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Depressive symptoms during childhood and cardiovascular risk factors in black and white men

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Abstract

Objective: Depressive symptoms and major depression predict cardiovascular disease (CVD) and CVD risk factors in adulthood. Evidence regarding the role of depression in the development of CVD risk in youth is minimal. The study evaluated the prospective relationship of depressive symptoms in childhood and adolescence with adult CVD risk factors in black and white men.

Methods: Health behaviors and medical history were measured in 165 black and 146 white men (mean age = 32); a subset in the Pittsburgh area had a fasting blood draw to measure metabolic syndrome and inflammation. Adult CVD risk factors were related to depressive symptoms and childhood socioeconomic status (SES) prospectively measured annually from ages 7 to 16 years, followed by adjustments for adult SES and depressive symptoms.

Results: Men with higher depressive symptoms ages 7 to 16 smoked more cigarettes, $B = 0.28$ (standard error, 0.12), $p = .015$, and ate fewer servings of fruits and vegetables, $B = -0.08$ (0.04), $p = .040$, as adults. The association for smoking was independent of adult depressive symptoms (concurrent) and childhood and adult SES as well as race. Depressive symptoms during childhood were unrelated to the metabolic syndrome or biomarkers of inflammation in adulthood.

Conclusions: Depressive symptoms in childhood may predict later adverse health behaviors in black and white men. No evidence was found for an association between childhood depressive symptoms with metabolic syndrome or inflammation markers at ages around 32 years. The nature of the sample and lack of measurement of depressive disorder diagnosis tempers the conclusions, and future research is needed to determine associations with biological measures at later life span phases.

Keywords

Depression; race; men; cigarette smoking; health behaviors; metabolic syndrome; inflammation; longitudinal

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INTRODUCTION

In 2015, American Heart Association commissioned an overview of the evidence that depressive symptoms, major depression, and bipolar disorder are associated with risk for premature atherosclerosis and risk factors for cardiovascular disease (CVD) in young adults (1). Cited in the review, several population-based studies showed those with a history of major depression and bipolar depression had higher CVD mortality rates and excess CVD prevalence (2, 3). On the other hand, in small samples, cross-sectional associations between depressive disorder and endothelial dysfunction were inconsistent (e.g., (4–6)), and in the Young Finns Study no prospective relationship between depressive symptoms and subsequent carotid intima medial thickness was observed (7). In general, the commission concluded that the evidence was encouraging but mixed, and future studies with larger sample sizes and adequate assessment of mood symptoms and disorders are needed.

Another approach to understanding the impact of depression or depressive symptoms on the developing individual is to investigate associations with cardiovascular risk factors. Most studies are on middle-aged and older adults and do find evidence that those with depression or elevated depressive symptoms are more likely to have the metabolic syndrome, obesity, elevated inflammatory biomarkers, and poor health behaviors (1, 8). The fewer studies available for youth do not yield a consistent picture. On the one hand, a review of 16 studies concluded that adolescents with high depressive symptoms are more likely to become overweight adults than are less depressed adolescents (9). Hungarian children with diagnosed major depression smoked more and were less active than their nondepressed siblings, albeit were similar in weight (10). Boys who had higher depressive symptom scores had greater insulin resistance in several studies (11, 12). On the other hand, in several large studies, only girls (not boys) with high depressive symptoms had greater insulin resistance and risk for Type II diabetes as adults (13, 14) and one study reported that adolescent boys higher on depressive-anxious symptoms had lower blood pressure (14).

Most studies are based on a single or onetime assessment of depressive symptoms. However, some evidence in adults suggests that repeated assessments of depressive symptoms or disorder are more predictive of CVD risk than one-time assessments. For example, recurrent major depression and high depressive symptoms on several occasions relate to prevalence of coronary calcification in middle-aged men and women (15), as well as to progression of coronary calcification in healthy women (16, 17). For studies of children, it may be particularly important to have a comprehensive assessment of depressive symptoms over key development periods, i.e., childhood and early and late adolescence, when substantial changes are occurring in emotional, physical, and social demands. In this way, one could ask whether overall depressive symptoms in youth over time or within key developmental stages are critical regarding when depressive symptoms may have a negative impact on CVD risk. Consistent with this notion are the results of a prospective study that found depressive disorder diagnosed in childhood (as opposed to adolescence or adulthood) predicted CVD events, partially due to the longer duration of exposure to depression across the study period (18).

The objectives of the present report are 3-fold: (a) to examine the relationship between repeated annual assessments of depressive symptoms from ages 7 to 16 with key cardiovascular risk factors, namely life style and biological risk factors for CVD, in adulthood; (b) to test whether obtained associations are due to early depressive symptoms predicting later depressive symptoms in adulthood, which confers its impact on CVD risk factors, or are obtained independent of depressive symptoms in adulthood; and (c) to explore the influence of race and childhood and adult socioeconomic status (SES) on the pattern of associations. To meet these objectives, we used data from a school-based sample of black and white men who had participated in the parent study starting in the first grade. We anticipated that men with elevated depressive symptom scores in childhood and adolescence would have elevated risk CVD risk factors and that the relationships would be independent of SES and adult depressive symptoms.

METHODS

Participants

Participants were recruited from the youngest cohort of the Pittsburgh Youth Study (PYS; (19)), a longitudinal study of 503 boys initially recruited from Pittsburgh Public Schools in 1987–1988 when they were in the first grade. Eight hundred forty-nine boys were randomly chosen to undergo a multi-informant (i.e., parent, teacher, child report) screening that assessed early conduct problems, with half the sample recruited from the top 30% of the screening measure, and the rest randomly selected from the remainder for a total of 503 boys in the longitudinal sample, hereafter called risk screening group. The boys' mean age at screening was 6.9, and racial composition was predominately White (40.6%) and Black (55.7%). Nearly all primary caregivers were biological mothers (92%), with 45.3% cohabiting with a partner and 16.9% completing less than 12 years of schooling. Over half of families (61.3%) were receiving public financial assistance (e.g., food stamps).

In adulthood (mean age = 32 years; range 30–34 years), we contacted eligible PYS men to participate in a study examining early developmental factors associated with risk for CVD. Exclusionary criteria were death (N=18), prior withdrawal from PYS (N=44), severe mental disability (N=4), and current incarceration (N=42). Of the remaining 395 men, 312 (79%) agreed to participate in some or all the protocol. Among those eligible but who did not participate, 22 declined participation; 19 failed to respond to messages or missed appointments; and 42 could not be located. Of the 312 men, 266 participated in the laboratory protocol as described below. The current sample of 312 did not differ from the original sample of 503 in terms of race, $p = .084$, or risk screening group, childhood SES (see definition below), and number of health problems in childhood, all ps > .250. One person was missing PYS data because the caregiver withdrew him from the study, but he rejoined after he became of majority age. Thus, the present analysis was based on a total possible of 311 men.

Overview of Protocol

Participants who lived within driving distance of Pittsburgh or planned to return for a visit were scheduled for a laboratory assessment. Upon arrival at the laboratory after an 8 hour

fast, they completed consent forms, had anthropometrics measured and a blood draw. After a light meal and resting for 10 minutes, they performed a series of challenging tasks while cardiovascular measures were taken. Finally, they were interviewed regarding sociodemographic characteristics, health history, and health behaviors, and completed questionnaires regarding stress and personal characteristics. After the laboratory portion, they wore an actigraph and completed daily diaries for a week to assess sleep. If participants did not live near Pittsburgh, plan to visit to Pittsburgh if they lived out of town, or were unwilling to complete the laboratory portion, they completed telephone interviews and online questionnaires. The study procedures were approved by the Institutional Review Board at the University of Pittsburgh.

Measures

Family socioeconomic status.—Each year (from 1988 until 1996), the caregiver completed a family information form to ascertain the educational attainment and current occupation of the parent(s) living in the home. These were coded into the Hollingshead occupational status and educational categories and summed after weighting the occupation relatively more (20). The same data manager coded occupations over all years; new occupations not on the original list were categorized by the data manager following group meetings to develop consensus on appropriate categories. For those currently unemployed, the job code from the prior annual visit was used. If the child was from a 2-parent family, the higher Hollingshead score was used. Higher scores indicate higher SES.

In adulthood, men were asked for their educational attainment and current occupation and coded into the same Hollingshead Index scores. If they were currently unemployed but receiving unemployment compensation, the job code was based on the prior PYS visit (about 3 years earlier). For those currently unemployed but not receiving unemployment compensation, the job code was assigned the lowest occupation if they were on other forms of public assistance or their income was based on illegal activities.

Depressive symptoms.—Depressed mood was assessed by the 13 item Recent Mood and Feelings Questionnaire (short form) completed by the boys at each annual assessment between the ages of 7 and 16 years (21). The items were as follows: you felt miserable or unhappy; did not enjoy anything at all; felt so tired that you just sat around and did nothing; were very restless; felt that you were no good anymore; cried a lot; found it hard to think properly; hated yourself; felt that you were a bad person; felt lonely; thought that nobody really loved you; thought that you could never be as good as others; felt that you did everything wrong. Items were rated on a 3-point scale, 0 = not true, 1 = sometimes true, and 2 true. The scale is correlated substantially with the scores from the Children's Depression Inventory and the Diagnostic Interview Schedule for Children depression scale and discriminated clinically referred psychiatric patients from pediatric controls (21). Confirmatory factor analysis shows a unidimensional factor structure across grades 1 – 10; factor loadings increased with age, suggesting the depressive symptom construct was measured with less error as the children matured (22). Of the 311 boys who participated as adults, between 290 and 309 boys completed the depressive symptom measures at each of the 10 annual assessments. For descriptive purposes only in Table 1, scores were averaged

across the entire time period; the Cronbach's alpha coefficient of .72. Higher the scores the greater the depressive symptoms.

The same measure was administered to the men to maintain comparability of assessment. An adult form is available and is almost the exact same wording as the child version. Cronbach's alpha was .88.

Health behaviors.—Participants were asked about their smoking use using PYS questions from earlier evaluations. They were asked if they had smoked cigarettes in the last year and average number of cigarettes smoked per day; responses were coded into never or not in the last year, ≤ 10 cigarettes per day; or > 10 cigarettes per day. The square root of the frequency of cigarettes was also examined. The Paffenbarger physical activity scale assesses average kilocalories expended in leisure activities, walking, and stair climbing per week (23). Because it was skewed, 50 was added to the scores and natural log transformed prior to analysis. Data on daily fruit and vegetable consumption were collected using six items from the Behavioral Risk Factor Surveillance System fruit and vegetable module (24). Because of the distribution, it was transformed by square root for analysis.

Adult biological risk factors.—Laboratory assessments for biological risk factors were completed for 260 men. Height and weight were measured and body mass index (BMI; kg/m^2) was calculated. Blood pressure was measured with a CARESCAPE Dinamap V100 Vita Signs Monitor (GE Medical Systems Information Technologies Inc) with a standard occluding cuff placed on the participants' nondominant arm. Two resting measures were taken at the start of the protocol and testing for calibration against simultaneous blood pressure using an auscultatory method with a second cuff placed on the dominant arm. The auscultatory cuff was removed before the beginning of the baseline period. Blood pressure collection occurred every two minutes during the 10-minute baseline and was averaged. Metabolic syndrome was based on the ATP-III definition, which includes resting blood pressure, ≥130 or ≥80 mmHg or use of anti-hypertensive medications; fasting glucose ≥100 mm/dl or use of anti-diabetic medications, triglycerides ≥ 150 mg/dl; HDL- cholesterol < 40 mg/dl, and waist circumference ≥ 40 inches (25). Due to the age of participants, we used the number of risk factors above the clinical cutoffs as our primary outcome, as opposed to the conventional definition of yes/no having three or more of the five indicators. A number of secondary analyses were also conducted: including HDL-C or triglycerides as above the clinical cutoff if participants were using lipid-lowering agents (N= 7); calculating a continuous measure based on Z-transformations of each component and averaging (without regard to medications for hypertension, lipids, or diabetes); and finally removing those who did not fast. None of these additional analyses changed the pattern of results and are not reported further. C-reactive protein (CRP) and interleukin 6 (IL-6) were the measures of inflammation. Participants were removed from the analysis of inflammation if they reported being ill in the last 3 days (N=23), were on anti-inflammatory medications (N=9), or had CRP values > 10 mg/L (N=15), which could indicate an acute infection, resulting in 224 men in the inflammation analysis. The values were log transformed prior to analysis. An analysis including the 15 men with CRP values > 10 mg/L showed the same pattern of results as the more restrictive sample.

Laboratory assays were conducted by the Heinz Laboratory at the University of Pittsburgh, which is a certified laboratory. High sensitivity CRP was measured turbidimetrically and IL-6 was measured in duplicate using a high sensitivity ELISA kit (R&D Systems, HS600) Total cholesterol and triglycerides concentrations were determined by coupled enzymatic methods. HDL-C was isolated based upon the method of Izawa. Glucose was measured by hexokinase-coupled reaction (Boehringer Mannheim Diagnostics).

Statistical Analyses—Sample characteristics for the full sample and differences by race were calculated by t-test or chi-square as appropriate. Correlations among the predictor and outcome variables were examined in the full sample. To test the major study hypotheses, models examined the overall association between the repeatedly assessed measures of depressive symptoms reported from ages 7 to 16 and cardiovascular health risk factors assessed at age 32 years using STATA 15. This was accomplished using regression models that treated each assessment of depressive symptoms (up to 10 assessment points per individual) as a separate observation within the dataset and robust standard errors that adjusted for the for the non-independence of the repeated observations on the predictors within individuals. This modeling strategy produces a regression coefficient that represents the overall association between the repeatedly assessed predictor indexing childhood depressive symptom and the cardiovascular health outcomes in adulthood. The same modeling was used to summarize repeated assessments of childhood SES ages 7 to 16 years. Advantages of this analytic approach is that it uses all available information to generate parameter estimates, resulting in more reliable, efficient, and precise estimates, and the models can accommodate multiple time-invariant and time-varying covariates.

Covariates in the initial model were risk stratification group and race, and time-varying childhood SES, followed by tests for interaction of depressive symptoms with race and family SES. To assess for potential developmental changes in the association between depressive symptoms assessed from ages 7 to 16 and the adult outcomes, we also tested for interactions with age and age^2. Finally, we also adjusted for adult depressive symptoms scores and adult SES in additional models. Post hoc power calculations indicated a power of .80 to detect correlations of .162, .174, and .189 for sample sizes of 300, 260, and 220, respectively, assuming three covariates and an alpha of .05.

RESULTS

Sample characteristics

The sample was composed of 311 men in their thirties, with SES scores in the lower to middle-class on average (Table 1). The depressive symptom scores averaged across ages 7 to 16 years were low overall. Nearly 29% smoked cigarettes more than 10 cigarettes per day, with more Whites than Blacks represented in that category. The sample was moderately active on average. About 40% were considered obese. Only 19% would be classified as having the metabolic syndrome, but nearly 25% had elevated CRP. Blacks were more likely to have elevated blood pressure or be on antihypertensive medications than Whites, whereas Whites were more likely to have elevated triglycerides.

Table 2 shows univariate correlations among key variables. Number of metabolic syndrome criteria in adulthood was correlated with inflammatory measures. SES in childhood and adulthood was moderately correlated as were depressive symptoms in childhood and adulthood. Higher family SES in childhood was correlated with less cigarette smoking, more consumption of fruit and vegetables, and more physical activity in adulthood. Higher depressive symptoms in childhood were associated with more frequent cigarette smoking and less consumption of fruit and vegetables in adulthood.

Childhood depressive symptoms and adult health behaviors

Both depressive symptoms and family SES ages 7 to 16 were associated with greater frequency of cigarette smoking in adulthood, adjusted for risk group and race (Table 3). This pattern of results was similar in nominal logistic regression analyses, with depressive symptoms predicting differences between the nonsmoking men and men who smoked more than 10 cigarettes per day, OR = 1.168, $p = .013$. Higher family SES was associated with more physical activity as an adult, and childhood depressive symptoms were related to fewer servings of fruits and vegetables as an adult.

To illustrate the meaning of these findings, model-predicted values (at means for risk group and childhood SES) were calculated using the sample-based scores of high (upper 25th percentile) and low (lower 25th percentile) levels of childhood depressive symptoms in relation to the probability of being in the three groups of nonsmokers, smokers averaging 10 cigarettes or less per day, and smokers averaging more than cigarettes per day (Figure 1), and average weekly servings of fruits and vegetables (Figure 2). Probability of being in the >10 per day cigarette smoking group was 32% and 24% for the high and low depressive symptom men respectively. Average servings (back transformed) differed by 1.5 servings per week for the high and low depressive symptom groups.

We next examined whether these effects were apparent after adjustments for adult depressive symptoms and adult SES. The results showed that the frequency of adult cigarette smoking was significantly associated with depressive symptoms in childhood, childhood family SES tended to be associated, and adult SES was significantly associated. Greater physical activity was no longer related to lower childhood SES but was related to both higher adult SES and less adult depressive symptoms. More servings of fruits and vegetables were no longer associated with childhood depressive symptoms; they were associated with higher adult SES.

Because the parent study collected cigarette smoking data starting when the boys were 12 to 13 years old, we repeated the above analyses excluding boys who reported smoking at least one cigarette when 12 or 13 years of age (N = 30). Our results were the same as reported above. It is of interest to note that the 30 boys had somewhat higher childhood depressive symptoms scores overall beginning at age 10 to 12 years of age than the remaining boys who had not started to smoke by that time, $ps < .010$.

We next examined whether there were significant interactions between childhood depressive symptoms and race for the health behaviors, adjusted for race, risk group, childhood and adult SES, and adult depressive symptoms. Only physical activity interacted with race, B =

−0.08, SE=03 p = .005, such that among whites, the more depressive symptoms in childhood the more physical activity they reported, B = .120 (SE, .041), p = .004; among blacks there was no relationship, B = −0.04 (.04), p = .36.

There were no significant interactions between age or age^2 and childhood depressive symptoms for any of the health behaviors, ps > .18.

Childhood depressive symptoms and adult cardiometabolic risk factors

Childhood depressive symptoms and childhood family SES were unrelated to number of metabolic risk factors, systolic blood pressure (SBP), or inflammation. Further adjustment with adult depressive symptoms and adult SES in the model showed that lower adult depressive symptoms and lower adult SES were related to higher IL6 levels. Further adjustment by BMI indicated that adult depressive symptoms and adult SES remained associated with IL6 (data not shown), p < .031. There were no significant interactions between age or age^2 and childhood depressive symptoms, ps > .21.

DISCUSSION

The present study addressed the long-term impact of depressive symptoms in childhood and adolescence on cardiovascular risk factors in men in their thirties. The results showed that men who reported higher depressive symptoms in childhood were more likely as adults to be cigarette smokers, independent of adult depressive symptoms and childhood and adult SES. They also reported eating fewer fruits and vegetables as adults but these effects became nonsignificant with adjustments for SES and depressive symptoms in adulthood. Findings did not detect a unique influence of depressive symptoms across development, perhaps in part because the associations of depressive symptoms averaged within developmental periods of ages 7 to 9; 10 – 12; and 13 −16 were moderate (data not shown). Overall, these results are consistent with cross-sectional analyses of health behaviors and depression in adulthood as well as the longitudinal analysis of cigarette smoking associated with pediatric depressive disorder (11).

In contrast to the associations of childhood depressive symptoms with health behaviors, results showed no significant associations with the number of metabolic risk factors or biomarkers of inflammation. The latter was somewhat surprising, given that multiple depressive episodes in children measured at ages 9 to 16, 19, and 21 predicted elevated CRP levels in the Great Smoky Mountains Study (26). The latter observation was due in part to body mass index, smoking behavior, and report of recent infections. However, in their analyses, cumulative depressive symptoms were unrelated to elevated CRP, suggesting that multiple exposures to depressive disorder may be key for understanding childhood origins of inflammation. Another complexity in understanding the long-term effect of childhood depression is that depression and inflammation may have bidirectional relationships and experimental data suggest that inflammation may proceed depression (27–28).

Our study took advantage of the racial composition of the study sample to see if the relationships varied by blacks vs whites. It showed only one relationship that varied by race, albeit, in an unexpected direction. Whites who had more childhood depressive symptoms

reported greater physical activity as adults, whereas among blacks there was no relationship. Typically, depressed individuals are less active and that was the case in the present sample: more active adults regardless of race concurrently reported less depressive symptoms. Why whites with higher childhood depressive symptom scores might be more active is not apparent, especially given the internal validity of the associations between more physical activity and higher SES in adulthood.

The study has a number of strengths and weaknesses. First, repeated measures of depressive symptoms were available and the same measure was used throughout the study. Second, the sample included both black and white men and the sample was similar in key characteristics to those enrolled in parent study. On the other hand, the study findings are restricted to men from an urban area, and the men were originally oversampled for risk for anti-social behavior. The study did not measure metabolic risk factors and inflammation throughout the study but only in adulthood.

In conclusion, our study indicates that depressive symptoms during childhood and adolescence predict worse adult cigarette smoking and less vegetable/fruit intake but not metabolic factors or inflammatory biomarkers. That low adult SES was related to adverse health behaviors and IL6 levels lends credence to the validity of the study findings, given the consistency in the literature of these relationships. It may be that depression in the developing individual initially impacts health behaviors and then later biological risk factors for CVD. Perhaps the case the key to CVD risk in early life is major depressive disorder as opposed to depressive symptoms. Further research is needed on the long-term sequelae of depressive disorder in childhood and adolescence.

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Abbreviations:

CVD	cardiovascular disease
SES	socioeconomic status
PYS	Pittsburgh Youth Study
BMI	body mass index
CRP	C-reactive protein
IL-6	interleukin 6
SBP	systolic blood pressure
DBP	diastolic blood pressure

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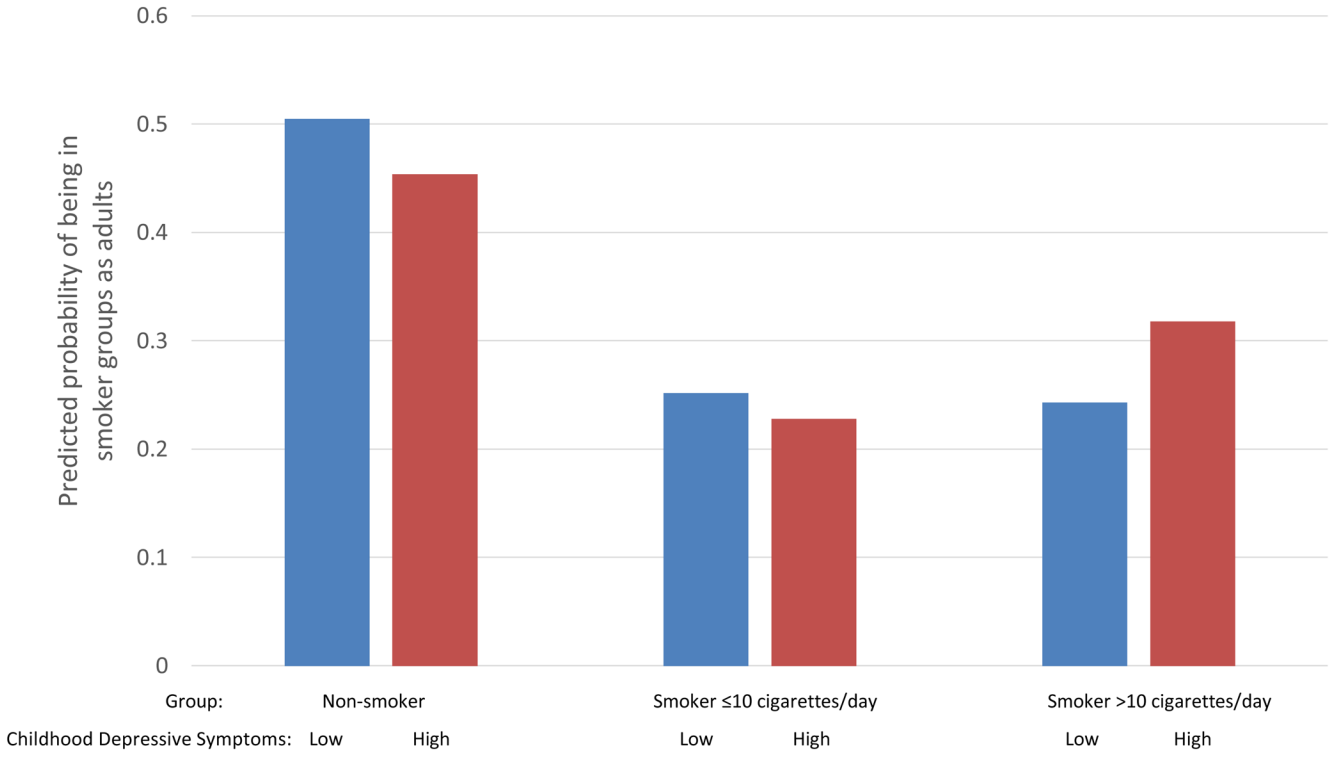

Figure 1.

Probability of being in each adult smoker group based on the model-specified values at low (25th percentile) and high (75th percentile) childhood depressive symptom scores, adjusted for mean risk group and childhood SES.

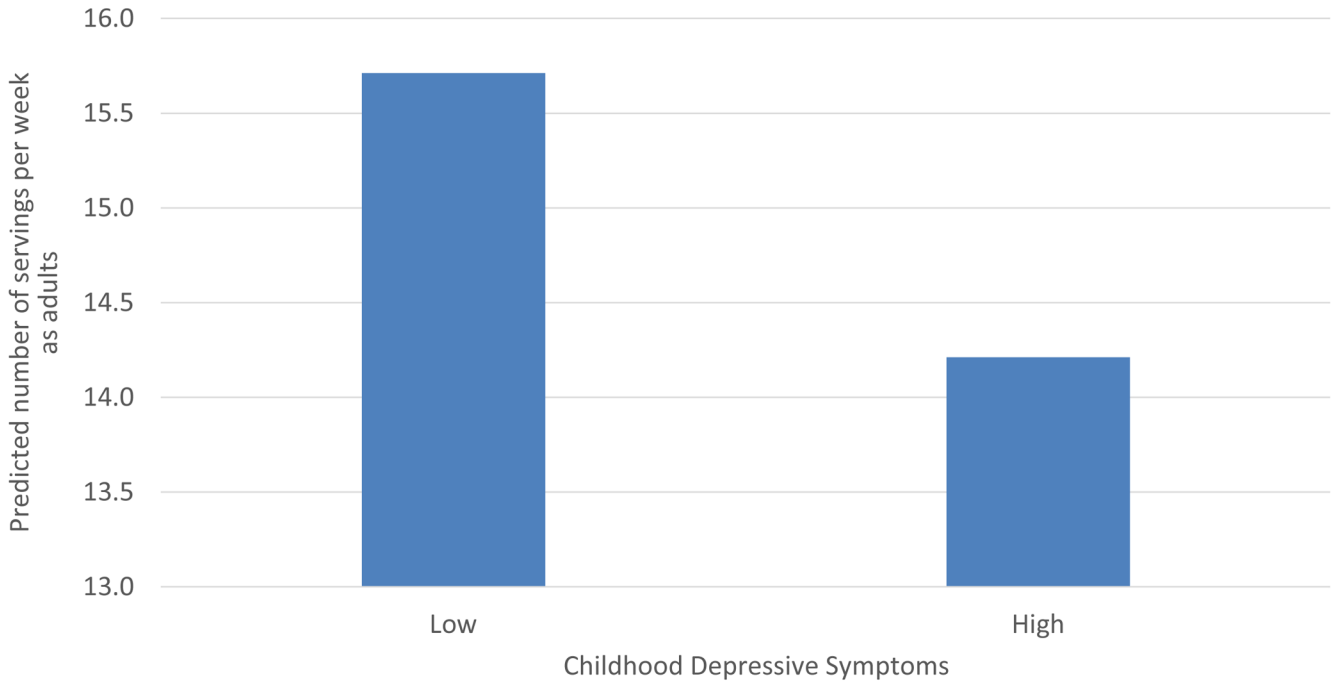

Figure 2.

Predicted mean weekly fruit and vegetable servings in adulthood based on the model-specified values at the low (25th percentile) and high (75th percentile) childhood depressive symptom scores, adjusted for mean risk group and childhood SES.

Table 1.

Sample Characteristics

Mean (SD) or N (%)	Black (N=165)	White (N=146)	Total (N=311)
Age	32.33 (1.01)	32.21 (.97)	32.27 (.99)
Hollingshead SES			
Average ages 7–16 l ***	35.95 (8.63)	40.86 (10.19)	38.26 (9.69)
Adult ***	28.45 (14.17)	36.45 (15.44)	32.23 (15.29)
Depressive symptoms			
Average ages 7-16	4.02 (2.63)	3.75 (2.35)	3.90 (2.51)
Adult	4.72 (4.54)	4.70 (4.82)	4.72 (4.67)
Cigarette smoking (N, %)			
Never ***	66 (40.0)	79 (54.1)	145 (46.6)
≤10 per day	56 (33.9)	20 (13.7)	76 (24.4)
≥10 per day	43 (26.1)	47 (32.2)	90 (28.9)
Physical activity kcal per week	1633 (2230)	1272 (1474)	1463 (1918)
Fruit and vegetable servings per week	17.24 (14.24)	16.63 (11.02)	16.95 (12.81)
BMI	29.58 (7.25)	29.40 (7.18)	29.50 (7.21)
<25 N (%)	45 (30.0)	25 (21.6)	70 (26.3)
25-30	44 (29.3)	49 (42.2)	93 (35.0)
≥30	61 (40.7)	42 (36.2)	103 (38.7)
Number metabolic syndrome criteria	1.30 (1.37)	1.46 (1.39)	1.37 (1.38)
None	54 (36.0)	39 (33.9)	93 (35.1)
1	42 (28.0)	24 (20.9)	66 (24.9)
2	29 (19.3)	26 (22.6)	55 (20.8)
3+	25 (16.7)	26 (22.6)	51 (19.2)
SBP mm Hg **	124.24 (13.46)	120.02 (10.83)	122.39 (12.53)
DBP mm Hg **	73.72 (10.23)	70.28 (8.17)	72.21 (9.52)
Individual metabolic syndrome criteria N:			
SBP ≥130 or DBP ≥85 or on medications *	46 (30.9)	21 (18.1)	67 (25.3)
Waist > 102cm	41 (27.5)	29 (25.0)	70 (26.4)
HDL < 40 mg/dl	43 (28.7)	43 (37.4)	86 (32.5)
Triglycerides ≥ 150 mg/dl ***	29 (19.5)	44 (38.3)	73 (27.7)
Lipid medications N	3 (2.0)	4 (3.4)	7 (2.6)
Glucose ≥100 mg/dl or on medications	35 (23.3)	31 (27.0)	66 (24.9)
CRP mg/l	2.00 (2.10)	2.30 (2.19)	2.13 (2.14)
N % <1	57 (45.2)	36 (36.7)	93 941.5)
1-3	44 (34.9)	33(33.7)	77 (34.4)
>3	25 (19.8)	29 (29.6)	54 (24.1)
IL6 pg/ml	2.02 (1.71)	1.90 (1.62)	1.97 (1.67)

* $p<.05$

** $p<.01$

*** $p<.001$ for race differences

Note: For CRP and IL6, subjects who were sick, taken anti-inflammatory medications and CRP >10 were excluded.

For purposes of this table only, depression and family SES were averaged across observations measured at ages 7 through 16.

Table 2.

Correlation coefficients among predictor and outcome variables.

	SES	Depressive symptoms								
	Adult	**Age 7-16**	**Adult**	**Cigarette smoking**	**Fruit/ vegetable**	**Physical activity**	**No. Metabolic syndrome criteria**	**SBP**	**CRP**	**IL6**
SES										
Age 7-16	.30***	−.09*	−.04	−.17***	.09*	.11**	.06	.04	−.01	−.03
Adult	--	−.05	−.17**	−.34***	.26***	.24***	.09	.03	−.04	−.21**
Depressive symptoms										
Age 7-16		--	.18***	.10***	−.07*	−.01	−.00	.00	.02	−.02
Adult			--	.07	−.11*	−.15**	−.04	−.001	.02	−.08
Cigarette smoking				--	−.16**	.12*	−.24***	−.21**	−.02	.06
Fruit/vegetable servings					--	.17**	.02	−.01	.05	−.04
Physical activity						--	−.06	−.001	−.07	−.05
No. Metabolic syndrome criteria							--	.59***	.39***	.20**
SBP								--	.24***	.13*
CRP									--	.44***

*$p<.05$

**$p<.01$

***$p<.001$

Note: For CRP and IL6, subjects who were sick, taken anti-inflammatory medications and CRP >10 were excluded. For purposes of this table, the regression coefficient for depressive symptoms and family SES were from analyses where each observation from ages 7 to 16 (up to 10 observations) was treated as a separate observation within person within the data set.

Table 3.

Regression estimates (standard errors) from multivariable models* of depressive symptoms and socioeconomic status on health behaviors per week.

	Cigarettes		Physical activity		Fruit and vegetable servings	
	***B* (SE)**	***p***	***B* (SE)**	***p***	***B* (SE)**	***p***
Model 1 (N=311)						
Childhood (age 7-16)						
Depressive symptoms	.28 (.12)	.015	.01 (.03)	.73	−.08 (.04)	.040
Family SES	−.06 (.02)	<.001	.01 (.004)	.010	.01 (.01)	.060
Race (Black = 1; White = −1)	−.10 (.27)	.71	.05 (.06)	.40	−.02 (.08)	.85
Model 2 (N=302)						
Childhood (age 7-16)						
Depressive symptoms	.24 (.11)	.032	.03 (.03)	.30	−.06 (.04)	.11
Family SES	−.03 (.02)	.053	.004 (.004)	.39	−.001 (.01)	.81
Race (Black = 1; White = −1)	−.39 (.27)	.15	.09 (.07)	.16	.07 (.08)	.43
Adult						
Depressive symptoms	.04 (.23)	.85	−.15 (.06)	.016	−.09 (.07)	.23
SES	−.10 (.02)	<.001	.02 (.005)	.001	.03 (.01)	<.001

*Adjusted for risk stratification group. For purposes of this table, the regression coefficient for depressive symptoms and family SES were from analyses where each observation from ages 7 to 16 (up to 10 observations) was treated as a separate observation within person in the data set. All three health behaviors are transformed prior to analysis.

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Table 4.

Regression estimates (standard errors) from multivariable models* of depressive symptoms and socioeconomic status on biological measures (N=261-263).

	No. metabolic syndrome risks		SBP		CRP		IL6	
	***B* (SE)**	***p***	***B* (SE)**	***p***	***B* (SE)**	***p***	***B* (SE)**	***p***
Model 1								
Childhood (age 7-16)								
Depressive symptoms	.01 (.04)	.88	.13 (.32)	.68	.02 (.03)	.60	−.01 (.02)	.47
Family SES	.01 (.01)	.41	.08 (.04)	.084	−.002 (.005)	.66	−.002 (.003)	.66
Race (Black = 1; White = −1)	−.09 (.09)	.30	2.34 (.77)	.002	−.10 (.08)	.20	.02 (.04)	.73
Model 2								
Childhood (age 7-16)								
Depressive symptoms	.01 (.04)	.84	.09 (.32)	.78	.01 (.03)	.65	−.004 (.02)	.79
Family SES	.003 (.01)	.68	.06 (.05)	.20	−.002 (.01)	.70	.0001 (.003)	.97
Race (Black = 1; White = −1)	−.07 (.09)	.46	2.51 (.78)	.002	−.11 (.08)	.16	−.01 (.04)	.90
Adult								
Depressive symptoms	−.05 (.08)	.50	.12 (.67)	.86	−.02 (.06)	.74	−.08 (.04)	.027
SES	.01 (.01)	.38	.04 (.05)	.45	−.005 (.01)	.40	−.01 (.003)	<.001

*Adjusted for risk stratification group. For purposes of this table, the regression coefficient for depressive symptoms and family SES were from analyses where each observation from ages 7 to 16 (up to 10 observations) were treated as a separate observation within the data set. Note: For CRP and IL6, subjects who were sick, taken anti-inflammatory medications and CRP >10 were excluded.