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Developmental associations of self-reported body mass and depressive symptoms: A longitudinal examination of the transition from adolescence to young adulthood

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A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Epidemiology and Biostatistics
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DEVELOPMENTAL ASSOCIATIONS OF SELF-REPORTED BODY MASS AND
DEPRESSIVE SYMPTOMS: A LONGITUDINAL EXAMINATION OF THE
TRANSITION FROM ADOLESCENCE TO YOUNG ADULTHOOD

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Abstract

The purpose of this thesis was to examine the longitudinal associations between body mass and depressive symptoms as adolescents transition into young adults, using secondary data analysis of the National Longitudinal Survey of Children and Youth. Adolescents (N = 1,895) were followed across five ages, between ages 17 and 25. Body mass and depressive symptoms were self-reported at each age. Latent growth modelling was used for all analyses. Results showed that the trajectory of body mass increased over time, while the trajectory of depressive symptoms decreased over time, for both males and females. Adolescent females with higher initial body mass levels reported a slower decrease in depressive symptoms over time. Conversely, adolescent males with higher initial depressive symptoms reported a slower increase in body mass over time. Public health implications, future research initiatives, and conclusions are further discussed.

Keywords

Acknowledgments

Table of Contents

List of Tables

List of Figures

List of Appendices

Chapter 1

1   Background

1.1   Body mass and depressive symptoms

Among Canadian adolescents, both body mass and depressive symptoms have become increasingly significant public health concerns, as respective increases in these health outcomes place individuals at heightened risk of becoming overweight/obese and developing clinically significant depressive symptoms[1] (Roberts, Lewinsohn, and Seeley, 1991; Shields, 2006). Over the past three and a half decades, the prevalence of obesity has increased substantially in economically developed countries such as Canada (Lobstein, Baur, & Uauy, 2004). Particularly alarming is the rate of increase in adolescents and young adults. According to a nationally representative sample of Canadians, the obesity rate for adolescents aged 12-17 tripled between 1978 and 2004, from 3% to 9% (Shields, 2006). Although obesity is increasing in all age groups, young adults aged 18 to 29 are experiencing the highest rate of increase (Mokdad, Serdula, Dietz, et al., 1999). In Canada, the obesity rate for 25-34 year olds more than doubled from 1978 (9%) to 2004 (21%; Tjepkema, 2006). In a 2007 national survey of Canadians, 6.4% of 18-19 year olds and 11% of 20-24 year olds were classified as obese (Shields, 2008). The obesity rate for 18-29 year olds in the United States increased from 7.1% in 1991 to 12.1% in 1998 (Mokdad et al., 1999). The increased prevalence of obesity in adolescence and young adulthood has been linked to the concurrent rise of many physical health problems normally associated with adults, including type 2 diabetes,

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[1] Note, there is a difference between depressive symptoms and clinically significant depressive symptoms. Depressive symptoms are defined as any one or more symptoms identified in validated depressive symptom scale(s) (e.g., decreased enjoyment of pleasurable activities, low self-esteem, hopelessness, apathy, difficulty sleeping, etc.,), with higher scores (across the entire continuum of scores) indicating higher depressive symptoms (Radloff, 1977). Clinically significant depressive symptoms are defined as an established cut point in depressive symptom scales used to distinguish a sufficient amount of depressive symptomology to closely resemble clinically depressed patients in treatment (Berkman, Berkman, Kasl, et al., 1986). For example, using the Center for Epidemiological Studies- Depression Scale (CES-D; Radloff, 1977), a cut point of greater than or equal to 22 for adolescent males and 24 for adolescent females is suggested to identify cases of Diagnostic and Statistical Manual III- Revised (DSM-III-R) major depression and dysthymia (Roberts, Lewinsohn, and Seeley, 1991).

cardiovascular disease, hypertension and stroke, and certain forms of cancer (World Health Organization [WHO], 2003).

In addition to increases in body mass, depressive symptoms during adolescence are also common (Birmaher, Ryan, Williamson, et al., 1996). In fact, late adolescence has been reported to be the period of the lifespan with the greatest experience of depressive symptoms (Wight, Sepúlveda, & Anashensel, 2004). Following a peak at about age 16 to 17 years of age, depressive symptoms tend to gradually decline into young adulthood (Ge, Natsuaki, & Conger, 2006; Wight et al., 2004). Depressive symptoms during adolescence and young adulthood include decreased enjoyment of pleasurable activities, low self-esteem, hopelessness, apathy, and difficulty sleeping (Radloff, 1977). At particular cutoffs, these symptoms have been shown to predict the development of major depression disorder (Georgiades, Lewinsohn, Monroe, & Seeley, 2006), which in turn has been associated with increased risk of coronary heart disease, myocardial infarction, and mortality (Birmaher, Brent, Bernet, et al., 2007; Birmaher, Williamson, Dahl, et al., 2004; Brent, 2006; Faith, Matz, & Jorge, 2002; Williamson, Birmaher, Brent, et al., 2000).

1.2   Adolescence and young adulthood

Keeping these particular health outcomes in mind, there has recently been growing awareness of the importance of the life course perspective in public health research. Increasingly, evidence has shown that factors operating at special critical periods earlier in life may influence health at later periods in the life course (Osler, 2006). By utilizing a life course perspective, the influence of a risk factor in one stage of life can be examined as individuals proceed into the next phase of life (Merten, Wichrama, & Williams, 2008).

Adolescence and young adulthood are both particularly important developmental periods to consider when examining the course and co-occurrence of both body mass and depressive symptoms. Adolescence is a sensitive period of development, during which the influence of positive or negative experiences may have a lasting effect throughout the lifespan (Bornstein, 1989). Developmental psychologists consider adolescence an important life-stage to study because of the critical changes that mark this period of

development, especially as a result of puberty (Dahl, 2004). More specifically, puberty is linked to physical changes in body size (Dahl, 2004), making adolescence a developmental period for increased weight, as well as the onset and persistence of obesity (Dietz, 1994). In addition to physical changes in body size, adolescence is also marked as a period of heightened emotions (Dahl, 2004). Because of this natural tendency toward high-intensity feelings, it is not surprising that there is an increase in the onset of internalizing problems, such as depressive symptoms, within this age group (Elliott, Huizinga, & Menard, 1989; Kandel & Davies, 1982).

Young adulthood, on the other hand, is a developmental stage characterized by increased educational endeavours, establishing employment, and establishing and maintaining interpersonal and intimate relationships (Merten et al., 2008). Young adulthood marks the point in time where major transitional changes occur. For example, across the lifespan young adulthood is the period with the highest level of fertility, the highest marriage rates, the highest rates for leaving school, and the highest residential migration rates (Rindfuss, 1991). For the majority of young adults, major decisions and role changes occur in most areas of life and are occurring in a relatively short and overlapping period, making this time in the life course particularly dynamic (Rindfuss, 1991). Individuals in this life stage have much more independence over important life choices, such as physical activity, dietary intake, and participation in healthy or unhealthy behaviors (e.g., binge drinking, smoking; Kemper, Post, Twisk, & van Mechelen, 1999), all of which affect physical health. With regards to mental health, young adults also acquire a significantly greater capacity for integrating thought and emotion, allowing for greater emotional regulation than they had as adolescents.

In addition to period-specific changes that occur during adolescence and young adulthood, large and complex changes also occur as adolescents *transition* into young adults. Increases in body mass among adolescents and young adults in particular is a major health concern because overweight and obese conditions in these developmental stages tend to persist into adulthood (Whitaker, Wright, Pepe, Seidal, & Dietz, 1997). For example, researchers have reported that more than 70% of obese adolescents become obese adults (Guo, Roche, Chumlea, Gardner, & Siervogel, 1994). In addition, daily

physical activity tends to decrease from adolescence into young adulthood, whereas fat mass gradually increases (Kemper et al., 1999). In a longitudinal study, Kemper and colleagues (1999), reported that while less than 10% of males were classified as obese at age 13-16, by the time these individuals were 32 years old, this percentage doubled to 20%. For females, increases in obesity went from 10% at ages 12-16 to 30% at age 32. Overall, the increase in body mass as well as the decrease in depressive symptoms that occur as adolescents become young adults, illustrate that the transition from adolescence to young adulthood with respect to these health outcomes is an important developmental period on which to focus academic attention (Sampson & Mrazek, 2001).

1.3 Objective and rationale

While the links between body mass and physical health have been established, the same is not true for relations with psychological health indices such as depressive symptoms (Hill, 2005). Recently however, the high prevalence of obesity and depression has led researchers to examine potential associations between the two. Although there is some research on the cross-sectional relationship between body mass and depressive symptoms (Baumeister & Härter, 2007; Faith et al., 2002; John, Meyer, Hapke, Rumpf, & Schumann, 2004), it is still unclear how these two health outcomes interact over time (Faith, Butryn, Wadden et al., 2011; Liem, Sauer, Oldehinkel, & Stolk, 2008).

In addition, there is no consistency in the literature regarding the role of sex on these associations, especially during the transition from adolescence to young adulthood. These are important research questions to address for two primary reasons. First, body mass and depressive symptoms are both dynamic developmental processes that change across the lifespan (Needham, Epel, Adler, & Kiefe, 2010), and thus should be examined prospectively within a developmental framework in order to identify important changes. Second, body mass (Shields, 2006) and depressive symptoms (Kuczmarski, Ogden, Guo, et al., 2002; Locke & Newcomb, 2001) are both differentially distributed based on sex, thus longitudinal associations between the two may drastically vary for males and females.

Studying how body mass and depressive symptoms independently change, as well as how they are associated over time, will aid in understanding the course and consequences of both health outcomes. Furthermore, an important research and public health question to address is whether there is a causal relation between both body mass and depressive symptoms in the transition from adolescence to young adulthood, and whether these associations are similar for males and females. In a recent review, Liem and colleagues (2008) suggested that identifying treatable predictors such as depressive symptoms could lead to better outcomes for preventing and treating overweight and obesity later in life. In similar respects, psychological issues may be preventable by addressing physical health and body weight issues early on. Understanding the sex differences in these associations may help determine when, and to whom, resources should be targeted at to effectively prevent negative health outcomes later in life. Overall, given the toll that both body mass and depressive symptoms have on an individual's quality of life and functioning, as well as the persistence and lifecourse stability both tend to have into adulthood (Bradley, Houts, Nader, et al., 2008), it is important to explore the potential links between them and target public health prevention strategies to help mitigate future adverse health outcomes (Calamaro & Waite, 2009).

With these public health implications in mind, the objective of this thesis is examine the longitudinal associations between body mass and depressive symptoms during the transition from adolescence to young adulthood, and to investigate if and how these associations differ for males and females. By examining the relationship between body mass and depressive symptoms over time, we can determine whether longitudinal associations exist, as well as whether one health outcome better predicts the other. As previously mentioned, understanding the longitudinal associations between body mass and depressive symptoms may be an important step in identifying risk factors to target in prevention strategies aimed at increasing the physical and psychological well-being of adolescents and young adults (Clark, Haines, Head, et al., 2007).

Few prospective studies have effectively evaluated this research question, and even fewer have focused on the developmentally important transition from adolescence to young adulthood. That is, while associations in adulthood have been heavily

researched (Bjerkeset, Romundstad, Evans, et al., 2008; Forman-Hoffman, Yankey, Hillis, Wallace, & Wolinsky, 2007; Gariepy, Wang, Lesage, & Schmitz, 2010; Needham et al., 2010; Roberts, Deleger, Strawbridge, & Kaplan, 2003), very little is known about the effect weight status has on the decrease in depressive symptoms across late adolescence and young adulthood, as well as the effect depressive symptoms have on the increase in weight across late adolescence and young adulthood. Thus, there is a need for well-formulated longitudinal research to examine the predictive direction of these associations across these developmental periods.

The rest of this thesis will be organized in the following order: groups of theoretical models accounting for the prospective association between body mass and depressive symptoms, longitudinal research on the association between body mass and depressive symptoms, current plan of study, methods, results, and discussion. First, two major groups of theoretical models accounting for the prospective association between body mass and depressive symptoms will be presented with a focus on the adolescent and young adulthood developmental periods. Next, a literature review covering longitudinal research that has assessed the directions of association between body mass and depressive symptoms will be presented, as well as a summary of the limitations with current research in this area. Following this literature review and summary, a plan of study, along with specific objectives and hypotheses will be presented. Next, methodological topics covering the data source, variables used, and analyses will be discussed. Finally, the results will be presented, along with a discussion of findings and recommendations for future research. Important sex differences in the association between body mass and depressive symptoms will be highlighted throughout all sections.

Chapter 2

2 Literature review

2.1 Theoretical models accounting for the prospective association between body mass and depressive symptoms

The purpose of this literature review is to examine the empirical evidence assessing the direction of association between body mass and depressive symptoms in adolescents and young adults. There are two major groups of theoretical models that explain the prospective association between body mass and depressive symptoms. The first group describes how increases in body mass may lead to increases in depressive symptoms over time. The second group describes how increases in depressive symptoms may lead to increases in body mass over time. Each of these models will be explained in detail below.

2.1.1 Body mass affecting depressive symptoms

There are many possible causal pathways, both psychologically and physically, that may lead adolescents and young adults with higher body mass to develop increases in depressive symptoms over time. These include the internalization of biased attitudes towards weight, weight-teasing, neurobiological causal pathways, and functional impairment. Each of these causal pathways will be explained in greater detail below.

2.1.1.1 Internalization of biased attitudes

According to the reflected self-appraisal hypothesis, an individual's self-concept (i.e., his/her internalized view of himself/herself) is socially constructed through self-appraisals and the judgments of others (Hayes & Ross, 1986; Ross, 1994). According to the social comparison theory, individuals engage in a continuous process whereby they compare themselves to people with whom they believe possess desirable traits (Festinger, 1954). Thus, the thoughts and feelings individuals have about their body are constantly affected by social influences and the comparisons they make with others.

Since overweight status is perceived as a less attractive and more stigmatizing characteristic in Western cultures (Hill, 2005), individuals with higher body mass may internalize the negative attitudes of society towards their weight, resulting in lower self-esteem and higher psychological distress (Ross, 1994). Studies on stereotyping have shown that obese individuals are viewed as being undisciplined, inactive, and unappealing (Hill, 2005). These stereotypes may be especially prominent during adolescence (Bradley et al., 2008) given the dramatic changes in cognitive functioning and physical development. In fact, these negative views tend to be held by people who are younger (Hill, 2005), placing heavier adolescents and young adults at a higher risk of being stereotyped by their peers. Thus, body mass may affect depressive symptoms through an internalization of biased attitudes from peer, family, and society towards the individual.

Internalization of biased attitudes may be more prominent in females compared to males (Wadden & Stunkard, 1987), especially during adolescence and young adulthood. For adolescent boys, most of the weight gain as a result of puberty is explained by increases in muscle tissue, whereas weight gain for adolescent girls is mostly explained by significant fat mass deposition in the abdomen, buttock, and thigh regions (Tanner, 1989). Overall, fat mass increase during this developmental period is significantly greater in females than males (Xie, Unger, Gallager, et al., 2010). Females also tend to place a great deal of subjective importance on their physical appearance (Wadden & Stunkard, 1987). This increase in fat mass, paired with high levels of self-consciousness and embarrassment about the physical changes that accompany puberty, may place adolescent girls at a higher risk for the development of body image issues as well as increases in depressive symptoms over time (Wadden & Stunkard, 1987).

2.1.1.2 Weight-teasing

Weight teasing toward heavier individuals has been reported among health professionals, peers, and family members (Cash, 1995; Neumark-Sztainer, Story, & Faibisch, 1998). In fact, 'fat-teasing' is reported by one in seven adolescents (Hill, 2005). One study reported that, for females, weight teasing in middle adolescence was associated with lower self-esteem, lower body satisfaction, and marginally higher

depressive symptoms in young adulthood (Eisenberg, Neumark-Sztainer, Haines, & Wall, 2006). For males however, weight-teasing in middle adolescence did not prospectively predict increases in depressive symptoms during young adulthood, although it did predict lower body satisfaction. Thus, depressive symptoms may develop through poorer peer relationships and an increased dissatisfaction with appearance. The resulting poor peer relationships are also associated with poorer mental health (Hill, 2005), making it more likely for adolescents to develop increases in depressive symptoms over time. Thus, increases in body mass may affect depressive symptoms through the stigma and social prejudices against these adolescents (Needham & Crosnoe, 2005).

These associations may be stronger for females than for males. Compared to boys, girls are stigmatized significantly more often for their body mass, and they face more teasing, bullying and marginalization in social relationships (Tang-Peronard, & Heitmann, 2008).

2.1.1.3   Neurobiological causal pathways

In addition to social and psychological influences, physical health pathways exist that may also explain the association between increases in body mass and increases in depressive symptoms over time. For example, one such pathway is inflammation. Increases in body mass can be regarded as an inflammatory state since weight gain has been shown to activate inflammatory pathways (Emery, Fondow, Schneider, et al., 2007; Shoelson, Herrero, & Naaz, 2007). This inflammation, in turn, has been associated with an increase in depressive symptoms (Bremmer, Beekman, Deeg, et al., 2008; Milaneschi, Corsi, Pennix, et al., 2009; Vaccarino, Johnson, Sheps, et al., 2007). In extreme cases of increased body mass, obesity may involve hypothalamic-pituitary-adrenal (HPA) axis dysregulation (Pasquali, Vicennati, Cacciari, & Pagotto, 2006; Walker, 2001), which is known to be associated with depressive symptoms (Belanoff, Kalehzan, Sund, Fleming Ficek, & Schatzberg, 2001; Holsboer, 2000). Using yet another example, obesity may lead to increases in depressive symptoms through the associated increased risk of diabetes mellitus and increased insulin resistance (Lee, Lee, Ser, Chen, & Chen, 2008), both of which may result in brain alterations (Huber, 2008) and increased risk for depressive symptoms (Ajilore, Haroon, Kumaran, et al., 2007).

2.1.1.4   Functional impairment

Functional impairment may also explain the association between higher body mass and depressive symptoms. Individuals with a high body mass who have greater difficulties with mobility and physical activity may be more likely to experience depressive symptoms over time (Bornstein, Schuppenies, Wong, & Licinio, 2006; Lago, Dieguez, Gomez-Reino, & Gualillo, 2007). For example, prospective studies have found an inverse association between physical activity and the development of depressive symptoms (Brown, Ford, Burton, Marshall, & Dobson, 2005; Camacho, Roberts, Lazarus, Kaplan, & Cohen, 1991; Farmer, Locke, Moscicki, et al., 1988; Lampinen, Heikkinen, Ruoppila, 2000; Paffenbarger, Lee, & Leung, 1994; Strawbridge, Deleger, Roberts, & Kaplan, 2002).

2.1.1.5   Summary

Overall, increases in body mass may affect depressive symptoms through many causal pathways, including an internalization of biased attitudes, weight-teasing, neurobiological causal pathways, and functional impairment. This prospective association may be stronger for female youth compared to male youth because females place greater importance on thinness as a popular ideal for feminine beauty (Wadden & Stunkard, 1987). Girls are also more likely than boys to ruminate about and internalize negative feelings associated with their weight status (Fredrickson & Roberts, 1997). Finally, weight-teasing is more likely to occur for adolescent girls than boys (Tang-Peronard, & Heitmann, 2008). These factors may all translate to stronger prospective associations between higher body mass and increases in depressive symptoms for females compared to males.

2.1.2   Depressive symptoms affecting body mass

In addition to higher body mass affecting depressive symptoms over time, depressive symptoms may also lead to changes in body mass across adolescence and young adulthood. The causal pathways through which this prospective association may be explained includes neurobiological causal pathways and affect regulation, and

impaired sleep quality and physical inactivity. Each causal pathway will be explained in greater detail below.

2.1.2.1   Neurobiological causal pathways and affect-regulation

Depressive symptoms may lead to increases in body mass through various neurobiological causal pathways. For example, increased levels of cortisol are observed during periods of stress and depression, which can in turn induce abdominal obesity (Björntorp, 2001). Furthermore, serotonin dysregulation may characterize both depressive symptoms as well as the risk for obesity, such that individuals with disturbances in serotonin regulation may attempt to regulate their serotonin levels by consuming an excessive amount of carbohydrate-rich foods rich (Wurtman, Wurtmen, Mark, et al., 1985). Similarly, according to the affect-regulation model, individuals with increased depressive symptoms may be more likely to intake food excessively as an attempt to alleviate their negative affect (Liem et al., 2008). Coping mechanisms for depressive symptoms may also include consuming substances, such as cigarettes (Hooshmand, Willoughby, & Good, 2011), that are associated with weight gain (Goodman & Whitaker, 2002), as well as unhealthy eating (Laitinen, Power, Ek, Sovio, Järvelin, & 2002). In sum, these unhealthy behaviors may explain the association between depressive symptoms and weight gain (Greenfield, Rehm, & Rogers, 2002).

2.1.2.2   Sleep quality and physical inactivity

Impaired sleep quality is another potential causal pathway, as poor sleep quality is common in individuals with clinically significant depressive symptoms (Lauer, Wiegand, & Krieg, 1992; Riemann, Berger, & Voderholzer, 2001), and inadequate sleep is a risk factor for weight gain (Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005). Depressive symptoms are also associated with lethargy, social withdrawal, and physical inactivity (Faith et al., 2011), and could therefore lead to weight gain; although, many authors report that adjusting for physical activity did not change the positive associations they initially found between depressive symptoms and body mass (Goodman & Whitaker, 2002; Needham & Crosnoe, 2005).

2.1.2.3   Summary

Overall, depressive symptoms may affect weight gain through a variety of causal pathways, including neurobiological causal pathways and affect-regulation, and impaired sleep quality and physical inactivity. In contrast to the sex differences for the prospective associations between body mass and subsequent depressive symptoms, there is no theoretical rationale that prospective associations between depressive symptoms and subsequent increases in body mass may be stronger for females compared to males.

2.2   Longitudinal research on the association between body mass and depressive symptoms

Given this understanding of the groups of theoretical models, the longitudinal research assessing these associations will be examined next. The three main objectives of this section are to: 1) present a summary of all studies that investigated this research question; 2) highlight the conceptual, methodological, and analytical differences among studies that may contribute to the disparity in the current state of literature; and 3) identify the major limitations of previous research that will be addressed in the present study.

Several exclusion criteria were applied when selecting studies to present in the literature review. First, since population-based associations examining change are of interest, the following literature review excludes clinical studies and studies that used clinical samples. Second, since depressive symptoms are of primary interest in the current study, the literature review excludes studies that used a diagnosis of depression. Third, only longitudinal studies in which the same participants were followed for two or more cycles of data collection will be examined, and studies that did not measure changing levels in the outcome of interest will be excluded (although these studies, when appropriate, are included in the Appendix, for more comprehensive coverage of the literature). Finally, in order to fully capture research on adolescents and/or young adults, the present literature review focuses solely on studies in which participants were younger than 30 years of age at the first cycle of data collection and older than 12 years of age at the last cycle of data collection (Rindfuss, 1991).

The following section will be divided into several component sections. Studies will first be organized into the direction of association they aimed to assess (i.e., body mass affecting depressive symptoms, and vice versa). Studies within each direction of association will be further organized into two subgroups: those that examined change across two points, and those that examined change across more than two points by using statistical techniques for longitudinal data (e.g., survival analysis, growth curve modeling). Summaries of research studies will then be presented in order to highlight the current disparities in the literature and provide reasoning, where possible, to explain these disparities. Following this literature review and summary, an overall rationale and objective for the direction of research in this area will be presented, as well as the limitations of past research that have prevented valid conclusions to be drawn concerning the direction of association between body mass and depressive symptoms. This section will conclude with a proposed model for examining the direction of these associations over time within the adolescent and young adult developmental periods.

Before describing studies, it is important to recognize that there have been vast differences in how depressive symptoms have been operationalized and measured in previous research. Depressive symptoms have been measured using a wide array of symptom scales, including the Center for Epidemiological Studies- Depression Scale (CESD-D; Radloff, 1977), the Children's Depression Inventory (CDI; Kovacs & Beck, 1977), the Hopkins Symptom Checklist (HSCL; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 2007), the Mood and Feeling Questionnaire (MFQ; Angold, Costello, Messer, et al., 1995), and Minnesota Multiphasic Inventory- Obvious Depression Scale (MMPI-ODS; Greene, 1991). In addition to the differences in scales used across studies, there were also several different versions of the same scale used across studies. With respect to the CES-D for example, different studies have utilized the CES-D-5 (Xie et al., 2010), the CES-D-9 (Merten et al., 2008), the CES-D-10 (Ball, Burton, & Brown, 2009), and the Modified CES-D-18 (Goodman & Whitaker, 2002).

In addition, while some studies measured depressive symptoms using a continuous measure of the scale (Boutelle, Hannan, Fulkerson, Crow, & Stice, 2010; Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Merten et al., 2008;

Pine, Cohen, Brook, & Coplan, 1997; Stice, Presnell, Shaw, & Rohde, 2005; Tanofsky-Kraff, Cohen, Yanovski, et al., 2006; Xie et al., 2010), others used cutoffs to identify clinically significant depressive symptoms (Ball et al., 2009; Clark et al., 2007; Goodman & Whitaker, 2002; Jansen, van de Looij-Jansen, de Wilde, & Brug, 2008; Rhew, Richardson, Lymp, et al., 2008), or both (Anderson, Murray, Johnson, et al., 2011; Barefoot, Heitmann, Helms, et al., 1998). All of these different operationalizations and measurements of depressive symptoms will be included for the purposes of presenting a comprehensive literature review, as well as to demonstrate the variability that currently exists across studies.

2.2.1 Body mass affecting depressive symptoms

Several studies have investigated whether increases in body mass affect subsequent depressive symptoms in adolescents and young adults. These studies are summarized below, as well as presented in greater detail in the Appendix. Studies are first organized into whether they measured change in depressive symptoms across two time points or more than two time points, then further organized into whether they reported a prospective association or not.

2.2.1.1 Measuring change in depressive symptoms across two time points

The majority of studies have measured change across two time points to estimate the effect of body mass on subsequent depressive symptoms, controlling for prior levels of depressive symptoms and other covariates. The following four studies reported significant associations. First, Anderson and colleagues examined these associations in a cohort of adolescent females from public schools across the USA at two time points: grades six and eight (Anderson et al., 2011). The authors operationalized clinically significant depressive symptoms as a CES-D score of 24 and over, and categorized BMI into obese and non-obese using the Center for Disease Control (CDC) reference guide. The authors found a significant interaction between race and body mass, such that obesity status predicted subsequent clinically significant depressive symptoms only for White females. Associations were not significant for Hispanics, or for non-Hispanic Blacks. The

authors reported consistent findings when they repeated their analyses with depressive symptoms (using continuous CES-D scores) and body mass (using continuous BMI z-scores).

Ball and colleagues examined these associations in a cohort of young women in Australia who were 22-27 years old at baseline, and re-examined them three years later (Ball et al., 2009). The authors operationalized clinically significant depressive symptoms as a CES-D-10 score of 10 or more, and used the World Health Organization (WHO) classifications to categorize BMI (i.e., into underweight, overweight, normal weight, and obese categories). They found that overweight and obese females, compared with normal weight females, had a greater risk for clinically significant depressive symptoms at follow-up.

Using a nationally representative cohort from the United States, Merten and colleagues (2008) examined whether adolescent obesity at baseline was associated with increases in depressive symptoms six years later. The authors used continuous scores from the CES-D-9 as a measure of depressive symptoms, and categorized BMI into obese and not obese groups using the CDC reference guide. They reported a significant sex interaction, such that obesity for female adolescents, but not for male adolescents, was associated with higher depressive symptoms in young adulthood.

Xie and colleagues (2010) used multiple group structural equation models to compare associations of obese status on subsequent depressive symptoms in a community sample of Asian and Hispanic middle school students in Los Angeles followed over one year. The authors used continuous scores from the CES-D-5 as a measure of depressive symptoms, and categorized BMI into overweight and not overweight groups using the CDC reference guide. They examined associations separately for males and females, for Asians and Hispanics, and for accultured and not accultured adolescents. They found that Asian males who were overweight at baseline had higher depressive symptoms at follow-up than non-overweight Asian males. Also, males with low acculturation who were overweight at baseline had higher subsequent depressive symptoms compared to their not

overweight counterparts. The authors did not find any other significant associations (i.e., for girls, high accultured boys, or Hispanic boys).

Only three studies that measured change in depressive symptoms across two time points did not report any significant findings. First, Clark and colleagues (2006) examined associations in a representative sample of adolescents (ages 11-14; re-examined two years later) attending schools in East London. The authors identified clinically significant depressive symptoms as a score of eight or higher, using the Short Mood and Feeling Questionnaire (SMFQ; Angold et al., 1995), and used the United Kingdom reference curve classifications to categorize BMI into overweight (i.e., having a BMI greater than the 85th percentile), and obese (i.e., having a BMI greater than the 95th percentile). They found that neither overweight nor obese status predicted subsequent clinically significant depressive symptoms.

Using a nationally representative cohort of US adolescents, Goodman and Whitaker (2002) examined the association between obesity in 7th-12th grade adolescents and clinically significant depressive symptoms one year later. The authors categorized clinically significant depressive symptoms as a Modified CES-D-18 score of 24 or more for females, and 22 or more for males, and used the CDC reference classifications for classifying BMI into obese and not obese categories. They found that being obese at baseline did not predict clinically significant depressive symptoms at follow-up.

Rhew and colleagues examined whether overweight status in sixth grade predicted depressive symptoms one year later in a cohort of middle school students in Seattle, Washington (Rhew et al., 2008). The authors examined continuous log-transformed depressive symptoms, measured using the MFQ (Angold et al., 1995), and categorized BMI into obese, overweight, and not overweight groups using CDC reference guides. They found that neither overweight nor obese status at baseline predicted depressive symptom scores at follow-up.

2.2.1.2   Measuring change in depressive symptoms using more than two time points

Only one study used more complex longitudinal analyses in order to examine changes across more than two time points. Using repeated measures multivariate logistic regression over four annual cycles of data, Boutelle and colleagues examined the influence of overweight or obesity status the previous year on depressive symptoms the subsequent year, in adolescent females from public and private schools in Austin, Texas (Boutelle et al., 2010). The authors examined continuous depressive symptoms using an average of past year severity ratings for each symptom in the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Chambers, Puig-Antich, Hirsch, et al., 1985). Response options for each symptom ranged from 1 (not at all) to 4 (severe). BMI was categorized into obese, overweight, and not overweight groups using CDC reference guides. The authors reported a significant positive effect of obesity (but not overweight) on subsequent depressive symptoms.

2.2.1.3   Summary of studies examining body mass affecting depressive symptoms

In summary, these studies all aimed to investigate the theoretical framework that body mass leads to increases in depressive symptoms through the previously mentioned causal pathways (i.e., internalization of biased attitudes, weight-teasing, neurobiological causal pathways, and functional impairment). Of the eight studies reviewed, almost half did not report finding any sort of an association between baseline body mass and subsequent depressive symptoms. This discrepancy in research findings highlights the high level of inconsistency in research. One explanation for this inconsistency may be that inconsistent findings reflect an inconsistent phenomenon- that is, that body mass may predict depressive symptoms for some individuals but not others (Friedman & Brownell, 1995). Although this may be the case, it is difficult to make this conclusion because the high level of variation among current studies makes cross-study comparisons challenging.

These variations occur at the conceptual, methodological, and analytical levels, and may explain a great deal of the disparity in results for studies investigating body

mass as a predictor of subsequent depressive symptoms. Conceptual differences across studies exist in terms of the developmental periods studied, the operationalization of body mass and body mass comparison groups, the operationalization of depressive symptoms, and differences in the populations studied.

With respect to the first conceptual variation, the majority of studies did not examine the developmental transition from adolescence to young adulthood, but instead focused solely on changes within adolescence (Anderson et al., 2011; Boutelle et al., 2010; Clark et al., 2007; Rhew et al., 2008; Xie et al., 2010), or young adulthood (Ball et al., 2009). Although all studies operationalized body mass using categorizations, one study used a continuous measure of BMI in addition to categorization (Anderson et al., 2011).

There were also large variations among studies in operationalizing body mass comparison groups. For example, some studies compared obese adolescents to not obese adolescents, where the not obese group included individuals classified as overweight, normal weight, or underweight (Anderson et al., 2011; Goodman & Whitaker, 2002; Merten et al., 2008). Other studies compared obese and/or overweight groups to not overweight groups, where the not overweight group included individuals classified as either normal weight or underweight (Boutelle et al., 2010; Clark et al., 2007; Rhew et al., 2008; Xie et al., 2010). Of note, only one study compared obese, overweight, and underweight groups separately with normal weight groups (Ball et al., 2009).

As previously mentioned, some studies operationalized depressive symptoms into categories by establishing clinically significant cutoffs from scaled continuous measures (Ball et al., 2009; Clark et al., 2007; Goodman & Whitaker, 2002). Other studies, however, operationalized depressive symptoms as a continuous scale, with higher scores indicating more depressive symptoms (Boutelle et al., 2010; Merten et al., 2008; Rhew et al., 2008; Xie et al., 2010). One study used both a continuous and categorized operationalization of depressive symptoms (Anderson et al., 2011).

Studies also differed in the composition of the populations they examined. While most studies sampled both males and females (Clark et al., 2007; Goodman & Whitaker,

2002; Merten et al., 2008; Rhew et al., 2008; Xie et al., 2010), some sampled females only (Anderson et al., 2011; Ball et al., 2009; Boutelle et al., 2010). Also, some studies used at-risk samples (Rhew et al., 2008), samples with one predominant race/ethnicity (Xie et al., 2010), or samples from outside of North America (Ball et al., 2009; Clark et al., 2007).

Methodologically, there are large variations among the length of intervals, source of height and weight measurements, reference tools for establishing body mass cutoffs, and tools for measuring depressive symptoms. In particular, studies differed on whether they examined associations over annual (Boutelle et al., 2010; Goodman et al., 2002; Rhew et al., 2008; Xie et al., 2010), biennial (Anderson et al., 2011; Clark et al., 2007), or longer (Ball et al., 2009; Merten et al., 2008) intervals. Additionally, when calculating BMI, some studies used measured height and weight (Anderson et al., 2011; Boutelle et al., 2010; Clark et al., 2007; Xie et al., 2010), while others used self-reported height and weight (Ball et al., 2009; Goodman & Whitaker, 2002; Merten et al., 2008).

With regards to reference tools for establishing body mass cutoffs, studies ranged in their use of categorizations based on CDC (Anderson et al., 2011; Boutelle et al., 2010; Goodman & Whitaker, 2002; Merten et al., 2008; Rhew et al., 2008; Xie et al., 2010), WHO (Ball et al., 2009), or other reference guides (Clark et al., 2007). Finally, every study used a slightly different measurement tool to assess depressive symptoms, ranging from K-SADS (Boutelle et al., 2010), MFQ (Rhew et al., 2008) or SMFQ (Clark et al., 2007), and different versions of the CES-D (Anderson et al., 2011; Ball et al., 2009; Merten et al., 2008; Goodman & Whitaker, 2002; Xie et al., 2010).

Analytically, studies varied in the number of measurement points used for analyses. While one study examined associations over three or more cycles (Boutelle et al., 2010), the majority of research in this area focused on assessing only two points (Anderson et al., 2011; Ball et al., 2009; Clark et al., 2007; Goodman & Whitaker, 2002; Merten et al., 2008; Rhew et al., 2008; Xie et al., 2010). Studies also varied in the statistical techniques they used to assess whether body mass predicted subsequent depressive symptoms, with some using logistic regression techniques (Ball et al., 2009;

Clark et al., 2007; Goodman & Whitaker, 2002), and others using linear regression techniques (Boutelle et al., 2010; Merten et al., 2008; Rhew et al., 2008; Xie et al., 2010).

Finally, there have been large discrepancies between each study with respect to the covariates controlled for during analyses. The table in the Appendix illustrates this discrepancy by listing all covariates considered for each study. Entering a different set of covariates into a model could impact results by changing the estimated effect that body mass has on subsequent depressive symptoms. Without this consistency in covariates used, cross-study comparisons are made much more difficult.

Certain patterns are evident with respect to these conceptual, methodological, and analytical variations among studies, in which certain types of studies may be more likely to report an effect than others. Restricting the literature review to only those two studies that examined, to some extent, the transition between adolescence and young adulthood, both studies investigated associations in both males and females, and reported nonsignificant associations for males (Goodman & Whitaker, 2002; Merten et al., 2008).

When focusing on adolescents as they transition to young adulthood, there is some evidence that body mass may be more predictive of moderate increases in depressive symptoms, as opposed to being predictive of clinically significant depressive symptoms. For example, Goodman and Whitaker (2002) used a CES-D cutoff for predicting clinically significant depressive symptoms in adolescents (Roberts et al., 1991), and reported that 12-19 year-olds who were obese at baseline did not have an increased risk of clinically significant depressive symptoms when measured one year later. On the other hand, using a continuous scale for depressive symptoms, Merten and colleagues (2008) examined the association between obesity at age 12-18 and depressive symptoms five years later and found that, among females, obesity in adolescence was associated with higher depressive symptom scores during young adulthood.

From these patterns, there is some evidence that, for adolescents transitioning to young adulthood, associations between body mass and subsequent depressive symptoms are nonsignificant for males; and also that increases in body mass may lead to higher levels of depressive symptoms, but not necessarily clinically significant depressive

symptoms. However, it is still difficult to compare findings across studies with two or even three similarities, as results may be confounded by the many other study differences (i.e., operationalization of body mass comparison groups, populations examined, statistical techniques, and covariates modelled). Overall, although greater precision in conceptualization, methodology, and analytical techniques across studies would potentially translate to less variety in results, the vast differences in the design of previous studies makes cross-study comparisons difficult.

2.2.2   Depressive symptoms affecting body mass

Several studies have also investigated whether depressive symptoms affect subsequent increases in body weight in adolescents and young adults. These studies are summarized below, as well as presented in greater detail in the Appendix. Studies are first organized into whether they measured change in body mass across two time points or more than two time points, then further organized into whether they reported a prospective association or not.

2.2.2.1   Measuring change in body mass with only two time points

Several studies have measured change across two time points in order to estimate the influence of depressive symptoms on subsequent body mass, controlling for prior levels of body mass and other covariates. The following five studies reported significant associations. First, Anderson and colleagues (2011) examined these associations in a cohort of adolescent females from public schools across the USA at two time points: grades six and eight. The authors operationalized clinically significant depressive symptoms as a CES-D score of 24 and over, and categorized BMI into obese and non-obese using the Center for Disease Control (CDC) reference guide. The authors found a significant interaction between depressive symptoms and race such that, having clinically significant depressive symptoms at sixth grade predicted subsequent obesity in White females, but not in Black or Hispanic females. They also repeated their analyses using continuous BMI scores and continuous depressive symptom scores and found similar results.

Using a nationally representative cohort of USA adolescents, Goodman and Whitaker (2002) examined the association between clinically significant depressive symptoms in 7th-12th grade adolescents and obesity one year later. The authors categorized clinically significant depressive symptoms as a Modified CES-D-18 score of 24 or more for females, and 22 or more for males, and used the CDC reference classifications for classifying BMI into obese and not obese categories. The authors found that those who had clinically significant depressive symptoms at baseline had a higher risk of obesity at follow-up, compared to those who did not have clinically significant depressive symptoms at baseline.

Franko et al. (2005) used two community-based cohorts of Black and White females from three US states to examine the influence of depressive symptoms (using continuous CES-D scores) on subsequent body mass. Body mass was analyzed as both a continuous and categorical variable (i.e., categorized into obese and not obese categories, using CDC reference guidelines). The first cohort was examined at age 16 and again at age 21; the second cohort was examined at age 18 and again at age 21. The authors reported that higher depressive symptoms at either age 16 or 18 predicted an increased risk of obesity at age 21. They also found a similar pattern of results when body mass was examined as a continuous variable, such that higher depressive symptoms at either age 16 or 18 predicted an increase in body mass at age 21.

Barefoot and colleagues examined associations between depressive symptoms and body mass in a cohort of North Carolina university students aged 16-25 at baseline, and again 23 years later (1998). Depressive symptoms were analyzed as both a continuous and categorical variable. As a categorical variable, the authors defined a high level of depressive symptoms as a Minnesota Multiphasic Inventory- Obvious Depression Scale (MMPI-ODS; Greene, 1991) score of 11 or higher for males, and 13 or higher for females[2]. The authors found a significant interaction between initial body mass and

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[2] Note, the categorization of high level of depressive symptoms identified by the authors does not reflect a level of clinically significant depressive symptoms, but only identifies those with a relatively large number of depressive symptoms (Barefoot et al., 1998).

depressive symptoms on weight change, such that individuals with a high level of depressive symptoms who were initially lean gained less weight (i.e., less of a change in BMI between two time points) than individuals with a low level of depressive symptoms who were initially lean. Furthermore, individuals with a high level of depressive symptoms who were initially heavy gained more weight than individuals without a high level of depressive symptoms. The authors reported similar results when a continuous measure of depressive symptoms was used instead of a dichotomized cutoff.

Fifth, Rhew et al., 2008 examined whether clinically significant depressive symptoms in sixth grade predicted body mass one year later in a cohort of middle school students in Seattle, Washington. The authors categorized clinically significant depressive symptoms as a MFQ score of 15 or higher for males, and 20 or higher for females, and used a continuous measure of self-reported BMI. They found a significant interaction between sex and depressive symptoms. Males with clinically significant depressive symptoms at baseline had lower subsequent body mass compared to males without clinically significant depressive symptoms. For females, clinically significant depressive symptoms at baseline did not predict subsequent body mass.

Two studies that measured change in body mass across two time points did not report any significant findings. First, Stice et al. (2005) examined associations between depressive symptoms at 11-15 years and development of obesity over a four-year period in a sample of adolescent girls from schools across metropolitan southwestern USA. The authors examined continuous depressive symptoms using an average of severity ratings for each symptom in the K-SADS. BMI was categorized into obese and not obese groups using CDC reference guides. The authors found that depressive symptoms at baseline did not predict the onset of obesity over a four-year period.

In another study, Jansen et al. (2008) examined associations between clinically significant depressive symptoms and subsequent overweight and obesity (using WHO classifications) in a cohort of youth from Rotterdam, ages 9-10 at baseline and 12-13 at follow-up. The authors categorized clinically significant depressive symptoms as a score of four or more using the Short Depression Questionnaire for Children (SDQC; Meier,

Mellenbergh, & de Wit, 1986), and used WHO classifications for categorizing BMI into not overweight, overweight, and obese categories. The authors found that clinically significant depressive symptoms at baseline did not predict overweight status or obese status three years later in this cohort of young adolescents.

2.2.2.2 Measuring change in body mass using more than two time points

Only one study testing the directional association between baseline depressive symptoms and subsequent body mass used more than two time points. Tanofsky-Kraff et al. (2006) used an average of 4.2 annual measurements of children from Maryland, USA, who were 6-12 years old at baseline, and applied mixed regression models with depressive symptoms as a fixed effect and the child as a random effect. The authors used CDI scores as a continuous measure of depressive symptoms, and used a dual-energy x-ray absorptiometry (DEXA) continuous scale to measure fat mass and change in fat mass over time. The authors found that baseline depressive symptoms did not predict increases in body fat mass over the course of the study, and that interaction effects with sex were not statistically significant.

2.2.2.3 Summary of studies examining depressive symptoms affecting body mass

In summary, although all of these studies investigated the theoretical framework that depressive symptoms lead to increases in body mass through the previously mentioned causal pathways (i.e., neurobiological causal pathways and affect-regulation, and sleep quality and physical inactivity), most did not make any explicit mention of a driving theoretical framework (Barefoot et al., 1998; Franko et al., 2005; Goodman & Whitaker, 2002; Jansen et al., 2008; Rhew et al., 2008; Tanofsky-Kraff et al., 2006;). Overall, there is some evidence that initial depressive symptoms are prospectively associated with an increase in body mass in adolescents and young adults. On the other hand, almost one half of the studies testing the directional association from depressive symptoms to body mass reported null findings. There was also a great deal of inconsistency regarding whether or not differential prospective associations between depressive symptoms and subsequent body mass exist for males and females. Of the five

studies that assessed sex differences, only one found a different pattern of results for males and females such that associations were inversely significant for males (i.e., clinically significant depressive symptoms predicted lower subsequent BMI; Rhew et al., 2008).

As was the case above, the inconsistencies in results of studies assessing the direction of association from depressive symptoms to increases in body mass reflect a disparity in the conceptual, methodological, and analytical frameworks among studies. Conceptual differences across studies examining whether depressive symptoms predict increases in body mass are similar to those studies examining the opposite direction of association (i.e., body mass to depressive symptoms). Differences across studies exist on multiple levels in terms of developmental periods studied, operationalization of body mass, and differences in the populations studied.

Only two studies examined the developmental transition from adolescence to young adulthood (Franko et al., 2005; Goodman & Whitaker, 2002). Instead, most studies focused solely on changes within adolescence (Anderson et al., 2011; Jansen et al., 2008; Rhew et al., 2008; Stice et al., 2005; Tanofsky-Kraff et al., 2006); or young adulthood (Barefoot et al., 1998).

With regards to operationalizing body mass into categories, most studies used cutoffs to define overweight and/or obese status (Goodman & Whitaker, 2002; Jansen et al., 2008; Rhew et al., 2008; Stice et al., 2005). A few studies, however, operationalized body mass as a continuous scale of BMI (Barefoot et al., 1998; Tanofsky-Kraff et al., 2006). One study used both a continuous and categorized operationalization of body mass (Anderson et al., 2011).

Studies also differed in the populations they examined. While most studies sampled both males and females (Barefoot et al., 1998; Goodman & Whitaker, 2002; Jansen et al., 2008; Rhew et al., 2008; Tanofsky-Kraff et al., 2006), some sampled females only (Anderson et al., 2011; Franko et al., 2005; Stice et al., 2005). There were also some differences across studies in the composition of the population from which participants were sampled. For example, a few studies used at-risk samples (Rhew et al.,

2008; Tanofsky-Kraff et al., 2006). Anderson and collegues (2011), as well as Goodman and Whitaker (2002) included only Hispanic, non-Hispanic Black, and non-Hispanic White adolescents in their studies. Franko and colleagues (2005), and Tanofsky-Kraff et al., (2006) only sampled Black or White participants from the United States. One study investigated associations in adolescents and young adults from the Netherlands (Jansen et al., 2008).

Methodologically, there are large variations among the length of intervals, source of height and weight measurements, reference tools for establishing body mass cutoffs, and tools for measuring depressive symptoms. In terms of interval length, most studies examined associations over annual (Goodman & Whitaker, 2002; Rhew et al., 2008; Stice et al., 2005; Tanofsky-Kraff et al., 2006) or biennial (Anderson et al., 2011; Franko et al., 2005) cycles, whereas others used intervals between three and six years (Franko et al., 2005; Jansen et al., 2008) or much longer (Barefoot et al., 1998).

When calculating BMI, most studies used measured height and weight (Anderson et al., 2011; Stice et al., 2005), while one used self-reported height and weight (Goodman & Whitaker, 2002). Of note, a few studies used inconsistent measurements between cycles (Barefoot et al., 1998; Franko et al., 2005; Jansen et al., 2008). That is, height and weight would be measured at one cycle and self-reported at another. One study used DEXA to directly measure fat mass (Tanofsky-Kraff et al., 2006). With regards to reference tools for establishing body mass cutoffs, almost all but one study (Jansen et al., 2008) used categorizations based on CDC.

Finally, almost every study used a slightly different measurement tool to assess depressive symptoms, including K-SADS (Stice et al., 2005), MFQ (Rhew et al., 2008), MMPI ODS (Barefoot et al., 1998), CDI (Tanofsky-Kraff et al., 2006), SDQC (Jansen et al., 2008), and different versions of the CES-D (Anderson et al., 2011; Franko et al., 2005; Goodman & Whitaker, 2002).

Analytically, studies varied in the number of measurement points used for analyses. While one study examined associations over four cycles (Tanofsky-Kraff et al., 2006), the majority of research in this area focused on assessing only two points

(Anderson et al., 2011; Franko et al., 2005; Goodman & Whitaker, 2002; Jansen et al., 2008; Rhew et al., 2008; Stice et al., 2005). Studies also varied in the statistical techniques they used to assess whether depressive symptoms predicted subsequent body mass, with some using logistic regression techniques (Goodman et al., 2002; Jansen et al., 2008; Stice et al., 2005), and others using linear regression techniques (Barefoot et al., 1998; Rhew et al., 2008; Tanofsky-Kraff et al., 2006).

As was the case for studies examining the effect of body mass on subsequent depressive symptoms, studies examining this direction of association have also varied greatly in their use of covariates controlled for during analyses. The table in the Appendix illustrates this discrepancy by listing all covariates considered for each study. As mentioned above, inconsistencies in covariates used make cross-study comparisons much more difficult.

Overall, there are potentially multiple combinations of differences across studies with respect to the operationalization of depressive symptoms and body mass, the populations studied, the number and length of intervals, the statistical technique for analyses, and the use of covariates that could drastically affect not only the findings of each study, but also the interpretation of the literature. For example, continuous BMI levels may be differentially associated with depressive symptoms than an obesity or overweight classification. Similarly, continuous depressive symptoms may predict body mass differentially than clinically significant depressive symptoms. Furthermore, differences in results may exist between males and females, across different races, or across samples selected from different parts of the world. Finally, entering a different set of covariates into a model could impact results by changing the estimated effect that depressive symptoms have on subsequent body mass. Overall, the multiple possibilities of study design makes comparisons difficult because it becomes impossible to tease out similarities among studies when there are yet so many levels of differences.

As opposed to the findings from studies investigating the direction of association from body mass to depressive symptoms, previous research examining the direction of association from depressive symptoms to body mass had few discernable patterns in

results with regards to conceptual, methodological, and analytical differences. That is, because there were such few similarities and such great differences among studies, it was not possible to identify certain types of studies that were more likely to find an effect than others. One evident pattern was that, of the two studies that examined both males and females and did not report a significant effect (Jansen et al., 2008; Tanofsky-Kraff et al., 2006), both controlled for sex in the analysis instead of testing whether results differed for males and females. Rhew and colleagues (2008) found that males clinically significant depressive symptoms at baseline had lower subsequent body mass, whereas females with clinically significant depressive symptoms at baseline had higher subsequent body mass. Thus, not accounting for this sex difference analytically may lead to null associations.

Overall, there is some evidence that with respect to depressive symptoms predicting body mass, differential associations may exist between males and females. However, because of the complexity and amount of conceptual, methodological, and analytical differences among studies, it is difficult to conclusively compare findings. As previously mentioned, greater precision in the methodologies across studies would translate to less variety in results. However, the vast differences in the current state of literature on depressive symptoms predicting body mass make cross-study comparisons difficult. A major goal for research in this area should be to work towards greater consistency by addressing the major limitations of previous research and providing a strong justification for the operationalization of concepts, as well as the methodologies and analytical techniques used.

2.2.3   Limitations in current literature

In addition to the above-mentioned conceptual, methodological, and analytical differences among studies, there are several key within-study limitations that may also contribute to mixed findings in the literature. The major limitations of previous research are: lack of consistency in measuring body mass and depressive symptoms over cycles, failure to assess longitudinal change in outcomes, using only two waves of data, failure to examine reciprocal relationships, and finally, lack of transitional research examining the

adolescent to young adult developmental period. Problems with these key limitations are explained in detail below. These within-study limitations are also summarized in Table 1.

Table 1: Within-study limitations of previous research.

First author, year	Inconsistent measurement tool for body mass and/or depressive symptoms	Did not control for or exclude baseline dependent variable	Two cycles	Did not examine reciprocal relationship	Did not examine adolescent to young adult transition
Anderson, 2011			X		X
Ball, 2008			X	X	X
Bardone, 1998*		X	X	X	
Barefoot, 1998	X		X	X	X
Boutelle, 2010				X	X
Clark, 2006			X	X	X
Duarte, 2010*		X	X	X	
Franko, 2005	X		X	X	
Goodman, 2002			X		
Goodwin, 2009*		X	X	X	
Herva, 2006*		X	X	X	
Jansen, 2008	X		X	X	X
Merten, 2008			X	X	
Pine, 1997*	X	X	X	X	
Rhew, 2008			X		X
Stice, 2005				X	
Tanofsky-Kraff, 2006				X	X
Xie, 2010			X	X	X

Note. *Not included in literature review because does not measure change in outcome.

2.2.3.1 Inconsistency in measurement tools for body mass and/or depressive symptoms

Many studies have used inconsistencies in the source of height and weight reports across cycles (Barefoot al., 1998; Franko et al., 2005; Jansen et al., 2008). For example, Franko and colleagues (2005) used self-reported height and weight to calculate participants BMI at the first cycle, but used measured height and weight during the subsequent cycle. Similarly, Jansen and colleagues (2008) and Barefoot and colleagues (1998) both used measured height and weight at baseline and self-reported height and weight at follow-up. Inconsistencies across cycles in the source of height and weight reports may bias results if sources are differentially accurate in their reports of height and weight (Goodman, Hinden, & Khandelwal, 2000).

2.2.3.2   Failure to assess longitudinal change in outcomes

Many studies did not assess longitudinal change in outcomes because they failed to control for baseline levels of the dependent variable (Bardone, Moffitt, Caspi, et al., 1998; Duarte, Sourander, Nikolakaros, et al., 2010; Goodwin, Sourander, Duarte, et al., 2009; Herva, Laitinen, Miettunen, et al., 2006; Pine et al., 1997). As is the case with all longitudinal analyses, controlling for initial levels of an outcome ensures that models are predicting change over time instead of stability in the health outcome over time (Stice & Bearman, 2001). For example, past depressive symptoms are known to predict future depressive symptoms (Eaton, Shao, Nestadt et al., 2008), thus, failing to control for initial levels of depressive symptoms makes it difficult to model the effects of body mass on the development of depressive symptoms.

2.2.3.3   Examining less than three cycles of data

The third major limitation of previous research is that most studies measured these health outcomes across only two waves (Anderson et al., 2011; Ball et al., 2009; Barefoot et al., 1998; Clark et al., 2007; Franko et al., 2005; Goodman & Whitaker, 2002; Jansen et al., 2008; Merten et al., 2008; Rhew et al., 2008; Xie, et al., 2010). Although technically measuring change, these studies assess development poorly (Rogosa, Brandt, & Zimowski, 1982) because two-panel designs do not allow for examination of developmental trajectories, and thus may only be appropriate when the growth process is irrelevant or known to be linear (Duncan, Duncan, & Strycker, 2006). However, neither body mass nor depressive symptoms are linear processes during the transition between adolescence and young adulthood (Needham et al., 2010). Thus, measuring less than three waves of data does not take into account the growth process involved nor does it allow for the identification of curvilinear trajectories for either health outcome.

2.2.3.4   Failure to examine reciprocal relationship

The majority of longitudinal studies (Ball et al., 2009; Barefoot et al., 1998; Boutelle et al., 2010; Clark et al., 2007; Franko et al., 2005; Jansen et al., 2008; Merten et al., 2008; Stice et al., 2005; Tanofsky-Kraff et al., 2006; Xie et al., 2010) that test the

association between body mass and depressive symptoms have focused on measuring only one direction of the relationship (i.e., body mass predicting depressive symptoms, or vice versa). Statistically measuring one direction does not test whether there are reciprocal associations between body mass and depressive symptoms within the same sample. Investigating reciprocal relationships over time is important in order to examine whether the predictive relationship is unidirectional, or whether there are reciprocal effects where either construct may predict growth in the other (Needham, et al., 2010). These alternative mechanisms would have different implications for etiology, prevention, and treatment.

2.2.3.5 Lack of adolescent to young adulthood transitional research

Finally, many studies focus on young adult populations (Ball et al., 2009; Barefoot et al., 1998; Forman-Hoffman et al., 2007; McCarty, Kosterman, Mason, et al., 2009; Needham et al., 2010; Roberts et al., 2003) or younger adolescents (Anderson et al., 2011; Boutelle et al., 2010; Clark et al. 2007; Jansen et al., 2008; Rhew et al., 2008; Tanofsky-Kraff et al., 2006; Xie et al., 2010). The association between body mass and depressive symptoms may be different for adolescents than for children or adults (Napolitano & Foster, 2008), and there is far less research on adolescents as they transition into young adults. Adolescence and young adulthood are critical periods of growth and maturation, and are associated with changes in diet, physical activity, sedentary behavior, as well as psychological health (Alberga, Sigal, Goldfield, Prud'homme, & Kenny, 2012). Thus, an understanding of the causal associations between mental and physical health during this transition is needed, and differentiating findings from earlier or later life stages is important for public health initiatives.

Only four studies in the present literature review examined prospective associations as adolescent's transition into young adults (Franko et al., 2005; Goodman & Whitaker, 2002; Merten et al., 2008; Stice et al, 2005). However, each of these studies present with limitations of their own, as identified in Table 1. For example, by only examining two data points, Merten and colleagues (2008) could not effectively examine the developmental growth in either health outcome. Also, having a six-year interval

between data points could potentially increase the chances of transitional life events confounding prospective associations. The second major limitation of the Merten and colleagues (2008) study is that they compared obese adolescents to not obese adolescents, such that the not obese group comprised of overweight, normal weight, or underweight adolescents. The mental health status of overweight and underweight adolescents may be systematically different from normal weight adolescents, and collapsing all three weight categories into a single comparison group may bias results if there are differences among these weight categories.

In another example, the study by Franko and colleagues (2005) is limited in its ability to generalize to the population since the authors only sampled Black and White females. They also had inconsistent reporting sources for height and weight, where self-reports were used at the first wave and measured height and weight at the second wave. Therefore, the increase in body mass at the second wave may reflect a true consistency in body mass that was not captured in the first wave because adolescents tend to under-estimate their weight and over-estimate their height, thus lowering their initial BMI (Danubio, Miranda, Vinciguera, Vecchi, & Rufo, 2008; Elgar, Roberts, Moore, & Tudor-Smith, 2005; Must & Strauss, 1999). Overall, among the very few studies that did examine the developmental transition from adolescence to young adulthood, inconsistencies in findings are evident among studies, and major methodological flaws exist within each study.

2.2.4   Summary of literature review

In summary, there are several conceptual, methodological, and analytical differences that exist in the present state of the literature. Despite many studies having examined this research question, the level and complexity of variation among existing research makes cross-study comparisons difficult, thus complicating interpretability of findings. These inconsistencies among studies combine with the various above-mentioned limitations within studies. These include: lack of consistency in measuring body mass and depressive symptoms, failure to assess longitudinal change in outcomes, using only two waves of data, failure to examine reciprocal relationships, and a lack of transitional research between adolescence and young adulthood. Taken together, as the

literature currently stands, it is difficult to draw concrete conclusions about the prospective associations between body mass and depressive symptoms across adolescence and young adulthood. With these limitations in mind, the next section will highlight the rationale for this study, as well as its objectives and hypotheses.

2.3 Plan of study, objectives, and hypotheses

The purpose of the present study is to assess the association between two developmental processes, body mass and depressive symptoms, as adolescent's transition into young adulthood. As can be deduced by the literature review presented above, several studies have already examined this research question. However, this study was conducted in order to address the major limitations and gaps of previous research in this area, as well as to contribute to some level of consistency that will help make cross-study comparisons possible in the future. Thus, strong conceptual, methodological, and analytical justifications were made throughout the following methods section.

The specific objectives of this study are to: 1) examine individual trajectories of body mass and depressive symptoms as adolescents transition into young adulthood; 2) examine if changes in body mass over time are associated with changes in depressive symptoms over time; 3) examine whether initial levels of body mass predict changes in depressive symptoms over time, and/or vice versa; and 4) examine whether any of the above associations differ between sex.

For objective (1), based on previous research (Ge et al., 2006**;** Kuczmarski et al., 2002; Needham et al., 2010), we expect to find an increase in body mass over time and a decrease in depressive symptoms over time. Additionally, we expected to see body mass levels to be consistently higher for males than females (Shields, 2006), and depressive symptom levels to be consistently higher for females than males (Kuczmarski et al., 2002; Locke & Newcomb, 2001), at all time points.

Given that there is little research examining the association between changes in both health constructs over time, and the inconsistencies in the literature on the direction and strength of this association for males and females, objectives (2), (3), and (4) were

examined in a purely exploratory manner. However, we expect that, if a prospective association is evident between initial body mass and subsequent growth in depressive symptoms, it would be stronger for females than males. This hypothesis is made on the grounds of both the theoretical and empirical evidence presented in the literature review. As previously stated, weight concerns (Fredrickson & Roberts, 1997; Wadden & Stunkard, 1987), a greater level of weight-related stigmatization (Tang-Peronard, & Heitmann, 2008), and weight-teasing (Eisenberg et al., 2006; Tang-Peronard, & Heitmann, 2008) are more common in adolescent females than males. Furthermore, in both longitudinal studies that examined body mass affecting depressive symptoms during the transition between adolescence and young adulthood, associations for males were consistently nonsignificant across both studies (Goodman & Whitaker, 2002; Merten et al., 2008).

Chapter 3

3   Methods

3.1   Data

3.1.1   Data source

This study is based on a secondary data analysis of the National Longitudinal Survey of Children and Youth (NLSCY; Statistics Canada, 2009). The NLSCY is a population-based longitudinal study of Canadian children that follows their development and well-being from birth to early adulthood. The NLSCY was collected as a joint project of Human Resources Development Canada and Statistics Canada, and is designed to evaluate the determinants of developmental outcomes in Canadian children. The objectives of the survey include determining the prevalence of various risk and protective factors, understanding how factors and life events influence development, and collecting biological, social, and environmental information about children as they grow (Statistics Canada, 2009).

3.1.2   Sampling design and data collection

The target population for the original cohort in cycle 1 was selected from the Statistics Canada Labour Force Survey (LFS) and the National Population Health Survey (NPHS). The LFS is a monthly survey that collects labour market data from a national sample. It is representative of the civilian, non-institutionalized population aged 15 or over in Canada's 10 provinces. The LFS excludes residents of Yukon, Nunavut, and the Northwest Territories, as well as people living on Indian reserves, full-time members of the Canadian Armed Forces, and inmates of institutions; thus excluding approximately 2% of the population 15 years of age and older. Similarly, NPHS target population includes household residents in all provinces and territories, and excludes persons living on Indian reserves, on Canadian Forces Bases, and in some remote areas. Please see NLSCY microdata file (Statistics Canada, 2009) for more detail on the LFS and NPHS sampling design.

The NLSCY consists of eight biennial cycles in total (data collection for cycles 1 to 8 occurred in 1994-95, 1996-97, 1998-99, 2000-01, 2002-03, 2004-05, 2006-07, and 2008-09, respectively). Households that reported having at least one child between the ages of 0 and 11 years were selected as the household sample for cycle 1. For the first cycle, the children comprising the sample were selected at random, up to a maximum of four children per household. Approximately 25,000 children, ranging in age from newborn to 11 years inclusive, were initially selected in the ten provinces. The analyses for this study are based on data collected from the original cohort aged 10-11 years at the first cycle; thus, the following discussion will be limited to the procedures and characteristics that pertain to this cohort from the NLSCY longitudinal sample.

Across collection cycles, several changes were implemented in the sampling strategy. At the second cycle, a maximum of two, instead of four, children were chosen for whom data were to be collected. Beginning in the third cycle, children who were not sampled included: deceased children, duplicate cases, children who were the wrong age for the survey, households that were not traceable in the second cycle, households that had moved permanently out of the country, children on Indian reserves, and households that were adamant refusals. Beginning in the fourth cycle, it was decided to exclude households after two consecutive cycles of non-response. Once respondents reach 18 years of age, it is the decision of the respondent, not the person most knowledgeable (PMK), to respond to the survey. In other words, the response burden for the 18-year-old is not as great as it may be for younger children. Thus, in order to retain more cases, at the beginning of the fifth cycle, children who were 18-19 years of age were only excluded if there were three consecutive cycles of non-response. Beginning in cycle 7, the PMKs history of non-response was ignored for returning children who were 18 or older.

At the first cycle, a household roster was completed for each household in the NLSCY asking about basic demographic information and dwelling conditions. Once this was completed, one child between the age of 0 and 11 was randomly selected from each household, and a question was asked about whom in the household was the person most knowledgeable (PMK) about the child. The PMK provided information about

him/herself, his/her spouse/partner, the selected child, and household members. Data from the PMK was collected in a face-to-face or telephone interview using computer-assisted telephone interviewing (CATI). After the PMK gave permission, the interviewer provided a separate questionnaire to each child aged 10 to 11 years. These children were encouraged to self-complete the questionnaire in a private setting. Once completed, the questionnaire was sealed in an envelope and the parent was not permitted to see the completed questionnaire. In addition to the household component, questionnaires were mailed to, and returned by, teachers and principals of school-aged children for whom parental consent had been given.

At subsequent cycles, similar procedures were followed as mentioned above. Namely, households that participated in the first cycle of the survey were contacted, both the PMK and spouse were asked questions, children older than 10 years were provided with self-complete questionnaires, and school-component questionnaires were mailed out to participating children's teachers and principals, when applicable.

3.1.3   Sample selection

The purpose of this study was to examine the association between body mass and depressive symptoms as adolescents transition into young adulthood. Late adolescence was chosen as a starting point as opposed to early adolescence or childhood because depressive symptoms in these earlier developmental periods have a different pattern of symptoms (Carlson & Kashani, 1988; Ryan, Puig-Antich, Ambrosini, et al., 1987; Mitchell, McCauley, Burke, & Moss, 1988) and correlates (Jaffee, Moffitt, Caspi, et al., 2002) compared to depressive symptoms in late adolescence and young adulthood. Younger adolescents with depressive symptoms are more likely to experience agitation and anxiety (Mitchell et al., 1988; Ryan et al., 1987), and older adolescents and young adults are more likely to experience psychomotor retardation (i.e., slower thought processes and physical activity; Ryan et al., 1987), which may be more closely related to weight gain. Thus, the population of interest is Canadian youth beginning in late adolescence and followed into young adulthood. Late adolescence is defined as 16-18 years, and young adulthood refers to 18-30 years (Rindfuss, 1991). Thus, for the purposes

of the present study, an ideal nationally-representative population would prospectively follow adolescents from 16 years of age to 30 years of age.

An initial longitudinal cohort of children (N = 2,488) participated in the NSLCY survey in 1994-95 when they were 10-11 years old, in 1996-97 when they were 12-13 years old, in 1998-99 when they were 14-15 years old, in 2000-01 when they were 16-17 years old, in 2002-03 when they were 18-19 years old, in 2004-05 when they were 20-21 years old, in 2006-07 when they were 22-23 years old, and in 2008-09 when they were 24-25 years old.

In order to match the selected sample to the population of interest as closely as possible, youth were selected from the NLSCY once they were 16-17 years of age (i.e., starting at cycle 4, in 2000-01). The sample size for this group is 1,895 (47.9% female), representing 76% of the original cohort of 10-11 year olds at cycle 1. These youth were followed into young adulthood, until age 24-25. Although, it would have been ideal to have youth followed until age 30, the NLSCY currently only has data available until ages 24-25. Although sampling procedures and data collection did not begin when these youth were 16-17 years of age, cross-sectional weights were used in all analyses (explained in detail below). Thus, the sample selected for the present study represents the Canadian population of adolescents aged 16-17 years as of January 1, 2001.

Statistics Canada provides information on the overall participation rates of the initial 10-11 year olds for only five out of the eight cycles (96.1% in cycle 1, 91.2% in cycle 2, 87.5% in cycle 3, 75.1% in cycle 6, and 61.9% in cycle 8). However, participation rates for the entire longitudinal cohort (i.e., 0-11 year olds at cycle 1), as compared to the first cycle, are as follows: 86.5%, 91.5%, 89.2%, 84.5%, 81.3%, 82.4%, 80.5%, and 68%, for cycles 1-8, respectively.

3.2   Measurement instruments

3.2.1   Body mass

Body mass was assessed by BMI, which was calculated as weight in kilograms divided by height in meters squared. Height and weight were self-reported by individuals

at all five ages (i.e., when participants were 16-17, 18-19, 20-21, 22-23, and 24-25 years of age).

BMI was kept as a continuous variable in all analyses for several reasons. First, as BMI does not measure fat directly, there is currently no consensus regarding cut-offs for defining obesity in adolescents (Sweeting, 2007). In contrast with adult populations, much less is known about the levels of risk associated with specific BMI levels in children and adolescents. Due to this uncertainty, statistical approaches have often been used in which the distribution for a population is determined and percentile values are arbitrarily chosen to distinguish those with the highest BMI from the rest of the population (e.g., 95th percentile). This approach has resulted in different reference groups that identify different BMI cutoffs for obesity and overweight status based on sex and age. For example, the CDC (Kuczmarski et al., 2002), the International Obesity Task Force (IOTF; Cole, Bellizzi, Flegal, & Dietz, 2000), and the WHO (WHO, 2006) have all identified different reference datasets for children and adolescents.

Second, retaining the continuity in BMI levels allows researchers to examine individual heterogeneity over time, as well as associations between depressive symptoms and normal variations in relative weight, as distributed throughout the population (Hasler, Pine, Gamma, et al., 2004). Taking a more inclusive approach to body mass by examining weight as a continuous variable may be more appropriate for understanding the adverse health consequences and risk factors associated with higher BMI levels (Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Li, Rana, Manson, et al., 2006; Ogden, Carroll, Curtin, et al., 2006; Rashid, Fuentes, Touchon, & Wehner, 2003). Thus, given the lack of research identifying validated cutoffs for youth, as well as the benefits of examining weight as a continuous variable, the present study has kept BMI as a continuous predictor and outcome so that readers can interpret results with reference data as they see fit and results may be more meaningful within a population health context.

3.2.2   Depressive symptoms

Depressive symptoms were measured using the Centre for Epidemiological Studies- Depression Scale (CES-D; Radloff, 1977). This scale is used to measure the

frequency of symptoms in the public at large. Questions focus on affective components such as depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, and sleep disorders (Radloff, 1977). Within the NLSCY, the original 20-item scale was reduced to 12 questions by Dr. M. Boyle of the Chedoke-McMaster Hospital, McMaster University. This short version CES-D scale was developed to assess levels of depressive symptoms during the past week, and has a Cronbach's alpha of .85 (Poulin, Hand, & Boudreau, 2005). Participants were asked to rate on a four-point scale, from 0 (*less than 1 day*) to 3 (*5-7 days*), the occurrence of 12 feelings, such as "I felt depressed" or "I felt hopeful about the future", during the previous week. Three items on the scale were reversed scored so that higher scores indicated greater depressive symptoms. Severity ratings for each symptom were summed to form a continuous depressive symptom composite at each age, ranging from 0 to 36. The internal consistency of the scale was similar across the five ages (α = .84, .83, .80, .80, .82, for ages 16-17 to 24-25, respectively).

Depressive symptoms were kept as a continuous variable in all analyses because depressive symptoms, like weight, occur on a continuum, and similar to those individuals who are overweight but not yet obese, individuals who do not meet clinically significant levels of depressive symptoms may still be at risk for mental health consequences (Napolitano & Foster, 2008). Thus, in order to capture these population-level variations in depressive symptomology, the present study focused on continuous depressive symptoms rather than clinically significant depressive symptoms.

3.2.3   Age and sex

Age and sex were both reported by the PMK at the very first cycle. Age at each subsequent cycle was derived by the child's date of birth (as reported by the PMK during the first cycle of data collection) and the date of interview. For ease of interpretation, going forward, NLSCY cycles will be referred to by assumed median ages, with age 17 representing cycle 4 (i.e., baseline), age 19 representing cycle 5, age 21 representing cycle 6, age 23 representing cycle 7, and age 25 representing cycle 8.

Response rates for variables of interest across all five ages are presented in Table 2. For BMI, response rates ranged from 64.6-78.4% for males and 66.7-80.0% for females. For CES-D scores, response rates ranged from 64.6-70.2% for males and 67.5-75.3% for females.

Table 2: Sample size across ages.

	Variable	Age 17 (%)	Age 19 (%)	Age 21 (%)	Age 23 (%)	Age 25 (%)
Males	BMI	681 (68.9)	775 (78.4)	647 (65.5)	698 (70.6)	638 (64.6)
(n = 988)	CES-D	653 (66.1)	671 (67.9)	643 (65.1)	694 (70.2)	638 (64.6)
	Age	988 (100.0)	782 (79.1)	650 (65.8)	700 (70.8)	641 (64.9)
Females	BMI	681 (75.1)	696 (76.7)	676 (74.5)	662 (80.0)	605 (66.7)
(n = 907)	CES-D	678 (74.8)	665 (73.3)	683 (75.3)	677 (74.6)	612 (67.5)
	Age	907 (100.0)	713 (78.6)	695 (76.6)	677 (74.6)	613 (67.6)

Note. BMI: Body mass index. CES-D: Centre for Epidemiological Studies- Depression Scale. Percentages reflect response rates for original cohort of 17 year olds selected from cycle 4.

3.3 Analysis

3.3.1 Modeling strategy

As previously mentioned, the purpose of the present study is to assess the relation between two developmental processes, body mass and depressive symptoms, as adolescents transition into young adulthood. Latent growth modeling (LGM) was the analytical technique used to assess this research question. The following section will provide a justification of LGM as this analytical technique best represents the model of development and best complements the conceptual and methodological components of the present thesis.

In LGM, a single underlying growth trajectory for a construct is estimated for each person across all ages (Curren & Bollen, 2001). In LGM, two latent factors are estimated: an intercept and a growth parameter. The intercept represents the starting point

(e.g., BMI at baseline). The intercept factor presents information about the mean, or average, starting levels for individual growth curves, as well as the variance in starting levels between individual growth curves (Duncan et al., 2006). The growth parameter represents the rate of change of an individual's trajectory. This growth parameter may represent a linear slope or a more complex non-linear growth. Like the intercept factor, the growth factor consists of two components: the average, and the variance. That is, the growth factor presents information about the mean, or average, rate of change, as well as the variance in the rate of change across individuals. Taken together, the means for the intercept and growth represent the average trajectory for all individuals, and the variances for the intercept and growth represent the variability in trajectories across individuals. More information on techniques for estimating latent factors can be found in Duncan et al. (2006).

Most previous research has examined the association between body mass and depressive symptoms using traditional panel models [e.g., autoregressive cross-lagged (ACL) approaches] to explore the temporal directionality of relations between depressive symptoms and body mass. Using ACL models, researchers have evaluated whether level of depressive symptoms at one point in time predicts body mass at a later point in time (and vice versa), while accounting for rank-order stability in variables over time (i.e., they examine the extent to which the rank-order standing of an individual within a group is stable or unstable over time; Duncan et al., 2006). Duncan and colleagues (2006) identified several criticisms of developmental models that incorporate these autoregressive effects.

First, these types of statistical techniques do not provide information about developmental trajectories and individual-level change over time. In order to uncover the nature of development, it is necessary to assess individual-level change over time (Singer & Willett, 2003). Both body mass and depressive symptoms are developmental constructs. That is, they both change over time and across the lifecourse. For example, depressive symptoms increase throughout the adolescent years, peak during late adolescence, and decline into young adulthood (Ge et al., 2006). Similarly, body mass dramatically increases across childhood and adolescence and then levels off into

adulthood (Kuczmarski et al., 2002). Only by examining developmental trajectories using individual-level analysis could these changes over time be uncovered. Knowing that neither body mass nor depressive symptoms are linear processes during the transition between adolescence and young adulthood (Needham et al., 2010), it would be largely disadvantageous to use a design that is only appropriate when the growth process is irrelevant or known to be linear (Duncan et al., 2006). LGM identifies the process of change, and is used in analytical situations where describing the growth of a construct is of pivotal interest (Duncan et al., 2006), which is of central importance to the objectives of this thesis. As previously mentioned, the major objectives of the present thesis include examining individual trajectories of body mass and depressive symptoms as adolescents transition into young adulthood, examining if changes in body mass over time are associated with changes in depressive symptoms over time, and examining whether initial levels of body mass predict changes in depressive symptoms over time, and/or vice versa.

A second criticism to ACL approaches is that they fail to provide adequate generalization for three or more time points. When more than two time points are included in a model, the best an autoregressive technique can do is examine change scores between any two points in time. This constraint is particularly important with respect to the present study, because, as highlighted above, both body mass and depressive symptoms are assumed to be nonlinear processes. In order to identify a nonlinear process, a minimum of three data points are required. Thus, an analytical process that is able to adequately capture changes across three or more data points, as LGM is able to do, is required.

Lastly, as autoregressive models are only able to predict changes in the rank order of observations over time, they may fail to identify significant changes at the individual and group level if rank order stays the same between two time points. While the ACL approach may determine whether body mass at age 17 predicts depressive symptoms at age 19, it cannot provide any indication of whether body mass predicts individual-level change over many years in depressive symptoms, or whether the trajectories of these two health indicators are related. According to Duncan and colleagues, "an appropriate developmental model is one that not only describes a single individual's developmental

trajectory, but also captures individual differences in these trajectories over time" (p. 3; 2006). Using a person-centered approach, such as LGM, does provide this information. Using LGM, body mass level at one point in time can be used to predict *change* in depressive symptom scores across an extended period of time, and vice versa. Furthermore, it is possible in LGM to assess whether the trajectories of body mass and depressive symptoms are related to one another. Overall, utilizing a LGM approach will provide the best match for the developmentally-focused research objectives of the present study.

Research using a LGM approach to examine the developmental associations between body mass and depressive symptoms is limited. In fact, only two studies have utilized this analytic approach for this research question (Needham et al., 2010; Stice & Bearman, 2001). While these studies represent important extensions in research on body mass and depressive symptoms by adopting LGM, they suffer from a number of limitations. Most notably, Needham and colleagues (2010) did not focus on a specific developmental period. Examining four cycles over 15 years is a particularly long time period of investigation, making associations vulnerable to confounding effects of developmental periods and life transitions. At the other extreme, Stice and Bearman (2001) examined only three cycles of data over two years, with 10-month intervals between data collection points. Measuring at short intervals may not allow enough time between cycles to effectively examine changes during a particular developmental period such as adolescence, which spans over six years. No study using LGM has looked specifically at the adolescent period and its transition into young adulthood. This is an important limitation, as the later years of adolescence and early young adulthood involve significant changes in both body mass and depressive symptoms (Ge et al., 2006; Kemper et al., 1999).

In the present study, LGM was used to estimate individual trajectories of body mass and depressive symptoms, as well as examine the association between body mass and depressive symptoms, across five ages. This modeling strategy complements the key objectives identified in the plan of study. The first objective was to examine individual trajectories of body mass and depressive symptoms as adolescent's transition into young

adulthood. By using data from five ages (i.e., from ages 17, 19, 21, 23, and 25), this first objective will be met by using LGM to model separate individual, or univariate, trajectories for body mass and depressive symptoms. In addition to linear changes over time, nonlinear changes for both body mass and depressive symptoms will be examined. This will allow us to investigate whether changes in either health outcome are stable over time, or if they plateau over time within the developmental period.

The second objective was to examine if changes in body mass over time are associated with changes in depressive symptoms over time, and the third objective was to examine whether initial levels of body mass predict changes in depressive symptoms over time, or vice versa. LGM allows us to enter both univariate trajectories into a single dual-trajectory model (i.e., a multivariate model), where intercept and growth parameters are estimated for both body mass and depressive symptoms. Specifying this multivariate model allows us to examine if the rate of change for body mass is associated with the rate of change for depressive symptoms, thus assessing if changes in body mass are associated with changes in depressive symptoms. This multivariate model also allows us to examine if the intercept for body mass predicts the growth for depressive symptoms, thus assessing if initial levels of body mass predict changes in depressive symptoms over time (and vice versa with the intercept of depressive symptoms predicting the growth of body mass).

These analytical processes will be explained in greater detail in the results section. Overall however, it is clear that using an LGM analytical technique would be the best approach to study development, and more specifically, to investigate the key objectives of the present thesis.

3.3.2   Data screening

Since all BMI scores were derived from self-reported height and weight measures, there were some errors in both height and weight that needed to be examined prior to data analysis. Checks were used to flag improbable heights over time, as well as biologically implausible weight and BMI values. The following steps were taken to clean the height variable before computing BMI for each age. First, each individual's height trajectories

were calculated from all eight cycles of data collection (n = 2,488). An assumption was made that height could not decrease over time. Thus, any individual whose height decreased at any adjacent point across the eight cycles of data collection was flagged. One thousand and sixty-eight of such individuals were flagged for one data point (416 for two data points, 90 for three data points, and 9 for four data points). Next, each height trajectory was individually examined in order to determine the point (or points) that most likely deviate from the rest of the trajectory. Once this point was flagged, an equation was used to replace it by applying an estimation using adjacent heights and age at time of interview.

In addition, heights were imputed using the same equation if an individual provided data about weight, but not height. This was done as an attempt to maximize available weight data in order to compute BMI, and 27 such cases were identified. All analyses were rerun without these imputations, and similar results were found.

Weight was checked by deleting any biologically implausible values based on an individual's age and sex (Beck, Schaefer, Nace, et al., 2012). Three such deletions were made. BMI and depressive symptoms across all ages exhibited acceptable skewness and kurtosis [i.e., skewness < 3 and kurtosis < 10 – Kline, 2005] for structural equation modeling.

3.3.3   Additional statistical considerations/adjustments

3.3.3.1   Model fit

Factor loadings are random, as each individual has a slightly different factor loading (explained below). Since maximum likelihood functions were used as a result of random factor loadings, traditional model fit indices, such as Comparative Fit Index (CFI), Tucker Lewis Index (TLI), and Root Mean Square Error of Approximation (RMSEA) could not be used. Instead, Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), and loglikelihood tests were used to compare models. More information on these fit indices may be found in (Burnham & Anderson, 2004). A loglikelihood chi square difference test was determined to be significant at the $\alpha = .01$ level.

3.3.3.2   Adjustment for variation in age at baseline

Ideally, the application of LGM is dependent largely on data collection whereby individuals are observed at the same point in time (Muthén, 2002). However, participants' ages ranged from 14.9 to 18.2 years at baseline. To calculate these age differences at time of interview for baseline estimates, participants' age at baseline was subtracted by the median age (assumed to be 17, for ease of interpretability). Since participants' ages at baseline in this study varied, these variations from the median age at baseline were controlled for, making the cohort homogenous with respect to starting age.

3.3.3.3   Adjustment for departure from balance design

An ideal application of LGM is also dependent on having equal spacing of assessments for all individuals (Muthén, 2002). However, for all cycles in the NLSCY, data collection spanned over two years. Thus, random factor loadings were used in each model in order to account for the variation between interview times among participants and across ages. For each age, factor loadings represented a participant's exact age at time of interview (in years) subtracted by the assumed median age at baseline (i.e., 17). Thus, at age 19, factor loadings would have an approximate mid-point of 2 (ranging between 1 and 3), at age 21, factor loadings would have an approximate mid-point of 4 (ranging between 3 and 5); at age 23, factor loadings would have an approximate mid-point of 6 (ranging between 5 and 7); and at age 25, factor loadings would have an approximate mid-point of 8 (ranging between 7 and 9). The advantage of using this method is that we do not need to make an assumption that every participant was measured at the exact same time. Instead we can statistically allow measurement periods for each person across each cycle, to vary, as they did in reality.

3.3.3.4   Missing data

There are two major forms of missing data in the present analysis. First, not everyone who was originally sampled as a part of the original cohort at the first cycle of NLSCY data collection participated at the fourth cycle. Since analyses for the present study began at this cycle, when participants turned 16-17, only participants who responded at age 16-17 were included from the original cohort of participants. Thus, for

the present cohort, cross-sectional weights were computed by dividing each person's baseline cross-sectional survey weighting by the cohorts' average survey weighting. Cross-sectional weights were used in all analyses so that estimates at baseline represent the Canadian population of 16-17 year olds as of January 1, 2001. Given the complexity in the NLSCY sampling design with regard to stratification, multiple stages of selection, and unequal probabilities of selection of respondents, cross-sectional weights must be used in order to avoid bias in survey estimates and analyses (Statistics Canada, 2009).

The second form of missing data was due to the longitudinal sequential design of the NLSCY. For example, after baseline, some participants did not complete the survey at all time periods. Missing data are assumed to be independent from values of the study measures; thus, it is reasonable to assume that these data are missing at random (Little & Rubin, 2002). Mplus offers superior handling of missing data (using Full Information Maximum Likelihood algorithm; FIML), for missing data points and for missing cases due to attrition. FIML uses all available data to generate maximum-likelihood estimates consisting of a vector of means and a covariance matrix among all variables in the data set (Wothke, 1996). FIML retains cases missing one or more cycles, and therefore avoids the use of biased parameter estimates that can occur with pair-wise or list-wise deletion (Arbuckle & Wothke, 1999; Schafer & Graham, 2002).

3.3.3.5   Power/sample size calculations

Very little is known about the exact estimates needed to generate strong power calculations for LGM because these types of analytic models are still considered relatively new techniques (Muthén, 2002). Furthermore, because of the complexity of the present model, as well as the large number of parameter estimates that are expected, more general guidelines identified by the literature for SEM will be used to argue a sufficiently large enough sample size. In general, the literature suggests that having about 200 subjects per group provides sufficient statistical power needed for any LGM analysis (Hoyle, 1995; Ullman, 1996). Since a separate growth model will be estimated for each sex on both health outcomes, a total sample size of 400 (i.e., 200x2) is needed for sufficient statistical power for one model. The sample size from the original cohort at baseline is 1,895, which indicates a large enough sample of adolescents and young adults

to conduct an in-depth analysis of body mass and depressive symptom trajectories, separately for males and females.

Chapter 4

4 Results

4.1 Descriptive results

This chapter begins with a discussion of the characteristics of the sample, including the average BMI and CES-D score at each age, as well as correlations between BMI and CES-D score at each age. These descriptive statistics will be presented separately for males and females. Following this, the findings from each research objective will be presented. As previously mentioned, the objectives of this study are to: 1) examine individual trajectories of body mass and depressive symptoms as adolescents transition into young adulthood; 2) examine if changes in body mass over time are associated with changes in depressive symptoms over time; 3) examine whether initial levels of body mass predict changes in depressive symptoms over time, and/or vice versa; and 4) examine whether any of the above associations differ across sex.

The total sample size for this study was 1,895 (47.9% female). Table 3 outlines the means and standard deviations for BMI and CES-D score across the five ages. As expected, for both males and females, average BMI increased from adolescence to young adulthood. For males, average BMI at age 17 was 23.0 and steadily increased by about 0.6 points at each age until age 23, where average BMI plateaus at around 24.9. When males were 25 years old, the average BMI increased by about only 0.2 points. Standard deviations in BMI for males tended to stay relatively consistent over time (i.e., at around 3.9 points). In females, compared to males, there was a more steady increase in average BMI over time. At age 17, the average BMI for females was 22.4. This average steadily increased to 22.8 at 19 years, 23.6 at 21 years, 23.9 at 23 years, and 24.5 at 25 years. Average BMI for females was consistently lower than males at every age. Furthermore, there were greater variances in average BMI for females than males at every age. Standard deviations in BMI for females tended to increase over ages, from 4.2 to 5.4.

Table 3: Means and standard deviations of study measures across five ages.

Variable	*Age 16* *M (SD)*	*Age 19* *M (SD)*	*Age 21* *M (SD)*	*Age 23* *M (SD)*	*Age 25* *M (SD)*
BMI					
Males	22.97 (3.92)	23.64 (3.96)	24.33 (3.92)	24.89 (3.91)	25.05 (4.05)
Females	22.43 (4.22)	22.77 (4.90)	23.57 (4.60)	23.86 (5.42)	24.55 (5.41)
CES-D					
Males	8.02 (5.72)	7.39 (5.58)	4.40 (4.59)	3.71 (4.04)	4.10 (4.37)
Females	10.29 (6.41)	8.49 (6.11)	5.91 (5.96)	5.13 (4.93)	4.91 (5.38)
Age	16.9y (7m)	18.7y (7m)	20.8y (7m)	22.8y (7m)	24.8y (7m)

Note. BMI: Body mass index. CES-D: Centre for Epidemiological Studies- Depression Scale. Higher scores for variables indicate higher BMI and CES-D scores. Scores reflect adjusted means.

For both males and females, average CES-D score decreased from adolescence to young adulthood. For males, average in CES-D score (out of a possible 36 points) was 8.0 at age 17. The average slightly decreased at age 19 to 7.4, and then drastically decreased at age 21 to 4.4. Following these ages, the average CES-D score plateaus at 3.7 and 4.1 at ages 23 and 25, respectively. Standard deviations in CES-D score for males tended to decrease over ages, from 5.7 to 4.1. For females, the average in CES-D score was higher than the average in CES-D score for males at every age. In addition, the average in CES-D score demonstrated a more gradual decline across ages for females than they did for males. At age 17, the average in CES-D score for females was 10.3. This average decreased to 8.5 at age 19, 5.9 at age 21, 5.1 at age 23, and 4.9 at age 25. Although standard deviations in CES-D score for females also tended to decrease across ages, these standard deviations were greater for females than they were for males, at all ages.

Table 4 outlines the correlations between BMI and CES-D score at each age, separately for males and females. BMI was highly and significantly correlated across ages, for both males and females. Adjacent age correlations for BMI were highest, with Pearson correlation coefficients ranging from .76 to .84 for males, and from .74 to .84 for females (all p's < .001). However, BMI at age 17 was still highly correlated with BMI at age 25 (.62 for males, and .67 for females; all p's < .001).

CES-D scores were also highly correlated and statistically significant across ages for both males and females, although to a lesser degree than BMI. As was the case with BMI, adjacent age correlations for CES-D scores was highest, with Pearson correlation coefficients ranging from .46 to .58 for males, and from .40 to .46 for females (all p's < .001). CES-D scores were also highly correlated between age 17 and age 25, with Pearson correlation coefficients of .43 for males and .38 for females (all p's < .001). Adjacent age correlations for CES-D scores were consistently higher for males than females, with the greatest difference between sexes occurring between ages 17 and 19, where CES-D scores were correlated at .58 for males and .40 for females.

The correlation between BMI and CES-D score across ages was drastically different for males and females. For males, BMI and CES-D score correlations at the first two ages were not statistically significant (Pearson correlation coefficient of .02 at age 17 and .04 at age 19; all p's > .05). However, during the next three ages, correlations were negative and statistically significant. These correlations also increased in magnitude across ages: -.08 at age 21, -.11 at age 23, and -.14 at age 25 (all p's < .01). The largest correlations for males were between BMI at age 21 and CES-D score at age 23 (-.21, $p <$ .001), between BMI at age 21 and CES-D score at age 25 (-.21, $p <$ .001), and between BMI at age 25 and CES-D score at age 23 (-.18, $p <$ .001).

For females, a completely different pattern of correlations emerged. BMI and CES-D score correlations within all ages were positive and statistically significant. The correlation magnitudes increased until age 21, with Pearson correlation coefficients of .08 at age 17 ($p <$ .05), .17 at age 19 ($p <$ .001), and .22 at age 21 ($p <$ .001). Following this peak, correlation magnitudes decrease slightly, with correlation coefficients of .08 at age

23 ($p < .05$), and .14 at age 25 ($p < .01$). The largest correlations for females were between BMI at age 17 and CES-D score at age 19 (.30, $p < .001$), between BMI at age 19 and CES-D score at age 21 (.29, $p < .001$), and between BMI at age 17 and CES-D score at age 21 (.25, $p < .001$).

The most striking sex differences between BMI score and CES-D score correlations were evident for the last three ages, where for males correlations were consistently negative and statistically significant, and for females, correlations were consistently positive and statistically significant. For example, the correlation between BMI score at age 21 and CES-D score at age 23 was -.21 for males and .13 for females (all p's < .01).

Table 4: Correlations between BMI and CES-D score across five ages (males/females).

Variable	BMI 19 years	BMI 21 years	BMI 23 years	BMI 25 years	CES-D 17 years	CES-D 19 years	CES-D 21 years	CES-D 23 years	CES-D 25 years
BMI 17 years	.76*** / .84***	.67*** / .79***	.68*** / .74***	.62*** / .67***	.02 / .08*	.18*** / .30***	-.03 / .25***	.02 / .04	-.04 / .13**
BMI 19 years	-	.80*** / .76***	.73*** / .74***	.74*** / .61***	-.04 / .14**	.04 / .17***	-.03 / .29***	-.10* / .14**	-.08* / .13**
BMI 21 years	-	-	.84*** / .81***	.76*** / .70***	-.11* / .14**	-.08 / .23***	-.08** / .22***	-.21*** / .13**	-.21*** / .18***
BMI 23 years	-	-	-	.81*** / .74***	-.06 / .10*	-.02 / .24***	-.10* / .24***	-.11** / .08*	-.15*** / .03
BMI 25 years	-	-	-	-	-.14**/.07	-.04 / .09*	-.12** / .14**	-.18*** / .14**	-.14** / .14**
CES-D 17 years	-	-	-	-	-	.58*** / .40***	.45*** / .33***	.48*** / .36***	.43*** / .38***
CES-D 19 years	-	-	-	-	-	-	.46*** / .42***	.44*** / .26***	.35*** / .22***
CES-D 21 years	-	-	-	-	-	-	-	.57*** / .46***	.42*** / .30***
CES-D 23 years	-	-	-	-	-	-	-	-	.57*** / .41***
CES-D 25 years	-	-	-	-	-	-	-	-	-

Note. BMI: Body mass index. CES-D: Centre for Epidemiological Studies- Depression Scale. Higher scores for variables indicate higher depressive symptoms and frequency of health risk behaviors.
† $p < .06$. * $p < .05$. ** $p < .01$. ***

4.2   Growth trajectories

4.2.1   Objective 1: Univariate growth trajectories

The first objective of this study was to examine individual trajectories of body mass and depressive symptoms as adolescents transition into young adulthood. This was done by using data from five ages (i.e., age 17, 19, 21, 23, and 25) to model separate univariate trajectories for BMI and CES-D scores.

The intercept and growth factors are each defined by setting factor loadings to specify time points in the model. Since the intercept is constant over time, it is modeled by constraining the loadings for each time point to equal 1. For each of the univariate trajectories in the present thesis, the intercept factor loading was set to 1 at all time points. This was done in order to estimate the starting point for each trajectory as the baseline (i.e., age 17).

The growth factor in a LGM represents change, and thus is modeled by a successive increase in factor loadings over time. For example, a linear increase in BMI over five time points may be modeled by constraining the loadings of the five time points to 0, 1, 2, 3, and 4, respectively. Growth factor loadings in the present thesis were set to 0 at baseline (i.e., age 17), and set to random factor loadings, with an approximate mid-point of 2 (at age 19), 4 (at age 21), 6 (at age 23), and 8 (at age 25). The exact growth factor loadings reflect the variation in interview times among participants across ages. They average at approximately 2, 4, 6, and 8 in order to reflect the approximate number of years each interview took place after the baseline interview at age 17. For example, a growth factor loading of 2 at age 19 represents an individual who was interviewed exactly two years after the first interview. As previously mentioned, the advantage of using random factor loadings is that they allow measurement periods for each person, across each age point, to vary, as they did in reality.

Age at baseline (centered at 17) was entered as a covariate on the intercept for both the BMI trajectory and the CES-D score trajectory. Age was centered at 17 (median age) for baseline for ease of interpretability. As previously explained, age was entered as

a covariate for the intercept of both univariate growth models in order to control for the variations in age at baseline, thus making the cohort homogenous with respect to starting age. Homogenizing the cohort was necessary as LGM applications assume that all individuals are observed at the same point in time (Muthén & Muthén, 2004).

Separate univariate trajectories were identified for BMI and CES-D scores by first testing each health outcome on a linear growth model followed by a quadratic growth model. Quadratic associations were examined to determine if BMI and CES-D scores followed a nonlinear growth trajectory from adolescence into young adulthood. Nonlinearity was examined because, as previously mentioned, body mass increases rapidly over childhood and adolescence and then plateaus during young adulthood (Kuczmarski et al., 2002). Furthermore, depressive symptoms peak during late adolescence and then decrease and plateau across young adulthood (Ge et al., 2006). Thus, in order to examine these nonlinear growths, quadratic associations were investigated. Chi square difference tests, using log likelihoods, were conducted to compare the linear model to the quadratic model for each health outcome. The linear growth model was chosen for all behaviours unless the χ^2 difference test between the linear model and the quadratic model indicated a significant difference for the model at $p < .01$. The model with the lowest AIC and BIC values was determined to be the better fitting model of the two. The model with the linear growth factor was the more parsimonious choice for BMI, $\chi^2 diff$ (6) = 8.96, $p > .05$. In contrast, the model with the quadratic growth indicated a better fit than the linear model for CES-D scores, $\chi^2 diff$ (6) = 54.14, $p < .001$.

Once univariate models were identified, sex differences were assessed (objective 4). To do this, a chi square difference test was used to compare two sets of models: an unconstrained model and a constrained model. The unconstrained model is the original multi-group model in which parameters were estimated separately for each sex. This model produced different parameter estimates (i.e., mean and variance for both the intercept and growth) for males and females. The constrained model constrains the variance for both the intercept and growth to be equal between males and females, thus allowing the model to calculate the same estimates for both males and females without

differentiating on sex. This constrained model is equivalent to examining trajectories of BMI and CES-D scores for everyone in the sample. The chi square difference test indicates if estimates conducted by separating sex are statistically significantly different from estimates conducted without separating by sex. A significant chi square difference test here would indicate that estimates from each model are different enough from one another to statistically validate presenting results separately by sex (i.e., using the multi-group model identifying sex differences). The chi-square difference test indicated that there were significant differences between the unconstrained model and the constrained model for CES-D scores, $\chi^2 diff$ (3) = 28.89, $p < .001$. However, the chi-square difference test indicated that there were not any significant differences between the unconstrained model and the constrained model for BMI, $\chi^2 diff$ (2) = 5.67, $p = .059$.

Overall, chi square difference tests were used to determine the most parsimonious model for identifying the trajectory of BMI and CES-D scores, and to determine whether any sex differences exist in these trajectories. For BMI, the model with linear growth was most parsimonious, and there were no statistically significant sex differences for the trajectory of BMI. For CES-D scores, the model with a quadratic growth had a significantly better fit than the model with a linear growth alone, and there were significant sex differences for the trajectory of CES-D scores. Although, chi square difference tests indicated no sex differences, BMI trajectories will still be separated by sex for ease of interpretability and consistency across all research objectives.

Results of univariate trajectories are shown in Table 5. In addition, fitted estimates for the univariate trajectories are presented in Figure 2. According to the univariate models, average BMI at onset (i.e., age 17) starts at 23.3 for males and 22.5 for females, and increases linearly across the study period, for both boys and girls, although boys have slightly elevated initial levels and growth over time than girls. There was a statistically significant amount of variance in the intercept and growth of BMI, for both males and females, indicating a large amount of inter-individual differences in starting points and growth of BMI for males and females. Although both variances were significant, there were larger variances in BMI for females than males, in both the intercept and growth.

According to the univariate trajectories, males have an average CES-D score of 8.6 at onset (i.e., age 17), whereas females have an average CES-D score of 10.4 at age 17. While average BMI increased over time, average CES-D scores started out high at age 17 and decreased over time. The quadratic trend indicates that, for this developmental period, this decrease slows down and plateaus at about age 23. CES-D scores started out higher, decreased at a slower rate, and plateau later for females compared to males. Variances in the intercept of average CES-D scores were statistically significant for both males and females, although these variances were larger for females compared to males. For both sexes, variances were statistically nonsignificant for CES-D linear and quadratic growth, indicating that there were few individual-level differences in the decline and plateau of CES-D scores.

In summary, the first objective aimed to examine individual trajectories of body mass and depressive symptoms as adolescents transition into young adulthood, by modeling separate univariate trajectories for BMI and CES-D scores across five data points from age 17 to 25. Average BMI growth followed a positive linear trajectory for both males and females, with males having slightly (but not statistically significant) higher initial levels and growth in BMI, compared to females. Average CES-D scores decreased over time for both males and females. The trajectory for average CES-D scores over time was nonlinear, such that there was a gradual decrease from age 17 to age 21, and then CES-D scores tended to plateau into young adulthood. The trajectory for CES-D scores was significantly different for males and females, such that, on average, females had both a higher starting level of CES-D scores, as well as a slower decline in CES-D scores, compared to males. The variances for both trajectories, with respect to intercept, were statistically significant, meaning that there was a great deal of individual-level variation from average in the starting point of BMI and CES-D scores. Variances for the linear growth of BMI were also statistically significant for both males and females, indicating a great deal of individual-level variation in the linear rate of change of BMI. These variances were consistently larger for females than males. However, nonsignificant variances for both growth elements of CES-D trajectories indicate small variation in the rate of change of CES-D scores, for both males and females.

Table 5: Parameter estimates of the multi-group univariate growth models of BMI and CES-D scores, by sex.

Variable		Intercept, Mean (Variance)	Linear growth, Mean (Variance)	Quadratic growth, Mean (Variance)
BMI	Males	23.29*** (11.50***)	0.27*** (0.06**)	-
	Females	22.47*** (15.25***)	0.25*** (0.12***)	-
CES-D	Males	8.63*** (10.00***)	-1.54*** (0.45)	0.12*** (0.01)
	Females	10.42*** (11.91***)	-1.36*** (1.35)	0.08** (0.03)

Note. BMI = body mass index. CES-D: Centre for Epidemiological Studies- Depression Scale. The total sample was N = 1793. All models had covariances between intercept and growth parameters (and linear and quadratic parameters, when applicable), as well as a path from age at baseline to intercept of health outcome.
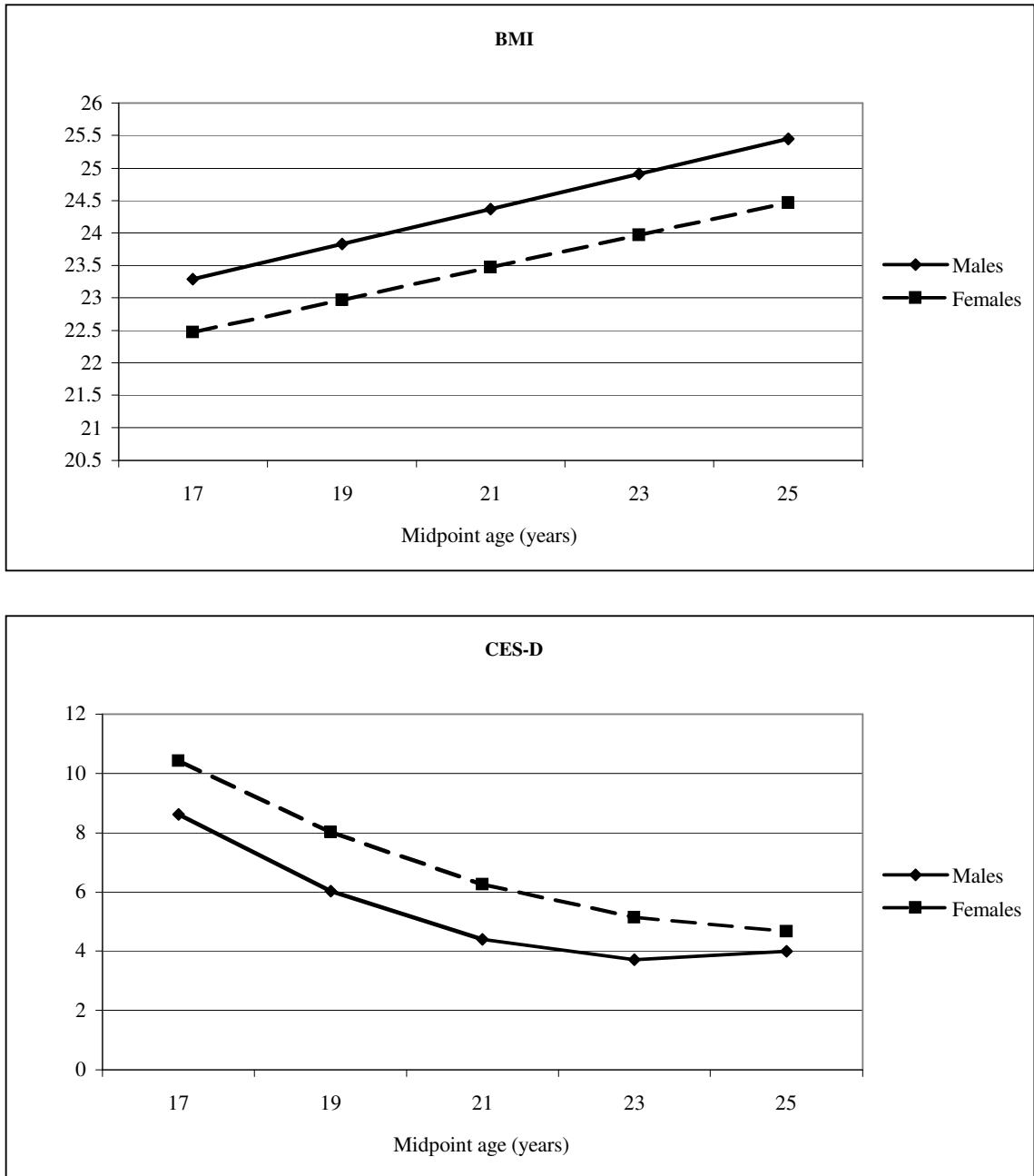
** $p < .01$. *** $p < .001$.

Figure 1: Fitted estimates for univariate trajectories.

4.2.2   Objectives 2 and 3: Multivariate growth trajectory

Objectives 2 and 3 will be presented together in the following section because they were both estimated within the same LGM. The second objective was to examine if changes in body mass over time were associated with changes in depressive symptoms over time.

Achieving this involved several analytical steps. First, the two separate univariate trajectories identified from the first objective (i.e., the linear multi-group BMI trajectory, and the quadratic multi-group CES-D scores trajectory) were both entered into a single dual-trajectory model (i.e., a multivariate model). This multivariate model estimated an intercept and growth for BMI, as well an intercept and growth for CES-D scores. Second, within this multivariate model, a path between the BMI and CES-D scores for each age was added, as well as a path between the intercept of BMI and the intercept of CES-D scores. These paths were added in order to control for contemporaneous associations between BMI and CES-D scores at each age. This control would ensure that any prospective associations are not simply due to the correlation of both health outcomes at each age (Stice & Bearman, 2001).

In addition, for both BMI and CES-D scores, a path was also added between the intercept to the growth parameter in order to control for baseline levels of each health outcome. Given the high stability of both health outcomes, this control would ensure that any changes seen in either BMI or CES-D scores over time were not due to their respective initial levels (Bradley et al., 2008).

Finally, a path was specified between the growth of BMI and the growth of CES-D scores. This path represents the covariance in the linear growth of both health outcomes, and allowed us to examine the second research objective of whether changes in BMI were associated with changes in CES-D scores, after controlling for contemporaneous associations at each age and initial levels of each health outcome.

The third objective was to examine whether initial levels of body mass predict changes in depressive symptoms over time, and/or vice versa. In order to do this, two

paths were simultaneously added to the same multivariate model: one in which the growth of CES-D scores was regressed onto the intercept of BMI, and one in which the growth of BMI was regressed onto the intercept of CES-D scores. These paths allowed us to examine whether the intercept of BMI predicted the growth, or rate of change, in CES-D scores, and whether the intercept of CES-D scores predicted the growth, or rate of change in BMI. More specifically, these two coefficients will estimate: 1) the degree to which initial levels of BMI at age 17 predict changes in the average decrease in CES-D scores over time; and 2) the degree to which initial level of CES-D scores at age 17 predict changes in the average increase of BMI over time.

Since age at baseline was significant in the BMI univariate trajectory, but not the CES-D scores univariate trajectory, a path from age at baseline was only specified to the intercept of BMI in the multivariate model. In addition, the variance for the quadratic growth term for CES-D scores was constrained to zero in the multivariate model since it was statistically nonsignificant in the univariate latent growth model.

Results from the multivariate latent growth model are presented in Figure 3, separately for males and females. For males, with regards to objective 2, the linear growth of BMI was not associated with the linear growth of CES-D scores ($p > .05$). For objective 3, the path from BMI intercept to CES-D scores linear growth was not significant, $\beta = -.018$, $p = .217$, indicating that average starting levels of BMI did not predict changes in CES-D scores over time, for males. However, the path from CES-D scores intercept to BMI linear growth was negative and significant, $\beta = -.016$, $p = .031$, indicating that, on average, adolescent males who had higher initial depressive symptoms reported a slower increase in body mass over time, than adolescent males who did not have higher initial depressive symptoms.

For females, with regards to objective 2, the linear growth of BMI was not associated with the linear growth of CES-D scores ($p > .05$). For objective 3, the path from BMI intercept to CES-D scores linear growth was negative and significant at a trend level, $\beta = -.026$, $p = .052$, indicating that, on average, adolescent girls who had higher initial body mass reported a slower decrease in depressive symptoms over time, than

adolescent girls who did not have higher initial body mass. However, the path from CES-D scores intercept to BMI linear growth was not significant, $\beta = -.009$, $p = .364$, indicating that, on average, starting levels of depressive symptoms did not predict linear changes in body mass over time, for females.

Variances in intercept and growth parameters for the multivariate model followed a similar pattern to variances for the univariate models. Namely, large and statistically significant variances were present in the intercept and linear growth of BMI, for both males and females, as well as the intercept of CES-D. In addition, all variances were larger in females than males.

4.2.3   Objective 4: Sex differences

As previously mentioned, the final objective was to examine whether associations between initial levels of body mass and changes in depressive symptoms, and vice versa, over time, vary across sex. In order to test whether the multivariate associations were invariant across sex, a multi-group analysis was performed in the same manner in which they were described for the univariate trajectories. Namely, invariance was tested by comparing the multivariate model in which all parameters were constrained to be equal between males and females to an unconstrained multivariate model in which all parameters were free to vary. The chi-square difference test indicated that there were significant differences between the unconstrained multivariate model and the constrained multivariate model, $\chi^2 diff\,(5) = 35.78$, $p < .001$, indicating that some of the associations for the dual trajectory model differ across sex. More specifically, the association between average initial BMI and average changes in CES-D scores, as well as the association between average initial CES-D scores and average changes in BMI, were both different for males and females (objective 3). However, the association between the growth of BMI and the growth of CES-D scores were not different between males and females (objective 2). As mentioned above, chi-square difference tests for univariate models indicated that there were significant differences between the unconstrained model and the constrained model for CES-D scores, but not for BMI (objective 1).

Males   **Females**
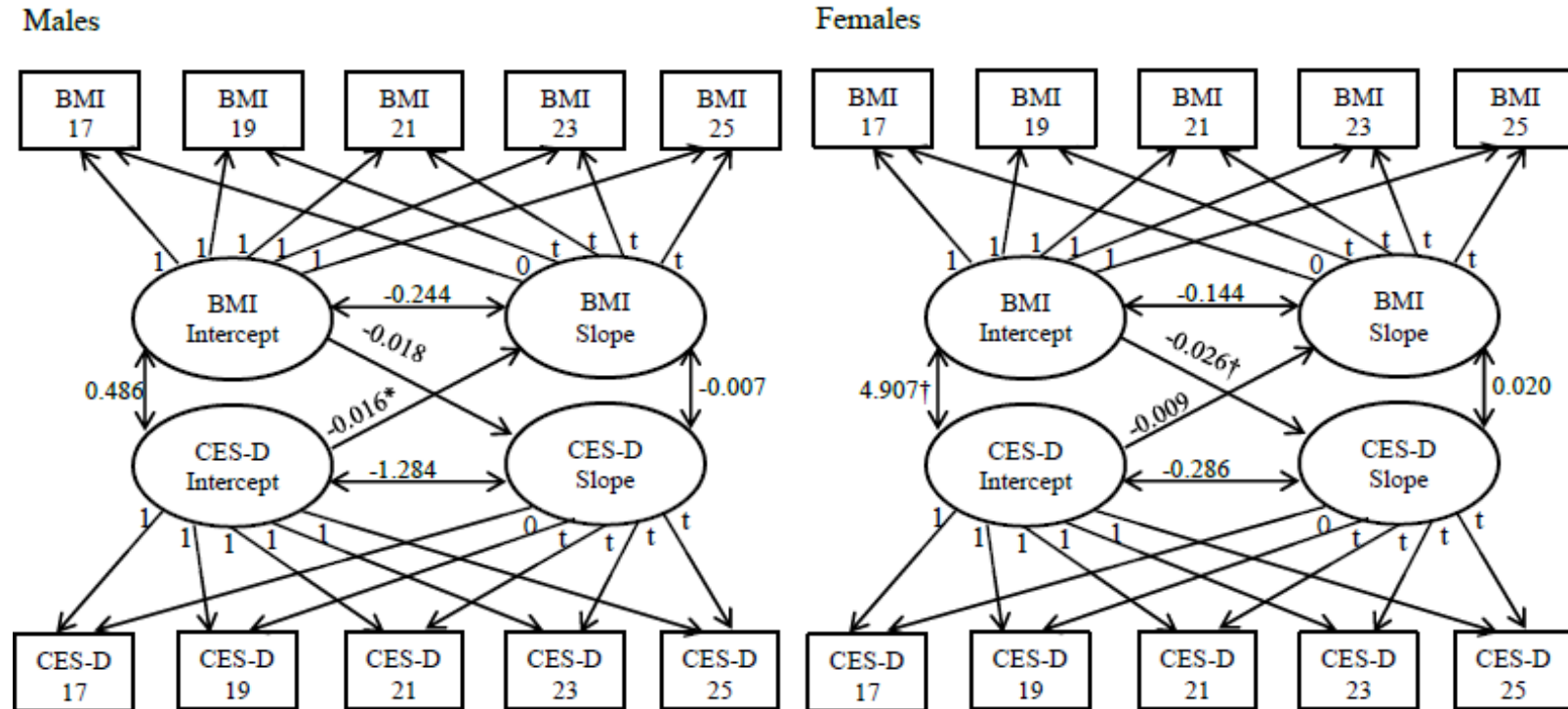
Note: BMI = body mass index. CES-D = Centre for Epidemiological Studies- Depression Scale. Multivariate latent growth model of BMI and CES-D across five ages. All values are unstandardized coefficients (as standardized coefficients cannot be estimated using random factor loadings). Not all residuals are shown. Quadratic variance was constrained to zero. Covariances between BMI and CES-D within each age also are not shown (e.g., BMI at age 17 and CES-D at age 17). Double-arrows refer to covariances among the factors. Although not shown, a path from age at baseline was added to the intercept of BMI.

† $p < .06$. * $p < .05$. ** $p < .01$.

Figure 2: Multivariate model testing bidirectional pathways between BMI and CES-D.

Chapter 5

5   Discussion

This chapter begins with a summary of the results. The summary includes a brief overview of the results, focusing on the main findings from the univariate and multivariate analyses pertaining to the study objectives. Following this summary, the results for each objective are discussed in context with previous research on adolescents and young adults. This discussion will provide a rationale and conceptual understanding of the associations examined in the present thesis, as well as the important public health implications of this study. The strengths and limitations of the current study, as well as the important conceptual, methodological, and analytical suggestions for future research are discussed next. Finally, this chapter concludes with an overall summary of the present thesis and the importance of research in this area.

5.1   Summary of main findings

The main goal of this study was to identify the growth trajectories of body mass and depressive symptoms as adolescent's transition into young adults, and to examine the associations between these growth trajectories. This main goal is composed of four specific objectives. These objectives were to: 1) examine individual trajectories of body mass and depressive symptoms as adolescent's transition into young adults; 2) examine if changes in body mass over time were associated with changes in depressive symptoms over time; 3) examine whether initial levels of body mass predicted changes in depressive symptoms over time, and/or vice versa; and 4) examine whether any of the above associations differed by sex.

With respect to the first objective of examining individual trajectories of body mass and depressive symptoms, results indicated that the average BMI trajectory increased linearly from age 17 to 25, for both males and females. Average BMI levels started out higher and increased at a faster rate for males than females, although this difference was not statistically significant. There was a statistically significant level of variation in both the starting levels and linear growth of BMI, especially for females,

indicating that a great deal of individual-level variation exists in BMI trajectories. Furthermore, although there were increases over time in average BMI for both males and females, it is important to note that average BMI was within the normal weight range at all ages for both males and females, with the exception that average BMI for males at 25 years of age was within the overweight range (Kuczmarski et al., 2002).

The model-estimated trajectory for CES-D scores was negative and quadratic. That is, CES-D scores started out higher, and decreased until about age 21 where scores plateaud for the next two ages. The trajectory for CES-D scores was significantly different for males and females, such that females had both a higher starting level of CES-D scores, and a slower decline in CES-D scores, compared to males. Average CES-D scores were within the minimal depressive symptoms range for both males and females, at all ages (Poulin et al., 2005). Statistically significant variance was only present in the intercept of CES-D scores for both males and females, and was greater for females, indicating a high degree of individual-level variation in starting levels of CES-D scores, particularly for females. On the other hand, variances in linear or quadratic growth for the CES-D trajectory were not statistically significant, indicating that the average decline and plateau for CES-D scores are relatively consistent across individuals.

With respect to the second objective of examining if changes in BMI were associated with changes in CES-D scores, results showed that the linear growth of BMI was not associated with the linear growth of CES-D scores, for either males or females. That is, the rate of change in BMI was not associated with the rate of change in CES-D scores.

For the third objective examining whether initial levels of body mass predicted changes in depressive symptoms over time, results showed that starting levels of BMI predicted change in CES-D scores over time for females, but not males. On average, adolescent girls who had higher initial BMI levels reported a slower decrease in CES-D scores over time, than adolescent girls who did not have higher initial BMI levels. This association was significant at a trend level. Furthermore, starting levels of CES-D scores predicted change in BMI over time for males, but not females. On average, adolescent

males who had higher initial CES-D scores reported a slower increase in BMI over time, than adolescent males who did not have higher initial CES-D scores.

Finally, with respect to the fourth objective, examining whether any of the above associations differ across sex, results from chi square difference tests indicated that there were significant differences between males and females in the univariate model for CES-D scores, and the multivariate model with BMI and CES-D scores. However, there were no significant gender differences in the univariate model for BMI.

5.2 Rationale for results

5.2.1 Objective 1

The results from the first objective were expected. As mentioned in the hypotheses above, we expected trajectories of body mass to increase over time, and trajectories of depressive symptoms to decrease and plateau from adolescence to young adulthood. We also expected that, at all ages, body mass would be higher for males than females, and depressive symptoms would be higher for females than males.

According to the CDC growth charts, body mass increases from childhood to age 20 are expected, and these increases follow a normal physical growth pattern (Kuczmarski et al., 2002). Body mass at these developmental periods is also expected to be higher for males than females, and reflects the fact that males generally have greater muscle mass than females (Tanner, 1989).

Research on depressive symptoms in adolescents and young adults indicates that during early adolescence, depressive symptoms increase, until they peak in late adolescence, at around 17 or 18 years of age (Ge et al., 2006). This peak in depressive symptoms during late adolescence is expected, as adolescence is a period of heightened emotions (Dahl, 2004). Following this peak, depressive symptoms tend to decrease into young adulthood (Needham et al., 2010). Adolescents tend to develop better emotion-regulation abilities as they age and develop into young adults (Kemper et al., 1999), thus accounting for the lower depressive symptoms we see over time. Although depressive

symptoms tend to generally decrease from adolescence into young adulthood, rates are still higher in females compared to males (Locke & Newcomb, 2001).

5.2.2   Objective 2

There is a paucity of previous multivariate LGM research on body mass and depressive symptoms, especially during adolescence and young adulthood. For these reasons, the second, third, and fourth objectives were examined in an exploratory way.

With respect to the second objective, changes in body mass over time were not related to changes in depressive symptoms over time. As there were no studies in the previous literature that examined this particular research question, inferences cannot conclusively be made from the results presented here. However, because body mass and depressive symptoms tend to follow very different individual trajectories from late adolescence to young adulthood (i.e., body mass increases linearly and depressive symptoms decrease nonlinearly), it may not be surprising that changes in one health outcome over time are unrelated to changes in the other health outcome over time. Regardless, further research needs to be done in order to validate the findings with respect to this second objective.

5.2.3   Objective 3

5.2.3.1   Body mass affecting depressive symptoms

Results from the third objective will be divided into two components: baseline body mass predicting change in depressive symptoms, and baseline depressive symptoms predicting change in body mass. For body mass affecting depressive symptoms in males, we found that, on average, BMI at age 17 did not predict growth in CES-D scores over time. This finding is similar to what was mentioned in the literature reviewed above; that, both studies which focused on the transition between adolescence and young adulthood, and investigated associations in both males and females, reported nonsignificant associations between body mass and subsequent depressive symptoms for males (Goodman & Whitaker, 2002; Merten et al., 2008).

Body mass during adolescence is largely driven by muscle mass in males (Tanner, 1989). This increased muscle mass may be explained through greater physical activity, including greater participation in organized sports and strength training. Furthermore, popularity in adolescence is attributed to athletics for males (Chase & Dummer, 1992). Thus, it would be highly unlikely that, for the general population of adolescent males, higher body mass would be associated with an increase in subsequent depressive symptoms (Anderson, Cohen, Naumova, Jacques, & Must, 2007).

For females however, the story is quite different. The present study found that, on average, adolescent girls with higher BMI at age 17 had a slower decline in CES-D scores compared to their peers. That is, although on average there was a reduction in depressive symptoms over time, females with higher body mass at age 17 were, on average, much slower at reducing their depressive symptoms than females without higher body mass at age 17; indicating that at each age, these females had higher depressive symptoms on average than their peers.

These results are fairly consistent with the pattern of findings reported in the literature review section pertaining to studies examining body mass affecting depressive symptoms. More specifically, for female adolescents transitioning to young adulthood, body mass may lead to higher levels of depressive symptomology. Merten and colleagues (2008), for example, examined the association between body mass at age 12-18 and depressive symptoms five years later and found that, among females, obesity in adolescence was associated with higher depressive symptoms in young adulthood. Similarly, Boutelle and colleagues (2010) found that for adolescent girls, obesity predicted depressive symptoms one year later.

Whereas popularity in adolescence is attributed to athletics for males, it is attributed to appearance for females (Chase & Dummer, 1992). Because females tend to place a great deal of subjective importance on their physical appearance (Wadden & Stunkard, 1987), female adolescents with higher body mass may be especially at risk for developing subsequent depressive symptoms through the internalization of biased attitudes (Boutelle et al., 2010). This internalization of biased attitudes may be the most

relevant causal pathway to explain the association between higher body mass and higher subsequent levels of depressive symptoms for females across adolescence and young adulthood.

Although the internalization of biased attitudes is one potential causal pathway explaining the association between initial body mass and subsequent depressive symptoms for females, there are many other possible causal pathways, as explained in the literature review. Functional impairment in females with higher body mass may also lead them to develop higher depressive symptoms compared to their peers (Bornstein et al., 2006; Lago et al., 2007). Physical activity tends to decline for adolescent girls, and this inactivity may prospectively lead to the development of higher depressive symptoms over time (Brown et al., 2005; Camacho et al., 1991; Farmer et al., 1988; Lampinen et al., 2000; Paffenbarger et al., 1994; Strawbridge et al., 2002).

While weight-teasing and neurobiological causal pathways to depressive symptoms may also play a role, they may not be as prominent in the present study because of the age-group studied, as well as the conceptualization of body mass and depressive symptoms. Weight-teasing tends to occur in childhood and young adolescence and rarely as much as older adolescents age into young adults (Hill, 2005).

Furthermore, neurobiological causal pathways may play a role in the association between obesity classification and subsequent clinically significant depressive symptoms, but not in the association between high BMI and subsequent depressive symptoms. For example, obesity, but not necessarily high BMI, has been shown to predict subsequent depression through inflammatory pathways (Emery et al., 2007; Shoelson et al., 2007), HPA dysregulation (Pasquali et al., 2006; Walker, 2001), and increased risk of diabetes mellitus (Ajilore et al., 2007).

5.2.3.2   Depressive symptoms affecting body mass

With regards to the opposite direction of association (i.e.. depressive symptoms affecting body mass), results indicated that, on average, high initial CES-D scores did not lead to higher BMI growth in males or females. Although we found that depressive

symptoms did not predict higher body mass in either males or females, the reasons for this may be different. For girls, the association between depressive symptoms and subsequent body mass may only be significant when considering clinically significant depressive symptoms, which were not considered in the present study. For example, Anderson, Cohen, Naumova, and Must, 2006 reported that a MDD diagnosis in childhood and adolescence predicted a steeper increase in weight for females.

In addition, dose-response studies have reported that the number of times an adolescent meets clinical criteria for a major depression diagnosis during adolescence predicts an increased risk of obesity in young adulthood (Richardson, Davis, Poulton, et al., 2003). These recurrent, or persistent, episodes of depression are much more common for females than males (Richardson et al., 2003), thus making it more likely for depression, when diagnosed at a clinical level, to lead to increases in body mass for females.

In the literature review above, studies that investigated associations between depressive symptoms and subsequent body mass in both males and females reported nonsignificant associations for males (Goodman & Whitaker, 2002; Tanofsky-Kraff et al., 2006). Furthermore, in line with our findings, there is some evidence that males are inclined to lose instead of gain weight when depressed (e.g., Carpenter, Hasin, Allison, & Faith, 2000). Carpenter and colleagues (2000) found that underweight status in men was associated with an increased risk of clinical depression and suicidal tendencies. In addition, using a community-based study of sixth grade students, Rhew and colleagues (2008) found that adolescent boys with clinically significant depressive symptoms at baseline reported lower subsequent BMI one year later, compared to their non-depressed peers. Finally, there has also been some cross-sectional research supporting an inverse relationship between depressive symptoms and body mass among adult men (Crisp & McGuiness, 1975; Crisp, Queenan, Sittampaln, & Harris, 1980; Kittel, Rustin, Dramaix, et al., 1978). Similarly, the correlational analyses of the present study showed significant and negative relations between body mass and depressive symptoms for adolescent and young adult males.

As previously mentioned, body mass during adolescence is largely driven by muscle mass for boys (Tanner, 1989), partially explained by greater physical activity and participation in organized sports. Popularity for adolescent boys is largely attributed to athletics and organized sport participation (Chase & Dummer, 1992) because males tend to prefer a large and muscular body as opposed to a thin one (Herva et al., 2006). Boys with depressive symptoms may be less likely to increase their body mass through participation in physical and muscle-building activities because these activities occur in highly social contexts during adolescence. Depressive symptoms also may be associated with a lack of athletic ability in boys, which may be indexed by a low body mass. Furthermore, a moderational process may occur, such that depressed males start out with lower body mass (Carpenter et al., 2000), and also tend to gain less weight over time (Barefoot et al., 1998).

Finally, for both males and females, the heterogeneity of depressive symptoms may make it difficult to prospectively affect increases in body mass. More specifically, depressive symptoms include either increased or decreased appetite, and there may be a sub-sample of depressed adolescents who report losing weight, or a failure to gain weight over time as opposed to gaining weight (Kendler, Eaves, Walters, et al., 1996). This inherent heterogeneity in the manifestation of depressive symptoms may confound associations between depressive symptoms and subsequent body mass, for both males and females.

5.3   Implications

The results of the present study have many academic and public health implications. One major such implication is the importance of considering sex in the associations between body mass and depressive symptoms in adolescents and young adults. These associations differ greatly for males and females, and these differences need to be taken into consideration in future research, as well as within a public health perspective. Females tend to place a greater importance on appearance and thinness than males, and they are also more like to experience negative feelings, such as depressive symptoms, associated with their weight status (Wadden & Stunkard, 1987). Thus for females, higher body mass could be indicative of future mental health problem. For males

however, it is particularly important to pay attention to lower weight status and depressive symptoms. Depressed males may be failing to attain the proper nutrients and exercise they need in order to build muscle mass. Thus, depressive symptoms in boys may lead to a failure to fully physically develop.

A population-based strategy targeting these two health outcomes would be most effective if it focused on preventing depressive symptoms in adolescent boys, and preventing overweight conditions in adolescent girls. For males, attenuating the risk factors for depressive symptoms early in the lifespan is critically important. In addition, males with higher depressive symptoms should be closely monitored with regards to diet and physical exercise in order to ensure proper development into young adulthood.

Since higher BMI levels in adolescent girls had an effect on their rate of change in CES-D scores as young adults, it is important for adolescent girls to take the necessary steps to achieving healthy body mass during the teenage years. Weight-related changes can be promoted by parental and societal influences. Parents of teenagers can help in many ways. First, since they are the primary models in developing the food preferences and habits for their household, they can begin by evaluating their own lifestyles in terms of health-related quality (Benton, 2004; Merten et al., 2008). Also, by practicing authoritative parenting (Baumrind, 1966), parents can promote positive self-regulation (Patock-Peckham, Cheong, Balhorn, & Nagoshi, 2001). This positive self-regulation in their teens allows them make healthier food and activity choices of their own, while still having guidance and monitoring from their parents (Merten et al., 2008).

On a societal level, interventions need to be developed addressing the discrimination of individuals with higher body mass in educational, professional, and social settings (Merten et al., 2008). These interventions are particularly important for women, as research has shown that recruiters and employers judge these individuals capabilities based on weight-related biases (Ding & Bornhop, 2005; Tunceli & Williams, 2006). Finally, school administrators and directors need to be aware of the detrimental effect that poor nutrition and exercise habits on an adolescent's psychological well-being over time (Merten et al., 2008).

Care providers should be aware of the extent to which body mass and depressive symptoms interact in some individuals. More specifically, they should be alert to the possibility of depressive symptoms in their patients with obesity, and should screen for these symptoms appropriately, as well as monitor their changes over time (Faith et al., 2011). Health care providers should also be aware of and monitor weight changes in patients that present with clinically significant depressive symptoms.

5.4   Strengths

The major strength of the present study was that it addressed the majority of within-study limitations of past research. These include: lack of consistency in measuring body mass and depressive symptoms over time, failure to assess longitudinal change in outcomes, using only two waves of data, failure to examine reciprocal relationships, and a lack of transitional research between adolescence and young adulthood. The present study used the same measures to assess body mass and depressive symptoms across all five ages. That is, at all ages, self-reported height and weight were used to calculate BMI, and self-reported CES-D scores were used to measure depressive symptoms. We assessed longitudinal change in both health outcomes by controlling for baseline levels of each outcome during analytical modeling. In order to effectively assess growth over time in both outcomes, we used five data-points over an eight-year period. The present study examined, simultaneously, the reciprocal prospective associations between body mass and depressive symptoms. Finally, in order to gain an understanding of how these developmental processes are associated as adolescents age, we assessed the prospective associations between these two health outcomes during the transition from adolescence to young adulthood.

In addition, strong conceptual, methodological, and analytical justifications were made throughout the methods section in order to contribute to a level of consistency that will ease cross-study comparisons in the future. First, late adolescence was chosen as a starting point because depressive symptoms in earlier developmental periods has a different pattern of symptoms (Carlson & Kashani, 1988; Ryan et al., 1987; Mitchell et al., 1988) and correlates (Jaffee et al., 2002) compared to depressive symptoms in late adolescence and young adulthood. Although, in order to fully capture young adulthood,

an ideal follow-up period would have been until age 30 (Rindfuss, 1991), the constraints of the data source used only allowed examination up to age 25. The NLSCY was chosen because it is the largest population-based longitudinal study of Canadian children that follows their development and well-being from birth to early adulthood (Statistics Canada, 2009).

Second, BMI was kept as a continuous variable in order to take a more inclusive population-based approach to body mass. Similarly, CES-D scores were measured as a continuous variable in order to capture heightened depressive symptoms in individuals who do not meet criteria for clinically significant depressive symptoms.

Third, in order to more accurately capture the developmental objectives of the study, the analytical procedure of LGM was used as opposed to an autoregressive modeling approach. No study to date has used LGM to examine, specifically, the adolescent period and its transition into young adulthood. This is an important limitation, as the later years of adolescence and early young adulthood involve significant changes in both body mass and depressive symptoms (Ge et al., 2006; Kemper et al., 1999).

Finally, many steps were taken during the data screening and analysis phase to ensure validity in results. These steps included: using checks to flag and correct improbable height and BMI over time; adjusting for variation in age at baseline; adjusting for the variation in interview times among participants and across ages; applying sample weights so that estimates represent the Canadian population of 16-17 year olds as of January 1, 2001; and using FIML to handle missing data by using all available data to generate maximum-likelihood estimates.

5.5 Limitations

Although the present thesis had major strengths over previous studies, there were also a few limitations. These limitations include: using BMI as a proxy for adiposity; use of self-reported height and weight to calculate BMI; the low response rate for both BMI and CES-D; and follow-up period and length of intervals. These limitations will be discussed in greater detail below.

The first limitation concerns using BMI as a proxy for adiposity. This limitation is particularly relevant for the adolescent period, as gains in both muscle and bone mass can be substantial (Tanofsky-Kraff et al., 2006). Thus, changes in BMI may reflect these increases in muscle and bone mass, and not necessarily changes in fat mass. Although, density-based methods (e.g., underwater weighting) and scanning methods (e.g., DEXA) are the gold standard for assessing overall body fat and fat distribution, factors other than accuracy in body fat estimation need to be considered when deciding on an ideal measure of body fat (Power, Lake, & Cole, 1997). Factors such as accessibility, simplicity, cost and ease of use, and acceptability are all important considerations. Thus, using gold standard measures such as DEXA would not be feasible in a large-scale population study since the test must be performed in a major medical facility, and the equipment is very expensive and must be operated by a skilled technician (Lobstein et al., 2004). In addition, the procedure itself takes about 20 minutes and requires a very cooperative subject (Lobstein et al., 2004). Overall, feasibility restrictions exist for all of the density-based and scanning methods, making them unsuitable as measures of body fatness for the present study.

BMI has been recommended as the measure of choice for epidemiologic and population-based research because it is relatively easy to implement (Dietz & Robinson, 1998). BMI also correlates well with direct measures of total body fat, including DEXA, as well as with health measures such as blood pressure, adverse lipoprotein profiles, atherosclerotic lesions, serum insulin levels, and diabetes mellitus in adolescent samples (Dietz & Robinson, 1998). Overall, research has documented that BMI is a valid measure of adiposity, especially for population-based research (Garrow & Webster, 1985).

A second major limitation concerns the use of self-reported height and weight to calculate BMI. Individuals tend to under-estimate their weight and over-estimate their height (Elgar et al., 2005; Danubio et al., 2008; Morrissey, Whetstone, Cummings, & Owen, 2006), potentially leading to an inaccurate estimate of BMI. This inaccurate estimate would be especially problematic if self-reported height and weight are differentially biased between depressed and non-depressed individuals (Rhew et al, 2008). In this specific situation, prospective associations between BMI and depressive

symptoms may be biased. For example, individuals who suffer from depression may be more prone to negative self-perceptions and greater self-criticality (Lewinsohn, Gotlib, & Seeley, 1997). This lower self-appraisal, in turn, may translate to more realistic height and weight self-reports than the height and weight reported by non-depressed individuals (Alloy & Abramson, 1988). Although a valid concern, there are several considerations that need to be made with respect to this particular limitation.

First, these inaccuracies in self-reported height and weight reflect the specific developmental period under examination (Rhew et al, 2008). Biased estimates from self-reported weights tend to be the greatest under age 14 (Himes & Faricy, 2001). Rhew and colleagues (2008) also concluded that the specific developmental period of early adolescence might be a time where self-reports are particularly inaccurate. Strauss (1999) found that in young adolescents aged 12-16, inaccuracies were more likely to occur for the younger aged youth compared to the older youth. Another study showed that BMI under-reporting is minimal in young adults between ages 20 and 40 (Schutz & Woringer, 2002). Anderson and colleagues (2006) further commented that self-reported height and weight tend to be more accurate in adults and older adolescents. Several studies have, in fact shown a high correlation between BMI calculated from self-reported height and weight and BMI calculated from measured height and weight for older adolescents and young adults (Attie & Brooks-Gunn, 1989; Galambos, Almedia, & Peterson, 1990; Himes, Hannan, Wall, & Neumark-Sztainer, 2005).

Second, the discrepancies between self-reports and physical measurements tend to mainly occur at the extremes of the BMI distributions (Kuskowska-Wolk, Bergstrom, & Bostrom, 1992; Sorensen, Stunkard, Teasdale, & Higgins, 1983)**.** Thus, a report bias of individuals under-reporting BMI at higher BMI levels would affect results by making estimates conservative (Ternouth, Collier, & Maughan, 2009).

Finally, several validation studies have stated that the bias of self-report BMI is too small to affect conclusions about associations in large-scale epidemiological studies (Stunkard & Albaum, 1981; Stevens, Keil, Waid, & Gazes, 1990). Validation studies also suggest that the self-report bias is unlikely to affect conclusions about associations

between body mass and psychological outcomes, such as depressive symptoms, particularly in longitudinal studies (Stunkard & Albaum, 1981; Stevens et al., 1990). Authors suggest that the self-reported BMI biases associated with depressive symptoms are most likely to operate in cross-sectional analyses instead (Hasler, Pine, Kleinbaum, et al., 2005).

These biases are likely not to impact longitudinal studies because the self-report measure is from the same individual over time, thus reducing the amount of bias to only one source of bias over all ages. That is, by consistently relying on self-reported height and weight throughout all ages, we have only a single source of bias. Thus, increases in BMI over time cannot be due to the tendency for individuals to under-estimate their weight and over-estimate their height, because the same individuals are providing estimates for their height and weight over time. As previously mentioned, Franko and colleagues (2005), had inconsistent reporting sources for height and weight, where self-reports were used at the first wave and measured height and weight at the second wave. Thus, the increase in BMI may reflect a true consistency in BMI that was not captured in the first wave because self-reports of height and weight would have had the effect of lowering their initial BMI (Elgar et al., 2005; Danubio et al., 2008).

The third major limitation was the low participant response rate throughout all ages. As Table 2 indicates, after age 17, participation rates dropped in subsequent cycles. From ages 19 to 25, participation rates were 79%, 71%, 73%, and 66%, respectively. The biggest decrease in participation occurred between age 17 and 19, where there was a 20% drop in participation. A decrease in retention is expected to occur within longitudinal studies (Hansen, Tobler, & Graham, 1990). In a meta-analysis on attrition rates, Hansen and colleagues (1990) suggested that studies with retention rates above 71.8% at 24 months, and above 67.5% at 36 months, would be judged to have acceptable and interpretable rates of retention. As retention rates were 79% at the age 19 (i.e., two years after baseline), this level of attrition occurring between age 17 and 19 of the current study is acceptable. Furthermore, at all ages within the current study, retention rates were higher than the acceptable rate that was defined by Hansen et al., (1990) for three years after baseline. These findings indicate that, although retention rates decreased over the

course of the present study, at eight years after baseline, retention rates were still within the acceptable range defined at three years after baseline. Although these retention rates were acceptable, there is a possibility that data is not missing at random if individuals with higher body mass and/or depressive symptoms were more likely to drop out of the study.

The final major limitation of the present study is that the starting age point examined, as well as the length in time of follow-up, may have affected our findings. That is, we began our analyses at age 16-17, and measured change up until age 24-25. Depressive symptoms at age 16 may not affect changes in body mass up until age 25. For example, Richardson et al., 2003 examined this research question using two cohorts; one cohort was 11-15 years old at baseline and 26 at follow-up, and the other was 18-21 years old at baseline and 36 at follow-up. The authors found that depression at ages 11-15 was not a significant predictor of obesity at age 26 for either males or females. However, females with depression at ages 18-21 had an increased risk of obesity at age 26. This study suggests that the effect of depressive symptoms on subsequent body mass may be more likely to occur when the time points are closer together rather than farther apart. Thus, examining the associations over an 8-year period, beginning at 16-17 years, may have been too long or too early to see an association between depressive symptoms and subsequent body mass.

These limitations, although not major, should be examined in greater detail in future studies researching the association between body mass and depressive symptoms. Namely, associations should be examined using gold standards for BMI assessment, including measured height and weight. In addition, analyses should be replicated on various ages and interval lengths between adolescence and young adulthood in order to best understand what is occurring during this developmental transition.

5.6   Future research

The public health implications presented above can only be drawn from tentative conclusions from the exploratory results reported in the present study. Several authors have concluded that better-designed studies are needed to draw more reliable conclusions

on the prospective association between body mass and depressive symptoms (Fabricatore & Wadden, 2004; Liem et al., 2008). For example, Friedman and Brownell, 1995, suggested that authors include large, nationally representative samples, consistent definitions and measurements of body mass, valid assessment tools, and appropriate control groups. Faith et al., 2001 also encouraged authors to use standardized and validated assessments of depressive symptoms, as well as control for baseline levels of the follow-up dependent variable.

As previously mentioned, the variations that occur at the conceptual, methodological, and analytical levels, may explain a great deal of the disparity in results of studies investigating the prospective association between obesity and depression. Conceptually, consistencies do not exist with regards to the developmental periods studied, the operationalization of body mass and body mass comparison groups, the operationalization of depressive symptoms, and the populations studied. Methodologically, there are large variations among the length of intervals, source of height and weight measurements, reference tools for establishing body mass cutoffs, and tools for measuring depressive symptoms. Analytically, studies varied in the number of measurement points used for analyses, the statistical techniques they used, and the covariates controlled for to assess prospective associations between body mass and depressive symptoms.

Thus, as it currently stands, public health implications can only be drawn from tentative conclusions. A major goal for future studies should be to strive toward greater consistency in this area of research. A higher level of precision in conceptualizations, methodologies, and analyses used across studies would effectively translate into less variety in reported results. Having less variety in results across studies would allow researchers to confidently state research conclusions and public health implications based on evidence. In order to build research towards this higher level of consistency between studies, authors should strive to provide strong justifications for the operationalization of concepts, as well as the methodologies and analytical techniques used.

In addition to greater consistency between studies, future research needs to examine more clearly the causal mechanisms involved in the prospective association between body mass and depressive symptoms (Luppino, de Wit, Bouvy, et al., 2010). Mediators should be selected based on the direction of the association examined and the theoretical rationale. As mentioned in the literature review above, potential causal pathways for body mass affecting depressive symptoms include the internalization of biased attitudes towards obesity, weight-teasing, neurobiological causal pathways, and functional impairment. The potential causal pathways through which depressive symptoms affects body mass include neurobiological causal pathways and affect regulation, and impaired sleep quality and physical inactivity. Once research has clearly established whether prospective associations between body mass and depressive symptoms exist, these causal pathways need to be explored in order to understand the mechanisms linking their association.

Several studies have also reported null results when examining the prospective association between body mass and depressive symptoms (Clark et al., 2007; Goodman & Whitaker, 2002; Jansen et al., 2008; Rhew et al., 2008; Tanofsky-Kraff et al., 2006). These studies are also important, and reasons for null results for a particular developmental group should be more closely examined. For example, one line of reasoning may be that perception of and dissatisfaction with weight may be of greater importance for predicting depressive symptoms than weight per se (Stice, Hayward, Cameron, Killen, & Taylor, 2000). For example, Jansen and colleagues (2008) reported that body weight perception, and not weight status (self-reported or measured), was associated with mental health indicators among 12-13 year-olds.

This association may be especially relevant for adolescent and young adult females than males, given the higher level of concern for being thin. According to Kittel and colleagues, adolescent girls aged 11-19 reported significantly less satisfaction with their bodies than adolescent boys (1978). In addition, Douty and colleagues found that 59% of normal-weight female college students reported low satisfaction with their bodies (Douty, Moore, & Hartford, 1974). In another study, approximately 50% of girls in grade 10-12 believed that they were overweight, and nearly two-thirds wanted to lose weight

(Stunkard, 1957). Most of the boys however were either satisfied with their weight or believed they were too thin. Interestingly, the prevalence of overweight status was identical in both groups. These findings highlight the importance of perception in the association between body mass and depressive symptoms, particularly for females.

Stice and Bearman (2001) investigated associations within a sample of female adolescents from northern California, using three cycles of data over 10-month intervals. The authors hypothesized that neither starting levels of BMI nor changes of BMI over time would affect changes in depressive symptoms over time. Instead it was hypothesized that other variables would explain the growth in depressive symptoms across adolescence, including perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms. Growth curve analysis was used to examine whether initial levels of BMI predicted change in depressive symptoms over time, and whether change in BMI predicted change in depressive symptoms over time. Neither of these associations were significant. However, all other variables hypothesized to explain changes in depressive symptoms were significant. The authors concluded that body image and eating disturbances, but not BMI, contribute to the growth in depressive symptoms among female adolescents. Overall, these mechanisms need to be explored more clearly in future research.

Finally, it would also be useful for future studies to examine associations across different developmental periods (Faith et al., 2011). We cannot currently infer causality because increases in body mass and depressive symptoms may have begun before age 17. Therefore, there may be a different temporal picture before these ages as well as after age 25. Future research is needed to apply the same methodologies to early adolescence as well as adulthood in order to better understand the predictive relationship between body mass and depressive symptoms. The association is most likely a bidirectional association, but different directional associations may apply depending on age group and sex. Although depressive symptoms in adolescence did not predict higher body mass in young adulthood in the present study, an individual's mental state may begin to influence weight status as they become older. For example, when examining women 22-27 years old at baseline, Ball and colleagues (2009) found that decreases in weight predicted increases in

depressive symptoms three years later. For this age group, body image issues may not be as prominent in females, and instead, decreases in weight could reflect the occurrence of stressful life events, resulting in weight loss and depressive symptoms. Overall, exploring the associations across the lifecourse is important in order to fully understand developmental influences on both body mass and depressive symptoms.

5.7   Conclusion

In conclusion, the goal of this thesis was to examine longitudinally the associations between body mass and depressive symptoms during the transition from adolescence to young adulthood, and to investigate if and how these associations differ for males and females. It was conducted in order to address the major limitations and gaps of previous research in this area, as well as to contribute to some level of consistency that will help make cross-study comparisons possible in the future. Results showed that, on average, adolescent girls with higher initial BMI levels reported a slower decrease in CES-D scores over time, than adolescent girls who did not have higher initial BMI levels. In addition, on average, adolescent males with higher initial CES-D scores reported a slower increase in BMI over time, than adolescent males who did not have higher initial CES-D scores.

Understanding the development and course of body mass and depressive symptoms requires a lifecourse perspective for many reasons. First, both of these health outcomes have determinants early in life, and can often persist from adolescence into adulthood (Bradley et al., 2008; Guo et al., 2002). They can develop into chronic conditions that most likely require lifelong monitoring and management (Schwartz, 1998). Thus, studying how body mass and depressive symptoms independently change, as well as how they are associated over time, will aid in understanding the course and consequences of both health constructs. Furthermore, understanding the predictive factors associated with changes in body mass, as well as the predictive factors associated with depressive symptoms, is important for preventing negative health outcomes across the lifecourse. Finally, preventing increases in either body mass or depressive symptoms will be most effective when it is informed by an understanding of the etiological pathways involved in their development (Anderson et al., 2006).

References

Ajilore, O., Haroon, E., Kumaran, S., Darwin, C., Binesh, N., Mintz, J., et al. (2007). Measurement of brain metabolites in patients with type 2 diabetes and major depression using proton magnetic resonance spectroscopy. Neuropsychopharmacology, 32(6), 1224-1231. doi: 10.1038/sj.npp.1301248

Alberga, A. S., Sigal, R. J., Goldfield, G., Prud'homme, D., & Kenny, G. P. (2012). Overweight and obese teenagers: Why is adolescence a critical period? Pediatr Obes, 7(4), 261-273.

Allison, D. B., Fontaine, K. R., Manson, J. E., Stevens, J., & VanItallie, T. B. (1999). Annual deaths attributable to obesity in the United States. JAMA, 282(16), 1530-1538.

Anderson, S. E., Cohen, P., Naumova, E. N., Jacques, P. F., & Must, A. (2007). Adolescent Obesity and risk for subsequent major depressive disorder and anxiety disorder: Prospective evidence. Psychosomatic Medicine, 69(8), 740-747. doi: 10.1097/PSY.0b013e31815580b4

Anderson, S. E., Cohen, P., Naumova, E. N., & Must, A. (2006). Association of depression and anxiety disorders with weight change in a prospective community-based study of children followed up into adulthood. Arch Pediatr Adolesc Med, 160(3), 285-291. doi: 10.1001/archpedi.160.3.285

Anderson, S. E., Murray, D. M., Johnson, C. C., Elder, J. P., Lytle, L. A., Jobe, J. B., et al. (2011). Obesity and depressed mood associations differ by race/ethnicity in adolescent girls. Int J Pediatr Obes, 6, 6.

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). The development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. International Journal of Methods in Psychiatric Research, 5, 237-249.

Arbuckle, J., & Wothke, W. (1999). AMOS 4 user's reference guide. Chicago: Smallwaters Corp.

Attie, I. & Brooks-Gunn, J. (1989). Development of eating problems in adolescent girls: A longitudinal study. Developmental Psychology, 25, 70–79.

Ball, K., Burton, N. W., & Brown, W. J. (2009). A prospective study of overweight, physical activity, and depressive symptoms in young women. Obesity (Silver Spring), 17(1), 66-71. doi: 10.1038/oby.2008.497

Bardone, A. M., Moffitt, T. E., Caspi, A., Dickson, N., Stanton, W. R., & Silva, P. A. (1998). Adult physical health outcomes of adolescent girls with conduct disorder, depression, and anxiety. Journal of the American Academy of Child and Adolescent Psychiatry, 37, 594–601.

Barefoot, J. C., Heitmann, B. L., Helms, M. J., Williams, R. B., Surwit, R. S., & Siegler, I. C. (1998). Symptoms of depression and changes in body weight from adolescence to mid-life. International Journal of Obesity and Related Metabolic Disorders, 22, 688–694.

Baumeister, H. & Harter, M. (2007). Prevalence of mental disorders based on general population surveys. Social Psychiatry and Psychiatric Epidemiology, 42(7), 537-546.

Beck, J., Schaefer, C. A., Nace, H., Steffen, A. D., Nigg, C., Brink, L., et al. (2012). Accuracy of self-reported height and weight in children aged 6 to 11 years. Prev Chronic Dis, 9, 120021. DOI: http://dx.doi.org/10.5888/pcd9.120021

Baumrind, D. (1966). Effects of Authoritative Parental Control on Child Behavior, Child Development, 37(4), 887-907.

Belanoff, J. K., Kalehzan, M., Sund, B., Fleming Ficek, S. K., & Schatzberg, A. F. (2001). Cortisol activity and cognitive changes in psychotic major depression. Am J Psychiatry, 158(10), 1612-1616.

Benton, D. (2004). Role of parents in the determination of the food preferences of children and the development of obesity. Int J Obes, 28, 858-869.

Berkman, L. F., Berkman, C. S., Kasl, S. V., Freeman, D. H., Leo, L., Ostfeld, A. M., et al. (1986). Depressive symptoms in relation to physical health and functioning in the elderly. Am. J. Epidem, 124, 372-388.

Birmaher, B., Brent, D., Bernet, W., Bukstein, O., Walter, H., Benson, R. S., et al. (2007). Practice parameter for the assessment and treatment of children and adolescents with depressive disorders. J Am Acad Child Adolesc Psychiatry, 46(11), 1503-1526. doi: 10.1097/chi.0b013e318145ae1c

Birmaher, B., Ryan, N.D., Williamson, D.E., Brent, D.A., Kaufman, J., Dahl, R.E., et al. (1996). Childhood and adolescent depression: A review of the past 10 years. Part I. J Am Acad Child Adolesc Psychiatry, 35(11), 1427–39.10.1097/00004583-199611000-00011

Birmaher, B., Williamson, D. E., Dahl, R. E., Axelson, D. A., Kaufman, J., Dorn, L. D., et al. (2004). Clinical presentation and course of depression in youth: Does onset in childhood differ from onset in adolescence? J Am Acad Child Adolesc Psychiatry, 43(1), 63-70. doi: 10.1097/00004583-200401000-00015

Bjerkeset, O., Romundstad, P., Evans, J., Gunnell, D., Bjerkeset, O., Romundstad, P., et al. (2008). Association of adult body mass index and height with anxiety, depression, and suicide in the general population: The HUNT study. Am J Epidemiol, 14(2), 193–202.

Björntorp P. (2001). Do stress reactions cause abdominal obesity and comorbidities? Obes Rev, 2, 73–86. doi: 10.1046/j.1467-789x.2001.00027.x.

Bornstein, M. H. (1989). Sensitive periods in development: Structural characteristics and causal interpretations. Psychological Bulletin, 105(2), 179-197. doi: 10.1037/0033-2909.105.2.179

Bornstein, S. R., Schuppenies, A., Wong, M. L., & Licinio, J. (2006). Approaching the shared biology of obesity and depression: The stress axis as the locus of gene-environment interactions. Mol Psychiatry, 11(10), 892-902. doi: 10.1038/sj.mp.4001873

Boutelle, K. N., Hannan, P., Fulkerson, J. A., Crow, S. J., & Stice, E. (2010). Obesity as a prospective predictor of depression in adolescent females. Health Psychol, 29(3), 293-298. doi: 10.1037/a0018645

Bradley, R. H., Houts, R., Nader, P. R., O'Brien, M., Belsky, J., & Crosnoe, R. (2008). The relationship between body mass index and behavior in children. The Journal of Pediatrics, 153(5), 629-634.e623. doi: http://dx.doi.org/10.1016/j.jpeds.2008.05.026

Bremmer, M. A., Beekman, A. T., Deeg, D. J., Penninx, B. W., Dik, M. G., Hack, C. E., et al. (2008). Inflammatory markers in late-life depression: Results from a population-based study. J Affect Disord, 106(3), 249-255. doi: 10.1016/j.jad.2007.07.002

Brent, D. A. (2006). Glad for what TADS adds, but many TADS grads still sad. J Am Acad Child Adolesc Psychiatry, 45(12), 1461-1464. doi: 10.1097/01.chi.0000237708.28013.2a

Brown, W. J., Ford, J. H., Burton, N. W., Marshall, A. L., & Dobson, A. J. (2005). Prospective study of physical activity and depressive symptoms in middle-aged women. Am J Prev Med, 29(4), 265-272. doi: 10.1016/j.amepre.2005.06.009

Burnham, K. P. & Anderson, D. R. (2004). Multimodel inference: understanding AIC and BIC in Model Selection. Sociological Methods and Research, 33, 261–304.

Calamaro, C. J., & Waite, R. (2009). Depression and obesity in adolescents: What can primary care providers do? The Journal for Nurse Practitioners, 5(4), 255-261. doi: http://dx.doi.org/10.1016/j.nurpra.2009.01.004

Camacho, T. C., Roberts, R. E., Lazarus, N. B., Kaplan, G. A., & Cohen, R. D. (1991). Physical activity and depression: Evidence from the Alameda County Study. Am J Epidemiol, 134(2), 220-231.

Carlson, G., & Kashani, J. (1988). Phenomenology of major depression from childhood through adulthood: Analysis of three studies. American Journal of Psychiatry, 145, 1222-1225.

Carpenter, K. M., Hasin, D. S., Allison, D. B., Faith, M. S. (2000). Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: Results from a general population study. Am J Public Health 90, 251.

Cash, T. F. (1995). Developmental teasing about physical appearance: Retrospective descriptions and relationships with body image. Social Behavior and Personality: An International Journal, 23(2), 123-130. doi: 10.2224/sbp.1995.23.2.123

Chambers W. J., Puig-Antich J., Hirsch, M., Paez, P., Ambrosini, P. J., & Tabrizi M. A. (1985). The assessment of affective disorders in children and adolescents by semi-structured interview: Test-retest reliability of the schedule for affective disorders and schizophrenia for school-aged children, present episode version. Archives of General Psychiatry, 42, 696–702.

Chase, M. A., & Dummer, G. M. (1992). The role of sports as a social status determinant for children. Research Quarterly for Exercise and Sport, 63, 418-424.

Clark, C., Haines, M. M., Head, J., Klineberg, E., Arephin, M., Viner, R., et al. (2007). Psychological symptoms and physical health and health behaviours in adolescents: A prospective 2-year study in East London. Addiction, 102(1), 126-135. doi: 10.1111/j.1360-0443.2006.01621.x

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. BMJ, 320(7244), 1240-1243.

Crisp, A. H. & McGuiness, B. (1976). Jolly fat: Relation between obesity and psychoneurosis in general population. Br Med J, 3;1(6000), 7–9.

Crisp, A. H., Queenan, M., Sittampaln, Y., & Harris, G. (1980). "Jolly fat" revisited. J Psychosom Res, 24, 233–41.

Curran, P. J. & Bollen, K. A. (2001), The best of both worlds: Combining autoregressive and latent curve models, in: A. Sayer and L. Collins (eds.), New methods for the analysis of change, American Psychological Association, Washington DC, 107135.

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. Keynote address. Ann N Y Acad Sci, 1021, 1-22. doi: 10.1196/annals.1308.001

Danubio, M. E., Miranda, G., Vinciguerra, M. G., Vecchi, E., & Rufo, F. (2008). Econ Hum Biol, 6, 181-90.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory, 14 (1), 25-39.

Dietz, W. H. (1994). Critical periods in childhood for the development of obesity. The American Journal of Clinical Nutrition, 59(5), 955-959.

Dietz, W. H. & Robinson, T.N. (1998) Use of the body mass index (BMI) as a measure of overweight in children and adolescents. Journal of Pediatrics, 132(2), 191–193.

Ding, C. & Bornhop, J. (2005). Overweight and school: Are there any perceived achievement consequences of overweight among American youth? J. Soc. Sci., 1, 118-125.

Douty, H. I., Moore, J. B., & Hartford, D. (1974). Body characteristics in relation to life adjustment, body-image and attitudes of college females. Perceptual and Motor Skills, 39, 499-521.

Duarte, C. S., Sourander, A., Niklakaros, G., Pihlajamaki, H., Helenius, H., Piha, J., et al. (2010). Child mental health problems and obesity in early adulthood. J Pediatr, 156, 93–97. doi: 10.1016/j.jpeds.2009.06.066.

Duncan, T. E., Duncan, S. C., & Strycker, L. A. (2006). An introduction to latent variable growth curve modeling: Concepts, issues and applications (2nd ed.). Mahwah, NJ.

Eaton, W. W., Shao, H., Nestadt, G., Lee, B. H., Bienvenu, O. J., & Zandi, P. (2008) Population-based study of first onset and chronicity in major depressive disorder. Arch Gen Psychiatry 65, 513–520.

Eisenberg, M. E., Neumark-Sztainer, D., Haines, J., & Wall, M. (2006). Weight-teasing and emotional well-being in adolescents: Longitudinal findings from Project EAT. J Adolesc Health, 38(6), 675-683. doi: 10.1016/j.jadohealth.2005.07.002

Elgar, F. J., Roberts, C., Moore, L., & Tudor-Smith, C. (2005). Sedentary behaviour, physical activity and weight problems in adolescents in Wales. Public Health, 119(6): 518–24.

Elliott, D. S., Huizinga, D., & Menard, S. (1989). Multiple Problem Youth: Delinquency, Drugs and Mental Health Problems. New York, NY: Springer.

Emery, C. F., Fondow, M. D., Schneider, C. M., Christofi, F. L., Hunt, C., Busby, A. K., et al. (2007). Gastric bypass surgery is associated with reduced inflammation and less depression: A preliminary investigation. Obes Surg, 17(6), 759-763.

Fabricatore, A.N. & Wadden, T.A. Psychological aspects of obesity. (2004). Clinics in Dermatology, 22(4), 332–337.

Faith, M. S., Butryn, M., Wadden, T. A., Fabricatore, A., Nguyen, A. M., & Heymsfield, S. B. (2011). Evidence for prospective associations among depression and obesity in population-based studies. Obesity Reviews, 12(5), e438-e453. doi: 10.1111/j.1467-789X.2010.00843.x

Faith, M. S., Matz, P. E., & Jorge, M. A. (2002). Obesity-depression associations in the population. Journal of Psychosomatic Research, 53(4), 935-942. doi: http://dx.doi.org/10.1016/S0022-3999(02)00308-2

Farmer, M. E., Locke, B. Z., Moscicki, E. K., Dannenberg, A. L., Larson, D. B., & Radloff, L. S. (1988). Physical activity and depressive symptoms: The NHANES I Epidemiologic Follow-up Study. Am J Epidemiol, 128(6), 1340-1351.

Festinger, L. (1954). A Theory of Social Comparison Processes. Human Relations, 7(2), 117-140. doi: 10.1177/001872675400700202

Forman-Hoffman, V. L., Yankey, J. W., Hillis, S. L., Wallace, R. B., & Wolinsky, F. D. (2007). Weight and depressive symptoms in older adults: Direction of influence? J Gerontol B Psychol Sci Soc Sci, 62(1), S43-51.

Franko, D. L., Striegel-Moore, R. H., Thompson, D., Schreiber, G. B., & Daniels, S. R. (2005). Does adolescent depression predict obesity in black and white young adult women? Psychol Med, 35(10), 1505-1513.

Fredrickson, B. & Roberts, S. (1997). Objectification theory: Toward understanding women's lived experiences and mental health risks. Psychology of Women Quarterly, 21, 173-206.

Friedman, M. A., & Brownell, K. D. (1995). Psychological correlates of obesity: Moving to the next research generation. Psychol Bull, 117(1), 3-20.

Galambos, N. L., Almeida, D. M., Petersen, A. C. Masculinity, femininity, and sex role attitudes in early adolescence: Exploring gender intensification. (1990). Child Development, 61, 1905–14.

Gangwisch, J. E., Malaspina, D., Boden-Albala, B., & Heymsfield, S. B. (2005). Inadequate sleep as a risk factor for obesity: Analyses of the NHANES I. Sleep, 28(10), 1289-1296.

Gariepy, G., Wang, J., Lesage, A. D., & Schmitz, N. (2010). The longitudinal association from obesity to depression: Results from the 12-year National Population Health Survey. Obesity, 18(5), 1033-1038.

Garrow, J. S. & Webster, J. Quetelets index (weight/height2) as a measure of fatness. (1985). Int J Obes, 9, 147-53.

Ge, X., Natsuaki, M. N., & Conger, R. D. (2006). Trajectories of depressive symptoms and stressful life events among male and female adolescents in divorced and nondivorced families. Development and Psychopathology, 18, 1–21.

Georgiades, K., Lewinsohn, P. M., Monroe, S. M., & Seeley, J. R. (2006). Major depressive disorder in adolescence: The role of subthreshold symptoms. J Am Acad Child Adolesc Psychiatry, 45(8), 936-944. doi: 10.1097/01.chi.0000223313.25536.47

Goodman, E., Hinden, B. R., Khandelwal, S. (2000). Accuracy of teen and parental reports of obesity and body mass index. Pediatrics, 11:52–58. doi: 10.1542/peds.106.1.52.

Goodman, E., & Whitaker, R. C. (2002). A prospective study of the role of depression in the development and persistence of adolescent obesity. Pediatrics, 110(3), 497-504.

Goodwin, R. D., Sourander, A., Duarte, C. S, Niemelä, S., Multimäki, P., Nikolakaros, G., et al. (2009). Do mental health problems in childhood predict chronic physical conditions among males in early adulthood? Evidence from a community-based prospective study. Psychological Medicine, 39, 301–311.

Greene, R. L. (1991). The MMPI–2/MMPI: An interpretative manual. Boston: Allyn & Bacon.

Greenfield, T. K., Rehm, J., & Rogers, J. D. (2002). Effects of depression and social integration on the relationship between alcohol consumption and all-cause mortality. Addiction, 97(1), 29-38.

Guo, S. S., Roche, A. F., Chumlea, W. C., Gardner, J. D., & Siervogel, R. M. (1994). The predictive value of childhood body mass index values for overweight at age 35 y. Am J Clin Nutr, 59, 810–819.

Hansen, W. B., Tobler, N. S., & Graham, J. W. (1990). Attrition in substance abuse prevention research: A meta-analysis of 85 longitudinally followed cohorts. Evaluation Review, 14, 677-685.

Hasler, G., Pine, D. S., Gamma, A., Milos, G., Ajdacic, V., Eich, D., et al. (2004). The associations between psychopathology and being overweight: A 20-year prospective study. Psychol Med, 34(6), 1047-1057.

Hasler, G., Pine, D. S., Kleinbaum, D. G., Gamma, A., Luckenbaugh, D., Ajdacic, V., et al. (2005). Depressive symptoms during childhood and adult obesity: The Zurich Cohort Study. Mol Psychiatry, 10(9), 842-850.

Hayes, D., & Ross, C. E. (1986). Body and mind: The effect of exercise, overweight, and physical health on psychological well-being. J Health Soc Behav, 27(4), 387-400.

Herva, A., Laitinen, J., Miettunen, J., Veijola, J., Karvonen, J. T., Laksy, K., et al. (2006). Obesity and depression: Results from the longitudinal Northern Finland 1966 Birth Cohort Study. Int J Obes (Lond), 30(3), 520-527. doi: 10.1038/sj.ijo.0803174

Hill, A. J. (2005). Psychological aspects of obesity. Psychiatry, 4(4), 26-30.

Himes, J. H. & Faricy, A. (2001). Validity and reliability of self-reported stature and weight of US adolescents. Am. J. Hum. Biol. 13, 255–260.

Himes, J. H., Hannan, P., Wall, M., & Neumark-Sztainer, D. (2005). Factors associated with errors in self-reports of stature, weight, and body mass index in Minnesota adolescents. Ann Epidemiol, 15, 272-278.

Holsboer, F. (2000). The corticosteroid receptor hypothesis of depression. Neuropsychopharmacology, 23(5), 477-501. doi: 10.1016/s0893-133x(00)00159-7

Hooshmand, S., Willoughby, T., & Good, M. (2011). Does the direction of effects in the association between depressive symptoms and health-risk behaviors differ by behavior? A longitudinal study across the high school years. Journal of Adolescent Health, 50(2), 140-147. doi: http://dx.doi.org/10.1016/j.jadohealth.2011.05.016

Hoyle, R. H. (1995). The structural equation modeling approach: Basic concepts and fundamental issues. In Structural equation modeling: Concepts, issues, and applications, R. H. Hoyle (editor). Thousand Oaks, CA: Sage Publications, Inc., pp. 1-15.

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. Archives of General Psychiatry, 59, 215–222.

Jansen, W., van de Looij-Jansen, P. M., de Wilde, E. J., & Brug, J. (2008). Feeling fat rather than being fat may be associated with psychological well-being in young dutch adolescents. J Adolesc Health, 42(2), 128-136.

John, U., Meyer, C., Hapke, U., & Rumpf, H. J., Schumann A., (2004). Nicotine dependence. quit attempts. and quitting among smokers in a regional population sample from a country with a high prevalence of tobacco smoking. Prev Med, 38, 350-8.

Kemper, H. C., Post, G. B., Twisk, J. W., & van Mechelen, W. (1999). Lifestyle and obesity in adolescence and young adulthood: Results from the Amsterdam growth and health longitudinal study (AGAHLS). Int J Obes Relat Metab Disord. 23 (Suppl 3), S34-40.

Kandel, D., & Davies, M. (1982). Epidemiology of depressive mood in adolescents. Archives of General Psychiatry, 39, 1205–1212.

Kendler, K. S., Eaves,. L. J., Walters., E. E., Neale. M. C., Heath, A. C., Kessler, R. C. (1996). The identification and validation of distinct depressive syndromes in a population-based sample of female twins. Archives of General Psychiatry, 53(5), 391-399. doi: 10.1001/archpsyc.1996.01830050025004

Kittel, F., Rustin, R. M., Dramaix, M., de Backer, G. & Kornitzer, M. (1978). Psycho-social-biological correlates of moderate overweight in an industrial population. Journal of Psychosomatic Research, 22, 145–158.

Kline, R. B. (2005). Principles and practice of structural equation modeling (2nd ed.). New York: Guilford Press.

Kovacs M. & Beck A. T. (1977). An empirical-clinical approach toward a definition of childhood depression. In: Schulterbrandt JG, Raskin A, editors. Depression in childhood: Diagnosis, treatment and conceptual models. New York, NY: Raven Press, pp. 1–25.

Kuczmarski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., et al. (2002). 2000 CDC Growth Charts for the United States: Methods and development. Vital Health Stat, 11(246), 1-190.

Kuskowska-Wolk, A., Bergstrom, R., & Bostrom, G. (1992). Relationship between questionnaire data and medical records of height, weight and body mass index. Int J Obes Relat Metab Disord, 16, 1–9.

Lago, F., Dieguez, C., Gomez-Reino, J., & Gualillo, O. (2007). Adipokines as emerging mediators of immune response and inflammation. Nat Clin Pract Rheumatol, 3(12), 716-724. doi: 10.1038/ncprheum0674

Laitinen, J., Power, C., Ek, E., Sovio, U., & Järvelin, M. R. (2002). Unemployment and obesity among young adults in a northern Finland 1966 birth cohort. International Journal of Obesity, 26(10). doi: 10.1038/sj.ijo.0802134

Lampinen, P., Heikkinen, R. L., & Ruoppila, I. (2000). Changes in intensity of physical exercise as predictors of depressive symptoms among older adults: An eight-year follow-up. Prev Med, 30(5), 371-380. doi: 10.1006/pmed.2000.0641

Lauer, C. J., Wiegand, M., & Krieg, J. C. (1992). All-night electroencephalographic sleep and cranial computed tomography in depression: A study of unipolar and bipolar patients. Eur Arch Psychiatry Clin Neurosci, 242(2-3), 59-68.

Lee, W.-J., Lee, Y.-C., Ser, K.-H., Chen, J.-C., & Chen, S. (2008). Improvement of insulin resistance after Obesity Surgery: A Comparison of Gastric Banding and Bypass Procedures. Obesity Surgery, 18(9), 1119-1125. doi: 10.1007/s11695-008-9457-3

Lewinsohn, P. M., Gotlib, I. H., & Seeley, J. R. (1997). Depression-related psychosocial variables: Are they specific to depression in adolescents? Journal of Abnormal Psychology, 106, 365-375.

Li, T. Y., Rana, J. S., Manson, J. E., Willett, W. C., Stampfer, M. J., Colditz, G. A., et al. (2006). Obesity as compared with physical activity in predicting risk of coronary heart disease in women. Circulation, 113(4), 499-506.

Liem, E., Sauer, P. J., Oldehinkel, A. J., & Stolk, R. P. (2008). Association between depressive symptoms in childhood and adolescence and overweight in later life: Review of the recent literature. Archives of Pediatrics & Adolescent Medicine, 162(10), 981-988. doi: 10.1001/archpedi.162.10.981

Little, R. J. A., & Rubin, D. B. (2002). Statistical Analysis with Missing Data, 2nd ed. Wiley, New York.

Lobstein, T., Baur, L., & Uauy, R. (2004). Obesity in children and young people: A crisis in public health. Obesity Reviews, 5, 4-85. doi: 10.1111/j.1467-789X.2004.00133.x

Locke, T. F., & Newcomb, M. D. (2001). Alcohol involvement and dysphoria: A longitudinal examination of gender differences from late adolescence to adulthood. Psychol Addict Behav, 15(3), 227-236.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., et al. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. Archives of General Psychiatry 67, 220–229.

McCarty, C. A., Kosterman, R., Mason, W. A., McCauley, E., Hawkins, J. D., Herrenkohl, T. I., et al. (2009). Longitudinal associations among depression, obesity and alcohol use disorders in young adulthood. Gen Hosp Psychiatry, 31(5), 442-450.

Meier, P., Mellenbergh, G. J., & deWit, C. A. M. (1986). De Korte Depressie Vragenlijst voor Kinderen, KDVK [The Short Depression Inventory for Children, SDIC]. Nederlands tijdschrift voor de psychologie, 41, 364–368.

Merten, M., Wickrama, K. A. S., & Williams, A. (2008). Adolescent obesity and young adult psychosocial outcomes: Gender and racial differences. Journal of Youth and Adolescence, 37(9), 1111-1122. doi: 10.1007/s10964-008-9281-z

Milaneschi, Y., Corsi, A. M., Penninx, B. W., Bandinelli, S., Guralnik, J. M., & Ferrucci, L. (2009). Interleukin-1 receptor antagonist and incident depressive symptoms over 6 years in older persons: the InCHIANTI study. Biol Psychiatry, 65(11), 973-978. doi: 10.1016/j.biopsych.2008.11.011

Mitchell, J., McCauley, E., Burke, P. M., & Moss, S. J. (1988). Phenomenology of depression in children and adolescents. Journal of the American Acarlemy of Child and Adolescent Psychiatry, 27, 12-20.

Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B. A., Marks, J. S., & Koplan, J. P. (1999). The spread of the obesity epidemic in the United States, 1991-1998. JAMA, 282(16), 1519-1522.

Morrissey, S. L., Whetstone, L. M., Cummings, D. M., & Owen, L. J. (2006). Comparison of self-reported and measured height and weight in eighth grade students. J School Health 76(10), 512-15.

Must, A. & Strauss, R. S. (1999). Risks and consequences of childhood and adolescent obesity. Int J Obes Relat Metab Disord, 23 Suppl 2:S2-11

Muthén, B. (2002). Beyond SEM: General latent variable modeling. Behaviormetrika, 29, 81–117.

Napolitano, M. A., & Foster, G. D. (2008). Depression and obesity: Implications for assessment, treatment, and research. Clinical Psychology: Science and Practice, 15(1), 21-27. doi: 10.1111/j.1468-2850.2008.00107.x

Needham, B. L., & Crosnoe, R. (2005). Overweight status and depressive symptoms during adolescence. Journal of Adolescent Health, 36(1), 48-55. doi: http://dx.doi.org/10.1016/j.jadohealth.2003.12.015

Needham, B. L., Epel, E. S., Adler, N. E., & Kiefe, C. (2010). Trajectories of change in obesity and symptoms of depression: The CARDIA study. Am J Public Health, 100(6), 1040-1046.

Neumark-Sztainer, D., Story, M., & Faibisch, L. (1998). Perceived stigmatization among overweight African-American and Caucasian adolescent girls. J Adolesc Health, 23(5), 264-270.

Ogden, C. L., Carroll, M. D., Curtin, L. R., McDowell, M. A., Tabak, C. J., & Flegal, K. M. (2006). Prevalence of overweight and obesity in the United States, 1999-2004. JAMA, 295(13), 1549-1555.

Paffenbarger, R. S., Lee, I. M., & Leung, R. (1994). Physical activity and personal characteristics associated with depression and suicide in American college men. Acta Psychiatrica Scandinavica, 89, 16-22. doi: 10.1111/j.1600-0447.1994.tb05796.x

Pasquali, R., Vicennati, V., Cacciari, M., & Pagotto, U. (2006). The hypothalamic-pituitary-adrenal axis activity in obesity and the metabolic syndrome. Ann N Y Acad Sci, 1083, 111-128. doi: 10.1196/annals.1367.009

Patock-Peckham, J. A., Cheong, J., Balhorn, M. E., & Nagoshi, C. T. (2001). A social learning perspective: A model of parenting styles, self-regulation, perceived drinking control, and alcohol use and problems. Alcohol Clin Exp Res, 25(9), 1284–92.

Pine, D. S., Cohen, P., Brook, J., Coplan, J. D. (1997). Psychiatric symptoms in adolescence as predictors of obesity in early adulthood: A longitudinal study. Am J Public Health, 87(8), 1303–1310.

Poulin, C., Hand, D., & Boudreau, B. (2005). Validity of a 12-item version of the CES-D used in the National Longitudinal Study of Children and Youth. Chronic Diseases in Canada, 26(2-3), 65-72.

Power, C., Lake, J. K., & Cole, T. J. (1997). Measurement and long-term health risks of child and adolescent fatness. Int J Obes, 21, 507-26.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement, 1(3), 385-401. doi: 10.1177/014662167700100306

Rashid, M. N., Fuentes, F., Touchon, R. C., & Wehner, P. S. (2003). Obesity and the risk for cardiovascular disease. Prev Cardiol, 6(1), 42-47.

Rhew, I. C., Richardson, L. P., Lymp, J., McTiernan, A., McCauley, E., & Stoep, A. V. (2008). Measurement matters in the association between early adolescent depressive symptoms and body mass index. Gen Hosp Psychiatry, 30(5), 458-466.

Richardson, L. P., Davis, R., Poulton, R., McCauley, E., Moffitt, T. E., Caspi, A., et al. (2003). A longitudinal evaluation of adolescent depression and adult obesity. Arch Pediatr Adolesc Med, 157(8), 739-745. doi: 10.1001/archpedi.157.8.739

Riemann, D., Berger, M., & Voderholzer, U. (2001). Sleep and depression--results from psychobiological studies: An overview. Biol Psychol, 57(1-3), 67-103.

Rindfuss. R. (1991). The young adult years: Diversity, structural change, and fertility. Demography, 28 (4).

Roberts, R. E., Deleger, S., Strawbridge, W. J., & Kaplan, G. A. (2003). Prospective association between obesity and depression: Evidence from the Alameda County Study. Int J Obes Relat Metab Disord, 27(4), 514-521.

Roberts, R. E., Lewinsohn, P. M., & Seeley, J. R. (1991). Screening for adolescent depression: A comparison of depression scales. Journal of the American Academy of Child and Adolescent Psychiatry. 30, 58–66.

Rogosa, D., Brandt, D., & Zimowski, M. (1982). A growth curve approach to the measurement of change. Psychological Bulletin, 92(3), 726-748.

Ross, C. E. (1994). Overweight and depression. J Health Soc Behav, 35(1), 63-79.

Ryan, N. D., Puig-Antich, J., Ambrosini, P., Rabinovich, H., Robinson, D., Nelson, B., et al. (1987). The clinical picture of major depression in children and adolescents. Arch Gen Psychiatry, 44, 854-861.

Sampson, S. M. & Mrazek, D. A. (2001). Depression in adolescence. Curr Opin Pediatr, 13(6), 586-590.

Schafer, J. L. & Graham, J. W. (2002). Missing data: Our view of the state of the art. Psychol Methods, 7, 147-77.

Schutz Y. & Woringer V. (2002). Obesity in Switzerland: A critical assessment of prevalence in children and adults. International Journal of Obesity, 26(supplement 2), S3–S11.

Shields, M. (2006). Overweight and obesity among children and youth. Health Rep, 17(3), 27-42.

Shoelson, S. E., Herrero, L., & Naaz, A. (2007). Obesity, inflammation, and insulin resistance. gastroenterology, 132(6), 2169-2180. doi: http://dx.doi.org/10.1053/j.gastro.2007.03.059

Singer, J. D. & Willett, J. B. (2003). Survival Analysis. In J. A. Schinka & W. F. Velicer (Eds.), Handbook of Psychology, Volume 2: Research Methods in Psychology, Chapter 22, pages 555-580 (New York, NY: John Wiley & Sons).

Sørensen, T. I., Stunkard, A. J., Teasdale, T. W., & Higgins, M. W. (1983). The accuracy of reports of weight: Children's recall of their parents' weights 15 years earlier. International Journal of Obesity, 7, 115-122.

Statistics Canada. (2009). Microdata user's guide: National longitudinal survey of children and youth, cycle 7. Ottawa: Statistics Canada.

Stevens, J., Keil, J. E., Waid, L. R., & Gazes, P.C. (1990). Accuracy of current, 4-year, and 28-year self-reported body weight in an elderly population. Am. J. Epidemiol., 132, 1156–1163.

Stice, E. & Bearman, S. K. (2001). Body-image and eating disturbances prospectively predict increases in depressive symptoms in adolescent girls: A growth curve analysis. Dev Psychol, 37(5), 597-607.

Stice, E., Hayward, C., Cameron, R. P., Killen, J. D., & Taylor, C. B. (2000). Body-image and eating disturbances predict onset of depression among female adolescents: A longitudinal study. J Abnorm Psychol, 109(3), 438-444.

Stice, E., Presnell, K., Shaw, H., & Rohde, P. (2005). Psychological and behavioral risk factors for obesity onset in adolescent girls: A prospective study. J Consult Clin Psychol, 73(2), 195-202.

Strawbridge, W. J., Deleger, S., Roberts, R. E., & Kaplan, G. A. (2002). Physical activity reduces the risk of subsequent depression for older adults. American Journal of Epidemiology, 156(4), 328-334. doi: 10.1093/aje/kwf047

Stunkard, A. F. & Albaum, J. M. (1982). The accuracy of self-reported weights. Am J Clin Nutr, 34, 1593–1599.

Sweeting, H. N. (2007). Measurement and definitions of obesity in childhood and adolescence: A field guide for the uninitiated. Nutr J, 6, 32.

Tang-Peronard, J. L. & Heitmann, B. L. (2008). Stigmatization of obese children and adolescents, the importance of gender. Obes Rev, 9(6), 522–534.

Tanofsky-Kraff, M., Cohen, M. L., Yanovski, S. Z., Cox, C., Theim, K. R., Keil, M., et al. (2006). A prospective study of psychological predictors of body fat gain among children at high risk for adult obesity. Pediatrics, 117(4), 1203-1209. doi: 10.1542/peds.2005-1329

Tjepkema, M. (2006). Adult Obesity. Health Reports. Vol. 17, no. 3. August. Statistics Canada Catalogue no. 82-003. p. 9–24. /studies-etudes/82-003/archive/2006/9276-eng.pdf (accessed May 10, 2010).

Ternouth A., Collier D., & Maughan B. (2009). Childhood emotional problems and self-perceptions predict weight gain in a longitudinal regression model. BMC Med, 7, 46.10.1186/1741-7015-7-46

Tunceli, K., Li, K., & Williams, L. K. (2006). Long-term effects of obesity on employment and work limitations among U.S. Adults, 1986 to 1999. Obesity (Silver Spring), 14(9), 1637-1646.

Ullman. J.B. (1996). Structural equation modeling (In: Using Multivariate Statistics, Third Edition, B.G. Tabachnick and L.S. Fidell, Eds.). HarperCollins College Publishers. New York, NY. pp. 709-819.

Vaccarino, V., Johnson, B. D., Sheps, D. S., Reis, S. E., Kelsey, S. F., Bittner, V., et al. (2007). Depression, inflammation, and incident cardiovascular disease in women with suspected coronary ischemia: the National Heart, Lung, and Blood Institute-sponsored WISE study. J Am Coll Cardiol, 50(21), 2044-2050. doi: 10.1016/j.jacc.2007.07.069

Wadden, T. A. & Stunkard. (1987). A. J. Psychopathology and obesity. Ann N Y Acad Sci, 499, 55–65.

Walker, B. R. (2001). Activation of the hypothalamic-pituitary-adrenal axis in obesity: Cause or consequence? Growth Horm IGF Res, 11 Suppl A, S91-95.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997) Predicting obesity in young adulthood from childhood and parental obesity. New Eng J Med 337, 869–873.

Wight, R. G., Sepúlveda, J. E., & Aneshensel, C. S. (2004). Depressive symptoms: How do adolescents compare with adults? Journal of Adolescent Health, 34(4), 314-323. doi: http://dx.doi.org/10.1016/j.jadohealth.2003.05.003

Williamson, D. E., Birmaher, B., Brent, D. A., Balach, L., Dahl, R. E., & Ryan, N. D. (2000). Atypical symptoms of depression in a sample of depressed child and adolescent outpatients. J Am Acad Child Adolesc Psychiatry, 39(10), 1253-1259. doi: 10.1097/00004583-200010000-00012

World Health Organization. (2003). Global strategy on diet, physical activity and health: Obsity and overweight. from http://www.who.int/dietphysicalactivity/strategy/eb11344/strategy_english_web.pdf

World Health Organization. (2006). The WHO child growth standards. The World Health Organization: Geneva.

Wothke, W. (1996). Models for multitrait-multimethod matrix analysis. In G. Marcoulides and R. Schumacher [Eds.] Advances structural equation modeling techniques. Mahwah, NJ: Lawrence Erlbaum Associates.

Wurtman, J., Wurtman, R., Mark, S., Tsay, R., Gilbert, W., & Growdon, J. (1985). d-Fenfluramine selectively suppresses carbohydrate snacking by obese subjects. Int J Eat Disord, 4(1), 89-99.

Xie, B., Unger, J. B., Gallaher, P., Johnson, C. A., Wu, Q., & Chou, C. P. (2010). Overweight, body image, and depression in Asian and Hispanic adolescents. Am J Health Behav, 34(4), 476-488.

Appendix

Appendix A: Supplementary Literature review tables.

Table 1. Studies examining body mass as a predictor of depressive symptoms.

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Anderson, Murray, Johnson, et al., 2011	Random sample from multi-center group-randomized intervention trial in middle-school girls from 6 states in the United States. Hispanic, non-Hispanic black, and non-Hispanic White were only included. *Sample size*: 918 *Baseline age (y)*: 11 *Follow-up*: 2 cycles over 2 years. *Sex*: Females	Measured H&W. *Categorical* (CDC reference-obese vs. not obese). *Continuous* (BMI z-score).	CES-D (SR). *Categorical* (scores ≥ 24 = clinically significant depressive symptoms). *Continuous* (depressive symptoms).	*Age, Time typically spent home alone after school, Lunch type* (free/reduced), *Race*. *Baseline dependent variable*. Controlled in analyses. Excluded in analyses. *Moderation analyses*: Obesity X Race	*Multivariate logistic regression*. Unadjusted and adjusted ORs and 95% CIs were calculated for obese status (obese vs. Not obese) at C1 on subsequent CSDS 2 years later. *Multivariate linear regression* using depressive symptoms scores. *Moderation analyses*: Sig (Race)	*Not adjusted for baseline dependent variable.* **White: 2.48 (1.93, 3.19).** Black: 1.03 (0.46, 2.31). Hispanic: 0.86 (0.38, 1.94).	*Controlled baseline dependent variable.* **White: 2.09 (1.44, 3.02).** Black: 1.29 (0.57, 2.88). Hispanic: 0.76 (0.31, 1.85). *Excluded baseline dependent variable.* **White: 2.5 (1.57, 3.98).** Black: 0.98 (0.16, 5.97). Hispanic: 0.72 (0.26, 1.95). Results using continuous variables were consistent with results using dichotomous variables (results not shown).	Obesity status predicted subsequent clinically significant depressive symptoms (CSDS) only for White females, both before and after covariate adjustement, and both when baseline CSDS were controlled for or when females with baseline CSDS were excluded.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Ball, Burton, & Brown, 2009	Community-based study of young women in Australia. *Sample size*: 6677 *Baseline age (y)*: 22-27 *Follow-up*: 2 cycles over 3 years. *Sex*: Females	SR H&W. *Categorical* (WHO classifications-underweight, normal weight, overweight, obese).	CES-D-10 (SR). *Categorical* (scores ≥ 10 = clinically significant depressive symptoms).	*Education, Marital status, Occupation, Smoking status, Parity status, Serious health problems/disability, Physical activity*. Excluded pregnant women. *Baseline dependent variable.* Controlled in analyses.	*Multivariate logistic regression*. Crude and adjusted ORs and 95% CIs were calculated for the influence of C1 weight status (underweight, overweight, obese, vs. normal weight) on subsequent CSDS.	*All ORs not adjusted for baseline dependent variable*. Underweight 1.26 (0.99,1.6): **Overweight 1.32 (1.14, 1.53); Obese 1.81 (1.50, 2.18)**.	Underweight: 1.09 (0.83, 1.42); **Overweight: 1.21 (1.03, 1.43); Obese: 1.33 (1.08, 1.65)**.	Overweight and obese females, compared with normal weight females, had higher risk for clinically significant depressive symptoms at follow-up, both before and after covariate adjustment. NS for underweight females.
Boutelle, Hannan, Fulkerson, et al., 2010	Community-based study of adolescents in metropolitan area of Austin, Texas. *Sample size*: 496 *Baseline age (y)*: ~13.5 *Follow-up*: 4 annual cycles *Sex*: Females	Measured H&W. *Categorical* (CDC reference-not overweight, overweight, obese).	K-SADS (SR). *Continuous* (depressive symptoms).	*Age, Early puberty status.* *Baseline dependent variable.* Controlled in analyses. Excluded in analyses.	*Linear regression.* Adjusted coefficients with GEE to calculate influence of obesity status (overweight, obese, vs. not overweight) the previous year on depressive symptoms the subsequent year.	Not presented.	*Controlled baseline dependent variable.* Overweight: β = 0.0016 (SE = .03, *p* = .96); **Obese: β = 0.17 (SE = .05, *p* .01)**. *Excluded baseline dependent variable.* Did not mention results.	Overweight status was not associated with subsequent depressive symptoms. Obese status was associated with subsequent higher depressive symptoms when compared to females who were not overweight.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Clark, Haines, Head, et al., 2007	Community-based sample of adolescents attending school in East London. *Sample size*: 1513 *Baseline age (y)*: 11-14 *Follow-up*: 2 cycles over 2 years *Sex:* Both	Measured H&W. *Categorical* (United Kingdom reference curve classifications- overweight: BMI > 95th percentile, obese: BMI > 85th percentile).	SMFQ (SR). *Categorical* (scores ≥ 8 = clinically significant depressive symptoms).	*Age, Gender, Eligibility for free school meals, Ethnicity, General health status, Long-standing illness, Smoking, Alcohol use, Drug use, Physical activity.* *Baseline dependent variable.* Controlled in analyses. Excluded in analyses. *Moderation analyses:* Gender X Age	*Multivariate logistic regression*. Crude and adjusted ORs and 95% CIs were calculated for the influence of C1 weight status (overweight, obese, vs. not overweight) on subsequent CSDS. *Moderation analyses:* NS (Gender)	*Not adjusted for baseline dependent variable.* Overweight: 1.20 (0.95, 1.53); Obese: 1.04 (0.80, 1.36).	*Controlled baseline dependent variable.* Overweight: 1.16 (0.90, 1.50); Obese: 0.99 (0.75, 1.30). *Excluded baseline dependent variable.* Similar results (not presented).	Neither overweight status nor obesity status predicted subsequent clinically significant depressive symptoms, either before or after covariate adjustement.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Goodman & Whitaker, 2002	Nationally representative cohort of adolescents in the US. Non-Hispanic White, non-Hispanic Black, and Hispanic were included only. *Sample size*: 9374 *Baseline age (y)*: 12-19 *Follow-up*: 2 cycles over 1 year *Sex*: Both	SR H&W. *Categorical* (CDC reference- not obese, obese; for participants 20 years and older at follow-up, obese: BMI $\geq$ 30).	Modified CES-D-18 (SR). *Categorical* (scores $\geq$ 24 for females and $\geq$ 22 for males = clinically significant depressive symptoms).	Level 1 covariates: *Age, Gender, Race (White non-Hispanic/ Black non-Hispanic/ Hispanic), Parent education, Two parents in the home vs. Other.* Level 2 covariates: *Self-esteem, Smoking, Delinquent behavior, Low physical activity.* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses*: Self-esteem X CSDS, Smoking X CSDS, Delinquent behavior X CSDS, Low physical activity X CSDS	*Multivariate logistic regression*. Unadjusted and adjusted ORs and 95% CIs were calculated for obese status (obese vs. Not obese) at C1 on subsequent CSDS 1 year later. *Moderation analyses*: NS (All)	9.9% of those obese at C1 had CSDS at follow-up, compared with 8.7 of those not obese at baseline (p = 0.43).	Level 1 covariates: 1.16 (0.81, 1.65). Level 1 and 2 covariates: NS (results not reported).	Those who were obese at baseline did not have increased risk of clinically significant depressive symptoms at follow-up.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Herva, Laitinen, Miettunen, et al., 2006	Community-based study of adolescents in two provinces of Finland (all Caucasian). *Sample size:* 8451 *Baseline age (y):* 14 *Follow-up:* 2 cycles over 17 years *Sex:* Both	SR H&W. *Categorical* (internal references- overweight: 85th-95th percentile, obese: BMI ≥ 95th percentile)	HSCL. *Categorical* (three different score cut- offs used = 1.55, 1.75, 2.01).	*Father`s social class, Family type, Chronic somatic disease, Smoking, Alcohol use.* *Baseline dependent variable.* Did not control for or exclude in analyses.	*Multivariate logistic regression.* Crude and adjusted ORs and 95% CIs were calculated for the influence of C1 weight status (overweight, obese, vs. not overweight) on subsequent CSDS status. *Moderation analyses:* Stratified by gender.	*All HSCL cut-offs for overweight:* NS. *HSCL cut-off of 1.55.* Males: obese 1.15 (0.79, 1.69). Females: obese 1.34 (0.98, 1.82). *HSCL cut-off of 1.75.* Males: obese 1.26 (0.80, 1.96). **Females: obese 1.63 (1.16, 2.29).** *HSCL cut-off of 2.01.* **Males: obese 1.94 (1.07, 3.52).** Females: obese 1.49 (0.89, 2.47).	All HSCL cut-offs for overweight: NS. *HSCL cut-off of 1.55.* Males: obese 1.08 (0.72, 1.61). Females: obese 1.33 (0.97, 1.83). *HSCL cutoff of 1.75.* Males: obese 1.18 (0.73, 1.89). **Females: obese 1.64 (1.16, 2.32).** *HSCL cutoff of 2.01.* **Males: obese 1.97 (1.06, 3.68).** Females: obese 1.55 (0.93, 2.59).	Overweight status at age 14 did not predict subsequent clinically significant depressive symptoms (CSDS) at any cut-off point. When using a HSCL cut-off of 1.75, obese females at age 14 had higher risk of CSDS at 31, compared with not overweight females. When using a HSCL cutoff of 2.01, males with obesity at 14 had higher risk of CSDS at 31, compared with not overweight males.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Merten, Wickrama, & Williams, 2008	Nationally representative cohort of US adolescents. *Sample size*: 7881 *Baseline age (y)*: 12-18 *Follow-up*: 2 cycles over 6 years *Sex*: Both	SR H&W. *Categorical* (CDC reference- not obese, obese).	CES-D-9 (SR). *Continuous* (depressive symptoms).	*Parental education, Family economic hardship, Young adult status attainment, Race.* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses*: Gender X Obesity, Obesity X Race	*Multivariate linear regression*. Adjusted coefficients to estimate influence of obesity status (obese vs. not obese) in adolescence on subsequent depressive symptoms in young adulthood. *Moderation analyses*: Sig. (Gender), NS (Race)	N/A	*Females.* **β = 0.03, SE = 0.19, *p* < .001**. *Males.* β = 0.01, SE = 0.14, NS.	Among females, obesity in adolescence was associated with higher depressive symptoms in young adulthood. No significant associations between obesity and depressive symptoms for males.
Rhew, Richardson, Lymp, et al., 2008	Community-based study of sixth grade students from public middle schools in Seattle, Washington. Oversampled students scoring high on depression or disruptive behavior. *Sample size*: 446 *Baseline age (y)*: ~12 *Follow-up*: 2 cycles over 1 year *Sex:* Both	SR H&W. Measured H&W (n=165). *Categorical* (CDC reference- not overweight, overweight, obese).	MFQ (SR). *Continuous* (depressive symptoms-log transformed).	*Gender, Household income, Education of primary caregiver, Physical development, Race (Black, Pacific Islander, Other).* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses*: Depression X Gender	*Multivariate linear regression*. Unadjusted and adjusted ORs and 95% CIs were calculated for overweight status (overweight vs. Not overweight and obese vs. not overweight) at C1 on subsequent depressive symptoms 1 year later. *Moderation analyses*: NS (Gender)	N/A.	*Self-report BMI*. β= -0.08, *p* = 0.52. *Measured BMI*. Similar results. Similar results when using a obese instead of overweight.	Neither overweight status nor obese status at baseline predicted subsequent depressive symptoms 1 year later, regardless of whether BMI was self-reported or measured.

Table 1. Studies examining body mass as a predictor of depressive symptoms (continued).

Author(s), Year	Sample Characteristics	Body Mass measure	Depressive Symptoms measure	Covariates	Analysis type	Unadjusted odds ratio (95% confidence interval) or other statistics reported	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Xie, Unger, Gallaher, et al., 2010	Community sample of middle school students in Los Angeles (Asians and Hispanics only). Data came from smoking prevention trial study. *Sample size*: 1155 *Baseline age (y)*: 12 *Follow-up*: 2 cycles over 1 year *Sex*: Both	Measured H&W. *Categorical* (CDC reference- overweight, not overweight).	CES-D-5 (SR). *Continuous* (depressive symptoms).	*Pubertal status, SES, Smoking status, Age, Ethnicity, Intervention group.* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses*: Gender X Overweight, Race X Overweight, Acculturation X Overweight	*Multivariate linear regression.* Multiple group SEMs to compare associations of overweight status (vs. not overweight status) on subsequent depressive symptoms; separately for males and females, Asians and Hispanics, and accultured and not accultured. *Moderation analyses*: Sig. (Gender, Race, Acculturation)	N/A	Standardized (unstandardized) parameter estimates. Girl, Low AC: 0.13 (0.07, NS). Girl, High AC: -0.14 (-0.07, NS). **Boy, Low AC: 0.22 (0.14, *p* < .05).** Boy, High AC: 0.06 (0.04, NS). Girl, Asian: -0.26 (-0.09, NS). Girl, Hispanic: 0.01 (0.01, NS). **Boy, Asian: 0.26 (0.15, p < .05).** Boy, Hispanic: 0.12 (0.11, NS)	Asian males who were overweight at baseline had higher depressive symptoms at follow-up than non-overweight Asian males. Boys with low acculturation who were overweight at baseline had higher subsequent depressive symptoms compared to their not overweight counterparts. No other significant associations.

CDC classifications: Obesity= greater than or equal to 95th percentile; Overweight = 85th
WHO classifications: Obesity = BMI≥30; Overweight = BMI 25-29.99; Normal weight = BMI 18.50-24.99; Underweight = BMI<18.5

SR: self-reported. H&W: height and weight. Sig: significant. AC: Accultured. OR: odds ratio. CI: Confidence interval. CSDS: Clinically significant depressive symptoms. SES: socioeconomic status. NS: not significant. N/A: not applicable or not analyzed. CDC: Center for Disease Control. BMI: body mass index. WHO: World Health Organization. CES-D: Center for Epidemiological Studies- Depression Scale. MFQ: Mood and Feeling Questionnaire. SMFQ: Short Mood and Feeling Questionnaire. HSCL = Hopkins Symptom Checklist-25. SE: standard error. K-SADS: Schedule for Affective Disorders and Schizophrenia for School-Age Children. GEE: generalized estimating equations. SEM: structural equation model.

Table 2 . Studies examining depressive symptoms as a predictor of body mass.

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Anderson, Murray, Johnson, et al., 2011	Random sample from multi-center group-randomized intervention trial in middle-school girls from 6 states in the US. Hispanic, non-Hispanic black, and non-Hispanic White were only included. *Sample size* : 918 *Baseline age (y)* : 11 *Follow-up* : 2 cycles over 2 years. *Sex* : Females	Measured H&W. *Categorical* (CDC reference-obese vs. not obese). *Continuous* (BMI z-score).	CES-D (SR). *Categorical* (scores ≥ 24 = clinically significant depressive symptoms). *Continuous* (depressive symptoms).	*Age, Time typically spent home alone after school, Lunch type* (free/reduced), *Race* . *Baseline dependent variable* . Controlled in analyses. Excluded in analyses. *Moderation analyses* : Obesity X Race	*Multivariate logistic regression* . Unadjusted and adjusted ORs and 95% CIs were calculated for CSDS status at C1 on subsequent obesity status 2 years later. *Multivariate linear regression* using BMI. *Moderation analyses* : Sig (Race)	*Not adjusted for baseline dependent variable.* **White: 4.13 (1.77, 9.64).** Black: 0.82 (0.40, 1.67). Hispanic: 1.66 (0.58, 4.73)	*Controlled baseline dependent variable.* **White: 3.68 (1.72, 7.87).** Black: 1.58 (0.65, 3.83). Hispanic: 1.64 (0.36, 7.49). *Excluded baseline dependent variable.* White: 3.11 (0.85, 11.32). Black: 1.56 (0.51, 4.72). Hispanic: 1.34 (0.12, 14.81). Results using continuous variables were consistent with results using dichotomous variables (results not shown).	Depression predicted subsequent obesity status only in White females, in both unadjusted and adjusted models, controlling for baseline obesity. In adjusted models that excluded females with baseline obesity status, depression did not predict subsequent obesity onset for any race.
Bardone, Moffitt, Caspi, et al., 1998	Cohort of adolescents from New Zealand. *Sample size* : 459 *Baseline age (y)* : 15 *Follow-up* : 2 cycles over 6 years *Sex* : Females	Measured H&W. *Continuous* (BMI).	DISC (Interview). *Categorical* (diagnosis of MDD or dysthymia = depression)	*SES, Age at menarche, Absence of a father figure, Parental smoking, Childhood health, Maternal health, Maternal BMI, Religiosity, Daughter of teenage mother.* *Baseline dependent variable.* Did not control for or exclude in analyses.	*Multivariate linear regression* . Unadjusted and adjusted coefficients to estimate influence of depression (depressed vs. Not depressed) at age 15 on subsequent BMI at age 21.	β = 0.06 (NS)	β = 0.05 (NS)	Depression at age 15 did not predict BMI at age 21, both before and after covariate adjustment.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Barefoot, Heitmann, Heims, et al., 1998	Cohort of University of North Carolina students. *Sample size* : 4673 *Baseline age (y)* : 16-25 *Follow-up* : 2 cycles over 23 years *Sex:* Both	Measured H&W at baseline. SR H&W at follow-up. *Continuous* (change in BMI).	MMPI ODS (SR). *Categorical* (scores ≥ 13 for females and ≥ 11 for males = clinically significant depressive symptoms). *Continuous* (depressive symptoms).	*Gender, Exercise, Smoking.* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses* : Gender X BMI, BMI X CSDS	*Multivariate linear regression* . Adjusted coefficients to estimate influence of CSDS status on BMI change between two cycles. *Moderation analyses* : Sig (All)	N/A	**β = -19.68,** *p* **< 0.001.**	Participants with clinically significant depressive symptoms who were initially lean gained less weight than lean participants who without clinically significant depresive symptoms. Participants with clinically significant depressive symptoms who were intitially heavy appear to have gained more weight than heavy participants without clinically significant depressive symptoms.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Duarte, Sourander, Nikolakaros, et al., 2010	National population-based cohort of children born in Finland in 1981. *Sample size* : 2209 *Baseline age (y)* : 8 *Follow-up* : 2 cycles over 10-15 years *Sex* : Males	Measured H&W. *Categorical* (WHO classification- normal weight, overweight, obese)	CDI (SR). *Categorical* (internal reference- < 50th percentile = no CSDS, 50th-90th percentile = moderate CSDS, > 90th percentile = severe CSDS. *Continuous* (number of symptoms).	*Mother's education, Age at follow-up.* *Baseline dependent variable.* Did not control for or exclude in analyses. Excluded underweight.	*Multivariate logistic regression* . Adjusted ORs and 95% CIs were calculated for the influence of childhood CSDS status on subsequent weight status (obese, overweight).	N/A	*Categorical depressive symptoms.* **Moderate CSDS → overweight: 1.3 (1.01, 1.7)**. Moderate CSDS → obesity: 1.0 (0.6, 1.6). Severe CSDS → overweight: 1.2 (0.8, 1.8). Severe CSDS → obesity: 0.6 (0.2, 1.4). *Continuous depressive symptoms* . Overweight: 1.1 (0.98, 1.2); Obese: 0.9 (0.7, 1.2)	Children with moderate clinically significant depressive symptoms (CSDS) at age 8 were more likely to be overweight in young adulthood compared to children without CSDS after covariate adjustement. Nothing significant for moderate CSDS predicting obesity, or severe CSDS predicting overweight/obesity. With continuous depressive symptoms, depressive symtpoms at age 8 years were not associated with being overweight or obese.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Franko, Striegel-Moore, Thompson, et al., 2005	Two community-based cohorts of Black and White girls in 3 US states (California, Maryland, Ohio). *Sample size* : 1554 *Baseline age (y)* : ~16.5 *Follow-up* : 3 cycles at ages 16, 18, and 21 *Sex* : Females	SR H&W at C1, C2. Measured H&W at C3. *Categorical* (CDC reference- not obese, obese; for participants 21 years and older at follow-up, obese = BMI ≥ 30). *Continuous* (BMI).	CES-D (SR). *Continuous* (depressive symptoms).	*Race (Black/ White), Site (Ohio/ Maryland/ California), Parental education.* *Baseline dependent variable* . Controlled in analyses. *Moderation analyses* : Race X Depressive symptoms	*Multivariate linear regression* . Adjusted coefficients to estimate influence of depressive symptoms at age 16 and 18 (separate models) on subsequent BMI at age 21. *Multivariate logistic regression.* Adjusted ORs and 95% CIs calculated for the influence of depressive symptoms at age 16 and 18 (separate models) on subsequent obesity at age 21.	N/A	*Multivariate linear regression.* **Depressive Symptoms at age 16: β = 0.03 (SE = .009, p = .002). Depressive Symptoms at age 18: β=0.02 (SE = .008, p = 0.01).** *Multivariate logistic regression.* **Depressive Symptoms at age 16: 3.11 (1.13, 5.12). Depressive Symptoms at age 18: 3.50 (1.26, 5.80).**	Higher depressive symptoms at age 16 or 18 predicted increased risk of obesity at age 21, after covariate adjustment. Same pattern when BMI was continuous.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Goodman & Whitaker, 2002	Nationally representative cohort of adolescents in the US. Non-Hispanic White, non-Hispanic Black, and Hispanic were included only. *Sample size* : 9374 *Baseline age (y)* : 12-19 *Follow-up* : 2 cycles over 1 year *Sex* : Both	SR H&W. *Categorical* (CDC reference- not obese, obese; for participants 20 years and older at follow-up, obese: BMI ≥ 30).	Modified CES-D-18 (SR). *Categorical* (scores ≥ 24 for females and ≥ 22 for males = clinically significant depressive symptoms).	Level 1 covariates: *Age, Gender, Race (White non-Hispanic/ Black non-Hispanic/ Hispanic), Parental obesity, Parent education, Two parents in the home vs. Other.* Level 2 covariates: *Self-esteem, Smoking, Delinquent behavior, Low physical activity.* *Baseline dependent variable* . Controlled BMI z-score in analyses. Excluded obese in analyses. *Moderation analyses* : Gender, Self-esteem X CSDS, Smoking X CSDS, Delinquent behavior X CSDS, Low physical activity X CSDS	*Multivariate logistic regression* . Unadjusted and adjusted ORs and 95% CIs were calculated for clinically significant depressive symptoms at C1 on subsequent obesity status 1 year later. *Moderation analyses* : Sig (Smoking), NS (All else)	12.4% of those with CSDS at C1 were obese at follow-up, compared with 9.4% of those without CSDS at baseline **(p = .048)**.	Level 1 covariates. **Controlled for baseline BMI z-score: 2.05 (1.18, 3.56)**. **Excluded obese at baseline: 2.05 (1.04, 4.06)**. Level 1 and 2 covariates. Controlled for baseline BMI z-score: did not present results. **Excluded obese at baseline: 2.39 (1.05, 5.45)**.	Those with clinically significant depressive symptoms (CSDS) at baseline had a higher risk of obesity at follow-up, compared to those without CSDS at baseline. Similar magnitude of effect between analyses that excluded obese at baseline and analyses that controlled for baseline BMI z-score. Those with CSDS who smoked heavily were at an increased risk for worsening obesity, compared to those who did not smoke.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Goodwin, Sourander, Duarte, et al., 2009	National population-based cohort of children born in Finland in 1981. Sample size: 2712 Baseline age (y): 8 Follow-up: 3 cycles over 10-15 years Sex: Males	Measured H&W. *Categorical* (physician diagnosis of obesity using ICD-10 criteria).	CDI (SR). *Categorical* (internal reference- < 50th percentile = no CSDS, 50th-90th percentile = moderate CSDS, > 90th percentile = severe CSDS. *Continuous* (number of symptoms).	*Mother's education, Somatic health problems at age 8.* *Baseline dependent variable.* Did not control for or exclude in analyses.	*Multivariate logistic regression* . Unadjusted and adjusted ORs and 95% CIs were calculated for the influence of childhood CSDS status on subsequent obesity in young adulthood.	Moderate CSDS: 1.4 (0.9, 2.3). Severe CSDS: 0.7 (0.3, 1.8).	Moderate CSDS: 1.4 (0.9, 2.3). Severe CSDS: 0.5 (0.2, 1.4).	CSDS in childhood did not predict increased risk of obesity in young adulthood, both before and after covariate adjustment. Similar results with continuous depressive symptoms.
Jansen, van de Looij-Jansen, de Wilde, et al., 2008	Community-based study of youth in Rotterdam (Netherlands). *Sample size* : 787 *Baseline age (y)* : 9-10 *Follow-up* : 2 cycles over 3 years *Sex* : Both	Measured H&W at C1, SR H&W at C2. *Categorical* (WHO classifications-not overweight, overweight, obese).	SDQC (SR). *Categorical* (score ≥ 4 = clinically significant depressive symptoms).	*Gender, Country of origin, Level of education.* *Baseline dependent variable* . Controlled in analyses. *Moderation analyses* : Gender X Weight Status	*Multivariate logistic regression* . Adjusted ORs and 95% CIs were calculated for the influence of childhood CSDS on subsequent weight status (obese, overweight). *Moderation analyses* : NS (Gender)	N/A	Did not present results.	Clinically significant depressive symptoms at C1 did not predict weight status 3 years later, after covariate adjustment.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Pine, Cohen, Brook, et al., 1997	Community-based study of an adolescent cohort in upstate New York. *Sample size* : 644 *Baseline age (y)* : 9-18 *Follow-up* : 2 cycles over 9 years *Sex* : Both	SR H&W. *Categorical* (internal reference-obesity = BMI > 80th percentile). *Continuous* (BMI).	DISC (from either mother or child report at C1, from child only at C2). *Continuous* (depressive symptoms).	*Age, Ethnicity, Parental social class, Parental sociopathy, Physical health status, Childhood IQ, Smoking and alcohol use.* *Baseline dependent variable.* Did not control for or exclude in analyses.	*Multivariate linear regression* . Unadjusted and adjusted coefficients to estimate influence of depressive symptoms during adolecence on subsequent BMI in young adulthood. *Multivariate logistic regression.* Adjusted ORs and 95% CIs calculated for the influence of depressive symptoms during adolecence on subsequent BMI in young adulthood.	*Categorical.* Males: 1.46 (0.78, 2.74). **Females: 3.06 (1.91, 4.91).** *Continuous .* Not presented.	Categorical. Not presented. *Continuous.* **β = 0.38 ± .19 (p < .05).** When conduct disorder entered into model: β = 0.15 ± .20 (NS).	Before covariate adjustment, depressive symptoms during adolescence predicted young adult obesity in females, but not males. In multivariate associations, depressive symptoms during adolescence predicted young adult BMI only when adolescent conduct disorder was not included in the model (males and females grouped together).
Rhew, Richardson, Lymp, et al., 2008	Community-based study of sixth grade students from public middle schools in Seattle, Washington. Oversampled students scoring high on depression or disruptive behavior. *Sample size* : 446 *Baseline age (y)* : ~12 *Follow-up* : 2 cycles over 1 year *Sex:* Both	SR H&W. Measured H&W. *Continuous* (BMI).	MFQ (SR). *Categorical* (scores ≥ 15 = clinically significant depressive symptoms).	*Gender, Household income, Education of primary caregiver, Physical development, Race (Black, Pacific Islander, Other).* *Baseline dependent variable.* Controlled in analyses. *Moderation analyses* : Depression X Gender	*Multivariate linear regression* . Unadjusted and adjusted ORs and 95% CIs were calculated for CSDS at C1 on subsequent BMI 1 year later. *Moderation analyses* : Sig (Gender)	Self-report BMI. Males - 0.48 (-1.64, 0.67). **Females: 3.16 (1.91, 4.40).** Measured BMI. Results not reported.	Self-report BMI. **Males: β= -1.18 (-1.99, -0.37).** **Females: β= 2.49 (1.44, 3.54).** Measured BMI. Males: β = 0.12 (-0.49, 0.73). Females: β = 0.54 (-0.42, 1.51).	For self-reported BMI, males with clinically significant depressive symptoms (CSDS) at baseline had lower subsequent BMI compared to males without CSDS, after covariate adjustement (although this association was not significant before adjustment). For females, CSDS at baseline predicted higher BMI, both before and after covariate adjustment (for self-reported BMI). When BMI was measured, depression did not predict subsequent BMI levels.

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Stice, Presnell, Shaw, et al., 2005	Community-based cohort of adolescent girls from 4 public and 4 private schools in a metropolitan area of the southwestern US. *Sample size* : 496 *Baseline age (y)* : 11-15 *Follow-up* : 5 annual cycles *Sex* : Females	Measured H&W. *Categorical* (CDC classification- obesity).	SADS (SR). *Continuous* (depressive symptom severity index).	*Dietary restraint, Compensatory behaviors, Perceived parental obesity* *Baseline dependent variable*. Excluded at baseline.	*Multivariate logistic regression* . Unadjusted and adjusted ORs and 95% CIs were calculated for depressive symptoms at C1 on subsequent obesity over the 4-year follow-up period.	**4.62 (1.67, 12.74)**	2.32 (0.62, 8.65)	After covariate adjustment, depressive symptoms at C1 did not predict obesity onset during 4-year follow-up (associations were significant before adjustment).

Table 2 . Studies examining depressive symptoms as a predictor of body mass (continued).

Author(s), Year	Sample Characteristics	Body mass measure	Depressive symptoms measure	Covariates	Analysis type	Unadjusted result	Adjusted odds ratio (95% confidence interval) or other statistics reported	Observed relationship
Tanofsky-Kraff, Cohen, Yanovski, et al., 2006	Cohort of Black and White children from 3 Maryland schools, US, at increased risk for adult obesity. Sample size: 134 Baseline age (y): 6-12 Follow-up: ~4 annual cycles (follow-up periods ranged from 0.1-7.9 years Sex: Both	Measured DEXA. *Continuous* (fat mass, change in fat mass).	CDI (SR). *Continuous* (depressive symptoms).	*Age at baseline, Gender, Race, SES, Pubertal stage, Time, Self-reported dieting, Binge eating, ChEAT total score.* *Baseline dependent variable* . Controlled in analyses. *Moderation analyses* : Gender X Race, Gender X Time	*Linear regression.* Mixed model with depression as fixed effect and child as random effect. Mixed regression model for predicting influence of depression at childhood on increase in body fat over course of follow-up. Moderation analyses: NS (All)	N/A	$\beta = 0.004$, SE = 0.005 (-0.007, 0.014), *p* = .45	Depressive symptoms at baseline did not predict increases in body fat mass over time, after covariate adjustment.

CDC classifications: Obesity= 95th percentile; Overweight = 85th percentile; Not overweight = <85th percentile.

WHO classifications: Obesity = BMI≥30; Overweight = BMI≥25; Normal weight = BMI 18.50-24.99; Underweight = BMI<18.5

CDI categories: scores <50th percentile = no depression, 50-90th percentile = moderate depression, ≥90th percentile = severe depression

SR: self-reported. H&W: height and weight. C1, C2, etc.,: cycle 1, cycle 2, etc., respectively. N/A: not applicable or not analyzed. SES: socioeconomic status. Sig: significant. NS: not significant. CSDS: Clinically significant depressive symptoms. OR: odds ratio. CI: confidence interval. IQ: intelligence quotient. CDC: Center for Disease Control. BMI: body mass index. DISC: Diagnostic Interview Schedule for Children. MDD: major depressive disorder. WHO: World Health Organization. CES-D: Center for Epidemiological Studies- Depression Scale. MMPI-ODS Minnesota Multiphasic Inventory- Obvious Depression subscale. CDI: Children's Depression Inventory. ICD-10: International Classification of Diseases-10. SADS: Schedule for Affective Disorders and Schizophrenia. DEXA: Dual-energy X-ray absorpitiometry. MFQ: Mood and Feelings Questionnaire. SDQC: Short Depression Questionnaire for Children.

Curriculum Vitae

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