonuga-Barke, E., Schachar, R., Logan, G.D., Wigal, T.,… Turkel, E. (2001). The ecological validity of delay aversion and response inhibition as measures of impulsivity in AD/HD: A supplement to the NIMH

multimodal treatment study of AD/HD. *Journal Abnormal Child Psychology, 29,* 215-28.

Spoor, S. T., Stice, E., Bekker, M. H., Van Strien, T., Croon, M. A., & Van Heck, G. L. (2006). Relations between dietary restraint, depressive symptoms, and binge-eating: A longitudinal study. *International Journal of Eating Disorder, 39,* 700-707.

Steinberg, L., & Morris, A. S. (2001). Adolescent development. *Annual Review of Psychology, 52,* 83–110.

Stepp, S. D., Burke, J. D., Hipwell, A. E., & Loeber, R. (2012). Trajectories of attention deficit hyperactivity disorder and oppositional defiant disorder symptoms as precursors of borderline personality disorder symptoms in adolescent girls. *Journal of Abnormal Child Psychology, 40,* 7-20.

Stice, E, & Bearman, S. K. (2001). Body image and eating disturbances prospectively predict increases in depressive symptoms in adolescent girls: A growth curve analysis. *Developmental Psychology, 37,* 597–607.

Stice, E., Hayward, C., Cameron, R., Killen, J. D., & Taylor, C. B. (2000). Body image and eating related factors predict onset of depression in female adolescents: A longitudinal study. *Journal of Abnormal Psychology. 109,* 438–444.

Stice, E., Marti, C. N., Shaw, H., & Jaconis, M. (2011). An 8-year longitudinal study of the natural history of threshold, sub-threshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology, 118,* 587–597.

Stice, E., Mazotti, L., Weibel, D., & Agras, W.S. (2000). Dissonance prevention program

decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. *International Journal of Eating Disorders, 27,* 206-217.

Stice, E., & Whitenton, K. (2002). Risk factors for body dissatisfaction in adolescent girls: A longitudinal investigation. *Developmental Psychology, 38,* 669–678.

Stice E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin, 128,* 825–848.

Stice, E., Chase, A., Stormer, S., & Appel, A. (2001). A randomized trial of a dissonance-based eating disorder prevention program. *International Journal of Eating Disorders, 29,* 247_262.

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology, 110,* 124-35.

Stoolmiller, M., Kim, H. K., & Capaldi, D. M. (2005). The course of depressive symptoms in men from early adolescence to young adulthood: Identifying latent trajectories and early predictors. *Journal of Abnormal Psychology, 114,* 331-45.

Story, M., French, S. A., Resnick, M. D., & Blum, R. W. (1995). Ethnic/racial and socioeconomic differences in dieting behaviors and body image perceptions in adolescents. *The International Journal of Eating Disorders, 18,* 173–179.

Striegel-Moore, R. H., & Franko, D. L. (2008). Should binge eating disorder be included in the DSM-V? A critical review of the state of the evidence. *Annual Review of Clinical Psychology, 4,* 305-324.

Striegel-Moore, R. H., Rosselli, F., Perrin, N., DeBar, L., Wilson, G. T., May, A., & Kraemer, H. C. (2009). Gender difference in the prevalence of eating disorder symptoms. *International Journal of Eating Disorders, 42,* 471–474.

Ströhle, A., Stoy, M., Wrase, J., Schwarzer, S., Schlagenhauf, F., Huss, M.,… Heinz, A. (2008). Reward anticipation and outcomes in adult males with attention-deficit/hyperactivity disorder. *Neuroimage, 39,* 966-972.

Surman, C. B. H., Randall, E. T., & Biederman, J. (2006). Association between attention-deficit/hyperactivity disorder and bulimia nervosa: Analysis of 4 case-control studies. *Journal of Clinical Psychiatry, 67,* 351–354.

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry, 68,* 714–723.

Tanofsky-Kraff, M., Cohen, M. L., Yanovski, S. Z., Cox, C., Theim, K. R., Keil, M.,… Yanovski, J. A. (2006). A prospective study of psychological predictors of body fat gain among children at high risk for adult obesity. *Pediatrics, 117,* 1203-1209.

Tanofsky-Kraff, M., Faden, D., Yanovski, S. Z., Wilfley, D. E., & Yanovski, J.A. (2005). The perceived onset of dieting and loss of control eating behaviors in overweight children. *International Journal of Eating Disorders, 38,* 112–122.

Tanofsky-Kraff, M., McDuffie, J. R., Yanovski, S. Z., Kozlosky, M., Schvey, N. A., Shomaker, L. B.,…Yanovski, J. A. (2009). Laboratory assessment of the food intake of children and adolescents with loss of control eating. *American Journal Clinical Nutrition, 89,* 738-745.

Tanofsky-Kraff, M., Shomaker, L. B., Olsen, C., Roza, C. A., Wolkoff, L. E., Columbo, K. M.,…Yanovski, J. A. (2011). A prospective study of pediatric loss of control eating and psychological outcomes. *Journal of Abnormal Psychology, 120,* 108–118.

Tanofsky-Kraff, M., Shomaker, L. B., Stern, E. A., Miller, R., Sebring, N., Della Valle, D.,…Yanovski, J. A. (2012). Children's binge-eating and development of metabolic syndrome. *International Journal of Obesity, 36,* 956-962.

Tanofsky-Kraff, M., Goossens, L., Eddy, K. T., Ringham, R., Goldschmidt, A., Yanovski, S. Z.,… Yanovski, J. A. (2007). A multisite investigation of binge-eating behaviors in children and adolescents. *Journal of Consulting and Clinical Psychology, 75,* 901–913.

Tanofsky-Kraff, M., Marcus, M. D., Yanovski, S. Z., & Yanovski, J. A. (2008). Loss of control eating disorder in children age 12 years and younger: Proposed research criteria. *Eating Behav*iors, *9,* 360–365.

Tanofsky-Kraff, M., Morgan, C. M., Yanovski, S. Z., Marmarosh, C., Wilfley, D. E., & Yanovski, J. A. (2003). Comparison of assessments of children's eating-disordered behaviors by interview and questionnaire. *International Journal of Eating Disorders, 33,* 213–224.

Thompson, V. L. S. (1996). Perceived experiences of racism as stressful life events. *Community Mental Health Journal, 32,* 223-233.

Touchette, E., Henegar, A., Godart, N. T., Pryor, L., Falissard, B., Tremblay, R. E., & Côté, S. M. (2011). Subclinical eating disorders and their comorbidity with mood and anxiety disorders in adolescent girls. *Psychiatry Research, 185,* 185–192.Van

Eck, K., Markle, R. S., Dattilo, L., & Flory, K. (in press). Peer tolerance and conduct problems in college mediate the relation between ADHD symptoms and substance use. *The Psychology of Addictive Behaviors.*

Van Eck, K., Markle, R. S., & Flory, K. (2012). Do conduct problems and sensation seeking moderate the association between ADHD and three types of stimulant use in a college population? *Psychology of Addictive Behaviors, 26,* 939.

Van Lier, P. A. C., Muthén, B. O., Van Der Sar, R. M., & Crijnen, A. A. M. (2004). Preventing disruptive behavior in elementary schoolchildren: Impact of a universal classroom-based intervention. *Journal of Consulting and Clinical Psychology, 72,* 467–478.

Van Lier, P. A., van der Ende, J., Koot, H. M., & Verhulst, F. C. (2007). Which better predicts conduct problems? The relationship of trajectories of conduct problems with ODD and ADHD symptoms from childhood into adolescence. *Journal of Child Psychology and Psychiatry, 48,* 601-8.

Vaughn, A., Epstein, J. N., Rausch, J., Altaye, M., Langberg, L., Newcorn, J. H.,… Wigal, T. (2011). Relation between outcomes on a continuous performance test and ADHD symptoms over time. *Journal of Abnormal Child Psychology, 39,* 853–864.

Wade, T. J., Cairney, J., & Pevalin, D. J. (2002). Emergence of gender differences in depression during adolescence: National panel results from three countries. *Journal American Academy of Child and Adolescent Psychiatry, 41,* 190-198.

Waschbusch, D. A., Pelham, W. E., Jennings, J. R., Greiner, A. R., Tarter, R. E., & Moss, H. B. (2002). Reactive aggression in boys with disruptive behavior disorders: behavior, physiology, and affect. *Abnormal Child Psychology, 30,* 641-656.

Wear, R. W., & Pratz, O. (1987). Test-retest reliability for the eating disorder inventory. *International Journal of Eating Disorders, 6, 767–769.*

Webster-Stratton, C. (1984). Randomized trial of two parent-training programs for families with conduct-disordered children. *Journal of Consulting and Clinical Psychology, 52,* 666-678.

Wertheim, E. H., Koerner, J., & Paxton, S. J. (2001). Longitudinal predictors of restrictive eating and bulimic tendencies in three different age groups of adolescent girls. *Journal of Youth and Adolescence, 30,* 69-81.

Werthamer-Larsson, L. A., Kellam, S. G., & Wheeler, L. (1991). Effect of first grade classroom environment on shy behavior, aggressive behavior, and concentration problems. *American Journal of Community Psychology, 19,* 585- 602.

Wetter, E. K., & Hankin, B. L. (2009). Mediational pathways through which positive and negative emotionality contribute to anhedonic symptoms of depression: A prospective study of adolescents. *Journal of Abnormal Child Psychology, 37,* 507–520.

Whiteside, S. P., &, Lynam, D. R. (2001). The five factor model and impulsivity: using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30,* 669-689.

Wiesner, M., & Kim, H. K. (2006). Co-occurring delinquency and depressive symptoms of adolescent boys and girls: A dual trajectory modeling approach. *Developmental Psychology, 42*, 1220-35.

Wilfley, D. E., Friedman, M. A., Dounchis, J. Z., Stein, R. I., Welch, R. R., & Ball, S. A. (2000). Comorbid psychopathology in binge-eating disorder: relation to eating disorder severity at baseline and following treatment. *Journal Consulting and Clinical Psychology, 68*, 641–49.

Wonderlich, S. A., Gordon, K. H., Mitchell, J. E., Crosby, R. D., & Engel, S. G. (2009). The validity and clinical utility of binge-eating disorder. *International Journal of Eating Disorder, 42,* 687–705.

Appendix A:

Questionnaires

List of Questionnaires Proposed for Use in Analyses

- *Baltimore How I Feel-Young Child, Child Report*
- *Eating Disorders Inventory: Bulimia Scale*
- *Teacher Observation of Classroom Adaptation-Revised*

Measure 1. *Baltimore How I Feel – Young Child, Child Report**

During the past two weeks,

1	I liked the way I look*
2	I felt that I was good*
3	I felt like crying
4	I did not like myself
5	I felt that nothing made me happy anymore
6	I felt very unhappy
7	I felt sad
8	I had a lot of fun*
9	I felt like there was no use in really trying
10	I felt that I was a bad person
11	I felt that I might as well give up
12	I felt that I would have good times in the future*
13	I felt nothing would ever work out for me
14	I felt like killing myself
15	I felt that I would have more good times than bad times*
16	I felt grouchy
17	I felt that I was as good as other kids*
18	All I could see in the future were bad things not good things
19	I felt that it was my fault when bad things happened

Response Scale:

1 = Never,
2 = Once in a while,
3 = Sometimes,
4 = Most Times

Note. Only items pertaining to depression symptoms are listed.

* Denote items that are reverse scored.

Measure 2. *Eating Disorders Inventory: Bulimia Scale*

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1. I eat when I am upset.
2. I stuff myself with food.
3. I have gone on eating binges where I felt that I could not stop.
4. I have the thought of trying to vomit to lose weight. *
5. I think about bingeing or overeating.
6. I eat moderately in front of others and stuff myself when they are gone.
7. I eat or drink in secrecy.
8. In your own opinion, what is your current body weight? *

Response Scale:

Items: 1-7:

1 = Never,
2 = Rarely,
3 = Sometimes,
4 = Frequently,
5 = Usually,
6 = Always

Item 8:

1 = Very Underweight,
2 = Underweight,
3 = Average,
4 = Overweight,
5 = Very Overweight

*These items were not included in final analyses. For more information please refer to the factor analyses of this construct in the measures section of the methods chapter.

Measure 3. *Teacher Observation of Classroom Adaptation-Revised*

First to Third Grade Interview Items	Sixth to Ninth Grade Checklist Items
Concentration	
1. Completes assignments 2. Concentrates 3. Poor effort * 4. Works well alone 5. Pays attention 6. Learns up to ability 7. Eager to learn 8. Works hard 9. Stays on task 10. Easily distracted *	1. Completed assignments 2. Concentrated 3. Stayed on task 4. Was easily distracted * 5. Had difficulty organizing tasks and activities *
Hyperactivity	
1. Can't sit still 2. Out of seat/runs around 3. Always on the go/driven by a motor	1. Can't sit still 2. Fidgeted and/or squirmed a lot 3. Always on the go/driven by a motor
Impulsivity	
1. Waits for turn * 2. Interrupts or intrudes on others 3. Blurts out answer before item is completed	1. Waits for turn * 2. Interrupts or intrudes on others 3. Blurted out answer before question was complete

Response Scale:

1 = Almost Never
2 = Rarely
3 = Sometimes
4 = Often
5 = Very Often
6 = Always

*Denotes items that are reverse scored. After creating a summary variable, the direction of Concentration was reversed to be consistent with Hyperactivity and Impulsivity before being combined with as one construct of ADHD symptoms.