Adolescents' Depressive Symptom Experience Mediates the Impact of Long-Term Exposure to Maternal Depression Symptoms on Adolescents' Body Mass Index

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Abstract

Background Obesity is a cardiovascular disease risk factor and affects approximately 13.7 million U.S. children and adolescents between the ages 2 and 19 years old in 2015–2016.

Purpose To determine the relationship between children's average long-term exposure to maternal depressive symptoms age 1 month to Grade 6 and adolescents' body mass index (BMI) *z*-score at age 15 mediated by the adolescents' depressive symptom experience.

Methods A total of 1,364 infants and their families from the Eunice Kennedy Shriver National Institute of Child Health and Human Development Study of Early Child Care and Youth Development were recruited.

Results Mediation analyses revealed a significant relationship between children's average long-term exposure to maternal depressive symptoms from age 1 month to Grade 6 and adolescents' BMI *z*-score at age 15 (total effect = 0.015, $p = .013$, 95% confidence interval (CI): 0.0032, 0.027). The adolescents' experience of depressive symptoms significantly mediated this relationship (indirect effect = 0.0021, bias-corrected bootstrapped 95%

CI: 0.0004, 0.0044), with this mediated relationship more pronounced in girls.

Conclusions Findings indicate the possible existence of a mediating role of adolescents' depressive symptoms experience in the pathway from average long-term exposure to maternal depressive symptoms during children's early life to adolescents' elevated BMI.

Keywords: Adolescent · Obesity · Depression · Maternal · Body mass index · Mediation

According to the Center for Disease Control and Prevention (CDC; 2019), obesity affected approximately 18.5% (or 13.7 million) of U.S. children and adolescents aged 2–19 years in 2015–2016 [1]. The obesity prevalence of children aged 6–11 years old (18.4%) to adolescents aged 12–19 years old (20.6%) in 2015–2016 demonstrated an increasing trend of obesity among children and adolescents' as they aged [1]. Children and adolescents with obesity have an increased risk for adult obesity and cardiovascular disease risk factors, such as high blood pressure, high cholesterol, and Type 2 diabetes in their later lives [2–5].

Critical period models postulate that early life environmental conditions may program a pattern of biological and behavioral responses that have a long-term impact [6]. Current or recent exposure models specify paths through which risk factors at any point in the life cycle can influence concurrent health status [6]. Previous studies have proposed that maternal depression may play a vital role in the development of childhood overweight or obesity [7, 8]. One hypothesized pathway is that children may be more affected by long-term maternal depressive symptoms as opposed to short-term depressive symptoms [9, 10]. Chronic depression can impact a

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mother's ability to care for herself and her child, which, in turn, contributes to the risk of overweight and obese children [11, 12].

Maternal depression has long been associated with parenting behaviors [13]. Maternal depressive symptoms, negative affect, and inactivity may have a direct impact on parenting for children with healthy weight [14]. In older children, maternal depressive symptoms have been linked with increases in children's television viewing [15–17] and decreases in their physical activity [18]. Moreover, Milgrom et al. reported a positive association between perinatal depression and children's weight outcomes, which included overall adiposity, central adiposity, overweight, and obesity from age 0 to 8 years [19]. However, the relationship between maternal depression and childhood obesity has not yet been fully explored, leaving many important questions not yet answered. Particularly, it is crucial to understand how maternal depression affects child obesity at different developmental phases.

Chronic depression, not episodic depression, has been found to be associated with greater risk for children being overweight [14]. Prior research has shown that maternal depressive symptoms could influence children's risks for both depression and obesity, but the mechanism of such relationship is not fully understood. We hypothesize that children's depression could play a potential mediating role in the linkage of maternal depression to adolescent's obesity. Extant research indicates that adolescent depression played a role in increasing adolescent body mass index (BMI) [14, 20]. However, researchers have also acknowledged that few studies have explored the long-term association of maternal depressive symptoms and children's overweight status across time [20].

In this study, we used existing data from the Eunice Kennedy Shriver National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (NICHD SECCYD) to evaluate the mediating role of adolescents' experience of depressive symptoms in the pathway from average long-term exposure to maternal depressive symptoms during children's early life to adolescents' later-elevated BMI [21]. The NICHD SECCYD was a four-phase, multisite, prospective, longitudinal study that examined the relationships between child development and child care during infancy, early childhood, middle childhood, and middle adolescence (<https://www.nichd.nih.gov/research/supported/seccyd/overview>) [21]. In the current study, we hypothesized that children's average long-term exposure to maternal depressive symptoms from the age of 1 month to Grade 6 (predictor) would be related to the adolescents' BMI z-score at age 15 (outcome). The adolescents' experience of depressive symptoms was tested as the mediator with adjustment of children's gender, mother's delivery age, mother's minority status (dichotomous),

mother's educational level (years), income-to-need ratio at Grade 5, and study sites as covariates in the analyses. In addition, we explored gender variations in this hypothesized mediation pathway.

Methods

Participants

The SECCYD study followed 1,364 infants and their families who were recruited at 1 month of age from 10 locations in the USA in 1991 [21]. Potential participants were selected from 8,986 mothers who gave birth in the study hospitals during selected sampling periods, which were established on a site-by-site basis to ensure an unbiased sample that was representative of the catchment area population at each site. During Phase I of the study (1991–1994), the initial cohort of 1,364 children and their families were studied intensively from age 1 month after the birth through age 3 years old. The initial cohort included 24% ethnic minority children (13% African Americans, 6% Hispanics, and 5% Others), 11% mothers who had not completed high school, and 14% single-parent mothers. Phase II (1995–1999) followed a cohort of 1,220 of the enrolled children and families through first grade. Phase III (2000–2004) followed a cohort of 1,077 of the enrolled children and families through sixth grade. Phase IV of the study (2005–2008) followed a cohort of 1,009 enrolled children and families through age 15. Prior to the start of formal schooling, data were collected on an age-based chronological schedule (Phase I and the beginning of Phase II). With the onset of formal schooling, data were collected on a year-in-school basis (end of Phase II, Phase III, and beginning of Phase IV). After the seventh-grade assessment, most of the data were collected on an age-based schedule. The sample attrition rate between ages 1 month and 9 years (Phases I–III) was 21%, and the cohort lost an additional 6.3% between ages 9 and 15 years, resulting in a total of 1,009 children at age 15 years.

In this paper, the analysis sample included 844 participants with complete data of adolescents' experience of depressive symptoms, as well as weight and height assessments at age 15 years old, which accounted for 61.9% of the initial cohort in Phase I of the SECCYD. Preliminary analyses were conducted comparing baseline general sample characteristics of participants available for analysis with those who were missing in the analysis sample to evaluate potential sampling biases. No statistically significant differences were observed in the distribution of gender ($p = .055$), ethnicity ($p = .34$), average scores of children's experience of depressive symptoms ($p = .21$), and children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6

($p = .61$). The analysis sample had a significantly lower level of income-to-need ratio (4.38 vs. 5.21, $p = .04$), a significantly higher mother's education in years (14.41 vs. 13.94, $p = .001$), and age at birth (28.53 vs. 27.23, $p < .0001$) than the participants who had missing cases and were not available for the analysis.

Measures

Weight and height measurements

Each measurement followed standardized procedures [21]. These measurements were used as indicators of adiposity and the risk of cardiovascular diseases in this sample. Overweight and obesity were defined based on BMI between 85th and 95th and 95th age- and gender-specific percentile cutoffs of the 2000 CDC Growth Charts of children and adolescents in the USA [22, 23]. BMI z -scores were used in the analysis.

Maternal depressive symptoms

Maternal depressive symptoms were measured by the Center for Epidemiological Studies Depression (CES-D) Scale, which included 20 items reported by mothers of their children across different ages (i.e., age 1, 6, 15, 24, 36, 54 months, 1st, 3rd, 4th, 5th, and 6th grade [$\alpha = .85-.91$]) [21]. Respondents rated the frequency of depressive symptom experience in the past week. Scores ranged from 0 to 60, with higher scores indicating greater levels of depression. The CES-D is one of the most widely used and validated measures of depressive symptomatology for nonclinical samples [24]. Because postpartum depression is common and known to be associated with adverse outcomes for children, the construct of maternal depression was considered important for inclusion in the study at all major measurement points [24]. Children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6 was calculated by the mean score of maternal depression score (i.e., sum of CES-D score) from 1 month after delivery to Grade 6 (10 assessments).

Adolescent depressive symptoms

The Children's Depression Inventory was used to assess adolescents' depressive symptoms at age 15 (mediator). Adolescents' experience of depressive symptoms was assessed as the sum of items 1–10 after recoding responses to 0 to 2 and reverse coding items 2, 4, 5, 6, and 10 [21]. The possible range of scores was from 0 to 20, with higher scores indicating greater adolescent depression ($\alpha = .81$) [21].

Covariates

Children's gender, mother's delivery age, mother's minority status (a dichotomous variable with values of

non-Hispanic Whites vs. other groups), mother's years of education, income-to-need ratio at Grade 5, and study sites were controlled for in the analysis. Income-to-needs ratio at Grade 5 was calculated with family income, the number of people living in the home, as well as the federal poverty guidelines for that year, was assessed [25].

Statistical Analysis

Descriptive statistics were derived from means and frequencies. Mediation analyses were conducted to determine the hypothesized mediating role of adolescents' experience of depressive symptoms in the pathway from children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6 to adolescents' later BMI z -score at age 15 years. Children's gender, mother's delivery age, mother's minority status, mother's educational level, income-to-need ratio at Grade 5, and study sites were controlled for as covariates in the analysis. Andrew Hayes Process Macro (2013) implemented in SAS (version 9.4) for mediation analysis was used to produce a bias-corrected bootstrapped 95% confidence interval (CI) of the mediation effect with 10,000 bootstrapped samples generated by the bootstrapped sampling approach [26]. Instead of using a p -value, a statistically significant mediation effect was identified when the bias-corrected 95% CI did not contain zero. Bootstrapped CIs have been used to identify statistical significance of the mediation effects because they better respect the irregularity of the sampling distribution of the mediation effects and yield inferences that are more likely to be accurate with higher statistical power compared to the test results derived from the normal theory approach [26, 27]. In addition to the analyses with raw data, we also adopted a multiple-imputation approach to handling missing data and we replicated all of the mediation analyses with imputed data sets for sensitivity analytic purpose (data and results are not included in this manuscript but are available upon request). The multiple-imputation approach with Markov Chain Monte Carlo (MCMC) method was implemented in SAS Proc MI [28] in order to generate 40 imputed data sets [29, 30]. These imputed data sets were then used to replicate all the mediation analyses and results were further summarized with SAS Proc MIANALYSIS for interpretation [28]. Consistent findings were observed by comparing results from the analyses with imputed data and the results from the analyses with raw data.

Results

Descriptive information of the study sample are presented in Table 1. Boys and girls represented 49.64% and 50.36% of the sample, respectively. The majority of the

mothers were non-Hispanic White (82.23%) and the rest (17.77%) consisted of various ethnic minority groups (i.e., 12.91% African Americans, 2.13% Hispanics, and

2.73% others). The proportion of overweight and obese adolescents at age 15 years old were both 15.52%.

A statistically significant total effect of children's average long-term exposure to maternal depressive symptoms from children's age 1 month to Grade 6 on adolescents' later-elevated BMI *z*-score at age 15 was observed (total effect = 0.015 with the standardized coefficient = 0.09, $p = .013$, 95% CI: 0.0032, 0.027; Table 2; Fig. 1). In the mediation analysis, the direct effect of children's average long-term exposure to maternal depressive symptoms on adolescents' BMI *z*-score at age 15 was statistically significant (direct effect = 0.013 with the standardized coefficient = 0.078, $p = .033$, 95% CI: 0.0010, 0.024; Table 2), while the significant mediation effect of adolescents' experience of depressive symptoms was observed (indirect effect = 0.0021 with the standardized coefficient = 0.0124, bias-corrected bootstrapped 95% CI: 0.0004, 0.0044). As an effect size indicator, the ratio of indirect to the total effect of childhood exposure to maternal depression on BMI *z*-score at age 15 was 14.3%. These findings indicate that the adolescents' experience of depressive symptoms partially mediated the effects of children's average long-term exposure to maternal depressive symptoms on later-elevated BMI in adolescence.

We further explored potential moderated mediations by gender. Specifically, gender difference in the previously observed mediation pathway from children's average long-term exposure to maternal depressive symptoms to the adolescents' depressive symptom experience and, in turn, to the adolescents' elevated BMI. As summarized in Table 3, a significant moderated mediation effect by gender was observed (bias-corrected bootstrapped 95% CI: 0.0002, 0.0098; Table 3; Fig. 2). The mediation effect of adolescents' depressive symptom experience on the pathway from children's average long-term exposure to maternal depressive symptoms during the early childhood to later adolescents' BMI *z*-score at age 15 was statistically significant among girls (mediation effect = 0.0038

Table 1. General characteristics of the analysis sample ($N = 844$)

	Mean	SD
Score of average long-term exposure to maternal depressive symptoms from age 1 month to Grade 6	9.50	6.08
Adolescents' BMI <i>z</i> -score age 15	0.57	0.99
Score of adolescents' experience of depressive symptoms	1.97	2.62
Income-to-need ratio Grade 5	4.38	3.73
Mother's delivery age	28.53	5.56
Mother's education level (years)	14.41	2.39
Adolescent's gender ^a		
Boy	419	49.64%
Girl	425	50.36%
Mother's minority status ^a		
Majority (non-Hispanic Whites)	694	82.23%
Minority (all except non-Hispanic Whites)	150	17.77%
Weight status at age 15 years old ^a		
Normal/underweight	582	68.96%
Overweight	131	15.52%
Obesity	131	15.52%

BMI body mass index; SD standard deviation.

^a N and % are reported for categorical variables.

Fig. 1. Children's average long-term exposure to maternal depression and adolescents' body mass index *z*-score mediation model.

Table 2. Results of mediation models of adolescents' depressive symptom experience at age 15 on the pathway from children's average long-term exposure to maternal depression to adolescents' later body mass index (BMI) *z*-score at age 15

	Effect ^a	Standardized coefficients	95% CI	<i>p</i> -value
Total effect	0.015	0.09	0.003, 0.027	.013
Direct effect	0.013	0.078	0.0010, 0.024	.033
Indirect effect	0.0021	0.0124	0.0004, 0.0044	.030*

^aEffect and 95% confidence intervals (CIs) were estimated based on bootstrapped sampling approach. Total effect represents the overall effects of children's average long-term exposure to maternal depressive symptom from age of 1 month to Grade 6 on adolescents' later BMI; direct effect represents the *c'* path in Fig. 1, which is the effect of children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6 on the adolescents' later BMI with the mediator (i.e., adolescents' experience of depressive symptom) in the model. Indirect effect represents the *a* and *b* paths in Fig. 1, which are the indirect pathways from children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6 to the mediator (adolescents' experience of depressive symptoms) and, in turn, to the final outcome (i.e., adolescents' later BMI).

**p*-value from Sobel test.

Table 3. Results of moderated mediation models of adolescents' depressive symptom experience at age 15 as a mediator of the relationship between children's average long-term exposure to maternal depression and adolescents' body mass index z-score at age 15 by gender

	Effect ^a	Standardized coefficients	95% CI	p-value
Moderated mediation (conditional effects by gender)	–		0.0002, 0.0098	–
Boy direct effect	0.021	0.10	0.0052, 0.037	.0095*
Girl direct effect	0.005	0.046	–0.0110, 0.020	.55*
Boy indirect effect	–0.0004	–0.0017	–0.0036, 0.0023	–
Girl indirect effect	0.0038	0.0272	0.0008, 0.0087	–

^aEffect and 95% confidence intervals (CIs) were estimated based on bootstrapped sampling approach.

*p-value from Sobel test.

Fig. 2. Children's average long-term exposure to maternal depression and adolescents' body mass index z-score gender interaction moderated mediation model.

with the standardized coefficient = 0.0272 bias-corrected bootstrapped 95% CI: 0.0008, 0.0087) but not for boys. As an effect size indicator, the ratio of indirect to the total effect of childhood exposure to maternal depression on BMI z-score at age 15 was 38.23% in girls, while this ratio was only 1.75% in boys. In contrast, significant direct effects of children's average long-term exposure on adolescents' later-elevated BMI was found in boys (direct effect = 0.021 with the standardized coefficient = 0.10, 95% CI: 0.0052, 0.037) but not in girls.

Discussion

Summary and Interpretation

Our findings revealed a prospective harmful effect of children's average long-term exposure to maternal depressive symptoms from age 1 month to Grade 6 on adolescents' later BMI z-score at age 15, which was partially mediated by adolescents' depressive symptom experience. These findings provide evidence that long-term exposure to maternal depressive symptoms during children's early life could lead to elevated levels of BMI in later adolescence either directly or indirectly through adolescents' depressive symptom experience. It has been reported that persistent maternal depressive symptoms may be associated with childhood obesity [20]. Chronic clinical depression can make it even more challenging for mothers to care of their children, thereby increasing their child's risk for obesity, an important risk for other

negative health outcomes later in life, such as cardiovascular diseases.

Evidence suggests that prenatal and postpartum depression may have adverse physiological and behavioral effects on their children [31]. Mothers experience hormonal changes, which alter their moods, sleep patterns, and weight changes, which, in turn, affects mental health status [32]. In addition, postpartum depression affects approximately 10–15% of women and can impair mother–infant interactions that are important for child development [33]. Maternal attachment, sensitivity, and parenting style are critical for a healthy development of an infant's social, cognitive, and behavioral skills [33]. Depressed mothers display less attachment, sensitivity, and more severe or disrupted parenting behaviors, which may contribute to negative child outcomes [33]. For example, Lovejoy et al. found that depressed mothers displayed significantly greater negative and disengaged behaviors toward children compared to nondepressed mothers [13].

Upon further analysis, we found a significant gender difference in the mediation effects of adolescents' depressive symptom experience on the pathway from average long-term exposure to maternal depression during early childhood to adolescents' later BMI. Specifically, boys showed a significant direct effect of average long-term exposure to maternal depression on adolescents' BMI z-score at age 15, whereas girls displayed a significant indirect pathway through adolescents' depressive symptom experience as the mediator. That is, our results suggest that the observed indirect effect through adolescents' depressive symptom experience would be significantly varied between boys and girls, hence, moderated mediation by gender. The observed gender variation could be related to the gender gap of depression risk, which is widely reported in the literature. Specifically, it was reported that about twice as many women as men experience depression and several socioeconomic and biological factors may increase a women's risk of depression [31].

In addition, gender variation on negative psychological effects of overweight status has been reported in several prior studies with more pronounced effects

observed in girls than in boys [34–37]. It seems that girls tend to internalize the negative exposure to their mother's depression and their own depression contributes more to their increased BMI. While boys' BMI is negatively affected as a direct result of their mother's depression, possibly due to the less than ideal care they have received. Also, the observed gender variations may also be related to girls experiencing puberty at an earlier age of onset. Also, boys may be gaining weight from the lack of supervision and neglect when mothers are depressed, whereas weight and body image are integrally tied to young women's self-esteem. When girls become depressed, they are more likely than boys to have disordered eating and body weight and image issues. Further research is necessary to investigate and explore these gender differences. Potential gender variations in psychological effects on obesity risks should be considered during the development and implementation of prevention and intervention programs.

The representativeness of the sample is limited by the nature of the study participants being primarily non-Hispanic White, which also limits the generalizability of our research findings. As the analysis sample accounted for 61.9% of the initial cohort in Phase I of the SECCYD, preliminary analyses were conducted comparing general sample characteristics of participants available for analysis with the original full SECCYD sample in order to evaluate potential sampling biases. Our analysis sample was not significantly different from the full SECCYD sample in terms of the major predictor (children's average long-term exposure to maternal depressive symptoms from age of 1 month to Grade 6), the mediator (average scores of children's experience of depressive symptoms), and covariates of gender and ethnicity. However, the analysis sample was significantly different from the full SECCYD sample in terms of participants' levels of income-to-need ratio, mother's education in years, and age at birth. Based on these results, the risk of sampling biases of the analysis sample could be reasonably low, even though we were not able to completely eliminate this risk. Nevertheless, we should still be cautious that the observed findings may not generalize to other groups. As the observed indirect effects were relatively small, we should also be cautious to interpret our results.

Furthermore, our analyses revealed that people who were retained in the study during follow-ups (i.e., the analysis sample) were more educated and less in need. More effective follow-up strategies, such as securing more community support with inclusion of community health workers, adopting monetary rewards as the incentives, and/or logistic assistance or subsidies (e.g., transportation) to boost study retention and follow-up assessment participation, may need to be considered to improve the

completion rate for the vulnerable group who received less education and were high in need.

Conclusion

The observed findings suggested a significant pathway from average long-term exposure to maternal depression during childhood to adolescents' depressive symptom experience, which, in turn, had an impact on adolescents' BMI. This significant pathway was observed in girls, not in boys. Our findings highlight the need to incorporate mental health efforts in pediatric obesity prevention. Maternal depressive symptoms continue to be associated with children's elevated BMI. Potential gender variations in psychological effects on obesity risks should be considered during the development and implementation of intervention. However, we also need to look at paternal mental health and how that relationship can impact their offspring. Fathers also need to address their mental health in order to be better role models and providers for their children. We need to continue addressing these issues so that adolescents can lead an active lifestyle and be emotionally healthy. Future studies are needed to examine the relationships of not only maternal but also parental, mental health, and adolescent BMI because these relationships have an impact on achieving healthy adult lives and minimizing the risk of cardiovascular disease.

Additionally, we also need to consider other sources of influence. For example, children and adolescents' exposure to a myriad of stressors in the family environment can also affect obesity [38–40]. It has been well documented that low socioeconomic status, family financial strains (e.g., high expense levels relative to income, limited assets, and an inability to pay bills or make needed purchases), and impaired family cohesion and lack of support (e.g., parent–child hostility, lack of parental involvement and emotional support, inadequate parental monitoring, and poor parent–child communication), and family disruption (e.g., parental divorce, cohabitation, and stepfamily relationships) are major familial stressors to adolescents [40–43]. Chronic exposure to these stressors in the family environment may result in increased risks of obesity in adolescents [40, 44]. Familial connectedness, parental warmth, expectation, and monitoring were founded in prior cross-sectional studies to be important protective factors by decreasing the frequency of unhealthy behaviors and emotional distress and increasing the presence of healthy behaviors and psychosocial well-being [45]. We call for additional studies in this area to gather evidence so we can develop more targeted prevention and intervention programs to help fight against childhood obesity.

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Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards All authors have indicated that they have no potential conflicts of interest to disclose. The authors have no financial relationships relevant to this article to disclose.

Authors' Contributions All authors were involved in manuscript writing and final approval of submitted versions.

Ethical Approval We presented a poster entitled “Adolescent depression mediates long-term impact of cumulative maternal depression exposure on adolescent’s body mass index” at the Society of Behavioral Medicine Conference 2017, where it won the Meritorious Abstract Award. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards..

Informed Consent Informed consent was obtained from all individual participants included in the study.

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