

Exposure to Parental Depression in Adolescence and Risk for Metabolic Syndrome in Adulthood

Katherine B. Ehrlich
University of Georgia

Edith Chen
Northwestern University

Tianyi Yu
University of Georgia

Gregory E. Miller
Northwestern University

Gene H. Brody
University of Georgia

The psychosocial consequences of living with a depressed parent have been well characterized. Less well known, however, is how this exposure is predictive of later physical health problems. The present study evaluated how parental depression across youths' adolescence (ages 11–18) was associated with youth metabolic syndrome at age 25 ($n = 391$). Youth self-regulation and health behaviors were considered as possible moderators of the link between parental depression and youth metabolic syndrome. Analyses revealed that parental depression in adolescence was associated with a composite score reflecting metabolic syndrome components in early adulthood. Furthermore, self-regulation and health behaviors moderated this link, such that links between parental depression and the metabolic syndrome existed only for youth with low self-regulation or unhealthy behaviors.

Parental depression affects an estimated 10% of mothers and about 7.5 million parents in the United States annually, resulting in about 15.6 million children who live with a depressed parent (England & Sim, 2009; Ertel, Rich-Edwards, & Koenen, 2011). Parental depression has been associated with a wide range of negative outcomes for children across development, including depression and anxiety, substance abuse and dependence, and difficulties with interpersonal functioning (Goodman & Gotlib, 1999; Goodman et al., 2011; Hammen, Brennan, & Keenan-Miller, 2008; Weissman et al., 2016). These children are thought to be at risk in part because of their elevated exposure to stress and chaos in the home, harsh parenting, and a lack of a stable and supportive caregiving environment (Goodman & Gotlib, 1999; Lovejoy, Graczyk, O'Hare, & Neuman, 2000).

Despite our broad understanding of how parental depression contributes to children's mental health and psychosocial functioning across the life span, there has been little research on the possible *physical health* consequences for children of depressed parents. Over the last three decades, however, evidence has accumulated to suggest that exposure to an accumulation of adverse childhood experiences (ACEs; e.g., poverty, maltreatment, mental illness in the family) has a lasting influence on adult physical health, particularly chronic diseases associated with aging, including diabetes, heart disease, arthritis, and some cancers (Felitti et al., 1998; Gluckman & Hanson, 2006; Miller, Chen, & Parker, 2011; Repetti, Taylor, & Seeman, 2002). Much of this research on ACEs takes a cumulative risk approach (for reviews, see Ehrlich, Miller, & Chen, 2016; Sameroff, 2000). Based on the cumulative risk model, various risk factors are distilled into scores that reflect the presence versus absence of risk, and this total risk score is often correlated with physical health.

This research was supported by Award R01HD030588 from the National Institute on Child Health and Human Development and Award P30DA027827 from the National Institute on Drug Abuse.

Correspondence concerning this article should be addressed to Katherine B. Ehrlich, Department of Psychology, University of Georgia, Athens, GA 30602. Electronic mail may be sent to kehrlich@uga.edu.

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DOI: 10.1111/cdev.13003

In the present study, we focused specifically on whether chronic exposure to parental depression across adolescence was predictive of youths' later metabolic syndrome, a cluster of interrelated metabolic abnormalities that includes high blood pressure and blood glucose, lipid dysregulation, and abdominal adiposity. Metabolic syndrome is of particular concern because it forecasts substantially increased risk for cardiovascular disease and diabetes (e.g., Cornier et al., 2008; Sperling et al., 2015). Although most research on psychosocial predictors of cardiometabolic health has focused on the accumulation of risks, there is some evidence to suggest that exposure to parental depression specifically may forecast later health problems for youth. For example, parental depressive symptoms have been shown to be a risk factor for obesity in young children (e.g., Gundersen, Lohman, Garasky, Stewart, & Eisenmann, 2008). Additionally, maternal depressive symptoms during pregnancy have been associated at the bivariate level with young children's blood pressure (van Dijk, van Eijsden, Stronks, Gemke, & Vrijkotte, 2012), although these findings were no longer significant after controlling for a large number of possible confounds (see also van Dijk et al., 2014 for similar evidence in a different sample of children). Among adults with a family history of a depressed parent, Mannie et al. (2013) found elevated levels of blood pressure, arterial stiffness, and decreased insulin sensitivity compared to adults without a family history of depression. The present study extends this research by using prospective measures of exposure to parental depression (rather than retrospective reports) and a measure of metabolic dysregulation in young adulthood (rather than blood pressure readings in healthy children).

Protective and Vulnerability Factors

Although some children who are exposed to parental depression may face increased risk for chronic physical health problems, many children who grow up with a depressed parent will not show evidence of health problems in adulthood. And conversely, some youth may be especially vulnerable to the stress associated with a depressed parent. In the present study, we investigated this possibility that youth may not be equally affected by parental depression by examining two young adulthood characteristics that might serve as protective or vulnerability factors. We focused on two possible moderators—youth self-regulation and unhealthy behaviors—that have been identified as important

factors that shape physical health (e.g., Kershaw, Mezuk, Abdou, Rafferty, & Jackson, 2010; Moffitt et al., 2011). Here, we will describe the ways in which these factors could shape the extent to which parental depression is associated with youth metabolic syndrome.

First, we examined the role of youths' self-regulation in young adulthood as a characteristic that may protect or exacerbate youths' vulnerability to poor physical health in adulthood. Self-regulation reflects individuals' ability to regulate and control their attention, emotions, and behavior (Kochanska, Coy, & Murray, 2001; Mischel, Shoda, & Rodriguez, 1989). Individuals with high self-regulatory capacities demonstrate enhanced abilities to plan for the future and persist in goal-directed behavior, and these skills have been associated with better functioning across development (e.g., Moffitt et al., 2011). Furthermore, some evidence suggests that high self-regulation can protect youth from negative outcomes in the face of adversity. Examples of such resilience have shown that self-regulation can protect youth from the negative psychosocial outcomes often associated with maternal depression and low socioeconomic status (SES; e.g., Buckner, Mezzacappa, & Beardslee, 2009; Silk, Shaw, Forbes, Lane, & Kovacs, 2006). Young adults who are high in self-regulation may engage in planful, health-protective behaviors that aid in stress management and protect against ongoing metabolic dysregulation, such as high blood pressure and elevated blood sugar. In contrast, some researchers have found that *low* self-regulation can be a vulnerability factor for children. For example, Dich, Doan, and Evans (2015) recently found that youth who were high in negative emotionality showed greater allostatic load (an indicator of dysregulation across physiological systems; McEwen & Stellar, 1993) only when they demonstrated low levels of self-regulation. We conducted exploratory analyses to examine the way in which youths' self-regulation served as a protective or vulnerability factor for youth metabolic syndrome in early adulthood.

We also considered the role of young adult health behaviors as a possible moderator of the link between parental depression and youths' metabolic syndrome at age 25. One possibility is that some youth who were exposed to a depressed parent in childhood can compensate in adulthood by striving toward a healthy lifestyle (e.g., eating healthy, exercising, avoiding drugs and alcohol). Consistent engagement in these behaviors may promote health, despite previous exposure to less than ideal family environments. On the other hand, it may be

that poor health behaviors actually accentuate the negative effects hypothesized to be associated with parental depression. Unhealthy diets and sedentary lifestyles are associated with inflammation (e.g., Kiecolt-Glaser et al., 2016; Yudkin, Kumari, Humphries, & Mohamed-Ali, 2000) and are reliable predictors of metabolic syndrome (Park et al., 2003). Some evidence suggests that high-fat diets and stress interact to promote greater inflammation than either factor does on its own (Kiecolt-Glaser, 2010), and similar patterns may emerge in the prediction of metabolic syndrome. Thus, these health-compromising behaviors might exacerbate vulnerabilities associated with parental depression.

The Present Study

We tested a series of research questions in a 14-year prospective study of rural African American youth and their primary caregivers. First, we tested whether parental depression over a 7-year period across youths' adolescence was predictive of youth metabolic syndrome at age 25. Then, we tested a series of research questions concerning possible protective and vulnerability factors. We examined the role of youth self-regulation and youth unhealthy behaviors in adulthood that could moderate the extent to which the experience of parental depression in adolescence was associated with metabolic syndrome in adulthood. We hypothesized that self-regulation would serve as a protective factor for youth; in contrast, we expected that youths' unhealthy behaviors would exacerbate the risk for metabolic syndrome for youth who lived with a depressed parent.

Finally, we conducted exploratory analyses to look at the interaction of parental depression, youth self-regulation, and youth unhealthy behaviors in the prediction of metabolic syndrome to examine the conditions under which youth may be most at risk for physical health problems in adulthood. There are several ways that these factors could interact to predict youth metabolic syndrome. For example, one possibility is that youth with high self-regulatory capacities will have low levels of metabolic syndrome (regardless of their other risk factors) because of the health-protective effects of self-regulation (Moffitt et al., 2011). Another possibility is that these three risk factors have a dose-response effect, wherein individuals who are low in risk across factors will show the lowest levels of metabolic syndrome, followed by youth who have elevated risk on one factor, with the highest levels of risk emerging for youth who were exposed to

high levels of parental depression across adolescence, show poor self-regulatory capacities, and high levels of unhealthy behaviors. Because of the various ways that these factors could interact to predict youth metabolic syndrome, we did not form specific hypotheses about the nature of the interaction.

Method

Participants

The sample for this study was taken from a larger sample of African American target youth and their primary caregivers, who participated in 11 waves of data collection across childhood and into young adulthood; the mean age of the target youth was 11.2 years ($SD = 0.3$) at the 1st assessment and 24.7 years ($SD = 0.7$) at the 11th assessment. At the first assessment (conducted between August 2001 and September 2002), 667 families who resided in nine rural counties in Georgia were selected randomly from lists of fifth-grade students that schools provided (see Brody et al., 2004 for a full description). Although the primary caregivers in the sample worked an average of 39.4 hr per week at the first assessment, 46.3% lived below federal poverty standards. When participants were 19 years old, we followed up with 500 of the original study participants (funding constraints limited our sample size). The age 25 data collection (which took place between November 2014 and December 2015) included 408 participants from the randomly selected 500 sample at age 19 (81.6% agreement rate). Of this subsample, 391 agreed to take part in the blood drawn for assaying metabolic syndrome, which constituted the sample in the present study. Compared to the larger sample, the sample providing biological data at age 25 had higher percentage of female (59.8% vs. 52.8%), their family experienced more SES disadvantage, and youth spent more time living with parents who were clinically depressed (all $ps < .05$).

Data Collection Procedures

All data were collected in participants' homes using a standardized protocol. Two African American field researchers worked separately with the primary caregiver and the target child in each family. Interviews were conducted privately, with no other family members present or able to overhear the conversation. Certified phlebotomists went to each participant's home to draw a blood sample

when the participants were approximately 25 years of age. Written informed consent was obtained at each assessment wave. Each family was paid \$100 after the assessment, and each participant was paid \$160 after the assessment and blood draw at age 25. The University of Georgia's Institutional Review Board reviewed and approved all study procedures.

Measures

Parental Depression

When participants were 11–13 and 16–18 years of age, parents reported their depressive symptoms on the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977), which is widely used with community samples. Primary caregivers rated each of 20 symptoms on the following scale: 0 (*rarely or none of the time*), 1 (*some or little of the time*), 2 (*occasionally or a moderate amount of time*), or 3 (*most or all of the time*). Alphas were ranged from .83 to .91 across six assessment waves. Consistent with psychometric studies of the CES-D, a score of 16 was used as the cutoff to identify clinically significant depression. Presence of clinically significant depression across the six assessment waves was summed to determine the number of years that youth lived with parents who were clinically depressed (hereafter referred to as parental depression; $M = 1.29$, $SD = 1.72$, range from 0 to 6). Almost half (49.6%) of youth lived with a parent with scores lower than 16 at each assessment, 18.9% of youth had a parent with an assessment of 16 or greater at one time point, 10.7% of youth had a parent with two scores of 16 or greater, and the remaining youth (20.7%) lived with a parent who had CES-D scores of 16 or greater at three or more time points.

Youth Self-Regulation

At age 25, youth completed the 23-item Self-Regulation Questionnaire (Brown, Miller, & Lawendowski, 1999). Each item was rated on a Likert-type scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Example items include, "once I have a goal, I can usually plan how to reach it," "I set goals for myself and keep track of my progress," and "if I make a resolution to change something, I pay a lot of attention to how I am doing." All items were summed to yield a self-regulation score. Cronbach's alpha was .92.

Youth Unhealthy Behaviors

Unhealthy behaviors, expressed as an index, were derived from measures of unhealthy diets, lack of physical activities, sleep problems, and substance use. At age 25, youth reported their diet habits and total hours of physical activity in last week on the Adult Health Behavior Questionnaire (Fernald et al., 2008). The diet instrument asked about seven food habits: fast food, fruits/vegetables (reverse scored), sweet drinks, protein (reverse scored), chips/crackers, desserts, and fats eaten during the past week. Items were rated on a Likert-type scale ranging from 1 (*less than one*) to 3 (*four or more*). The responses were summed to form a diet habits score, where higher values represented less healthy diet habits. The physical activity instrument asked about times spent being physically active on three intensity levels (vigorous, moderate, or walking) during the past 7 days. Participants reported days per week and total minutes per day they engaged in physical activity across each of the three levels. Responses were summed to form a physical activity score that reflects total minutes of activity per week, which was then reverse scored. Youth reported their sleep problems during the past month on Medical Outcome Study Sleep scale (MOS, Hays & Stewart, 1992). The Sleep Problem Index includes nine items to compute an overall sleep problem summary. This index contains questions about sleep disturbance (the ability to fall asleep and maintain restful sleep), sleep adequacy (the sufficiency of sleep in terms of sleeping enough to provide restoration of wakefulness, reverse scored), and somnolence (daytime drowsiness or sleepiness). Cronbach's alpha was .75. Youth reported their past month cigarette, alcohol, and marijuana use and their excessive drinking on a widely used instrument from the Monitoring the Future Study (Johnston, O'Malley, Bachman, & Schulenberg, 2007). Responses to these four items were summed to form a substance use composite, a procedure that is consistent with our own and others' prior research (Brody & Ge, 2001; Newcomb & Bentler, 1988). The unhealthy behaviors index was calculated by summing the number of unhealthy behavior indicators (unhealthy diets, lack of physical activities, sleep problems, and substance use) on which each youth scored in the top quartile of risk; possible scores ranged from 0 to 4 ($M = 0.93$, $SD = 0.89$). In this sample, 37.9% reported no behaviors in the top quartile, 37.1% engaged in one unhealthy behavior, 19.7% were in the top quartile

for two behaviors, and the remaining participants (5.4%) reported unhealthy behaviors in three or four categories.

Youth Metabolic Syndrome

When each participant was age 25 years, a certified phlebotomist went to the participant's home to draw a fasting blood sample. Participants were asked not to eat or drink after midnight prior to the blood draw. Blood was drawn into Serum Separator Tubes (Becton-Dickinson, Franklin Lakes, NJ), and centrifuged on site according to the manufacturer's instructions. Serum was harvested and frozen immediately on dry ice. At the end of the study, glucose was measured photometrically using a UV test on a Roche/Hitachi cobas c502 analyzer (Roche Diagnostics, Indianapolis, IN). The average intra- and interassay coefficients of variation were 0.7% and 1.8%, respectively. This assay has a dynamic range of 2–750 mg/dl. High-density lipoproteins (HDL) and triglycerides were measured on a Roche/Hitachi cobas c701 analyzer. The average intra- and interassay coefficients of variation for these assays were below 1.6% and 2.4%, respectively. The assay's detection ranges are 8.85–885 mg/dl (triglycerides) and 3–120 mg/dl (HDL). Resting blood pressure was monitored with a Critikon Dinamap Pro 100 (Critikon, Tampa, FL) while the youth sat reading quietly. Three readings were taken every 2 min, and the average of the last two readings was used as the resting index. This procedure yields highly reliable indices of chronic resting blood pressure (Kamarck et al., 1992). The field researcher recorded participants' waist circumferences.

The presence of adult metabolic syndrome was defined by the International Diabetes Federation guidelines (Cornier et al., 2008). To qualify, an individual must show central adiposity, defined by ethnic and sex-specific cutoffs for waist circumference (for individuals of African descent, cutoffs are ≥ 94 and ≥ 80 cm for men and women, respectively). At least two of four additional components must also be present. They include (a) high blood pressure (systolic pressure ≥ 130 or diastolic pressure ≥ 85), (b) raised triglyceride levels (≥ 150 mg/dl), (c) raised fasting glucose levels (≥ 100 mg/dl), and (d) low HDL levels (< 40 mg/dl in men and < 50 mg/dl in women). For analyses, we constructed two outcome variables. One was a binary variable reflecting whether the participant met the International Diabetes Federation case definition for metabolic

syndrome ($n = 67$, 17.1%). The other was a weighted metabolic syndrome composite score based on the factor analysis of six components of metabolic syndrome (see Pladevall et al., 2006): central adiposity, defined by waist circumference; systolic and diastolic blood pressure; fasting blood triglyceride; fasting blood glucose; and fasting blood HDL. Scores for the standardized composite ranged from -2.60 to 4.19 .

Youth Life Stress

To account for factors that could provide plausible rival explanations, the analyses also controlled for life stress at age 25. Youth assessed their life stress using a checklist of 12 events (e.g., acute economic stress, death of a friend, parental divorce, serious injury or illness; Brody, Chen, & Kogan, 2010), indicating whether each event had occurred during the previous 6 months. The number of endorsed items was summed ($M = 1.14$, $SD = 1.54$). Less than half (42.5%) of the sample experienced zero stressful life events in the past 6 months, 29.9% experienced one stressful life event, 13.6% experienced two stressful events, and the remaining youth (14.1%) experienced three or more stressful life events.

Family Socioeconomic Disadvantage

When participants were 11–13 and 16–18 years of age, caregivers provided data on their families' SES. Six dichotomous variables formed a socioeconomic disadvantage index. A score of 1 was assigned to each of the following: family poverty based on federal guidelines, primary caregiver unemployment, receipt of Temporary Assistance for Needy Families, primary caregiver single parenthood, primary caregiver education level less than high school graduation, and caregiver-reported inadequacy of family income. The scores were summed to form the index at each wave and were averaged across the six assessment waves. The resulting index ranged from 0 to 6 ($M = 2.33$, $SD = 1.20$).

Youth Depression

As a final control for plausible alternative explanations, we measured youth depressive symptoms at age 25. Youth completed the CES-D, as described earlier for parental depression ($\alpha = .84$). Scores ranged from 0 to 20 ($M = 3.08$, $SD = 3.50$).

Results

Parental Depression and Youth Metabolic Syndrome

Table 1 presents descriptive statistics for the sample, along with bivariate correlations. Our initial analysis was designed to determine whether parents' depressive symptoms across youths' adolescence was associated with youth metabolic syndrome at age 25. Parental depression across ages 11–18 was correlated with higher metabolic syndrome composite at age 25, but this effect was no longer significant after controlling for possible confounds (i.e., gender, youth life stress, socioeconomic disadvantage, and youth depressive symptoms). At age 25, men had a higher metabolic syndrome composite than women. Women, however, were more likely to have diagnosable metabolic syndrome than men. (This difference is due to the fact that women had greater central adiposity than men, which is a requirement for metabolic syndrome diagnosis.) Family SES disadvantage assessed at ages 11–18 was associated positively with both metabolic syndrome diagnostic status and metabolic syndrome composite at age 25. High self-regulation was associated with low probabilities of having

diagnosable metabolic syndrome, even after controlling for possible confounds.

Parental Depression, Self-Regulation, and Metabolic Syndrome

This analysis evaluated whether parental depression across ages 11–18 interacts with youth self-regulation at age 25 to forecast youth metabolic syndrome at age 25. A logistic regression model (for metabolic syndrome diagnosis) and a linear regression model (for metabolic syndrome composite) were executed to test the study hypothesis. Both models included main effects of parental depression and youth self-regulation, and a product term representing the interaction of the two variables. All interaction analyses were conducted based on the conventions that Aiken and West (1991) prescribed, whereby the variables are first mean centered and interactions are calculated as the product of the centered variables. Gender, family SES disadvantage, youth life stress, and youth depressive symptoms were included as covariates. The results (see Table 2) of the logistic regression model predicting metabolic syndrome diagnosis did

Table 1
Descriptive Statistics and Correlations Among Study Variables (N = 391)

Variable	M (SD) or n (%)	Correlations								
		1	2	3	4	5	6	7	8	9
1. Gender (male = 1, female = 0)	157 male (40.2%)	—								
2. Family SES disadvantage (ages 11–18)	2.335 (1.196)	-.021	—							
3. Life stress (age 25)	1.138 (1.536)	-.034	.090	—						
4. Youths' depression (age 25)	12.338 (7.820)	-.049	.088	.207***	—					
5. Parental depression (ages 11–18)	1.286 (1.723)	-.030	.395***	.054	.123*	—				
6. Self-regulation (age 25)	56.205 (7.790)	-.041	-.089	-.157**	-.482***	-.011	—			
7. Unhealthy behaviors (age 25)	0.928 (0.894)	.037	.033	.147**	.330***	.005	-.274***	—		
8. Metabolic syndrome composite (age 25)	0 (1.000)	.134**	.176***	-.024	.007	.128*	-.086	.059	—	
9. Metabolic syndrome diagnosis (age 25)	67 (17.1%)	-.123*	.128*	.016	-.002	.084	-.107*	.093	.506***	—

Note. Correlations with gender and metabolic syndrome diagnosis are Spearman's correlation; others are Pearson's correlations. SES = socioeconomic status.
*p < .05. **p < .01, ***p < .001.

not reveal a significant interaction between parental depression and youth self-regulation. The results of the linear regression model predicting metabolic syndrome composite, however, revealed a significant interaction, $F(1, 383) = 6.210, p = .013, \Delta R^2 = .015$. To interpret this interactive effect, we plotted the estimated levels of youth metabolic syndrome composite by number of years living with a depressed parent at low (1 *SD* below the mean), medium (at the mean), and high (1 *SD* above the mean) levels of youth self-regulation. As shown in Figure 1, exposure to parental depression across ages 11–18 was associated with youth metabolic syndrome composite at age 25 among youth with low self-regulation (simple slope = 0.129, $SE = .046, p = .005$). Parental depression was not associated with youth metabolic syndrome composite among youth with medium (simple slope = 0.048, $SE = .031, p = .128$) or high self-regulation (simple slope = $-0.033, SE = .044, p = .453$). Furthermore, using the Johnson–Neyman technique (which identifies meaningful values of the moderator for which the simple slope of the regression line is significant; Preacher, Curran, & Bauer, 2006), we found that parental depression was associated with youth metabolic syndrome when their self-regulation values were 0.19 *SD* or lower than the mean.

Parental Depression, Health Behaviors, and Metabolic Syndrome

The analysis examined whether parental depression across ages 11–18 interacted with youth health behaviors at age 25 to forecast youth metabolic syndrome at age 25. Analyses revealed a significant interaction between parental depression and youth unhealthy behaviors in predicting the metabolic syndrome composite, $F(1, 383) = 5.738, p = .017, \Delta R^2 = .014$, after controlling for gender, family SES disadvantage, youth life stress, and youth depressive symptoms (see Table 3). We plotted the estimated levels of youth metabolic syndrome composite by number of years living with a depressed parent at 0, 1, and 2 unhealthy behaviors. As shown in Figure 2, more exposure to parental depression across ages 11–18 was associated with youth metabolic syndrome composite at age 25, but only among youth with two or more unhealthy behaviors (simple slope = 0.131, $SE = .049, p = .007$). Parental depression was not associated with youth metabolic syndrome composite among youth without unhealthy behaviors (simple slope = 0.028, $SE = .043, p = .518$) or with only one unhealthy behavior (simple slope = 0.052, $SE = .032, p = .102$). Similarly, analyzing the

Table 2
Parental Depression and Self-Regulation as Predictors of Metabolic Syndrome

	Metabolic syndrome composite (age 25)					
	B	SE	β	B	SE	β
1. Gender, male	.250	.101	.123*	.249	.101	.122*
2. Family SES disadvantage (ages 11–18)	.123	.045	.148**	.127	.045	.152**
3. Life stress (age 25)	-.033	.033	-.051	-.032	.033	-.049
4. Youths' depression (age 25)	-.006	.007	-.043	-.006	.007	-.045
5. Parental depression (ages 11–18)	.045	.032	.077	.048	.031	.082
6. Self-regulation (age 25)	-.012	.007	-.096	-.013	.007	-.105
7. Parental Depression \times Self-Regulation	—	—	—	-.010	.004	-.123*

	Metabolic syndrome diagnosis (age 25)					
	B	SE	Exp (B)	B	SE	Exp (B)
1. Gender, male	-.785	.306	0.456*	-.798	.308	0.450**
2. Family SES disadvantage (ages 11–18)	.232	.127	1.261	.236	.126	1.266
3. Life stress (age 25)	-.083	.102	0.920	-.084	.104	0.919
4. Youths' depression (age 25)	-.030	.021	0.971	-.030	.022	0.970
5. Parental depression (ages 11–18)	.101	.082	1.106	.091	.083	1.096
6. Self-regulation (age 25)	-.053	.021	0.948*	-.052	.021	0.949*
7. Parental Depression \times Self-Regulation	—	—	—	-.013	.012	0.987

Note. B = unstandardized regression coefficient; SE = standard error.
* $p < .05$. ** $p < .01$.

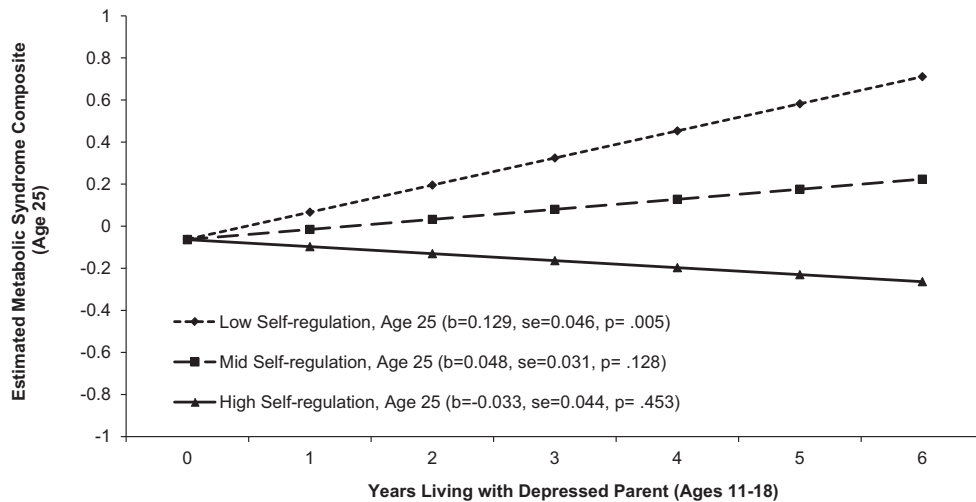


Figure 1. Young adults' metabolic syndrome composite at age 25 as a function of exposure to parental depression at ages 11–18 and youth self-regulation at age 25 (low = 1 *SD* below the mean; mid = mean; high = 1 *SD* above the mean). The lines represent the results of regression analyses at low, mid, and high levels of youth self-regulation, and the numbers in parentheses refer to the simple slopes.

significant interaction term with the Johnson–Neyman technique revealed that the association between parental depression and youth metabolic syndrome was significant when unhealthy behaviors were greater than 1.15 *SD* from the mean (which corresponds to two or more unhealthy behaviors). The logistic regression model predicting metabolic syndrome diagnosis did not reveal a significant interaction between parental depression and youths' unhealthy behaviors, however (see Table 3).

Parental Depression, Self-Regulation, and Health Behaviors, and Metabolic Syndrome

Finally, we conducted an exploratory test to examine the three-way interaction of parental depression, youth self-regulation, and youth health behaviors in the prediction of metabolic syndrome diagnosis and the composite score (see Table 4). Analyses also controlled for possible confounds. Analyses were nonsignificant in the model predicting youth metabolic syndrome diagnosis, but in contrast, a three-way interaction was significant in predicting the metabolic syndrome composite, $F(1, 379) = 9.055, p = .003, \Delta R^2 = .021$. We plotted the estimated levels of youth metabolic syndrome composite scores at low (1 *SD* below the mean) and high (1 *SD* above the mean) levels of parental depression and youth self-regulation for youth without unhealthy behaviors and those with two or more unhealthy behaviors. As shown in Figure 3, more exposure to parental depression across ages

11–18 was associated with youth metabolic syndrome composite at age 25 among youth with low self-regulation and two or more unhealthy behaviors (simple slope = 0.300, $SE = .064, p < .001$). Of particular importance was the finding that, during adolescence, youth who spent more time living with depressed parents did not evince high levels of metabolic syndrome composite during young adulthood if they demonstrated high levels of self-regulation or avoided unhealthy behaviors.

Discussion

This study presents the first prospective evidence showing that exposure to parental depression across adolescence, when coupled with low self-regulation and high levels of unhealthy behaviors, is associated with components of the metabolic syndrome in adulthood. To the extent that African American youth had relatively low self-regulation or high levels of unhealthy behaviors in adulthood, more exposure to parental depression was associated with higher scores on a composite measure of the metabolic syndrome. However, for youth with average or high levels of self-regulation and who refrained from unhealthy lifestyle behaviors (e.g., poor diet, lack of exercise) in adulthood, exposure to parental depression was not associated with the metabolic syndrome composite score at age 25.

These findings offer a promising outlook on the extent to which exposure to parental depression forecasts negative health outcomes for young

Table 3
Parental Depression and Unhealthy Behaviors as Predictors of Metabolic Syndrome

	Metabolic syndrome composite (age 25)					
	B	SE	β	B	SE	β
1. Gender, male	.256	.102	.126*	.264	.101	.130**
2. Family SES disadvantage (ages 11–18)	.129	.045	.154**	.131	.045	.157**
3. Life stress (age 25)	-.033	.033	-.051	-.035	.033	-.054
4. Youths' depression (age 25)	-.002	.007	-.017	-.001	.007	-.009
5. Parental depression (ages 11–18)	.042	.031	.072	.046	.031	.079
6. Unhealthy behaviors (age 25)	.069	.059	.062	.067	.059	.060
7. Parental Depression × Unhealthy Behaviors	—	—	—	.079	.033	.119*

	Metabolic syndrome diagnosis (age 25)					
	B	SE	Exp (B)	B	SE	Exp (B)
1. Gender, male	-.757	.304	0.469*	-.754	.304	0.470*
2. Family SES disadvantage (ages 11–18)	.254	.127	1.289*	.255	.127	1.290*
3. Life stress (age 25)	-.085	.098	0.918	-.086	.098	0.918
4. Youths' depression (age 25)	-.015	.019	0.986	-.014	.019	0.986
5. Parental depression (ages 11–18)	.085	.081	1.088	.084	.081	1.087
6. Unhealthy behaviors (age 25)	.316	.158	1.372*	.311	.160	1.364
7. Parental Depression × Unhealthy Behaviors	—	—	—	.019	.083	1.019

Note. B = unstandardized regression coefficient; SE = standard error.
*p < .05. **p < .01.

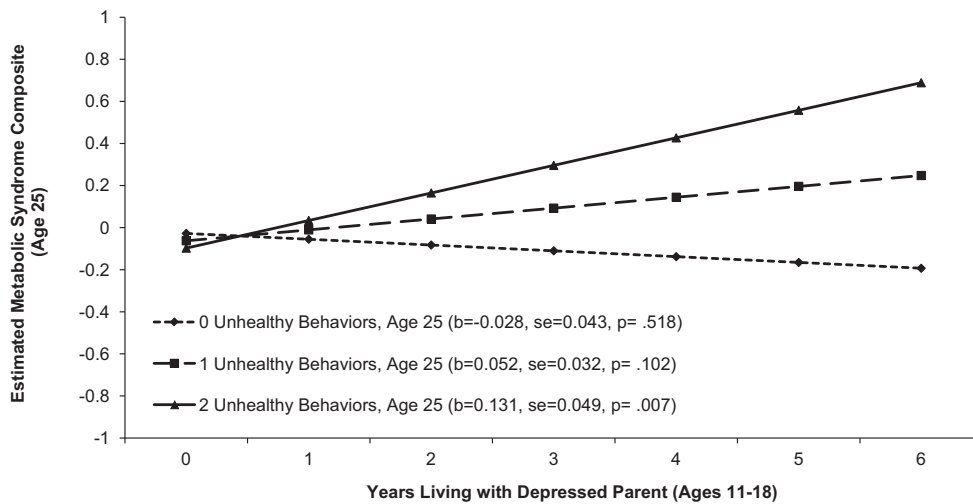


Figure 2. Young adults' metabolic syndrome composite at age 25 as a function of exposure to parental depression at ages 11–18 and youth unhealthy behaviors at age 25. The lines represent the results of regression analyses at none unhealthy behavior, one unhealthy behavior, and two unhealthy behaviors, and the numbers in parentheses refer to the simple slopes.

adults. One reason for this optimism is that self-regulation and health behaviors are malleable. Although these characteristics often take shape in childhood and adolescence (Umberson, Crosnoe, & Reczek, 2010), individuals can work to promote healthy lifestyles, by avoiding excessive alcohol use,

smoking, and poor diets, and adding exercise and good sleep habits into their daily routines. Self-regulatory capacities continue to develop across adolescence and early adulthood (Steinberg et al., 2009), suggesting that planning and self-control behaviors can also change over time. Moreover,

Table 4
Parental Depression, Self-Regulation, and Unhealthy Behaviors as Predictors of Metabolic Syndrome

	Metabolic syndrome (age 25)					
	Diagnosis			Composite		
	B	SE	Exp (B)	B	SE	β
1. Gender, male	-.864	.314	0.422**	.235	.099	.115*
2. Family SES disadvantage (ages 11–18)	.240	.129	1.271	.124	.044	.148**
3. Life stress (age 25)	-.083	.103	0.920	-.029	.032	-.045
4. Youths' depression (age 25)	-.036	.022	0.965	-.006	.007	-.045
5. Parental depression (ages 11–18)	.086	.086	1.090	.042	.031	.072
6. Self-regulation (age 25)	-.045	.021	0.956*	-.012	.007	-.094
7. Unhealthy behaviors (age 25)	.223	.171	1.250	.039	.058	.035
8. Parental Depression × Self-Regulation	-.016	.012	0.984	-.010	.004	-.124*
9. Parental Depression × Unhealthy Behaviors	-.017	.095	0.983	.062	.033	.092
10. Self-Regulation × Unhealthy Behaviors	-.027	.020	0.974	-.021	.007	-.149**
11. Parental Depression × Self-Regulation × Unhealthy Behaviors	-.011	.013	0.989	-.013	.004	-.148**

Note. B = unstandardized regression coefficient; SE = standard error; SES = socioeconomic status.
p* < .05. *p* < .01.

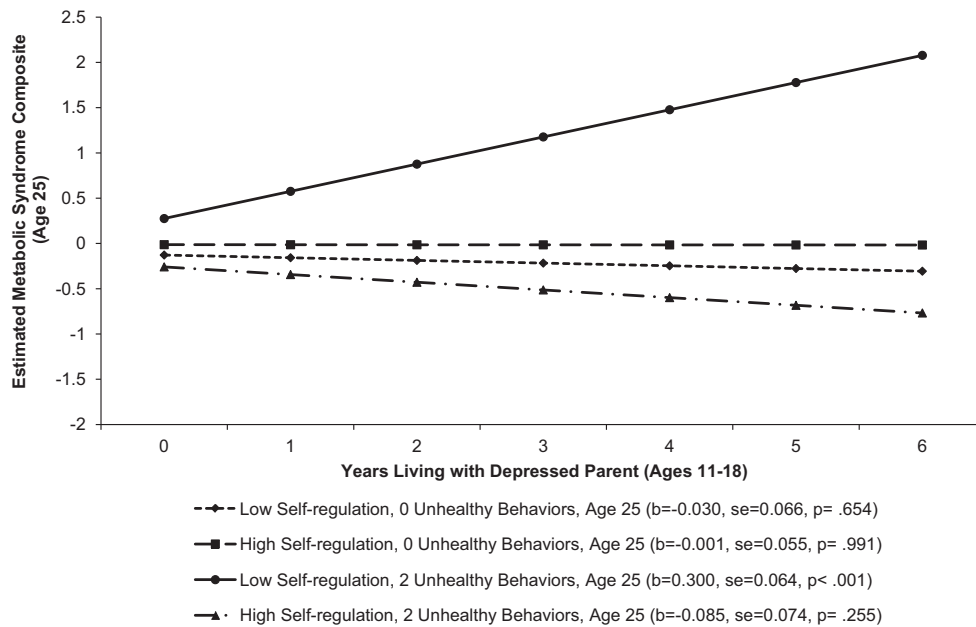


Figure 3. Young adults' metabolic syndrome composite at age 25 as a function of exposure to parental depression at ages 11–8 and youth self-regulation and unhealthy behaviors at age 25. The lines represent the results of regression analyses for different levels of self-regulation (low: 1 SD below the mean; high: 1 SD above the mean) and unhealthy behaviors (none vs. 2). Numbers in parentheses refer to simple slopes.

these young adult behaviors were unrelated to parental depression in the present study (see Table 1), which suggests that parental depression is not prescriptive of unhealthy behaviors that ultimately lead to metabolic dysregulation. Thus, for young adults who were exposed to a depressed parent in adolescence, efforts to promote self-regulation and

healthy behaviors (which were modestly correlated in this sample) may be particularly important.

This study adds to the growing evidence that exposure to various stressful experiences within the family in childhood and adolescence may heighten risk for physical health problems in adulthood (e.g., Felitti et al., 1998; Repetti et al., 2002). One

intriguing question that these findings raise is: To what extent could therapeutic interventions to improve parents' mental health also improve youths' physical health? Recent findings (Miller, Brody, Yu, & Chen, 2014) showed that, relative to controls, youth who participated in a psychosocial intervention with their parents had lower levels of inflammation 8 years later, and some of these reductions in inflammation were associated with improvements in parenting. Our findings suggest that some youth (i.e., those with low self-regulation and high levels of unhealthy behaviors) had greater metabolic syndrome composite scores when their parents' reported high levels of depressive symptoms across adolescence. Might these vulnerable youth benefit from improvements in parents' mental health? Furthermore, do youth benefit from parents' participation in mental health interventions even if parents continue to experience residual symptoms of depression, or do youth only benefit when parents experience long-lasting reductions in their symptoms? These important questions should be addressed in future research.

Of note is that while parental depression was associated with a continuous measure that reflects the components of metabolic syndrome, parental depression did not predict metabolic syndrome diagnosis. There have been differing opinions about the optimal way to define metabolic syndrome, and several medical organizations have released variations in the criteria required for a diagnosis (e.g., Grundy et al., 2004). Although these definitions capture similar risk factors that are viewed as evidence of metabolic dysregulation (e.g., heightened blood pressure, lipids, central adiposity), they differ in the relative weight placed on individual risk factors and the thresholds required for what is considered high risk. For example, we implemented the International Diabetes Federation guidelines for diagnosis, which require elevated central adiposity to receive a diagnosis, regardless of whether individuals are high on other indicators. One advantage of using a continuous measure of metabolic syndrome in analyses is that it reflects the fact that scores just below the cutoff ranges on individual risk factors may still confer some risk for chronic disease. These subclinical levels of metabolic risk may be medically relevant, especially if attempts to improve health are not pursued. Furthermore, from a statistical perspective, there are often costs to dichotomizing continuous variables (e.g., Cohen, 1983), and the use of a binary measure may dampen power to detect effects. (Indeed, effect sizes were comparable across

models.) Nevertheless, we are careful to note that parental depression did not predict metabolic syndrome diagnosis. Rates of metabolic syndrome are on the rise for both adolescents and adults (Duncan, Li, & Zhou, 2004; Ford, Giles, & Mokdad, 2004), so efforts to understand risk and protective factors are especially timely.

Several study limitations should be considered. First, we relied on self-reports of unhealthy behaviors, and future studies should consider incorporating additional tools to assess these activities. For example, the use of actigraphy may better capture physical activity and sleep, and daily diaries may provide more accurate information about diet compared to a self-report questionnaire. Similarly, our measures of stressful life events and self-regulation relied on youth reports, and future studies could augment this approach with interviews, behavioral tasks, or additional informants who could provide unique information.

In addition, we assessed exposure to parental depression across adolescence, but it is possible that exposure to parental depression in early childhood (or even prenatally) could have an effect on youth physical health in early adulthood. Future studies that assess parental depression across infancy, childhood, and adolescence could test hypotheses about whether there are particular windows across development in which youth are especially vulnerable to parental depression. In addition, our study relied on parental self-reports of depressive symptoms, and clinical interviews of depression might yield additional insight into parents' symptomatology.

Our sample included assessments of depression from only the primary caregiver, so we are unable to assess how exposure to parental depression across multiple caregivers relates to youth health. Of note, however, is that almost 60% of the families in our sample were single-parent families at baseline, so a different sample may be required to test hypotheses about how maternal and paternal depression may differentially predict youth metabolic dysregulation. These studies could help shed light on whether access to a nondepressed parent can protect youth against negative outcomes associated with exposure to a depressed parent, or whether youth who have two depressed parents show more health problems compared to youth with only one depressed parent. Similar questions remain about whether the presence of a depressed sibling or other family member living in the home represents a risk factor for metabolic dysregulation.

Additionally, because our study was initially designed to examine socioeconomic disadvantage and substance use outcomes in adolescents, we did not measure physical health in the early waves of this study, which limits our ability to examine change in metabolic dysregulation. Given that our study utilized correlational data, however, we are unable to make claims about the temporal order or causal status of the associations between parental depression and youths' health.

We note that the effects observed in the present study were small, so it is likely the case that additional protective factors exist that may shield youth from negative health outcomes associated with parental depression. Researchers who are exploring the consequences of parental depression should consider additional protective factors, such as facets of youths' temperament or environmental and contextual factors (e.g., strong community support). These efforts will help inform the development of interventions and prevention programs. In addition to considering other protective factors, it is important to investigate factors that may increase youth risk for metabolic syndrome. We focused on parental depression in the present study, but there may be other parental mental health problems (e.g., parent substance abuse, anxiety) that are predictive of youth physical health as well.

Finally, our study focused on youth from rural African American families, many of whom were living with considerable socioeconomic disadvantage and chronic stress. One question that remains is whether similar effects would emerge in samples with other sociodemographic groups (e.g., in samples with other racial/ethnic groups, or in middle class samples). Given the unique characteristics of this sample, we are hesitant to speculate about the generalizability of these findings, but we encourage researchers with access to ongoing longitudinal samples to consider these questions in their research.

In summary, our findings suggest that youths' exposure to parental depression across adolescence is associated with a composite of metabolic syndrome in adulthood, but this association was disrupted when youth possess high levels of self-regulatory skills or low levels of unhealthy lifestyle behaviors. Efforts to understand the mechanisms through which these processes unfold across development will be important to consider in future research. Some possible mechanisms, such as harsh parenting, parent-adolescent conflict, or family chaos, may serve as important points of intervention in families with a depressed parent.

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