The Longitudinal Relationship Between Obesity and Depression in Children

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Abstract

Much of the existing literature evaluating the bidirectional associations between obesity and depression has been conducted in an adult population. We highlight the existing evidence and potential mechanisms contributing to the reciprocal relationship in various developmental stages across childhood, adolescence, and early adulthood, and propose new conceptual models of obesity and depression in children and adolescents. The purpose of this study was to evaluate the relationship between depression symptoms of children ages 2-11 (and their mothers) and weight status cross-sectionally and longitudinally. Ninety families (children ages 2-11, and their mothers) who completed measures of depression (PHQ-9, BASC-2) and had their height and weight measured at two time points (baseline and 3-month follow-up) were included in this study. Bivariate correlation and linear regression were used to evaluate the cross-sectional relationship between child depression symptoms and child weight status. Bivariate correlations and odds ratio analyses were used to assess the longitudinal, bidirectional relationship between child depression symptoms and child weight status. We also used bivariate correlation to evaluate the longitudinal relationship between baseline maternal depression symptoms and child weight change. Results indicate that both cross-sectional and longitudinal, bidirectional relationships exist between depression symptoms and weight in children ages 2-11. Additionally, baseline maternal depression symptoms and baseline maternal weight status related to child depression symptoms and child weight status at 3 months, but only in boys. Baseline maternal and child depression increases odds of child overweight/obese weight status at 3 months, and baseline child overweight/obese weight status increases odds of "at risk" or "clinically significant" depression symptoms at 3 months. Findings suggest that a relationship between weight and mood exists both cross-sectionally, and longitudinally in children ages 2-11.

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Introduction

Comorbid Depression and Obesity

Obesity and depression are two chronic disorders with high prevalence rates that affect morbidity and mortality (van Baal et al., 2008; Katon et al., 2003). Adult obesity prevalence has been estimated to be 34.9% (Ogden et al., 2014), and pediatric obesity prevalence for children and adolescents aged 2 through 19 is estimated to be at 16.9% in the United States (Ogden et al., 2010). The prevalence of current major depressive disorder (meeting criteria for DSM-IV) has been estimated to be 4.1% in adults (Centers for Disease Control and Prevention, 2010), and prevalence of depressive disorders ranges from 2.2% in 9-16 year olds (Costello, et al., 2003) to 11.2% in 13-18 year olds (Merikangas et al., 2010). While obesity and major depression each have high prevalence rates, the high prevalence rates of comorbid depression and obesity have prompted cross-sectional and longitudinal research on the comorbid conditions.

Cross-sectional research in adults has thus far revealed that obesity is associated with nearly two times the likelihood of past-month depression; severe (Class 3) obesity is related with nearly five times the likelihood of past-month depression (Onyike et al., 2003). Other studies have found a U-shaped relationship in adult primary care patients, where higher prevalence of depression is observed in underweight and obese patients, such that prevalence of depression was 24% in underweight patients, and 23% in obese patients, but only 11% of "normal" weight patients (Carey et al., 2014). A review of longitudinal studies shows that the relationship between obesity and depression is bidirectional in adults; obese individuals have 55% increased risk for depression, and depressed individuals have 58% increased risk for obesity (Luppino et al., 2010).

Relationship of Weight and Depression in Children and Adolescents

Several reviews have evaluated the relationship between obesity and depression in longitudinal studies, but these studies all excluded children and adolescents (Fabricatore et al., 2011; Luppino et al., 2010; Markowitz, Friedman, & Arent, 2008). Some studies with children do exist, however. One meta-analysis explored how depression in adolescence impacts weight as an adult (Blaine, 2008). A new review (Mühlig et al., 2016) assessed a number of cross-sectional and longitudinal studies of associations between obesity and depression in children and adolescents; however, they only required anthropometric data at one time point for inclusion in the review. They concluded that more research is needed to clarify the longitudinal, reciprocal relationship of obesity and depression in children and adolescents are lacking, and are much needed in order to clarify the longitudinal nature of the relationship between the two factors. Another article broadly reviewed the similarities in clinical presentation and presented possible mechanisms of the disorders in children on a more theoretical basis (Reeves, Postolache, & Snitker, 2008), and suggests that more longitudinal studies are needed to find predictors and factors in childhood that contribute to later depression and obesity in adulthood. An additional review evaluated factors impacting the correlation of weight and appetite disturbance in children as risk factors for depression along the developmental trajectory in cross-sectional studies (Maxwell & Cole, 2009), and suggested a developmental model relating adolescent depression to later weight change and appetite disturbance into adulthood. Characteristics of selected studies reviewed can be found in Table 1. In conclusion, there are no reviews, to date, evaluating the bidirectional *longitudinal* relationship between obesity and depression in *children*.

Effects of Weight Gain and Obesity on Mood and Depression in Children and Adolescents

Most studies have found a relationship between baseline obesity and later depression in

females and not males, with evidence that this association often exists only in white, adolescent females (Anderson et al., 2011; Marmorstein, Iacono, & Legrand, 2014), and especially in older, adolescent females (Felton et al., 2010). Additionally, the hazard ratio for subsequent major depressive disorder (MDD) was found to increase with incremental increases in BMIz in obese females (Anderson et al., 2007). Two studies found support for baseline obesity relating to later depression in both males and females (Herva et al., 2006; Loth et al., 2011). However, Loth et al. (2011) found that while the increase in depressive mood from early to mid-adolescence was found in all adolescents (both male and female, and both healthy and overweight), the increase in depressive mood was significantly greater in overweight versus healthy weight males. Mustillo et al. (2003) found that obesity was associated with later depression only in males who were considered to be "chronically obese," or obese at each wave of the longitudinal study spanning over eight years.

In a few studies, baseline obesity was not significantly associated with later onset of depression. However, obesity was associated with greater cross-sectional depression symptoms in white females for one study (Goodman & Must, 2011), and in both males and females in another study (Hammerton, Thapar, & Thapar, 2014). In another study (Rawana & Morgan, 2014), BMI was not found to significantly impact the trajectory of depression, though weight management was associated with lower initial levels of depression symptoms, and low self-esteem was associated with depressive symptoms in females only. Additionally, obesity may impact poor perceived mental health, despite any direct impacts on mood (Roberts & Hao, 2013).

A review of the literature reveals that obesity appears to account for some variance later depression. The evidence for this association appears to be stronger in females, particularly white

females, and older adolescents, though chronic obesity over a large portion of childhood and adolescence appears to account for later depression in males. Additionally, the hazard ratio for subsequent depression appears to be further raised in adolescents with very high BMIz. These results suggest that the association between obesity and later depression may be stronger in males with chronic obesity; additionally, the relationship between these issues may be more common in older, white, female adolescents, particularly those with greater BMIz. Results also suggest that decreases in depression symptoms parallel decreases in BMI in the year following bariatric surgery (Sysko et al., 2012), providing support for weight loss potentially improving overall depressive symptoms in the timeframe immediately following extreme weight loss.

Effects of Mood and Depression on Weight Gain and Obesity in Children and Adolescents

The association between baseline depression and later weight gain was found only in females in a handful of studies. This association was found only in white, adolescent females for two studies (Anderson et al., 2011; Marmorstein, Iacono, & Legrand, 2014). Some studies found support for depression relating to later weight gain in both males and females (Goodman & Whitaker, 2002; McClure et al., 2012; Roberts & Duong, 2013). Two studies suggest that the association between baseline depression and later weight gain is found only in males, particularly in childhood versus adolescence (Aparicio et al., 2013; Korczak et al., 2014). Other studies found that baseline depression was not significantly associated with later weight gain (Kubzansky, Gilthorpe, & Goodman, 2012; Larsen et al., 2014), and a single study found that treating depression symptoms did not affect weight status (Mansoor et al., 2013). However, cross-sectional levels of distress (Kubzansky, Gilthorpe, & Goodman, 2012) and depressive symptoms (Larsen et al., 2014) were associated with higher baseline BMI.

Review of studies suggests that depression appears to account for later obesity, though

findings appear somewhat mixed. Evidence for this association exists for both males and females, and for females of multiple ethnicities (White, African American, Puerto Rican) (Goodman & Must, 2011; Merten, Wickrama, & Williams, 2008). However, there is some evidence suggesting that depression in childhood and adolescence may relate to later obesity differently in males versus females. For example, the finding that greater depressive symptoms were associated with greater later BMI in pre-adolescent boys (Aparicio et al., 2013; Korczak et al., 2014), and the finding that greater depressive symptoms were associated with greater later BMI in adolescent females suggest that sex differences vary dependent on age. Studies with participants with younger baseline ages found support for depression leading to later obesity in males only, or in both males and females. On the other hand, studies that found evidence for the relationship only among females were also studies with a baseline age of late childhood or adolescence. Pubertal factors that relate to higher rates of depression in females, beginning in late childhood and adolescence, may account for differences in these differential findings. Results also suggest that a decrease in depression symptoms via short-term treatment of treatment-resistant depression with anti-depressant medication does not decrease weight or BMI. However, early onset of treatment-resistant depression, endorsement of greater distress and depressive symptoms, were associated with greater baseline BMI (Mansoor et al., 2012). This may indicate that onset of depression that occurs earlier within the developmental trajectory, and with greater endorsement of distress and depressive symptoms, may impact weight more.

Changing Factors Within the Developmental Trajectory

The developmental trajectory has several key transition points that highlight times in which different factors may impact the relationship between weight and mood. Key shifts include the transition between early childhood and the school-age period, during which many

social and environmental factors may change. Another shift includes the transition between the school-age period and adolescence, during which physiological, neurobiological, cognitive changes may occur in addition to more social and environmental changes in the process of puberty. Moderating and mediating variables highlight the need for separate child and adolescent conceptual models.

Moderating Variables

Both mediators and moderators have been mentioned by researchers exploring the relationship between weight and depression. Moderators that influence this relationship which have been noted are gender, age, ethnicity, depression severity, BMI, and biological factors (including genetics and hormones).

The longitudinal association between obesity and depression is moderated by multiple factors, and provides support for gender as a moderator for this relationship, specifically, females having greater risk for depression as a result of obesity. However, the chronicity of obesity is a moderator that appears to heighten the risk for depression in males. Contrary to what has been previously summarized in the literature, the relationship between baseline depression and later obesity appears to be less strongly moderated by gender; in fact, several studies indicated support for this association in males only.

Paralleling the impact of chronicity of obesity, the literature reveals that earlier onset of depression, with greater depression severity impacting later obesity. There is also support for BMI being a moderator of obesity leading to depression, such that the risk for subsequent depression is greater in adolescents with very high BMIz. Age appears to moderate the relationship between obesity and later depression such that older adolescents are more prone to development of depression. This is consistent with literature highlighting the estimated

prevalence of MDD to be 2% in school-age children, and 2-8% in adolescents (Birmaher et al., 1996). Incidence of depression increases in both sexes during adolescence with onset of puberty (Angold & Costello, 2006); however, the Substance Abuse and Mental Health Services Administration (SAMHSA) notes that sex differences in depression rates also emerge during this time, such that rates of depression triple in girls versus boys between the ages of 12 and 15 (SAMHSA, 2012). Due to these sex differences that appear during adolescence, it would be useful to evaluate the relationship between depression and obesity differently in children versus adolescents. Some of the findings provide further support for the value in separating children from adolescents when reviewing this relationship.

Racial/ethnic group appears to be an inconsistent moderator of the relationship between obesity and depression. One prospective study (Anderson et al., 2011) found bidirectional associations between depression and obesity that were moderated by racial/ethnic group, such that the associations were seen in White girls, but not Black or Hispanic girls. However, the study noted that confidence intervals for Black and Hispanic girls were wide, indicating uncertainty surrounding this conclusion; additionally, more variability among ethnicities and including both ethnicities in the same sample could have also contributed to this finding. Another study found that obese non-Hispanic White males and females had significantly greater levels of depression symptoms after two years than non-Hispanic Blacks (Goodman & Must, 2011). Though, yet another study found an overall relationship between baseline obesity and later depression in females, with no differences between White and African American participants (Merten, Wickrama, & Williams, 2008). Such findings indicate that racial/ethnic grouping may not be a consistent moderator, potentially due to mediation of effects by cultural and social factors; other moderators and mediators may more reliably influence the relationship between

obesity and depression.

Mediating Variables

In addition to moderators that influence the relationship between weight and mood, mediators have also been previously noted in the literature. Mediators that explain and account for this relationship include multiple psychosocial factors, such as social interactions (including parenting and teasing), body dissatisfaction, self-efficacy, eating behaviors, diet, and lack of physical activity.

Several mediators are notable, including level of self-esteem, presence of weight-management efforts, perceived mental health, smoking behaviors, and adolescent overweight status. Low self-esteem in girls appears to explain the increased risk for depressive symptoms across adolescence into young adulthood (Rawana & Morgan, 2014) and is a predictor of later depressed mood in obese adolescents (Goodman & Whitaker, 2002). This is consistent with previous findings from a prospective study that demonstrated that obese children tend to have decreasing levels of self-esteem that tracks into adolescence, and correlates with increasing feelings of sadness and loneliness that were also linked with health behaviors such as smoking and alcohol consumption (Strauss, 2000). Such findings suggest that self-esteem plays a role in mediating the bidirectional relationship between obesity and depression in adolescents.

The literature also showed that engagement in weight management efforts was associated with lower levels of depressive symptoms (Rawana & Morgan, 2014). Possible explanations offered include possible engagement in healthier lifestyle behaviors, such as better dietary consumption and healthy levels of exercise. These findings support the utility of therapeutic lifestyle changes and interventions, such as healthy diet and physical activity, which have been shown to reduce risk for depression (Stathopoulou et al., 2006; Wiles et al., 2012).

Poor perceived mental health may be a mediator that consists of various components, including depression, where obesity increases risk for poor perceived mental health, which may translate into later increased risk for mood disturbances (Roberts & Hao, 2013). In the same article, the authors argued that overweight or obese youths are at minimal risk for future social and mental health consequences; however, they also acknowledge that they did not examine other possible mediators, such as social relationships, teasing and bullying, and body image. It is possible that these other factors may play additional mediating roles.

Smoking behavior may also partially explain the relationship between depression and later obesity. Findings suggest that depressed mood is related to smoking, and depressed, obese youth who are heavy smokers are at increased risk for later worsening obesity (Goodman & Whitaker, 2002). This evidence is consistent with prior findings of the bidirectional relationship that is present between depression and smoking (Vogel et al., 2002) and the relationship between obesity and smoking (Healton et al., 2006).

While certain biopsychosocial factors may not directly impact later obesity, one study noted that depression symptoms do not directly predict later obese BMI in adulthood, but indirectly impact later obesity through overweight BMI status in adolescence, which then persists into adulthood (McClure et al., 2012). Such findings argue for the importance of viewing the relationship between obesity and depression from a developmental perspective.

Conceptualizing a Model of Childhood and Adolescent Depression and Obesity

Existing Models

Several models can be implemented in order to conceptualize the longitudinal relationship between depression and obesity. One model is the social ecological model, which incorporates personal and environmental factors. Social ecological models have been proposed in

the realm of obesity (Davison and Birch, 2001; Story et al., 2008), where individual characteristics such as biology, demographic background, temperament, and cognition play a role in the context of the concentric circles representing hierarchical levels of environmental influences. Criticisms of existing social ecological models are that these models are primarily theoretical outlines of complex systems and do not suggest causal relationships or pathways between factors, which make them unsuitable for outlining longitudinal relationships.

Another model (Lytle, 2009), outlines a transdisciplinary social ecological model of obesity-related risk factors in the etiology of childhood obesity that was implemented in a longitudinal cohort study following youth between the ages of 10-16 at baseline over a span of three years. This model was effective in narrowing the focus of previous social ecological models by limiting the number of factors involved within the model, while emphasizing causal pathways. Lytle's model includes several categories of factors, including immutable factors (demographics, family history, family structure), contextual factors (individual/psychosocial, socio-environmental, and physical environment), behavioral/biological factors (diet and eating patterns, sleep, substance use, weight control, physical activity, metabolism, pubertal status, blood chemistry), and obesity risk (BMI, body composition). While Lytle's model has been applied to youth between the ages of 10 and 16, they noted that it may be helpful to have other conceptual models focusing on trajectories earlier in childhood or later in adolescence to explore other factors that may influence trajectories of weight gain.

Others have proposed a developmental symptom model with feedback for depression (Maxwell & Cole, 2009), in which changes in weight and appetite are displayed as symptoms of depression that act back on depression. In their model, they build off of a simple symptom model in which changes in weight and appetite ad symptoms of depression. They argue for a model that

displays feedback between appetite and weight and between weight and depression. By combining these concepts, and by keeping in mind developmental third variables, which may affect depression, appetite, and weight, they created a developmental symptom model with feedback (Figure 1). It is important to note that in Maxwell and Cole's developmental symptom model with feedback groups, variables that are affected by developmental factors (i.e.: puberty) are categorized as developmental third variables, which cause "spurious effects." The authors also express that it is likely that these third variables also serve as moderators impacting the relationship between depression and symptoms such as changes in appetite and weight. These authors do an exceptional job of highlighting the third variables that influence weight, appetite, and depression in adolescence and their relation to puberty, such as psychological and behavioral variables (exercise, body dissatisfaction, dieting, eating disorders, stress), hormones, hypothalamic control, and reward/regulation systems (serotonin, dopamine, norepinephrine, endogenous opioids). These factors may be useful to consider as moderators when creating a model for the reciprocal relationship between depression and obesity in adolescents.

Proposed Developmental Stage Model of Mechanisms

Current causal models highlighting mechanisms linking both obesity and depression in adults have been proposed. Of note, mediator/moderator models (Markowitz, Friedman, & Arent, 2008; Napolitano & Foster, 2008) have been useful in highlighting several moderators and the various behavioral, social, cognitive, and physiological mediators (Figure 2) linking obesity to later depression. Napolitano and Foster (2008) also proposed a separate, but basic mediator/moderator pathway for adolescents (Figure 3), where the directionality of the causal relationship is flipped to highlight the pathway leading from depression to later obesity. At the time of the proposed model, however, one of the key commentaries noted by Napolitano &

Foster was that the existing scientific knowledge for adolescents was not as advanced as the evidence for adults, and that there were more mediators and moderators waiting to be uncovered. These models argued for a unidirectional relationship of obesity to later depression in adults and depression to obesity in adolescents, but did not propose any direct reciprocal relationship between obesity and depression for both adults and adolescents.

Since 2008, more research has been conducted in adolescents exploring the bidirectional relationship between obesity and depression, giving the opportunity to expand and modify this adolescent model to specify more mediators and moderators. There is sufficient support for a separate model for children who are pre-pubescent, where sex does not play a moderating role. This is supported by evidence that indicates that age of onset, chronicity, and severity play a larger role in childhood depression and obesity leading to later obesity or depression. Sex differences do not seem to emerge until adolescence. Given that the relationship between obesity and depression differs dependent on developmental stage, it is important to look at this comorbidity and the causal pathways from a developmental stage perspective. Also, considering the focus of existing literature and models on adolescence and later childhood, it may be appropriate to create a developmental model that is stratified by developmental status (e.g. pre/post puberty). The proposed models for childhood and adolescence are outlined in Figures 4 and 5.

The relationship between depression and later obesity is characterized in both children and adolescents by several mediators (heavy smoking, HPA axis disruption, negative affect, social support, emotional eating) and moderators (earlier onset, depressive symptom severity). The relationship between obesity and later depression in both children and adolescents is depicted through several mediators (diet, exercise, poor perceived mental health, weight-based

teasing, heavy smoking) and moderators (chronicity of high BMI, severity of obesity).

While there is significant overlap between the child and adolescent models, the proposed child model of the reciprocal relationship between depression and obesity highlights prenatal and maternal factors that serve as risk factors for depression or obesity. The proposed adolescent model also includes risk factors, which include presence of depression symptoms or high BMI in childhood that carries over into adolescence. The adolescent model also includes pubertal factors as a moderator for both depression leading to obesity and obesity leading to depression, gender and age as moderators of obesity leading to depression, and self-esteem as a mediator of obesity leading to depression.

Remaining Questions

Presently, the youngest baseline age for studies evaluating the longitudinal effect of obesity on depression is seven, with the majority of studies having a baseline age between 11-18 years. The youngest baseline age for studies evaluating the effect of depression on later obesity is four years with most studies having a baseline age between 11-21 years. In order to better understand the developmental trajectory of the relationship between depression and obesity, it is important to study the relationship in all age ranges within childhood and adolescence, especially because results appear mixed regarding the effects of age and gender. Additionally, in order to better evaluate the relationship between obesity and psychological distress, several articles in this review (Kubzansky, Gilthorpe, and Goodman, 2012; Larsen et al., 2013; Marmorstein, Iacono, & Legrand, 2014; McClure et al., 2012) have suggested that it may be useful to study younger children. The need for studies in younger children is also supported by prior findings showing that weight trajectories appear to be static in adolescence (Singh net al., 2008; Crimmins et al., 2007). Additional differences between childhood and adolescent depression are highlighted by

differing neurobiological correlates and arrays of risk factors (Jaffee et al., 2002; Kaufman et al., 2001).

Current literature confirms the reciprocal relationship in both boys and girls, though it appeared that stronger findings in adolescent females were noted in several studies. It is possible that sex differences in the reciprocal relationship between obesity and depression may appear in adolescence, then disappear in adulthood. Considering that chronicity, severity, age of onset, and sex differences are key moderators impacting the trajectory of obesity and depression, it is important to evaluate this relationship longitudinally in children of all ages, and by stratifying children from adolescents.

Most of the recent research evaluating the relationship between obesity and depression has focused on the adolescent age range. While there is ample evidence for many changes that occur within adolescence, there is growing evidence supporting the importance of evaluating the chronicity and severity of obesity and depression that begins earlier within the developmental trajectory, such as in early childhood and the school age period. Current literature on obesity in early childhood also includes early, prenatal risk factors such as chronic maternal depression (Lampard, Franckle, Davison, 2014). Additionally, some preliminary rodent studies indicate the possibility of obesity inducing inflammatory states during pregnancy and lactation that may contribute to behavioral or cognitive changes in offspring (Bilbo & Tsang, 2010; Kang et al., 2014).

Given that research on this topic in younger groups of children is lacking, it would be valuable to explore the longitudinal relationship in a younger sample that is prepubescent (ages 2-11). Also, given that much of the existing literature in this area has focused on older children and adolescents who are more independent and autonomous, studying parents and prenatal risk

factors in younger groups of children is also warranted. More studies are needed to further elucidate the mediators and moderators underlying mechanisms associated with this reciprocal relationship between obesity and depression, particularly in children and adolescents. Continued research may lead to clarification of the proposed models, in addition to proposal of expanded models that can highlight differences in risk factors, mediators, and moderators that contribute to this relationship between infancy/early childhood and school age children. Opportunities for exploring new and existing clinical interventions targeted at this comorbidity still exist; to date, questions proposed years ago have still not been fully answered. Clinical questions noted by Markowitz, Friedman, and Arent in 2008 still remain: "Does weight loss predict mood improvement, or vice versa, does mood improvement predict weight loss?" and how does development affect this?

The Present Study

Considering that nearly all prior research evaluating the relationship between obesity and depression has been conducted in children ages 11 and up, more information is needed on children ages 2-11. Also, prior research has often lacked sufficient data points to assess the longitudinal nature of this relationship, and to do so prospectively. To fill this identified gap in the literature, this prospective study of children ages 2-11 evaluated whether a relationship between depression symptoms and weight status exists, and assessed how baseline depression symptoms relate to weight gain and how weight gain relates to change in depression symptoms.

Methods

Participants

The sample consisted of 93 families (children, and their mothers) from behavioral pediatric clinics within the Kansas City metropolitan area (University of Kansas Medical Center

and Children's Mercy Kansas City) who enrolled in the study. Of these families, 90 families completed measures for inclusion within our study analyses. Recruitment of participants was restricted based the following exclusionary criteria: child age <2 or ≥ 12, adopted children, developmental disorder (e.g.: Autism Spectrum Disorders, Down Syndrome, Prader Willi), medically diagnosed eating or feeding disorder (other than binge-eating disorder), medically diagnosed serious physical disease impacting physical development (e.g.: Cerebral Palsy, cancers, Cystic Fibrosis) or appetite (e.g.: hypo or hyperthyroidism), any condition impacting physical mobility, or steroid medications.

Measures

Behavior Assessment System for Children—Second Edition (BASC-2). The BASC-2 consists of a series of instruments including Parent Rating Scales (PRS), Teacher Rating Scales (TRS), Self-Report of Personality (SRP and SPR-I) designed to evaluate emotional/behavior disorders in children and adolescents. Forms are available for preschool (ages 2-5), child (ages 6-11) and adolescent (ages 12-21). This measure is helpful in evaluating mood concerns, such as depression in children as young as 2 years of age (Reynolds & Kamphaus, 2004), and is available in both English and Spanish languages. Previous studies have found that the parent report version of the BASC-2 produces results most sensitive to change (McClendon et al., 2011). The BASC-2 behavioral scales include atypicality, depression, hyperactivity, and somatization; the depression scale was used to evaluate depressive symptoms. T-scores for the BASC-2 are normed with an average of 50 and a 10-point standard deviation, with T-scores >70 noted to be "Clinically Significant." The BASC-2 PRS has excellent reliability and validity and Cronbach alphas for the depression scale on the child and adolescent versions range from .77 to .90. Test-retest reliabilities have average correlations in .80s for composite scores and between

the .70s and .80s for individual scales across all age groups. BASC-2-PRS was used to evaluate emotional/behavior of children in this study at both time points.

The Patient Health Questionnaire (PHQ-9). The PHQ-9 is a 9-item self-report questionnaire designed to screen, diagnose, monitor, and measure the severity of depression in adults. The PHQ-9 can be administered repeatedly over a span of time to evaluate improvement or worsening of depression symptoms, and is available in both English and Spanish languages. A score of 5=mild, 10=moderate, 15=moderately severe, and 20=severe depression (Kroenke, Spitzer, & Williams, 2001). PHQ-9 was administered to mothers at both time points.

Body Mass Index (BMI). Trained research staff measured child and mother heights and weights at both time points using standardized protocols while participants were dressed in light street clothes without shoes. Height was measured to the nearest 1 mm using a portable stadiometer. Body weight was measured to the closest 0.1 kg using a digital scale. These values were used to calculate body mass index using the formula BMI=[kg/m^2]. For children and adolescents, BMI is age- and sex-specific, and BMI was be plotted onto CDC growth charts to obtain a BMI percentile which was then converted to BMIz with standard formulas. BMIz is a standard deviation score indicating how much of a standard deviation a child's BMI is above or below the average BMI value for their age group and sex.

Demographic data, medical/psychological diagnoses, and medications. At the first time point, parents were asked to fill out a brief form on the child and family demographics in addition to any medical/psychological diagnoses the child had, and any medications that were taken.

Human Subjects

Institutional Review Board (IRB) Review and Informed Consent

The proposed protocol, and any subsequent modifications, were reviewed and approved by

the IRB at Children's Mercy Kansas City. A request to rely on the IRB at Children's Mercy Kansas City was approved by the University of Kansas Medical Center.

Procedures

Children and their mothers were recruited from behavioral pediatric clinics within the Kansas City metropolitan area (University of Kansas Medical Center and Children's Mercy Kansas City) through poster flyers and in-person while families were waiting in clinic waiting rooms prior to their scheduled visits. Parents who responded to advertising or who were interested when presented with study information were screened for eligibility criteria. Those who meet eligibility criteria provided written consent (via process approved by Children's Mercy Kansas City Institutional Review Board) and began their first visit (Time 1). Heights and weights of the child and mother were measured using standardized protocol. Parents completed BASC-2-PRS and PHQ-9. Three months ($\pm$2 weeks) following the first visit, families were contacted and scheduled for their second visit (Time 2), during which height and weights were measured, and parents completed BASC-2-PRS and PHQ-9. For the Time 2 visit, families had the choice of coming into their primary clinic for height and weight measurements, or for study staff to conduct a home visit during which these measurements were completed. Upon completion of each visit, families were compensated with small toys/trinkets for the child (<$5 value) for their time. Each study visit took 15-20 minutes. Participant flow through study is summarized in the CONSORT Flow Diagram in Figure 6.

Data Analysis

Data were analyzed using SPSS 22.0. BMIz and BASC-2 depression T scores were examined as continuous variables.

Establishing a Correlation Between Child Depression Symptoms and Child Weight Status.

Data at both time points were collapsed into a single pool of data and bivariate correlation was used to evaluate whether a relationship existed between depression symptoms and BMIz. Additionally, to accommodate for a possible curvilinear relationship, BMIz was plotted as absolute values to evaluate whether this better illustrated the relationship, and linear regression was used to assess how much BMIz accounts for variance in BASC-2 depression T-scores.

Assessment of Relationship Between Baseline Child Depression and Child Weight Trajectory.

We assessed the relationship between baseline child depression symptoms and change in child BMIz over 3 months using bivariate correlation. Additionally, we evaluated the relationship between baseline child depression symptoms and child BMIz at 3 months using bivariate correlation. We also assessed the risk of later overweight/obese weight status if a child had an "at risk" or "clinically significant" BASC depression score.

Assessment of Relationship Between Baseline Maternal Depression and Child Weight Trajectory.

We assessed the relationship between baseline maternal depression symptoms and change in child BMIz over 3 months using bivariate correlation, and also ran the bivariate correlation with data stratified by maternal depression severity. Furthermore, we collapsed data across time points and split the analyses by child gender to evaluate the relationship between child BMIz, child depression symptoms, maternal BMI, and maternal depression symptoms. We also assessed the risk of later overweight/obese weight status if the child's mother had a PHQ-9 score of 10 or greater (moderate-severe depression).

Assessment of Correlation Between Change in Weight Status and Change in Depression Symptoms.

We used correlation analysis to examine the relationship between change in child BMIz and child depression symptom change. Additionally, we evaluated the relationship between baseline child BMIz and child depression symptoms at 3 months using bivariate correlation. We also assessed the risk of later "at risk" or "clinically significant" BASC depression score if a child was overweight/obese weight status at baseline.

Results

Children were 2- 11 years old (M = 7.58, SD = 2.67), 58.9% male, and primarily Caucasian (84.4%). Mothers were 22-52 years old (M = 36.46, SD = 6.38), with 45.6% reporting a history of a depression diagnosis, and 38.9% reporting a current depression diagnosis. Comparison of follow-up completers (N=72) versus follow-up dropouts (N=18) revealed that families who dropped out at the three month follow-up time point were less educated ($p=0.003$), less likely to report history of depression diagnosis ($p<0.001$), less likely to report current depression diagnosis ($p=0.007$), but more likely to report greater depression symptoms on the PHQ-9 ($p=0.035$). Additionally, families who dropped out were less likely to be Caucasian ($p<0.001$). See Table 2 and Table 3 for complete participant demographics, including measures of depression and weight status at both baseline and three-month time points.

Cross-sectional Relationship Between Depression and BMIz

Bivariate correlations revealed a significant relationship between child depression symptoms and child BMIz ($r=.168$, $p=.034$) when data were collapsed across time points. Additionally, when BMIz data were plotted as absolute values, this relationship was maintained ($r=.313$, $p<0.001$), indicating that it is possible that both underweight and overweight BMIz are related to greater depression symptoms. Through linear regression, findings also suggest that BMIz does significantly explain 9.8% of variance in BASC-2 depression T-scores ($R^2=.098$,

F=16.62, p<0.001).

Relationship Between Baseline Child Depression and Child Weight Trajectory

Bivariate correlation between baseline BASC-2 depression T-scores and child BMIz change across 3 months was nonsignificant (r=.035, p=.771), meaning that baseline child depression was not related to change in BMIz over the 3 month period. However, bivariate correlation between baseline BASC-2 depression T-scores and child BMIz at 3 months was significant (r=.303, p=.009), suggesting a relationship between baseline child depression and BMIz at 3 months. Results also indicate that children who score in the "at risk" or "clinically significant" range on the BASC-2 depression scale at baseline are 2.26 times more likely to be overweight/obese at 3 months (OR=2.26; CI=.85, 6.02).

Relationship Between Baseline Maternal Depression and Child Weight Trajectory

Bivariate correlation between baseline maternal PHQ-9 scores and child BMIz change across 3 months was nonsignificant (r = -.107, p = .367), meaning that baseline maternal depression is not related to change in BMIz over the 3 month period. However, when data were stratified by maternal depression severity (based on PHQ-9), bivariate correlation revealed significant relationships between baseline maternal PHQ-9 scores and child BMIz change across 3 months when maternal depression was moderate (r = -.801, p = 0.017, N=8) and moderately severe (r = -1.00, p <0.001, N=2); this indicates that when maternal depression is more severe, children gain less weight over the 3 months. When we collapsed data across time points and split the bivariate correlations by child gender, we found that maternal depression symptoms have significant relationship to depression symptoms in boys (r=.223, p=.029, N=96) but not girls (r=.137, p=.282, N=66). We also assessed the risk of later overweight/obese weight status if the child's mother had a PHQ-9 score of 10 or greater (moderate-severe depression), and found that

children of mothers with clinically significant PHQ-9 scores (≥10) at baseline are 2.05 times more likely to be overweight/obese at 3 months (OR=2.05; CI=.59, 7.17).

Relationship Between Maternal Weight Status and Weight Status Children

We also found that when we collapsed data across time points and split the bivariate correlations by child gender, maternal BMI has a significant relationship to BMIz in boys (r=.392, p<0.001, N=96) but not girls (r=.172, p=.167, N=66).

Relationship Between Change in Weight Status and Change in Depression Symptoms

Bivariate correlation between change in child BMIz and child depression symptom change was non-significant (r = -.057, p=.637), suggesting that there is no relationship between change in child weight status and change in child depression symptoms across 3 months. However, bivariate correlation between baseline child BMIz and child depression symptoms at 3 months revealed a significant relationship (r=.278, p=.018), meaning that there is relationship between baseline child weight status and child depression at 3 months. Additionally, we found that children who are overweight or obese at baseline are 2.87 times more likely to score in the "at risk" or "clinically significant" range on the BASC-2 depression scale at 3 months (OR=2.87; CI=1.01, 8.07).

Discussion

Our aim for this study was to evaluate the cross-sectional and longitudinal relationship between depression symptoms and weight status of children ages 2-11 across a 3 month period. Our findings provide evidence for a cross-sectional relationship between depression and weight status in children. Our data also suggest that both low and high BMIz are associated with greater child depression symptoms, and that BMIz significantly accounts for nearly a tenth of the variance in depression symptom scores that we measured. Our results illustrate a relationship

between depression symptoms and BMI in children ages 2-11 that is consistent with prior findings in children ages 8-13 (Dockray, Susman, & Dorn, 2009) and also in adolescents (Sjoberg, Nilsson, & Leppert, 2005). Additionally, our findings highlight a relationship between both low and high BMIz and greater depression symptoms, which mirrors prior findings observed in adults (Carey et al., 2014), and adolescents (Cortese et al., 2009; Revah-Levy et al., 2011). These results help to establish that there is a relationship between depression and weight status in children, and provide a foundation for further analyses to assess the longitudinal relationship between these variables.

While prospective associations were not significant between baseline child depression and change in BMIz across 3 months, we did find that baseline child depression symptoms were related to weight status at 3 months, and children who scored in "at risk" or "clinically significant" ranges of depression symptoms were more than twice as likely to be overweight or obese 3 months later. These results provide preliminary support for childhood depression symptoms relating to later weight concerns. This is a particularly important first step in understanding how development may impact the relationship between depression and later obesity. Since prior research has primarily focused on this relationship in adolescence, this finding supports that the relationship exists in children between the ages of 2-11. Depression has been found to be associated with increased risks in the development and persistence of obesity (Goodman & Whitaker, 2002). These findings argue for earlier identification of depression symptoms to prevent chronic obesity.

Prospective associations revealed that a number of maternal factors may impact weight and depression symptoms in children. Our outcomes suggest that maternal depression relates to decreased weight gain over 3 months, especially in children of mothers experiencing moderate to

moderately severe depressive symptoms. Additionally, our findings also suggest that maternal factors may play more of a role in development of weight and mood problems in boys, and overall, children of mothers with clinically significant depression symptoms were more than twice as likely to be overweight or obese 3 months later. These findings suggest that assessment of maternal depression may be useful in early identification of potential weight and mood problems in children ages 2-11, especially in boys. This is consistent with prior findings showing sex differences in the association between maternal depressive symptoms and child BMIz (Duarte et al., 2012).

While prospective associations were not significant between change in child depression and change in BMIz across 3 months, we did find that baseline child BMIz was related to child depression symptoms at 3 months, and children who were overweight or obese were nearly three times more likely to be in the "at risk" or "clinically significant" ranges of depression symptoms 3 months later. These data provide preliminary findings in support of childhood weight status relating to later changes in depression symptoms (Faith, Matz, & Jorge, 2002) and increases knowledge regarding the relationship between obesity and later depression in children between the ages of 2-11, particularly since existing research has primarily evaluated this relationship in adolescence. These findings provide support for the importance of early interventions for childhood overweight and obesity to prevent later depression symptoms.

Our research has some limitations. We hypothesized a linear relationship between depression and weight status based on data collected at two time points 3 months apart. It is possible, however, that the relationship between depression and weight status is not linear, that it differs between age groups and other demographic factors, and that this relationship cannot be captured using only two time points. Future studies may choose to add additional time points to

allow for hierarchical linear modeling, allowing for both within and between subject comparisons. With two time points, the study remains novel and contributes to the literature by demonstrating the existence of the relationship between depression and weight status. While we did not anticipate concerns with recruitment during our study, we did encounter a problem with retention of families, due to the small window during which we required families to participate in the 3 month follow-up visit; however, despite some initial concerns about retention of families, we were able to maintain within our expected 20% rate of attrition.

Future Directions

Limited research has been examined the longitudinal relationship between depression and obesity in children between the ages of 2-11, and no research has yet been published in this area for young children ages 2-4. This prospective study provides a substantial contribution to current literature by expanding the scope of research in this area to include children of younger age, and is the first to explore this relationship in children between ages 2-4. While this study attempts to expand the current literature by providing information about this cross-sectional and longitudinal, bidirectional relationship in children ages 2-11, more longitudinal research (especially with larger sample sizes and longer follow-up periods) is needed to explore mediators and moderators that may be involved, and how these factors change throughout a child/adolescent's development. Identifying such factors will clarify areas for treatment focus, and may assist with identifying children and adolescents who are at most risk for later depression or obesity.

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Included Studies (Obesity --> Depression)

Author	Sample Size	Participant Sex	Baseline Age (years)	Follow-up length	Depression Measure	Findings
Anderson et al., 2007	701	Both	9-18	20 years	Clinical diagnosis	O --> D in females
Anderson et al., 2011	918	Female	12	2 years	Depressive symptoms	O --> D in white females
Boutelle et al., 2010	496	Female	11-15	3 years	Clinical diagnosis	O --> D in females
Felton et al., 2010	215	Both	10-13	4 months	Depressive symptoms	O --> D especially in older adolescents and females
Frisco, Houle, & Lippert, 2012	5243	Female	13-18	4 years	Depressive symptoms	O --> D in females
Goodman & Must, 2011	102	Both	7-12	3 years	Depressive symptoms	O -/-> D
Hammerton, Thapar, & Thapar, 2014	289	Both	9-17	29 months	Clinical diagnosis	O -/-> D
Herva et al., 2006	8451	Both	14	17 years	Clinical diagnosis; depressive symptoms	O --> D
Loth et al., 2011	2516	Both	12-15	5 years	Depressive symptoms	O --> D
Mansoor et al., 2013	296	Both	12-18	24 months	Clinical diagnosis; depressive symptoms	O not affect D tx
Marmorstein, Iacono, & Legrand, 2014	731	Both	14	10 years	Clinical diagnosis	O --> D in females
Merten, Wickrama, & Williams, 2008	7881	Both	12-18	6 years	Depressive symptoms	O --> D in females only (no difference in white vs black)
Mustillo et al., 2003	991	Both	9-16	8 years	Clinical diagnosis	O --> D in males only
Rawana & Morgan, 2014	4359	Both	12	10 years	Depressive symptoms	O -/-> D
Roberts & Duong, 2013	3134	Both	11-17	1 year	Clinical diagnosis; depressive symptoms	O -/-> D
Roberts & Hao, 2013	4175	Both	11-17	1 year	Clinical diagnosis	O -/-> D
Sysko et al., 2012	101	Both	14-18	15 months	Depressive symptoms	decrease in O --> improvements in D sxs

Included Studies (Depression --> Obesity)

Author	Sample Size	Participant Sex	Baseline Age (years)	Follow-up length	Depression Measure	Findings
Anderson et al., 2006	820	Both	9-18	18-20 years	Clinical diagnosis	D --> O in females
Anderson et al., 2011	918	Female	12	2 years	Depressive symptoms	D --> O in white females
Aparicio et al., 2013	229	Both	10	3 years	Depressive symptoms	D --> O in males
Gaysina et al., 2011	4559	Both	15	37 years	Depressive symptoms	D --> O in females
Goodman & Whitaker, 2002	9374	Both	13-21	1 year	Depressive symptoms	D --> O
Korczak et al., 2014	3294	Both	4-16	17 years	Clinical diagnosis	D --> O in males only for childhood, in females only for adolescence
Kubzansky, Gilthorpe, & Goodman, 2012	1528	Both	14-15	4 years	Depressive symptoms	D -/-> O
Larsen et al., 2014	2051	Both	11-17	3 years	Depressive symptoms	D -/-> O
Mansoor et al., 2013	296	Both	12-18	24 months	Clinical diagnosis; depressive symptoms	tx D not affect O
Marmorstein, Iacono, & Legrand, 2014	932	Both	14	10 years	Clinical diagnosis	D --> O in females
McClure et al., 2012	510	Both	10-14	12 years	Depressive symptoms	D --> OW at 14 yo --> O
Richardson et al., 2003	938	Both	11-21	5-15 years	Clinical diagnosis	D --> O in female
Rofey et al., 2009	285	Both	8-18	3 years	Clinical diagnosis	D --> sustained O weight trajectory in females
Roberts & Duong, 2013	3134	Both	11-17	1 year	Clinical diagnosis; depressive symptoms	D --> O

Table 1. Characteristics of studies included in review.

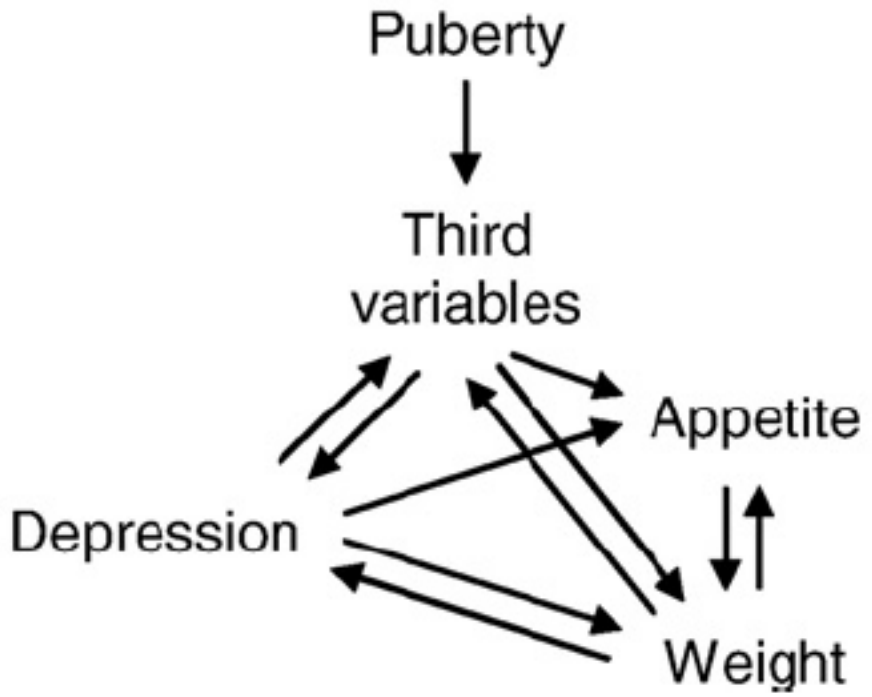

Figure 1. Maxwell and Cole's developmental symptom model.

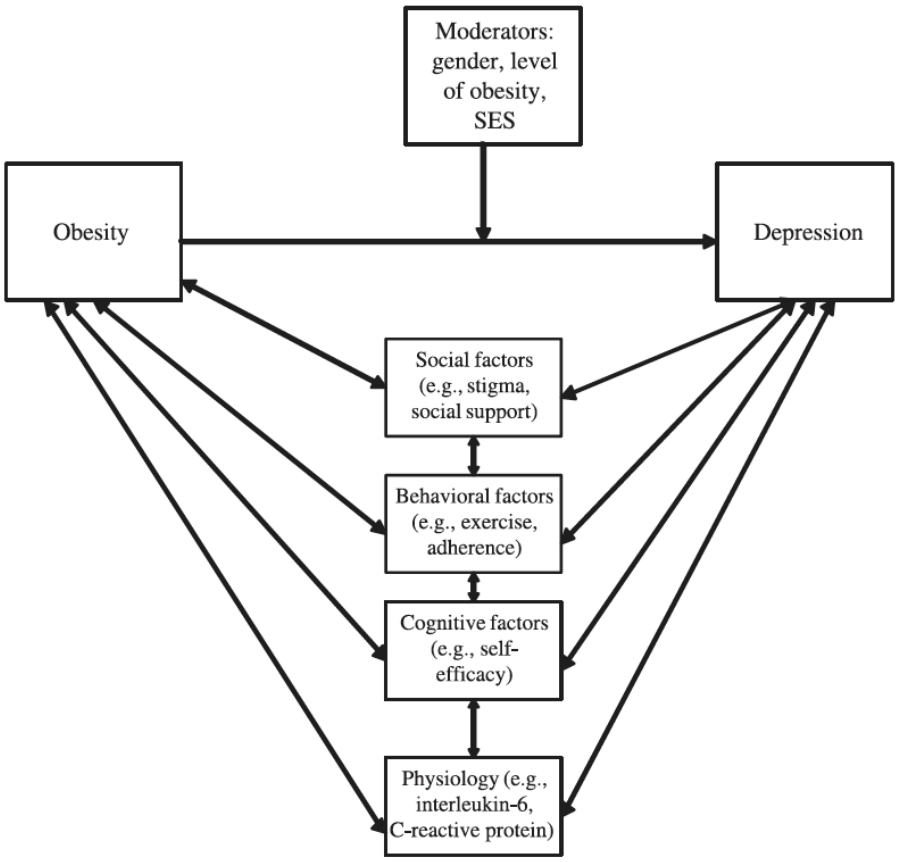

Figure 2. Napolitano & Foster's proposed mediator/moderator pathway for the obesity-depression relationship in adults.

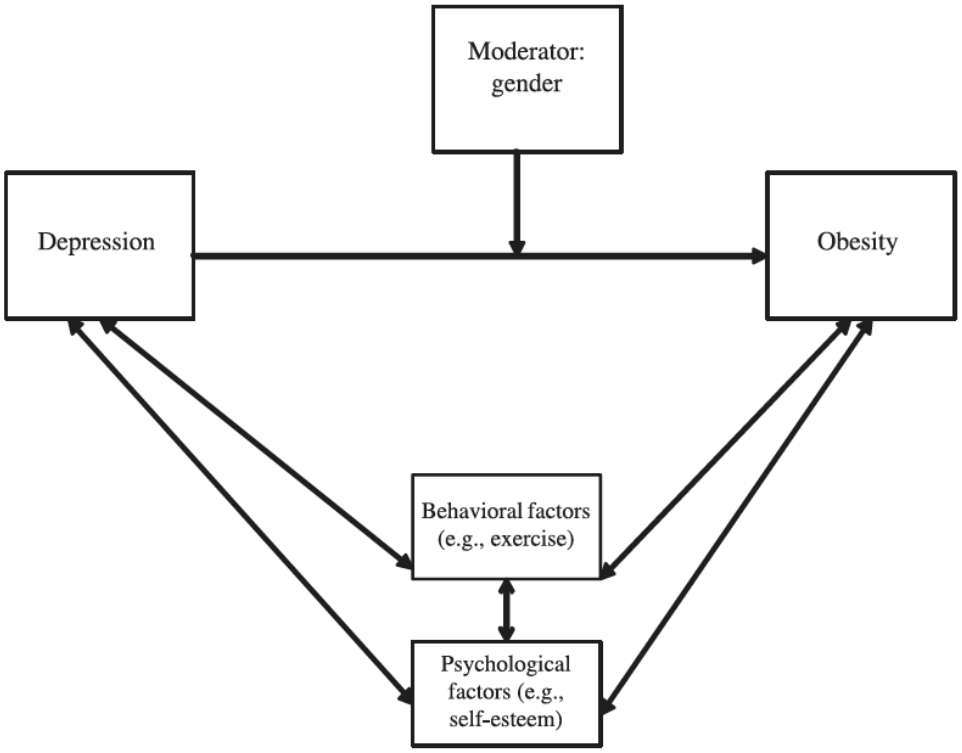

Figure 3. Napolitano & Foster's proposed mediator/moderator pathway for the depression-obesity relationship in adolescents.

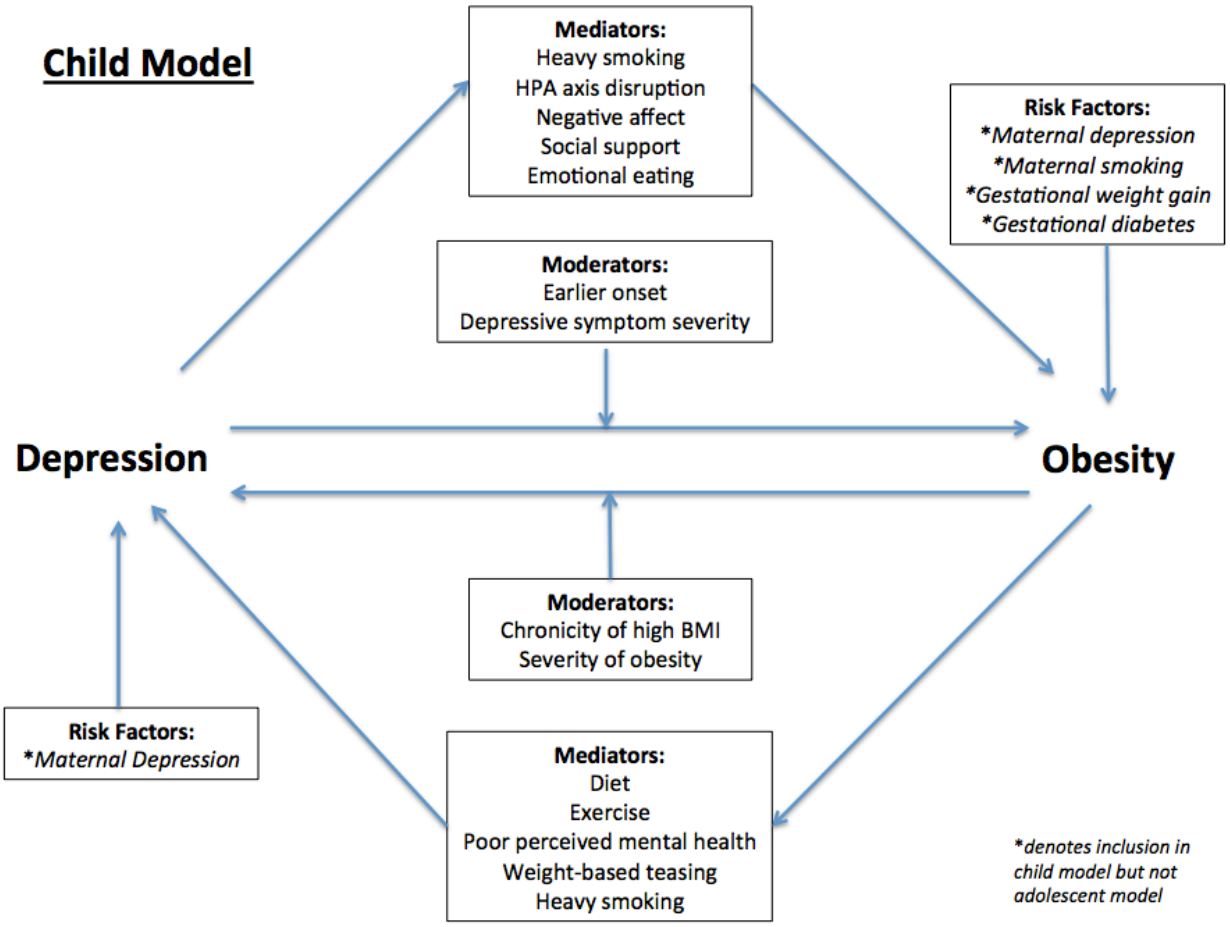

Figure 4. Proposed conceptual model of the reciprocal relationship between depression and obesity in children.

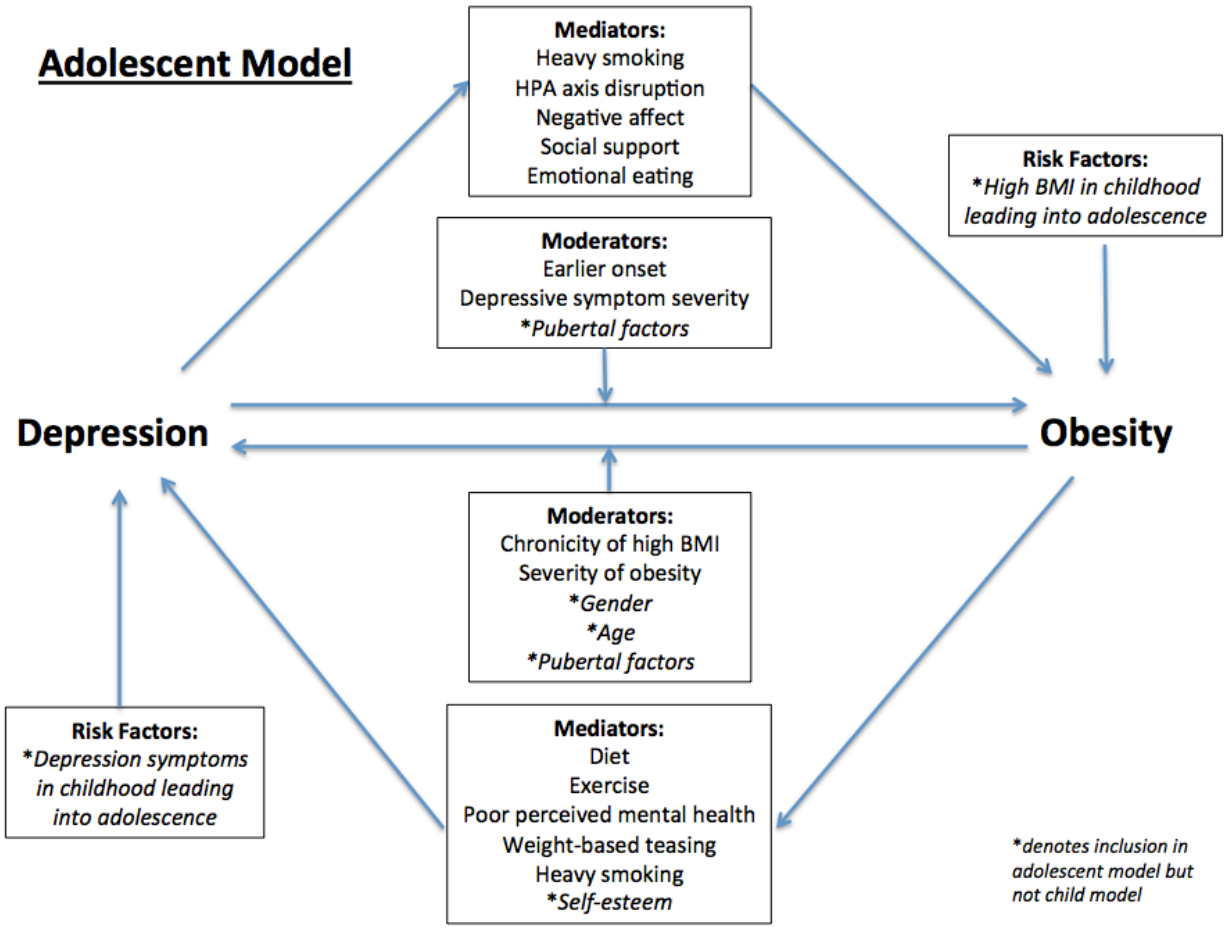

Figure 5. Proposed conceptual model of the reciprocal relationship between depression and obesity in adolescents.

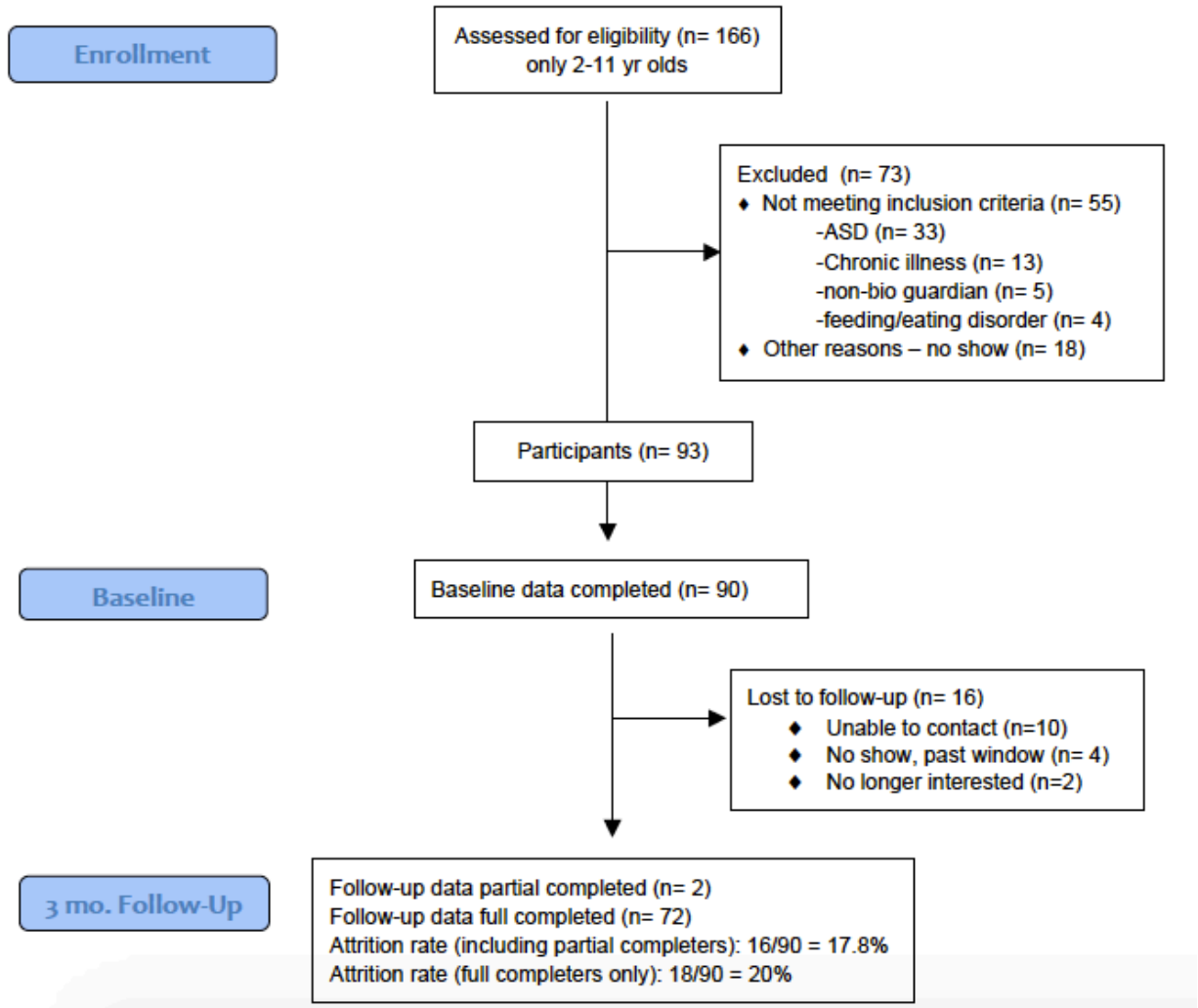

Figure 6. CONSORT Flow Diagram of progress participants within the study.

	All Baseline Completers n= 90	**Follow-up Completers n= 72**	**Follow-up Dropouts n= 18**
Child Age (years)	7.58 (SD=2.67) range = 2.16-11.98	7.74 (SD=2.72) range = 2.17-11.98	6.97 (SD=2.45) range = 2.39-10.41
Gender			
Female	37 (41.1%)	29 (40.3%)	8 (44.4%)
Male	53 (58.9%)	43 (59.7%)	10 (55.6%)
Ethnicity			
Caucasian	76 (84.4%)	64 (88.9%)	12 (66.7%)
Hispanic	6 (6.7%)	3 (4.2%)	3 (16.7%)
African American	3 (3.3%)	2 (2.8%)	1 (5.6%)
Asian	3 (3.3%)	2 (2.8%)	1 (5.6%)
Other/mixed	2 (2.2%)	1 (1.4%)	1 (5.6%)
Maternal Age (years)	36.46 (SD=6.38) range = 22.34-52.43	37.08 (SD=6.25) range = 22.34-52.43	33.99 (SD=6.45) range = 24.65-49.81
Parental Education			
9th grade	4 (4.4%)	1 (1.4%)	3 (16.7%)
HS degree	13 (14.4%)	10 (13.9%)	3 (16.7%)
Some college	25 (27.8%)	20 (27.8%)	5 (27.8%)
College degree	35 (38.9%)	33 (45.8%)	2 (11.1%)
Graduate degree	13 (14.4%)	8 (11.1%)	5 (27.8%)
Parental Income			
$0-19,999	13 (14.4%)	8 (11.1%)	5 (27.8%)
$20,000-39,999	13 (14.4%)	8 (11.1%)	5 (27.8%)
$40,000-59,999	18 (20.0%)	16 (22.2%)	2 (11.1%)
$60,000-79,999	14 (15.6%)	13 (18.1%)	1 (5.6%)
$80,000-99,999	8 (8.9%)	6 (8.3%)	2 (11.1%)
$100,000+	24 (26.7%)	21 (29.2%)	3 (16.7%)
Parent History of Depression			
No	49 (54.4%)	35 (48.6%)	14 (77.8%)
Yes	41 (45.6%)	37 (51.4%)	4 (22.2%)
Parent Current Depression			
No	55 (61.6%)	42 (58.3%)	13 (72.2%)
Yes	35 (38.9%)	30 (41.7%)	5 (27.8%)
Parent BL PHQ-9	5.46 (SD=5.00) range = 0-25	5.18 (SD=4.47) range = 0-20	6.56 (SD=6.76) range = 0-25

Table 2. Description of sample at Baseline.

	Baseline (N=90) M (SD) Range (low – high)	**3 Mo Follow-up (N=72)** M (SD) Range	**Change (N=72)** M (SD) Range
Child BMIz	.27 (1.17) -3.88 – 4.06	.35 (1.07) -3.54 – 2.32	.061 (.43) -1.02 – 1.41
Child BASC-2 Depression T-Score	59.09 (13.52) 37 – 102	59.76 (12.70) 37 – 92	.083 (5.41) -13 – 13
Parent BMI	29.4 (8.30) 15.8 – 60.9	29.21 (8.52) 15.6 – 62.0	.11 (.99) -1.8 – 4.2
Parent PHQ-9	5.46 (5.00) 0 – 25	5.51 (4.23) 0 – 18	.36 (4.58) -13 - 13

Table 3. Mean weight status, depression score, and change values at baseline and 3 month follow-up.