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Childhood Depression and Obesity:

Role of the Hypothalamic-Pituitary-Adrenal (HPA) Axis Activity

Childhood overweight and obesity is a major public health concern, and greater understanding of the development of childhood weight problems is imperative. Over the past 30 years, the prevalence of adolescent overweight has increased from 5% to at least 17% (Martyn-Nemeth et al., 2009) with some estimates being over 50% (Frisco, Houle, & Martin, 2009). Obesity represents a public health issue of great significance because it is related to insulin resistance, cardiovascular disease, and early mortality (Dockray, Susman, & Dorn, 2009). Furthermore, the risk of elevated blood pressure ranges from 2.5-3.7 times higher for overweight children than for non-overweight children (Daniels, 2006). Overweight is generally caused by over-eating and lack of exercise, and yet researchers are finding it hard to understand why children today are over-eating and failing to exercise. This study is designed to try to answer that question. The purpose of this study is to test a model of two possible contributing factors to the development of childhood weight problems: depression and hypothalamic-pituitary-adrenal (HPA) axis activity.

Depression and Obesity

Depressed adolescents may overeat in an attempt to distract themselves from their troubles. Some adolescents use eating as a way to provide comfort and distraction from negative emotions (Haines et al., 2007; Jenkins, Rew, & Sternglanz, 2005). Comfort eating, also known as emotional eating, may lead to overweight because it frequently takes place in the absence of hunger (Nguyen-Michel, Unger, & Spruijt-Metz, 2007) and therefore represents unnecessary calorie intake. Past studies have investigated the use of emotional eating as a coping mechanism in response to stressful situations and perceived stress, although these studies more frequently

include adults rather than children (Jenkins, Rew, & Sternglanz, 2005). Adult women with eating disorders have been found to have poor problem-solving skills (Jenkins, Rew, & Sternglanz, 2005). Adolescents with low self-esteem and avoidant coping (i.e. coping with a problem through distraction) tend to have unhealthy eating behavior (Martyn-Nemeth et al., 2009). The use of food as a coping strategy may lead depressed children to overeat and therefore place them at risk for overweight.

Unfortunately, there have been relatively few studies of depression and obesity in young children. Researchers have found that adolescent depression, body dissatisfaction, and other personal factors are associated with increased risk of binge-eating behavior, which can lead to increased weight gain (Haines et al., 2007). Depression in 8 to 13-year-old children is associated with their higher body mass index (BMI; Dockray, Susman, & Dorn, 2009), a ratio of bodyweight to height (kg/m^2) that is commonly used to assess weight problems (Hughes & Reilly, 2008). Depression may make children and adolescents more susceptible to eating disorders like obesity, and may lead these youth to eat food in an effort to alleviate this emotional turmoil. Alternatively, overweight children and adolescents may have an increased likelihood of depression because their higher weight leads them to feel poorly about themselves. Unhealthy core beliefs (i.e. feeling like one is not good enough) are found to be characteristic of both depression and eating disorders (Blissett & Meyer, 2006). Thus, relations between depressive symptoms and obesity may be bidirectional.

The link between depression and obesity may be stronger for girls than for boys. De Wit et al. (2010) found a significant positive association between depression and obesity in the general population, but a greater association for women than men. Similar studies have discovered that female gender may increase the chances of depression in some obese individuals

(Faith, Matz, & Jorge, 2002). Pesa, Syre, and Jones (2000) noted that the relation between depression and body weight in female adolescents is influenced by self-esteem and body image, with overweight females having a lack of feeling connected with others. This lack of connectedness may increase overweight female adolescents' risk for depression since these females will not feel good about themselves or believe that they have relationships with people with whom they could share their concerns. Other research has shown that young females' perceptions of their body weight may place them at greater risk for depression as compared to their actual weight (Frisco, Houle, & Martin, 2009). Research on pubertal status has shown that depressive symptoms increased with pubertal development for both girls and boys, but that girls had a greater increase throughout puberty (Richardson, Garrison, Drangsholt, Mancl, & LeResche, 2006). Similarly, girls' level of physical activity tends to decrease with pubertal development, which may contribute to their likelihood of becoming overweight (Dockray, Susman, & Dorn, 2009). Based on this prior research, this study examines sex and pubertal status as moderators of relations between depression and obesity; it is proposed that associations will be stronger for girls than for boys and stronger for children later in puberty.

The Hypothalamic-Pituitary-Adrenal (HPA) Axis

The current study further advances research on the development of eating and weight problems through examination of HPA activity. The HPA axis is one of the primary biological systems which allows organisms to adapt to physical and psychosocial changes in their environments. HPA activity has received increased attention in biopsychosocial models of developmental psychopathology, resulting in greater predictive ability of these models (Gunnar & Fisher, 2006). The integration of HPA activity into a model of childhood weight and eating

problems therefore offers the opportunity to improve understanding of how and why psychosocial stress (e.g., depression) may lead to the development of eating and weight issues.

In humans, perceived stress activates the central nervous system (CNS), causing the release of corticotropin releasing hormone (CRH) from the anterior pituitary and cortisol from the adrenal cortex (Burke, Davis, Otte, & Mohr, 2005). Cortisol is a steroid hormone that acts on the central nervous system, where it inhibits the release of CRH and adrenocorticotropin hormone (ACTH) through a negative feedback system in the hippocampus. Cortisol regulates blood pressure, blood glucose, and inflammation, and inhibits subsequent HPA activation. Cortisol is secreted in response to social evaluation, novelty, threat, and challenge (Gold & Chrousos, 2002). There are deleterious consequences of over- or under-activation of the HPA axis for physical, neuropsychological, and psychological functioning, including fatigue, infection, asthma, obesity, internalizing and externalizing symptoms (Fries et al., 2005; Heim et al., 2000; Susman, 2006). Adaptive HPA responding to environmental stress is thus characterized by efficient onset and termination of HPA activity (Stansbury & Gunnar, 1994).

Maladaptive HPA axis activity is related to the development of symptoms of depression and anxiety. Stress is a risk factor for depression, and it has been found that depression in adults and adolescents increases HPA activity (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). Elevated morning cortisol levels may be an indication of vulnerability to depression and are associated with anhedonia (Dougherty, Klein, Olino, Dyson, & Rose, 2009). Among adolescents with internalizing problems, increases in basal cortisol and heightened cortisol response to interpersonal stressors have been found (Natsuaki et al., 2009). These changes in cortisol activity could be a risk factor for depression because they are a measure of response to uncontrollable stress (Scarpa & Luscher, 2002). HPA activity is also linked to weight problems. High levels of

cortisol can lead to an increase in abdominal fat (Patrick, 2009). Increases in cortisol levels in response to stress may lead to a tendency to overeat (Farag et al., 2008). However, it is still unclear what role cortisol may play in associations between depressive symptoms and obesity (George, Khan, Briggs, & Abelson, 2010).

Cortisol release is closely connected with puberty and sex. Although the impact of sex hormones on the HPA axis at the onset of puberty is still poorly understood, sex and stress hormones are thought to be strongly interconnected. Sex and stress steroids are so closely related that some steroids act as end-products of both the HPA and hypothalamic-pituitary-gonadal (HPG) axes, and most sex steroids are responsive to stress (Natsuaki et al., 2009). Research has shown that the large changes in sex steroids at the onset of puberty influence HPA axis activity, which may contribute to vulnerability for depression in adolescent girls. As sex steroids and puberty are associated with HPA activity, researchers must seek to understand differences in cortisol reactivity based on sex and puberty (Natsuaki et al., 2009).

Proposed Model

This study proposes that childhood depression and cortisol will be related to children's eating behavior and body mass index. Specifically, we propose that cortisol will mediate relations between depression and weight-related outcomes in children. We expect that children with greater depressive symptoms will exhibit higher baseline cortisol and either cortisol hypo- or hyper-reactivity. In turn, maladaptive cortisol activity will be associated with eating behavior and weight problems. We also propose that cortisol will moderate relations between depression and weight-related outcomes in children; we expect that depression will be more strongly related to obesity and eating for those children with maladaptive HPA activity. In addition, we expect that sex and pubertal status will moderate relations. It is hypothesized that girls and children later

in puberty will have stronger relations among depressive symptoms, HPA activity, and weight-related outcomes.

Method

Participants

The current study is part of a larger study of families and children. Participants were two parent families with children between the ages of 6 and 12 from Lexington and the surrounding area. Parents were over the age of 21, had been living with a romantic partner for at least 2 years, and were regular drinkers of some amount of alcohol. Children were excluded if they had a chronic illness, if they were taking certain medications (e.g. stimulants or sedatives), or if they were sick at the time of the study. Also, children with a developmental delay (e.g., Autism), were ineligible for the study. Families were recruited through cold calling, referrals, posting fliers, handing out fliers at after-school programs, sending out postcards, media advertisements, and sending letters through the school systems. Children completed measures via interview by an experimenter to prevent confounds due to reading ability.

There were 50 participating families in the study. Ninety-four percent of the parents were married and 6% were cohabiting. The mean for how long couples have lived together was 12.12 years (SD = 5.38). The mean age for the mothers was 36.83 (SD = 6.95), the mean age for fathers was 38.68 (SD = 6.69), and the mean age for the children was 8.10 (SD = 1.69). Forty percent of the participating children were girls and 60% were boys. Of the participants, 76.3% were Caucasian, 18.3% were African American, and 5.4% were of mixed or other race. Participants were asked to select their household income using a range format. The mean response was $55,000 to $74,999. However, responses ranged from less than $17,000 (N = 2) to greater than

$125,000 (N = 3). Mean level of education was 16.07 years (SD = 2.46), and ranged from 12 to 21 years.

Procedures

The study was conducted with the approval of the institution's review board (IRB). The families came to the laboratory for a one-time 2 to 2½ hour visit and completed various questionnaires. Parents provided informed consent, and children provided informed assent. Next, children completed the Trier Social Stress Test (TSST; Dockray, Susman, & Dorn, 2009), in which they were asked to come up with their own ending to a story stem in 5 minutes, to tell an experimenter their ending for 4 minutes, and were told that their stories would be compared to other children's stories. Children were then asked to count backwards by 7 from the number 758 for children aged 9 to 12 and backwards by 3 from the number 100 for children aged 6 to 8. Children were asked to do this for 5 minutes, and if they made a mistake they were asked to start over. The TSST is designed to elicit HPA reactivity. During this time, the parents were filling out questionnaires about themselves and their children and participating in other tasks. Each child had a total of four saliva samples collected throughout the visit. The Baseline saliva sample was collected at the beginning of the session immediately after assent had been provided, the Post 1 sample was collected immediately after the TSST, the Post 2 sample was collected 20 minutes after the end of the TSST, and the Post 3 sample was collected 40 minutes after the end of the TSST. Saliva samples were collected using the passive drool method in which participants were instructed to stop swallowing their saliva and passively drool as close to 5mL of saliva as possible into a vial for a period of 5 minutes. Once samples were collected, they were immediately frozen at -20° and stored until assay. After all of the saliva samples had been

collected and all of the questionnaires were completed, participants were debriefed and given compensation for participating in the study.

Measures

Depression. Children completed the revised version of the Children's Depression Inventory (CDI-R; Kovacs, 1981). The CDI-R is a 15-item self-report measure with each item including three choices (yes, sometimes, or no) indicating the level of depressive symptoms. Responses are averaged to provide an overall depression score, with higher scores indicating greater depression. An example item is "I feel like crying many days." The CDI-R is widely used and has excellent psychometric properties. The internal consistency in the current study was good, $\alpha = 0.84$.

Eating Behavior. Two questionnaires were used to assess children's eating behavior. The Eating Pattern Inventory for Children (EPIC) is a 20-item child-report questionnaire including subscales for: (1) Dietary Restraint - reduced or wishes to reduce eating, usually for weight loss reasons; (2) External Eating - eating in response to external stimuli; (3) Parental Pressure to Eat; and (4) Emotional Eating - eating as a form of coping with emotional distress (Schacht et al., 2006). Children completed this questionnaire and each item had three choices (yes, sometimes, or no, coded as 2, 1, or 0) indicating how often they exhibited certain eating behaviors. Scores were computed by averaging responses. The internal consistency in the current study for each of the subscales was good: $\alpha = 0.88$ for dietary restraint, $\alpha = 0.78$ for external eating, $\alpha = 0.63$ for parental pressure to eat, and $\alpha = 0.84$ for emotional eating.

The Children's Eating Behavior Questionnaire (CEBQ; Wardle, Guthrie, Sanderson, &Rapoport, 2001) is a 26-item parent-report measure with five subscales: (1) Satiety Responsiveness/slowness in eating – the degree to which children eat slowly and stop eating

when they are full; (2) Food Responsiveness – food seeking behavior, even when full; (3) Enjoyment of Food; (4) Emotional Under-Eating; and (5) Emotional Over-Eating. Parents completed this questionnaire and the instructions prompted them to indicate how frequently each of the statements applied to their child's eating. Each item had five choices (never, seldom, sometimes, often, or always; coded 0 - 4). Item responses were averaged for each subscale, with higher scores representing more of each construct. The internal consistency in the current study for four of the subscales was good: $\alpha = 0.84$ for Food Responsiveness, $\alpha = 0.87$ for Enjoyment of Food, $\alpha = 0.81$ for Emotional Under-Eating, and $\alpha = 0.81$ for Emotional Over-Eating. The internal consistency for Satiety Responsiveness ($\alpha = 0.52$) was somewhat low.

Weight Problems. Children's height and weight were assessed using a physician's scale in the laboratory. Height and weight was used to compute body mass index (BMI) based on the standard formula: (lb x 703)/inches^2. BMI is a common measure used to determine weight problems. Children can be classified as underweight, healthy weight, overweight, or obese based on their BMI, age, and gender using criteria developed by the Centers for Disease Control (www.cdc.gov). Children's BMI and percentile for their age and gender were examined.

Pubertal Status. Parents completed the Pubertal Development Scale (PDS; Petersen et al., 1988) appropriate for the gender of their child. This parent-report scale includes questions about children's growth, height, and weight; body hair growth; skin changes such as an increase in acne; facial hair and deepening of voice in boys; and breast development in girls. The PDS is the most widely used questionnaire measure of pubertal development and is preferred over physical examination because it is less intrusive.

Cortisol. All saliva samples were assayed for salivary cortisol using a highly sensitive enzyme immunoassay US FDA (510k) cleared for use as an in vitro diagnostic measure of

adrenal function (Salimetrics, State College, PA). Assays were performed by the University of Kentucky Center for Clinical and Translational Science CR-DOC Core Laboratory. Due to time constraints, assay results were only available for the first 29 study participants. The test used 25 μl of saliva (for singlet determinations). Further, the test had a lower limit of sensitivity of 0.007 μg/dl, and a range of sensitivity from 0.007 to 1.8 μg/dl, and average intra- and inter-assay coefficients of variation between 1.3% and 5.5%. Each of the saliva samples had one freeze-thaw cycle on the morning of transfer to the assay laboratory. The cortisol variables included in analyses were: (1) Baseline Cortisol; (2) Post 1 Cortisol; (3) Post 2 Cortisol; (4) Post 3 Cortisol; (5) Change in cortisol from Baseline to Post 1, computed as a residualized change score; (6) Change in Cortisol from Baseline to Post 2, computed as a residualized change score; and (7) Change in Cortisol from Baseline to Post 3, computed as a residualized change score.

Analysis Plan

Initial analyses estimated bivariate correlations among variables. Subsequent analyses involved OLS multiple regression for tests of mediation and moderation, which assumes homoscedasticity and normality of residuals. Preliminary analyses examined data characteristics, including tests for potential problems with homoscedasticity, and for outliers (> 3 SD away from mean). Outliers were removed.

Tests for mediation were conducted following Baron and Kenny (1986) (See Figure 1). Separate models were fit for the different measures of eating and weight problems. First, the direct effect ("c") was tested in models in which weight and eating problems were predicted by Child Depression. Next, models tested whether Child Depression predicted Cortisol ("a"). Finally, models were fit in which both Child Depression ("c prime") and Cortisol ("b") were predictors of eating and weight problems.

Tests for moderation were conducted following Aiken and West (1991). Predictor variables were centered before computing cross products. Interactions between Child Depression and Cortisol were tested for the prediction of eating and weight variables (moderation of "c"). Interactions between Child Depression and Sex and between Child depression and Puberty were examined in the prediction of eating and weight problems (moderation of "c") and Cortisol (moderation of "a"). Interactions between Cortisol and Sex and between Cortisol and Puberty were examined in the prediction of eating and weight problems (moderation of "b"). Probing of interactions was performed using an online calculator (Preacher, Curran, & Bauer, 2006). Significant interactions were probed and plotted at +/- 1 SD of the predictor variables.

Results

Preliminary Analysis

Means and standard deviations are provided in Table 1. An examination of the data indicated two cases were consistent outliers on cortisol measures; these cases were removed for data analysis. No significant skew was observed in any study variable.

Bivariate Relations among Variables

Pearson correlations indicated that Child Depression was significantly associated with greater child-report of External Eating, $r(48) = .46$, $p < .01$, but lower mother-report of Emotional Over-Eating, $r(46) = -.38$, $p = .01$. There were no associations between Child Depression and any cortisol measures.

Several significant associations between cortisol and the eating and weight variables were observed. Higher child Post 1 Cortisol was related to lower mother-report and father-report of child Food Responsiveness, $r(25) = -.51$, $p < .01$ and $r(24) = -.39$, $p < .05$, respectively. Greater child Post 2 Cortisol was associated with lower mother-report of child Food Responsiveness,

r(25) = -.48, p < .05, and lower mother-report of Emotional Over-Eating, $r(25) = -.41$, $p < .05$. Greater child Post 3 Cortisol was related to higher child-report of Dietary Restraint, $r(24) = .39$, $p = .05$, greater child-report of Emotional Eating, $r(24) = .41$, $p < .05$. Greater increases in Cortisol from Baseline to Post 1 were associated with lower mother-report of child Food Responsiveness, $r(25) = -.43$, $p < .05$, lower mother-report of child Emotional Over-Eating, $r(25) = -.54$, $p < .01$, and lower father-report of child Food Responsiveness, $r(24) = -.43$, $p < .05$. Greater increases in Cortisol from Baseline to Post 2 were associated with lower mother-report of child Emotional Over-Eating, $r(25) = -.50$, $p < .01$. Greater increases in Cortisol from Baseline to Post 3 were related to lower mother-report of child Emotional Over-Eating, $r(24) = -49$, $p < .05$.

Cortisol as a Mediator and Moderator of Associations

Because Child Depression was not related to any cortisol variables, cortisol cannot mediate associations between Child Depression and eating and weight variables. Thus, no further mediation analyses were conducted. Tests of moderation were conducted in which cortisol variables were considered as moderators of the associations between Child Depression and the eating and weight variables. Several significant interactions were observed. The interaction between child Baseline Cortisol and Child Depression predicted Dietary Restraint, $\beta = .51$, $p < .05$. Simple slopes analysis indicated that the association between Child Depression and increased Dietary Restraint was significant only for children exhibiting higher levels of child Baseline Cortisol (Figure 2A). The interaction between child increases in Cortisol from Baseline to Post 3 was a significant predictor of Parental Pressure to Eat, $\beta = .53$, $p < .05$. Parental Pressure to Eat scores were similar in the context of lower Child Depression. In the context of higher Child Depression, children who exhibited greater increases in Cortisol from Baseline to

Post 3 reported greater Parental Pressure to Eat than children who exhibited less pronounced increases in Cortisol (Figure 2B). The interaction between child changes in Cortisol from Baseline to Post 3 also predicted mother-report of child Enjoyment of Food, $\beta = -.61$, $p < .05$. In the context of lower Child Depression, children's Enjoyment of Food was similar. In the context of higher Child Depression, children who exhibited less pronounced increases in Cortisol from Baseline to Post 3 had greater Enjoyment of Food than children exhibiting more pronounced increases in Cortisol (Figure 2C).

Child Sex as a Moderator of Relations

Next, child sex was considered as a moderator of relations between (1) Child Depression and eating and weight variables, (2) between Child Depression and cortisol variables, and (3) between cortisol variables and eating and weight variables. No significant interactions were observed for relations between Child Depression and the eating and weight variables or for relations between Child Depression and cortisol variables. Child Sex interacted with child Baseline Cortisol to predict father-report of Child Satiety Responsiveness, $\beta = .74$, $p < .01$. Baseline Cortisol was only associated with increased father-reported Child Satiety Responsiveness for girls (Figure 3A). Child sex also interacted with increases in child Cortisol from Baseline to Post 1 in the prediction of mother-report of child Food Responsiveness, $\beta = -.44$, $p < .05$. The association between change in Cortisol from Baseline to Post 1 was negatively associated with Food Responsiveness only for girls (Figure 3B). Similarly, child sex interacted with increased child Cortisol from Baseline to Post 2 in the prediction of mother-report of child Food Responsiveness, $\beta = -.51$, $p < .05$. The association between change in Cortisol from Baseline to Post 2 was negatively associated with Food Responsiveness only for girls (Figure 3C).

Pubertal Status as a Moderator of Relations

Finally, child pubertal status was considered as a moderator of relations between (1) Child Depression and eating and weight variables, (2) between Child Depression and cortisol variables, and (3) between cortisol variables and eating and weight variables. No significant interactions were observed for relations between Child Depression and the eating and weight variables or for relations between Child Depression and cortisol variables. However, numerous significant interactions between Child Pubertal Status and cortisol variables were observed. Child Pubertal Status interacted with Post 1 Cortisol in the prediction of mother-reported Child Satiety Responsiveness, $\beta = -.51$, $p < .05$. Higher Post 1 Cortisol levels were associated with lower Child Satiety Responsiveness only for children later in Pubertal Development (Figure 4A). Child Pubertal Status interacted with Post 2 Cortisol in the prediction of child-reported External Eating, $\beta = .69$, $p < .05$. Higher Post 2 Cortisol was associated with higher External Eating only for children higher in Pubertal Status (Figure 4B). Child Pubertal Status interacted with Post 2 Cortisol in the prediction of mother-reported Child Satiety Responsiveness, $\beta = -.67$, $p < .05$. Higher Post 2 Cortisol was related to higher child Satiety Responsiveness only for children lower in Pubertal Status (Figure 4C).

Child Pubertal Status interacted with change in cortisol from Baseline to Post 1 in the prediction of mother-reported Enjoyment of Food, $\beta = .49$, $p < .05$, and mother reported Emotional Under-Eating, $\beta = -.51$, $p < .05$. Increases in cortisol from Baseline to Post 1 were associated with greater Enjoyment of Food only for children higher in Pubertal Status; associations are very similar to those shown in Figure 4B. There was a marginally significant association between changes in cortisol from Baseline to Post 1 and lower Emotional Under-Eating only for children higher in Pubertal Status; there was no association between these two

variables for children lower in Pubertal Status. The pattern of associations is very similar that shown in Figure 4C. Child Pubertal Status interacted with change in cortisol from Baseline to Post 2 in the prediction of mother-reported child Emotional Under-Eating, $\beta = -.76$, $p < .05$. In the context of less pronounced changes in Cortisol from Baseline to Post 2, Emotional Under-Eating scores were similar. In the context of more pronounced increases in Cortisol from Baseline to Post 2, children lower in Pubertal Status had higher Emotional Under-Eating than children higher in Pubertal Status (Figure 4D). Child Pubertal Status interacted with change in Cortisol from Baseline to Post 3 in the prediction of mother-reported child Food Responsiveness, $\beta = .73$, $p < .05$. The interaction is similar to that presented in Figure 4B. For children higher in Pubertal Status, there was a significant positive association between increases in Cortisol from Baseline to Post 3 and greater Food Responsiveness. For children lower in Pubertal Status, there was a significant negative association between increases in Cortisol from Baseline to Post 3 and lower Food Responsiveness. Child Pubertal Status interacted with change in Cortisol from Baseline to Post 3 in the prediction of mother-reported child Emotional Over-Eating, $\beta = .58$, $p < .05$. The interaction is similar to that presented in Figure 4B. For children lower in Pubertal Status, more pronounced increases in Cortisol from Baseline to Post 3 were related to lower Emotional Over-Eating. For children higher in Pubertal Status, there was no significant association between change in Cortisol from Baseline to Post 3 and Emotional Over-Eating.

Discussion

The current study proposed that childhood depression and cortisol would be related to children's eating behavior and weight problems. We proposed that cortisol would mediate relations between depression and weight-related outcomes in children. We expected that children with greater depressive symptoms would exhibit either cortisol hypo- or hyper-reactivity, and

that maladaptive cortisol reactivity would be associated with eating behavior and weight problems. We also proposed that cortisol would moderate relations between depression and eating behavior and weight problems in children; child depression and eating variables would be more strongly related in the context of maladaptive activity. In addition, we expected that sex and pubertal status would moderate relations. We hypothesized that girls and children later in puberty would have stronger relations among depressive symptoms, HPA activity, and weight-related outcomes.

The study's first hypothesis that child depression would be linked to eating and weight issues was not consistently supported. We found that greater depression was related to greater external eating, but that it was also associated with lower emotional overeating. Thus, the directions of the effect were not consistent. In the first association, child depression was linked to worse eating behavior, but in the second association depression was related to better eating behavior. We also did not find any associations between child depression and eating that were moderated by sex or pubertal status. However, associations between depression and eating behavior were found for certain levels of HPA activity.

The second hypothesis was that HPA activity would mediate relations between child depression and eating and weight problems. This study found that child depression was not related to eating and weight issues. Child depression was also not associated with any of the cortisol variables (e.g., Baseline Cortisol, Post 1 Cortisol, change in Cortisol from Baseline to Post 2). These associations between HPA activity, depression, and eating and weight problems were not present for either sex or for different levels of pubertal status. Thus, findings did not support the hypothesis. It is possible that HPA activity may be more connected to weight or

depression in adults rather than in children. However, small sample size and the fact that this was a community sample may also have affected estimates of associations.

The third hypothesis that HPA activity would moderate associations between child depression and eating and weight problems was supported. Child depression was related to restraint only for children higher in baseline cortisol. Child depression was also associated with parental pressure to eat for children greater in cortisol reactivity at post 3, but not for children lower in cortisol reactivity at post 3. Child depression was linked to higher enjoyment of food only for children lower in reactivity at Post 3. Based on this evidence, it appears as though higher baseline cortisol and cortisol hyper-reactivity may be related to decreases in children's amount of eating. These children are restraining their eating, being pressured by their parents to eat more, and are enjoying their food less. They may be experiencing the anhedonic aspects of depression, such as withdrawal from pleasant activities including eating and having flat affect (Dougherty, Klein, Olino, Dyson, & Rose, 2009).

The fourth hypothesis that child sex would moderate relations between depression, cortisol, and eating and weight issues was partially supported. Relations were found only for girls and only when cortisol was a predictor. For girls, higher baseline cortisol was linked to greater satiety responsiveness, greater cortisol reactivity at post 1 was related to lower food responsiveness, and greater cortisol reactivity at post 2 was linked to lower food responsiveness. This was expected because girls seem to be more influenced by body image and feeling connected to others than boys; they may also be more likely to use food as a coping strategy (Jenkins, Rew, & Sternglanz, 2005). This evidence is also consistent with some prior research showing that higher cortisol levels were associated with less eating (Frisco, Houle, & Martin, 2009), but is inconsistent with other studies showing hyper-cortisolism is linked to over-eating

(Farag et al., 2008). The latter studies were done primarily with adolescents and adults, emphasizing the importance of not extrapolating findings from the adult literature to early childhood.

The fifth hypothesis that child pubertal status would moderate relations between depression, cortisol, and eating and weight issues was partially supported. No significant interactions were observed for relations between child depression and the eating and weight variables or for relations between child depression and cortisol levels. Child pubertal status did moderate relations between cortisol and eating and weight variables. The most significant associations were for children higher in pubertal status. For example, higher cortisol at post 1 was associated with lower child satiety responsiveness only for children later in puberty. Higher post 2 cortisol was related to higher external eating only for children higher in pubertal status. There was a significant positive association between increases in cortisol from baseline to post 3 and greater food responsiveness for children higher in pubertal status. Thus, food seems to matter more for children later in pubertal status, but only in the context of higher cortisol levels (Palmert, Radovick, & Boepple, 1998). These findings are consistent with hypotheses. Perhaps changes in cortisol levels that occur with puberty are somewhat driving children's increased appetites and greater enjoyment of food (Spiegel et al., 2004).

This study has shed additional light on relations between child depression and eating and weight problems in childhood. However, there limitations to the study. First, the study is cross-sectional in design. The direction of associations cannot be determined. While it is plausible that depression plays a causal role in the development of eating behavior, it is also possible that eating behavior has implications for the development of depression. Future longitudinal research is needed to explicate possible bidirectional associations. Also, future research including a

clinical sample and other developmental periods is needed to further understand relations among depression, cortisol, and eating and weight issues.

Despite these limitations, results showed that HPA activity may play an important role in the development of eating behavior. Greater cortisol seems to promote under-eating in the context of child depression, but this is not likely to be the case for children later in pubertal development. Associations were also stronger for girls, who are at greater risk for depression. Preventative measures must be taken to attempt to reduce children's risk for depression and eating and weight issues. Practitioners should be aware that links between depression and eating are different for various age groups, and should be particularly concerned about under-eating in early childhood.

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Table 1.

Variable	*M*	*SD*
CDI Score	36.42	5.80
Child External Eating Score	9.85	2.91
Child Dietary Restraint Score	18.60	4.93
Child Parental Pressure to Eat Score	5.35	1.19
Child Emotional Eating Score	9.54	2.81
Mother-Reported Child Food Responsiveness Score	10.91	4.26
Mother-Reported Child Enjoyment of Food Score	14.57	3.51
Mother-Reported Child Emotional Under-Eating Score	10.98	3.28
Mother-Reported Child Emotional Overeating Score	8.22	3.03
Mother-Reported Child Satiety Responsiveness Score	25.67	4.38
Father-Reported Child Satiety Responsiveness Score	26.07	4.22
Father-Reported Child Food Responsiveness Score	10.80	4.27
Father-Reported Child Enjoyment of Food Score	12.54	2.99
Father-Reported Child Emotional Under-Eating Score	10.61	3.69
Father-Reported Child Emotional Overeating Score	7.87	2.95
Child Raw BMI	18.68	3.78
Child BMI Percentile	69.25	31.11
Child Baseline Cortisol	0.06	0.03
Child Post 1 Cortisol	0.04	0.02
Child Post 2 Cortisol	0.04	0.02
Child Post 3 Cortisol	0.03	0.01
Change in Cortisol from Baseline to Post 1	0.00	0.01
Change in Cortisol from Baseline to Post 2	0.00	0.02
Change in Cortisol from Baseline to Post 3	0.00	0.01
Puberty Score	1.28	0.33

Note: Cortisol measures are in μ/mL; Change in cortisol is a residualized change score that adjusts for baseline levels.

Figure Captions

Figure 1. Example Mediation Model

Figure 2. Significant Interactions between Cortisol and Child Depression.

Figure 3. Significant Interactions between Child Sex and Cortisol

Figure 4. Significant Interactions between Child Pubertal Status and Cortisol

Figure 1.

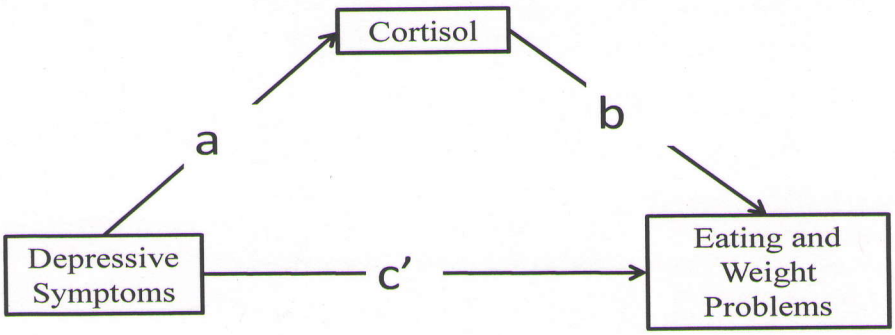
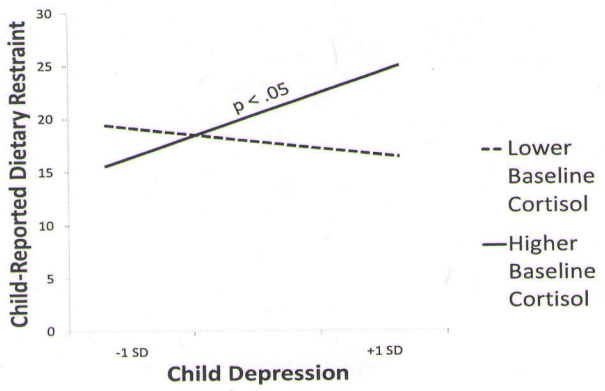
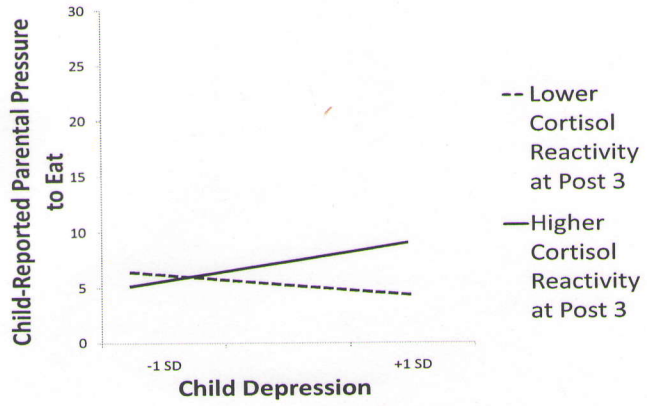

Cortisol
a
b
Depressive Symptoms
c'
Eating and Weight Problems

Figure 2.

A.


Child-Reported Dietary Restraint
30
25
20
15
10
5
0
p < .05
-1 SD
+1 SD
Child Depression
-- Lower Baseline Cortisol
— Higher Baseline Cortisol

B.


Child-Reported Parental Pressure to Eat
30
25
20
15
10
5
0
-1 SD
+1 SD
Child Depression
-- Lower Cortisol Reactivity at Post 3
— Higher Cortisol Reactivity at Post 3

C.

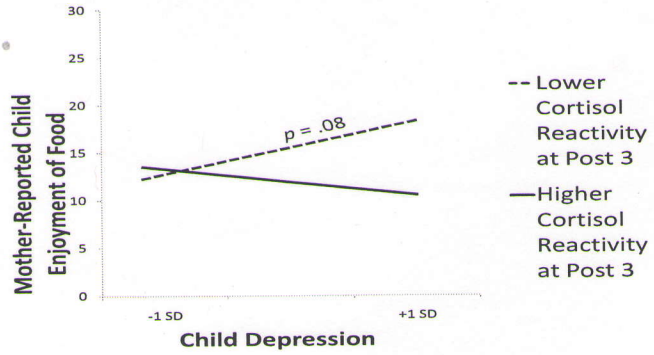
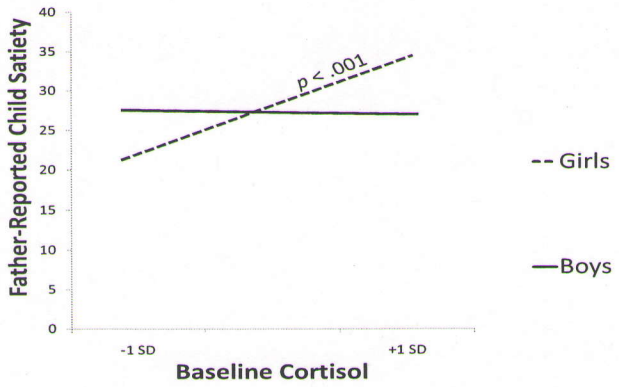
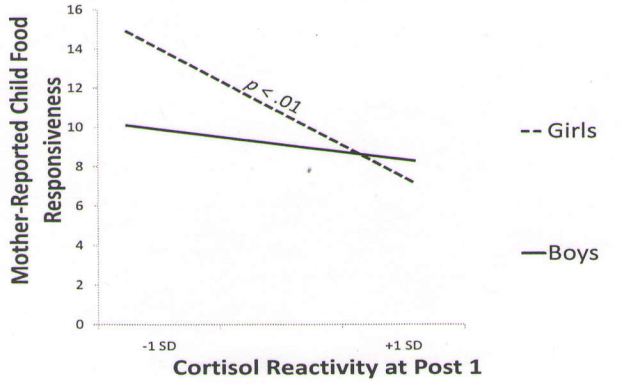

Mother-Reported Child Enjoyment of Food
30
25
20
15
10
5
0
p = .08
-1 SD
+1 SD
Child Depression
-- Lower Cortisol Reactivity at Post 3
— Higher Cortisol Reactivity at Post 3

Figure 3.

A.


Father-Reported Child Satiety
40
35
30
25
20
15
10
5
0
p < .001
-1 SD
+1 SD
Baseline Cortisol
-- Girls
—Boys

B.


Mother-Reported Child Food Responsiveness
16
14
12
10
8
6
4
2
0
p < .01
-1 SD
+1 SD
Cortisol Reactivity at Post 1
-- Girls
—Boys

C.

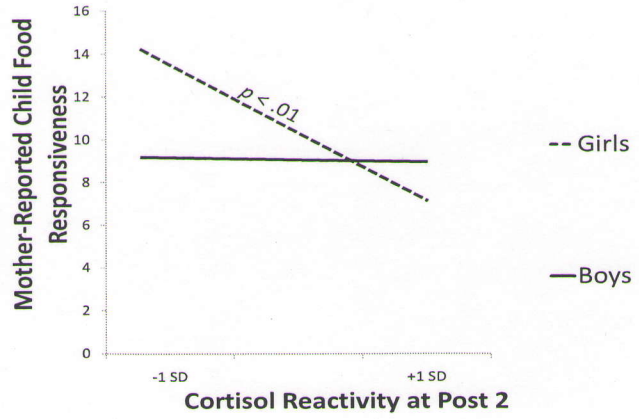
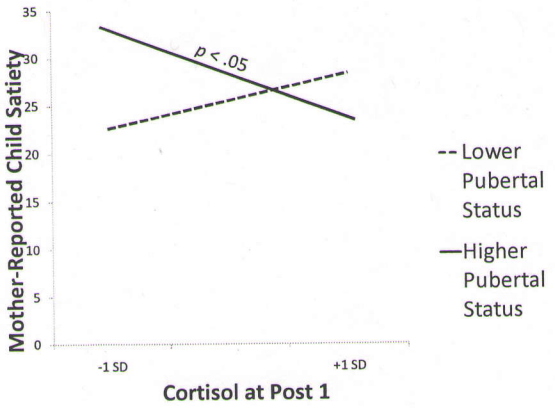
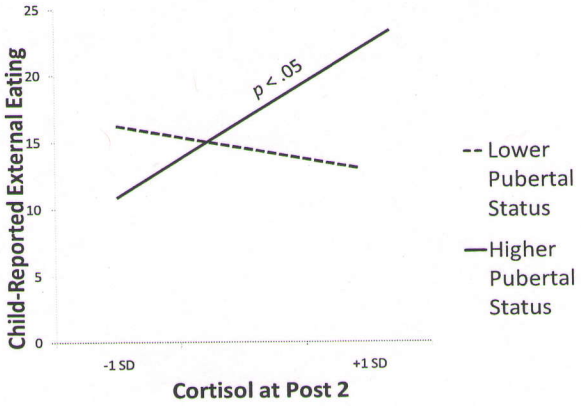

Mother-Reported Child Food Responsiveness
16
14
12
10
8
6
4
2
0
p < .01
-1 SD
+1 SD
Cortisol Reactivity at Post 2
-- Girls
—Boys

Figure 4.

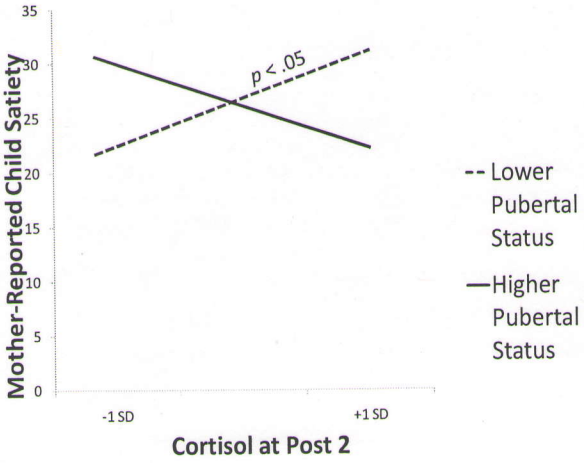
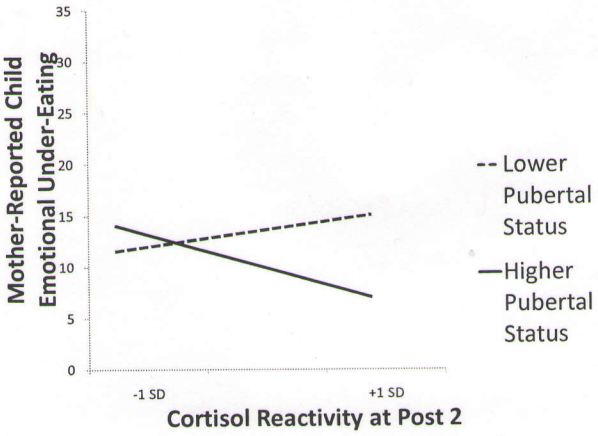
A.



B.



C.



D.