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Depression, Cortisol Reactivity and Obesity in Childhood and Adolescence

Samantha Dockray, Ph.D.¹,

Department of Epidemiology and Public Health, University College London

Elizabeth J. Susman, Ph.D., and

Department of Biobehavioral Health, The Pennsylvania State University

Lorah D. Dorn, Ph.D.

Division of Adolescent Medicine, Cincinnati Children's Hospital Medical Center and University of Cincinnati College of Medicine

Abstract

Purpose—Depression in childhood is associated with higher body mass index (BMI), a relative measure of overweight, and overweight is associated with cortisol reactivity, indexed by heightened secretion of cortisol in response to a stressor. The current study uses a mediation model to examine the associations between symptoms of depression, cortisol reactivity and BMI in a cross-sectional study.

Methods—Children (N=111) 8 to 13 years old and a parent completed structured interviews. The Child Behavior Checklist was used to assess symptoms of depression, and cortisol reactivity to the Trier Social Stress Test for Children was measured. Physical examinations were used to determine BMI (kg/m²) and pubertal stage.

Results—Depression was positively associated with BMI in both sexes. Age and pubertal stage were not significantly associated with BMI, nor was physical activity and BMI in a model including depression. In girls, but not in boys, the association between depression and BMI was mediated by cortisol reactivity.

Conclusions—The current findings attest to the significance of psychological states as potential components in models of childhood obesity, and provide conceptual and empirical support for the inclusion of cortisol reactivity in these models.

Introduction

Obesity and depression represent critical public health challenges of particular significance in children and youth. Obesity is associated with poor health outcomes that include insulin resistance, cardiovascular disease and early mortality. Rising in prevalence in the pediatric population, obesity has significant psychosocial and socioeconomic sequelae, and confers an increased risk of chronic disease [1]. The secular trend in overweight and obesity in US

children is paradoxical: weight status has increased whereas there has been little change in vigorous physical activity [2] or mean caloric intake above recommended intake for age [3]. A number of models of childhood obesity have examined emotional functioning, specifically depression as a factor, although obesity is usually posited as a cause rather than consequence of emotional state. The recent integration of neuroendocrine processes, for example increased cortisol secretion, into traditional models of obesity holds promise for illuminating mechanisms by which psychological processes may influence the development of obesity and indicate new directions in understanding and preventing childhood obesity [4]. The objectives of the current report are to determine the relations between depression, physical activity and body mass index in a sample of children and adolescents, and to assess the role of cortisol reactivity in mediating the relationship between depression and body mass index (BMI).

Traditionally, obesity has been viewed as an antecedent to depression, with social stigmatization producing shame, guilt and peer isolation leading to depression, especially in early life [5]. Recent studies provide support for a model wherein obesity is but a physical manifestation of the neuroendocrine processes associated with depression [6]. What has not been intensively examined are the specific neuroendocrine processes that mediate the association of depression and obesity. As convincing evidence that depression may be a causal factor in obesity, recent prospective studies demonstrated an association between childhood depression and obesity in later life [7]. Depressed children were more likely to be obese at follow-up, however, obese children were not at any increased likelihood of being depressed [3,8]. The increased likelihood of obesity for depressed children remained even after controlling for physical activity and other factors such as parental body mass index (BMI). In sum, both depression and obesity may have multiple biological and social determinants, and there is mounting evidence that childhood depression is associated with overweight and obesity, both in childhood and in later life.

Products of the stress system are a putative prime agent in the development of obesity. The physiological mechanism linking depression and BMI is proposed to be repeated activation of the hypothalamic-pituitary-adrenal (HPA) axis mediated stress response with the accompanying increase in cortisol secretion. High cortisol levels are associated with obesity, especially abdominal obesity [9] and to compound this problem, individuals who are overweight have a greater stress elicited change in cortisol secretion than normal weight individuals [10]. In addition, children with high cortisol reactivity may be less likely to engage in physical exercise [11]. Thus, we hypothesize that there will be an association between depression and BMI, but that this association will be mediated by cortisol reactivity to a laboratory stressor.

The sex differences that exist in overweight status and obesity are also relatively unexplored. The underlying rationale for such sex differences may be a function of the endocrine milieu, gender role socialization or psychosocial factors. Different socio-cultural norms and expectations with regard to weight gain are applied to girls, and there are numerous reports of relationships between depression and obesity being stronger in females than males [12]. A recent meta-analysis of longitudinal studies by Blaine ([3] shows compared to non-depressed people, depressed people are significantly more likely to be obese at a follow-up, and this association was more pronounced in adolescent girls. Blaine states that adolescent girls with depression are two and a half more likely to be obese at follow-up than their non-depressed peers. Thus, we expect that the association between depression and obesity will be stronger in girls than in boys.

Methods

Participants

Participants were 111 healthy children and adolescents and a parent or caregiver (90% mother, 8% father and 2% grandmother) who are participating in a longitudinal study of puberty and behavior. To ensure educational and income-level heterogeneity in the sample, there were several recruitment strategies; One strategy was to obtain a list of names of all the children from designated ZIP codes from the American Student List (ASL), a commercial enterprise that provides lists of names of school-age children. The list of names was generated by ASL from ZIP codes supplied by the investigator. The ZIP codes were chosen from the county in which the research lab was located and adjacent counties that had easy accessibility to the lab. The list from ASL included the name, address, and phone number of 966 children enrolled in the education system in the designated ZIP codes. A letter was mailed to the parents of the 966 children on the list, and then researchers telephoned each family to inquire whether the adolescent was interested in participating in the study. Eighty-five children were enrolled on the basis of responses from the parents of the children on the list. Of the remaining children, research staff were unable to contact 584 (e.g., returned letters, no forwarding address), and 89 were ineligible on the basis of study criteria. Other reasons for nonparticipation included discomfort with research procedures ($n = 11$); family problems ($n = 3$), such as death in the family; lack of time ($n = 41$); not interested ($n = 39$); or multiple reasons ($n = 1$). The remaining participants were obtained from flyers distributed throughout the community and from telephone responses to e-mails distributed to staff at a large university. These efforts resulted in an additional 48 families contacting the research project; of these, 26 participated. If the adolescent and a parent or guardian were interested in participating, the parent was administered a telephone screening interview by a pediatric nurse or a graduate student to establish the adolescent's eligibility for the study. If the eligibility criteria for participation were met, a visit was scheduled for the adolescent and 1 parent or guardian at a General Clinical Research Center (GCRC) of a research university. The visits were scheduled at 4:00 p.m. (± 1.5 hr) depending on the adolescent's school schedule. The study protocol was approved by the Institutional Review Board of a university. All methods and procedures were executed in accordance with a written protocol. Upon arrival at the GCRC, the parent and adolescent were read an explanation of the study, given an opportunity to ask questions, and asked to signed a consent or assent form.

Data from the first wave of measurement were used in the current analyses, so the data is cross-sectional. Girls were aged 8, 10 or 12 years and boys are aged 9, 11 or 13 years and all participants were free from chronic health problems, and were not using any medications known to interfere with hormone levels (e.g. oral steroids) or influence weight gain (e.g. SSRIs). Eight of the girls had reached menarche. Demographic characteristics of the sample appear in Table 1.

Measures

Depression—Symptoms of depression were assessed using the Child Behavior Checklist/4-18 (CBCL), a norm-referenced behavior rating scale completed by parents [13]. Parents use a three-point rating scale for 113 behavioral and emotional problems that have occurred during the past six months. The subscale of Anxious/Depressed behaviors was used in the current analysis ($\alpha = .67$) but in order to secure a normal distribution, log 10 transformed values were used in the analyses.

Cortisol Reactivity—Cortisol reactivity was assessed using change in salivary cortisol levels following the Trier Social Stress Test for Children (TSST-C). The TSST-C [14] is

used to elicit a stress response in the laboratory environment, and includes both a cognitive and social evaluative challenge in the presence of two judges. The participants are instructed to provide the ending for a story beginning they are given, and that their story must take at least 5 minutes to tell. Participants are also told that the judges will evaluate the story in relation to the story of other children their age. The TSST has proven to be a reliable stressor across different age groups and populations [15].

Salivary cortisol reflects HPA axis activity, and is the most effective modality for determining a stress response in laboratory settings [16]. Participants were instructed not to eat, drink (except water) or vigorously exercise in the 2 hours preceding the visit. Sessions were conducted at 4 p.m. $\pm$ 1 hour. Participants rinsed their mouths with water immediately before passively drooling into a 5 ml tube for 5 minutes for each sample. Samples were transferred to a -70 ° C ultra low freezer until assayed using a highly sensitive enzyme immunoassay specifically designed for use with saliva (Cat. No.1-0102/1-0112 Salimetrics, PA). The test has a range of sensitivity from 0.007 to 1.2 μ g/dl, and average intra- and interassay coefficients of variation (CV) of 5.34% and 9.86%, respectively. All samples and controls were assayed in duplicate and the average used in analyses. A total of five saliva samples were collected (Sample 1 at 0 minutes, Sample 2 at 20 minutes, Sample 3 immediately post TSST, Sample 4 at 10 minutes post TSST and Sample 5 at 20 minutes post TSST). Using the method of Pruessner and colleagues [17] we calculated change in salivary cortisol as Area Under the Curve Increase (AUC_I), in contrast to total area under the curve, this formula deletes the distance from zero for all measurements, thus changes subsequent to an event are emphasized. We averaged Samples 1 and 2 to provide a baseline measure, and then entered Samples 3, 4 and 5 as post-stressor, and to assure a normal distribution, the data were log 10 transformed prior to analyses. For descriptive purposes, the raw values are presented.

Physical Activity—Participation in physical activity was ascertained using the Child Health and Illness Profile – Parent Form (CHIP-P), a well-standardized generic health status questionnaire [18]. The Physical Activity scale is comprised of 6 items ($\alpha = .74$ in this sample) tapping the child's engagement in physical exercise and fitness, for example "In the past four weeks how often did your child play hard enough to sweat or breathe hard?". Higher scores reflect higher levels of achievement and participation in physical activity and exercise. The mean T score (49) and standard deviation (9.8) indicate that the current sample is comparable in physical activity to the general population.

Pubertal stage—Pubertal Stage was assessed using Tanner criterion of genital stage for boys and breast stage for girls [19] by a trained pediatric research nurse. If the adolescent did not consent to the physical examination (boys = 5 girls = 2), the adolescent's self rating of his/her stage of pubertal development was substituted for the nurse rating. The correlation between nurse rating and adolescent ratings of Tanner stage (genital/breast) in this sample was .76 ($p < .01$).

BMI

Measures were obtained by the research nurse with participants wearing light clothing without coats, jackets or shoes. Height was measured using a stadiometer (SECA Equipment) and standard techniques [20]. Participants were weighed on a digital scale (Model 8430, Cardinal Scale Manufacturing). The average of three measures of height and weight was used and BMI for age percentile was calculated using the standard formula of weight (kg)/height² (m).

Statistical Analyses

The hypotheses were tested using hierarchical regression analysis, following Baron and Kenney's [19] recommendations for testing mediation. According to their criteria for mediation: (1) there must be a significant association between the predictor and the outcome variable; (2) in an equation including both the mediator and the outcome variable, there must be a significant association between predictor and mediator variable, and the mediator variable must be a significant predictor of the outcome variable. More recently the requirement for a significant association between the predictor and outcome has been discounted ([21], and so a priori we decided to continue the analyses even without a significant association between depression and BMI. Post-hoc, the test power was analysed using the methods described by Cohen et al 2003 by [22] and show that for the final model, in both boys and girls, power was greater than 90, with alpha at .05. Age and socioeconomic status (SES) were controlled by entering both into the regression analysis at the first step. Model 1 regresses BMI on pubertal stage (breast for girls and genital for boys) in the first step followed by depression and physical activity. Model 2 regressed cortisol reactivity on pubertal stage in the first step followed by depression and physical activity. Model 3 tested the effect of the inclusion of cortisol reactivity as a mediator of depression and BMI. Models were tested separately for girls and boys given the hypothesized sex differences.

RESULTS

Twenty-seven percent (26.7%) of the sample was overweight, or at risk for overweight, with 9.5% of the children between the 85th and 95th percentile, and 17.1% above the 95th percentile for weight according to the CDC growth charts [6,7,23]. No children were underweight. The high number of participants with high percentiles reflects the prevalence of overweight and obesity in this age group in the USA [24] and the allows for the testing of the conceptual model. The demographic characteristics of the sample are presented in Table 1, There were no significant sex differences in measures of depression, physical activity, cortisol reactivity or BMI, and the whole group means derived from the raw scores are presented in Table 2. As the cortisol values and depression scores were not normally distributed, log transformed values were used in all subsequent analyses. The correlations between the variables are shown in Table 3. In girls, depressed mood was positively associated with greater cortisol reactivity and higher BMI, but negatively associated with physical activity. In addition, higher cortisol reactivity was associated with higher BMI, but lower levels of physical activity. In boys, depressed mood was associated with greater cortisol reactivity and higher BMI. Cortisol reactivity and BMI were positively associated. Physical activity had no significant association with any other variables in boys.

Associations between depression, cortisol reactivity and BMI

Multiple regression analyses were conducted to test the hypothesis relating depression, cortisol reactivity and BMI. Model 1 regressed BMI on the block of background variables of age and pubertal stage, then on physical activity and finally on the CBCL depression subscale. In both sexes depression was positively associated with BMI. Model 2 regressed stress reactivity (AUC₇) on the block of background variables, that is age and pubertal stage, then on physical activity, and finally on the CBCL depression subscale. In girls, age but not pubertal stage, had a significant positive association with stress reactivity. There was no association between age or pubertal stage for boys. In both boys and girls, depression was significantly and positively associated with cortisol reactivity (see Table 4). Physical activity was not associated with cortisol reactivity in either girls or boys. These models confirm the positive associations between symptoms of depression and cortisol reactivity, and between symptoms of depression and BMI, allowing further tests for mediation.

Cortisol reactivity as a mediator of the association between depression and BMI

Hierarchical multiple regression was used to test the full direct-indirect mediational effect model of the predictor, depression, controlling for its association with physical activity, followed by entry of the mediator, AUCI. The specified model accounted for 41% and 28% of the variance in BMI for girls and boys, respectively (see Table 4). In both boys and girls depression was associated with higher cortisol reactivity but only in girls was cortisol reactivity in turn associated with BMI. In addition, levels of physical activity did not have a significant effect on cortisol reactivity or on BMI for either boys or girls. The size of the mediated effect of depression on BMI was almost 4 times greater in girls (.16) than in boys (.04) where the effect size was calculated as the product of the path between depression and cortisol reactivity, and the path between cortisol reactivity and BMI [25]. The drop in the coefficients of the direct path from depression to BMI after the inclusion of stress reactivity in the model was significant according to Sobel's test ($Z = 2.18, p < .03$) [26] indicating that cortisol reactivity carries the influence of depression to BMI.

Thus, cortisol reactivity is a partial mediator of the association between depression and BMI in girls but not boys. The significant direct effect of depressed mood on BMI, found in girls, although diminished remained significant. In summary, for girls the effect of depressed mood on higher BMI is mediated and accentuated by higher cortisol responses to a stressor. However, in boys depressed mood is directly associated with BMI and cortisol reactivity, but cortisol reactivity is not in turn associated with BMI, and so therefore does not mediate the direct effects of depressed mood.

DISCUSSION

The current study demonstrates the psychosocial correlates of obesity in adolescence by showing that BMI relates to depressed mood, and indicates that this association may be mediated by cortisol reactivity in girls. The association between cortisol reactivity and body mass in girls parallels findings of predictors of adult obesity [27] and the commonalities in etiology indicate opportunities for early intervention. What is unique about the current findings is that cortisol reactivity was identified as a mediator of the association between depressed mood and obesity in girls. Consistent with previous research, we found that depressed mood is associated with cortisol reactivity and obesity; however, in contrast to other models of weight status, there is no robust association between physical activity and body mass. This pattern of findings underscores the role of psychological processes, in this case depression, in the development of obesity during adolescence.

The sex differences reported here extend existing knowledge of sex differences in the predictors of obesity and of cortisol reactivity, by identifying a mediator in the association between depression and obesity. The correlations between depressed mood and cortisol reactivity, and depressed mood and BMI, held across the sexes and are not surprising given the breadth of literature that has established links between these factors in adults [28]. The lack of a robust correlation between physical activity as measured here and BMI in girls, and the absence of an association between these factors in boys is counter-intuitive but not surprising given the inconsistencies in other cross-sectional studies [10].

Depression and physical activity

The oft-reported negative correlation between depressed mood and physical activity was indicated only in girls. One of the usual symptoms of depression is a change in levels of physical activity, especially a decrease in vigorous activities. The anomalous finding in boys, however, suggests that there may be motivators that over-ride a disinclination to exercise, such as internalized beliefs about sex appropriate exercise and ways to deal with

distress. Overall, girls' engagement in physical activity declines with pubertal development, and for many boys, advanced puberty is associated with greater participation in vigorous exercise [29]. These sex differences were paralleled in the correlations between physical activity and cortisol reactivity, with a negative association only indicated for girls. Adolescents with psychological problems are likely to have different health behaviors to healthy adolescents, including different exercise patterns [30,31]. A pattern of lack of exercise may be exacerbated as participation in school and community based exercise programs continues to decline [32]. The current results indicate different pathways between psychological states and physical health for boys and girls, and highlight the need for age and sex appropriate interventions to reduce obesity.

Depression and BMI

The confirmation of an association between depression and obesity is not surprising given the breadth of previous work in adults. Inconsistencies in the literature are notable however, especially with respect to sex differences [33-36]. The sex differences in depression-obesity associations revealed in the current analyses remained after controlling for age, pubertal stage and SES. Much of the previous work examining depression and obesity has drawn on populations of adults, or measured outcomes in adulthood, rather than in the pubertal years when interventions may be most effective for preventing or reducing later obesity. Depression has usually been associated with decreased appetite or food intake but there may be a sub-group of children or adolescents for whom increased, as in atypical depression, rather than decreased appetite is a sign of depression [30,37]. Our findings support the accumulating evidence indicating that depression and obesity share a common neurobiological pathway, specifically, the HPA axis, and are both associated with the over secretion of cortisol [38,39]. It is plausible that neurobiological pathways and the socio-environmental and familial milieu may contribute equally to predict and maintain both depression and obesity.

Mediating role of cortisol reactivity

A central objective of this study was to test the hypothesis that cortisol reactivity mediated the link between depression and BMI. The analyses confirmed that cortisol reactivity did mediate the association, indicated by the association of cortisol reactivity with depression, and the link between depression and BMI. Of note is that the association between cortisol reactivity and BMI was only significant in girls. In boys, although depressed mood was associated with cortisol reactivity, cortisol reactivity was not associated with BMI. This result is in keeping with the suggestion that there are sex differences in the psychosocial correlates of obesity [34,40], including in the years of early life. There are several plausible mechanisms for this sex difference, both physiological and behavioral. Persistent or repeated elevations in cortisol, as in response to a stressor, may influence the HPG axis and influence estrogen secretion patterns, and thus subsequent health outcomes in girls including those directly linked to fat deposition [41]. 'Stress-eating' may be a response perceived as more behaviorally appropriate for girls, and pubertal girls receive messages, both subliminal and overt, that women respond to stress by turning to high calorie foods. This behavior may then be reinforced by a pathway of central opioids or other physiological reward/regulatory pathways [42].

Limitations

Physical activity and depression were assessed using parental report measures as no equivalent child report measures were available. Although these measures have been validated, the use of child report measures, or objective measures of activity, may allow a more refined test of the model. The potential confound of psychosocial functioning and SES of the parental respondent with reported child depression was not indicated in this sample as

there was no significant associations between symptoms of psychological problems of the parent (as indicated by the Brief Symptom Inventory) and the child measures described. Further, this sample of adolescents is somewhat heterogeneous, and the sample size is relatively small and it is possible that the power to detect significant relationships may have been compromised, although estimates of power were good ($> .72$). Studies in psychology and other related fields have routinely documented power in the ranges of from .20 to .50 for detecting small to medium treatment effects.

In conclusion, this study provides unique insight into the etiology of obesity in late childhood and early adolescence. Cortisol reactivity appeared to be a mediator of depression and obesity, however, no causal implications can be drawn from the current report given its cross sectional nature. Future research in this area should examine these issues in a prospective fashion, especially given the contrasting findings regarding the role of depression as a cause or consequence of obesity in adolescence. The development of models that encompass the interacting biological and socio-environmental determinants of obesity has never been more pertinent. The current findings attest to the significance of psychological states as potential components in models of childhood obesity, and provide conceptual and empirical support for the inclusion of stress reactivity in these models. An understanding of the multiplicity of pathways shared by obesity and depression will inform the development of appropriate prevention and intervention programs for a condition of childhood so strongly associated with long term morbidity.

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Table 1

Demographic characteristics of the sample.

	Girls	Boys
N	55	56
Age (years)		
Mean	10.49 (1.5)	11.44 (1.6)
Tanner Stage (breast/genital) (N)		
1	21	26
2	11	12
3	8	7
4	5	5
5	0	1
Refused	5	2
Family SES		
Mean(SD)	46.2 (10.6)	47.2 (10.6)
Race (N)		
White/Non-Hispanic	49	52
Hispanic	3	1
African American	2	0
Asian American	1	2
American Indian	0	1

Note: Socioeconomic status (SES) determined by the Hollingshead [1] scale ranging from 8 (low) to 66 (high).

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Table 2

Means and standard deviations for measures of physical activity, depression, AUCI and BMI.

Variables	Girls Mean (SD)	Boys Mean SD
Depression	2.25 (2.13)	2.11 (2.2)
Physical activity	3.94 (.42)	4.25 (.50)
AUC _I	13.91 (12.92)	14.40 (12.31)
BMI	19.60 (4.51)	20.41 (4.57)

Note: AUC_I = Area Under the Curve Increase, BMI = Body Mass Index

Table 3

Correlations by sex between depression, physical activity, cortisol reactivity and BMI.

	1	2	3	4	5	6
1. Age	-	.62**	.29*	.02	.34*	.35*
2. Pubertal Stage	.66**	-	.14	.18	.31	-.11
3. CBCL Depression	.08	.19	-	-.37*	.37**	.31*
4. Physical Activity	-.28	-.41	-.06	-	-.41*	-.29
5. AUC _I	.47**	.19	.43**	.06	-	.52**
6. BMI	.16	.47**	.38**	.10	.29*	-

*Note.**
p=.05;**
p=.01

Correlations below the diagonal are for boys (N=56) and correlations above the diagonal are for girls (N=55).

Table 4
Regression models by sex of cortisol reactivity and BMI on age, pubertal stage, physical activity and depression.

	Girls				Boys			
	Model 1 (BMI)	Model 2 (AUCI)	Model 3 (BMI)	Model 1 (BMI)	Model 2 (AUCI)	Model 3 (BMI)	Model 1 (BMI)	Model 3 (BMI)
Age	.09*	.11*	.08*	.07	.13	.09*		.09*
Pubertal Stage	.14*	.07	.12	.09*	.02	.17*		.17*
Physical activity	.09	.02	.06	.06	.04	.03		.03
AUCI	-	-	.46*	-	-	.11		.11
Depression	.41**	.35*	.23*	.29*	.41*	.29*		.29*
F (df)	3.95* (4, 36)	2.97** (4, 36)	3.22* (5, 34)	2.53* (4, 38)	2.75* (4, 38)	2.53* (5, 37)		2.53* (5, 37)
R ²	.38*	.18	.41**	.26	.20*	.28		.28

Note:

* -p<.05;

** =p<.01;

BMI is Body Mass Index and AUCI is cortisol area under the curve increase.