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Childhood Socioeconomic Circumstances and Depressive Symptom Burden Across 15 Years of Follow up During Midlife: Study of Women's Health Across the Nation (SWAN)

Joyce T. Bromberger, PhD¹, Laura L. Schott, PhD², Karen A. Matthews, PhD^{2,3}, Howard M. Kravitz, DO, MPH⁴, Siobán D. Harlow, PhD⁵, and Jennifer Karas Montez, PhD⁶

¹Department of Epidemiology and Department of Psychiatry, University of Pittsburgh, Pittsburgh, Pennsylvania

²Department of Epidemiology, University of Pittsburgh, Pittsburgh, Pennsylvania

³Department of Psychiatry and Department of Psychology, University of Pittsburgh, Pittsburgh, Pennsylvania

⁴Department of Psychiatry and Department of Preventive Medicine, Rush University Medical Center, Chicago, Illinois

⁵Department of Epidemiology, University of Michigan, Ann Arbor, Michigan

⁶Department of Sociology, Syracuse University, Syracuse, New York

Abstract

Purpose—Childhood socioeconomic disadvantage may contribute to adult depression.

Understanding pathways by which early socioeconomic adversity may shape adult depression is important for identifying areas for intervention. Studies to date have focused on one potential pathway, adult socioeconomic status (SES), and assessed depression at only one or a few time points. Our aims were to examine (a) the association between childhood SES (low vs high) and depressive symptom burden in midlife and (b) whether adult socioeconomic, psychosocial, and physical health characteristics are important pathways.

Methods—Using annual data from a cohort of 1109 black and white U.S. women recruited in 1996–97, we evaluated the association between childhood SES and depressive symptom burden across 15 years in midlife and whether adult characteristics—financial difficulty, lower education, stressful events, low social support, low role functioning, medical conditions, and bodily pain—mediated the association. Depressive symptom burden was estimated by calculating area under the curve of annual scores across 15 years of the Center for Epidemiological Studies-Depression (CES-D).

Results—In unadjusted models, low childhood SES was associated with greater depressive burden ($P=0.0002$). Each hypothesized mediator, individually, did not reduce the association.

*Address correspondence to Joyce T. Bromberger, PhD, Department of Epidemiology, 3811 O'Hara Street, University of Pittsburgh, Pittsburgh, Pennsylvania 15213.

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However, when 5 of the hypothesized mediators were included together in the same analysis, they explained more than two thirds of the association between childhood SES and depressive symptom burden reducing the P-value for childhood SES to non-significance ($P=0.20$).

Conclusions—These results suggest that childhood SES influences midlife depression burden through a cluster of economic stress, limited social resources and physical symptoms in adulthood.

Keywords

Childhood Socioeconomic Status (SES); Depression Burden; Economic Stress; Longitudinal; Midlife Women

Introduction

Stressful environments in early life may contribute to long-term alterations in stress-sensitive biological systems and to behaviors that are detrimental to mental and physical health (1), leading to an increased risk for depression in adulthood, especially among women who are particularly susceptible to depression. Prior studies data suggest that childhood socioeconomic disadvantage may be a contributor to later depressive mood (2–4). Indeed, several large studies have reported that low childhood SES (measured in diverse ways) and low adult SES (often measured by educational attainment) are each independently associated with depressive symptoms in adulthood (3–5). What remain largely unknown are the mechanisms that explain the relationship of such distal phenomena. The research on this question has focused primarily on adult SES, for which the most common indicator is educational attainment, as a potential mediator between childhood SES and adult depressive symptoms, with mixed results (4; 31).

Many, but not all (6), previous studies of childhood SES and adult depression suggest an important and enduring role of disadvantaged childhood environments (2;7). For example, the Tromso Study of female employees of the City of Helsinki reported that childhood economic difficulties and mother's low education (but not father's) were associated with anxiety/depression symptoms (4). In the Health and Retirement study, in addition to being poor in childhood, having a mother and a father with less than eight years of education was associated with elevated depressive symptoms in middle-aged women. Luo and Waite (2). Results from both studies were independent of participants' own education.

Previous research using cross-sectional or longitudinal designs have typically measured depressive symptoms at one point in time. Single time-point evaluations do not measure cumulative burden of depression. Recurrent or persistent depressive mood is more likely to be associated with decrements in quality of life and functioning than is a single episode (32; 33).

Possible pathways, such as few psychosocial resources and poor physical health, may also be important but have rarely been examined. Compromised health and functioning, experiencing stressful events, and having low social support as well as financial difficulties in adulthood have been associated with childhood socioeconomic adversity (1) and are risk factors for depression (8;9), raising the possibility that they serve as pathways between child

SES and adult depression. Knowledge about intermediary factors is important as it would enhance our understanding of the process by which early exposures influence later life mental health and functioning and identify targets, for interventions.

Given the increased vulnerability of women to depressive symptoms during midlife (8;10), this paper considers the impact of early childhood SES adversity on the cumulative burden of depressive symptoms during midlife and the potential pathways linking the two. In the current analyses, we addressed the limitations in the previous literature by using annually collected data on depressive symptoms and retrospective reports of childhood SES from 1,109 black and white middle-aged women. Specifically, we sought to evaluate as potential mediators not only participants' own SES as adults but also adult physical health and psychosocial factors that may be critical mediators of early adversity and later depression. We further extend the literature by focusing on the total burden of depressive symptoms in midlife. We hypothesized that (1) adverse childhood SES would be associated with elevated risk of high depressive symptoms burden over 15 years in midlife, independent of potential confounders such as race, childhood health, age, and menopausal status; and, (2) the association between childhood SES and adult depressive symptom burden would be mediated by baseline measures of participants' SES (educational attainment, financial strain), psychosocial resources (social support, role functioning), stressful life events, and indicators of physical health (number of medical conditions, bodily pain).

METHODS

Participants and study design

This paper uses data from midlife U.S. women participating in the Study of Women's Health Across the Nation (SWAN), a large multi-site, multi-racial/ethnic community based study of the menopausal transition and aging. Specifically, the analysis is based on data from the original parent study and data collected as part of the Childhood Context Ancillary Study conducted at four (Boston, Chicago, the Detroit area, and Pittsburgh) of seven sites participating in the SWAN. Eligibility criteria for SWAN included age 42–52 years, being premenopausal (no change in regularity of menses) or early perimenopausal (change in regularity of menses). Each site recruited white women and a predetermined minority group; the four sites participating in the ancillary study recruited participants self-identifying as non-Hispanic white or black. Written informed consent was obtained from all participants in accordance with the sites' institutional review board requirements. Of the 1399 active participants at these four sites at follow-up visit 13, the analytic sample consists of the 1109 women (79%) who completed the Childhood Context Questionnaire (CCQ).

The SWAN study design and sampling procedures have been described previously (11). Women were evaluated at study entry and approximately annually with similar protocols. Women provided extensive reproductive and medical history, health, psychosocial, lifestyle, physical and psychological symptom and biologic data as part of the larger SWAN.

Measures

All measures used in this study were assessed at the baseline visit with the exception of the CCQ administered at follow-up visit 13 and the assessment of depressive symptoms at baseline and each visit.

Childhood socioeconomic and health circumstances during the first 18 years of life were assessed with the 10-item CCQ. Specifically, participants were asked to indicate their mother's and father's highest education level from seven categories (e.g., less than high school, post-graduate degree), and whether, as a child or teenager, their family owned a car, owned their home, ever received public assistance or had difficulty paying for food or rent. The reliability of the questionnaire was evaluated by comparing responses on the CCQ that had been administered previously to 259 women at SWAN follow-up visit 07 at the Pittsburgh site. The concordance of the individual questions was high, varying from 86.1% to 96.9%.

Based on their childhood SES responses, three distinctive subgroups were identified using latent class analyses following recommended procedures (12;13). We used latent classes of childhood SES disadvantage as the primary predictor of adult depressive symptoms burden as some studies suggest that a composite index may provide a better indicator of early social and economic environment (14). Class 1 is the lowest SES, consisting of women with low-educated and poor parents, e.g., family on public assistance (26% of women). Class 2 contains women with low educated but non-poor parents (50% of women). Class 3 is the highest SES, with highly educated and non-poor parents (24% of women) (15). These latent classes have been successfully used in previous SWAN studies to examine their association with inflammation and metabolic syndrome (15;16).

As the depressive symptom burden outcomes (described below) for the women classified in childhood SES Classes 2 and 3 were similar, these two classes were combined for all analyses to facilitate the mediation analysis and are referred to as the high childhood SES group (non-poor regardless of parental educational level). To evaluate childhood health, participants were asked to rate their childhood health as excellent, very good, good, fair or poor. Responses were dichotomized as those having very good/excellent childhood health versus good/fair/poor health.

Depressive symptom burden

Depressive symptom burden was based on scores from the Center for Epidemiologic Studies Depression (CES-D) scale, a 20-item questionnaire asking about frequency of being bothered by depressive symptoms during the previous week on a 4-point scale of 0 (rarely) to 3 (most or all of the time) (17). The CES-D was completed at baseline and each follow-up visit. Number of visits with CES-D data ranged from 2 to 14, with 91% of women contributing 10 or more visits with CES-D scores. The CES-D has been shown to be valid and reliable in diverse racial/ethnic populations (18–20).

To define the cumulative burden of depressive symptoms in midlife, we summarized the CES-D scores from all available baseline and follow-up visit data with a single value reflecting an area under the curve (AUC). AUC allows repeated, changing measurements

over time, with variable intervals to be summarized as a single cumulative value that reflects the overall extent and duration of burden (although not the shape and trajectory) (21). We calculated an AUC by graphing the participant's scores across time using Riemann sums. Specifically, the width of each rectangle is the length of time between measurements and the height is the mean of two consecutive measurements. The area of the rectangle is calculated according to the trapezoidal rule and the areas of all the rectangles for each participant are then summed together to estimate the area under the curve (21). To investigate whether AUC values for participants reflected depressive symptom levels across time, we compared the mean AUC for 1,2,3, or 4+ SWAN visits with a possible depressive "episode" (i.e., CES-D 16). Results showed a significant linear association between the number of "episodes" and the mean AUC, ranging for no episode ($m=52.7$) to 4+ episodes ($m=266.88$) ($p<.0001$), confirming that AUC was a reliable and reasonable approach to estimating depressive symptom burden. Because AUC values were not normally distributed, values were log transformed and scaled for a non-negative value by adding 1.

Potential mediators from baseline evaluation

Adult socioeconomic characteristics are measured by educational attainment and financial difficulty. Participants' educational attainment was categorized as less than high school, high school diploma or equivalent, some college/technical/vocational schooling, 4-year college degree, or post-graduate degree. Difficulty paying for basics was dichotomized as very hard/somewhat hard versus not very hard indicating the presence/absence of financial strain.

Physical health characteristics are measured by medical conditions and bodily pain. History of 13 pre-specified medical conditions was dichotomized as 0–1 versus 2 or more conditions. Bodily pain was evaluated by the *Medical Outcomes Study* Short Form (MOS) SF36 subscale comprising the sum of two items that assess pain severity during the past 4 weeks (none, very mild, mild, moderate, severe, or very severe), and how much pain interfered with normal work including work outside the home and housework (not at all, slightly, moderately, quite a bit, or extremely) (22). We used the 25th percentile of the sample as the cut point for high bodily pain as previously established (23).

Psychosocial factors included stressful life events, social support, and three types of role functioning. Stressful life events were assessed with a checklist of 18 life events and then rated according to how upsetting they were (24). Stressful events were dichotomized as 0–1 versus 2 or more "very upsetting" events in the past year. Social support was a summed score of frequency of availability of four types of needed emotional and instrumental supports (25). Three subscales from the Medical Outcomes Study Short Form (MOS) SF36 (22) were assessed, including role physical (role limitations related to physical health), role emotional (role limitations related to emotional problems), and social function (effect of physical health or emotional problems on normal family or social activities). Each scale was dichotomized using the bottom 25th percentile of the sample as the cut point for impaired functioning as previously established (23).

Statistical analysis

Distributions of baseline characteristics and depressive symptoms burden were summarized for the entire sample and separately for the two childhood SES groups. To assess whether there was multicollinearity among the hypothesized mediators, we examined Spearman Correlation Coefficients and the variance inflation factor (VIF). As we expected, the highest correlations were between subscales of the SF36, ranging from Role Emotional and Bodily Pain ($r=.21$) to Role Emotional and Social Functioning ($r=.46$). There was minimal correlation for all other predictors ($r < .23$). Importantly, the mean VIF was 1.22 with all VIF < 1.57 indicating no collinearity among mediators and covariates in all models. All remaining analyses controlled for SWAN site, race, childhood health, and study entry (baseline) age and menopausal status. The basic linear regression model examined the association between childhood SES groups and depressive symptoms burden while controlling for these covariates.

Baseline variables considered as potential mediators of the association between childhood SES group and depressive symptoms burden in midlife were participants' financial strain, education, number of medical conditions, bodily pain, stressful life events, low social support, and the three role functioning subscales. To evaluate whether each of these variables mediated the association between childhood SES and depressive symptoms burden, we conducted two sets of preliminary analyses. In the first, we examined in individual models, the association between mediator and depressive symptoms burden with childhood SES in the model. In the second set of analyses, we evaluated the association between the predictor (i.e. childhood SES group) and each hypothesized mediator (significant in the first analyses at $p < .05$) using linear regressions for continuous mediator outcomes and logistic regressions for dichotomous mediator outcomes. For each mediator, we determined the indirect effect by the product of coefficients method (Sobel, 1982), the proportion of the effect of the predictor explained by the mediator (calculated as $(\beta_{\text{unadjusted}} - \beta_{\text{adjusted}})/\beta_{\text{unadjusted}}$), and whether the mediator attenuated the relationship between childhood SES and depressive symptoms burden. Mediators with a significant indirect effect were then together added to the basic linear regression model in a multiple mediation model. Estimates of indirect effects were verified with path analyses utilizing bootstrap and Monte Carlo confidence intervals (26). Because SWAN is a multiethnic study we also examined whether mediators were differentially important for white versus black women. Analytic sample size varied slightly (1074 to 1086) for each mediation model because of random missing data of particular baseline characteristics. All analyses were run with SAS version 9.3 and STATA 9.

RESULTS

At the baseline visit, women had a mean age of 46 years (Table 1). Fifty-four percent of the sample was white, 54% were premenopausal and 46% were early perimenopausal. Compared to women with high childhood SES, women with low childhood SES had worse childhood health, and in adulthood a lower percentage with a college education themselves, a greater percentage who had a somewhat or very hard time paying for basics, lower social support, greater body pain and impairment in functioning, and higher average depressive

symptoms burden. Overall and in each of the two childhood SES groups, women had an average of nearly 13 visits across 15 years.

In the basic multivariable model (Table 2, Basic Model), lower childhood SES was associated with greater depressive symptom burden, independent of confounders, age, race, menopausal status, SWAN site, and childhood health (estimate=0.25, CI: 0.12, 0.38., $P=0.0002$). Being early perimenopausal at baseline and reporting poorer childhood health were each also positively associated with depressive symptoms burden while baseline age was inversely associated.

Mediation analyses

All hypothesized mediators were significantly associated with depressive symptoms burden with childhood SES in the model ($P<.05$). Low childhood SES was positively associated with three of these potential mediators ($p<.05$): difficulty paying for basics, high bodily pain, low social function, and negatively associated with participants' education and social support. Each of these five mediators had a significant indirect effect on the childhood SES relationship with depressive symptoms burden. Indirect effects were not significant for number of medical conditions, stressful events, role physical, or role emotional, and, thus, these characteristics were not considered further as mediators. There were no significant interactions between race and any of the potential mediators.

In Table 2, Models 1–5, we show the results from separate analyses of each of the five mediators with statistically significant indirect effects. Each Model shows that the individual mediator was a significant predictor of depressive symptoms burden independent of all other variables in the model and that the estimate for childhood SES (i.e. strength of association between childhood SES and depressive burden) was attenuated slightly with the addition of each mediator independently. However, childhood SES remained statistically significant at $P=0.01$. The bottom row shows the proportion of the association between childhood SES and adult depressive symptoms burden accounted for by each mediator. The proportions ranged from 0.18 (educational attainment) to 0.36 (social function).

The multiple mediation model (Table 3) included all five mediators identified above—education, paying for basics, social support, social function, and bodily pain. Collectively, these psychosocial factors explained more than two thirds of the association between childhood SES and total depressive burden. These five mediators together attenuated the association between childhood SES and depressive burden to statistical non-significance (estimate=0.08, CI: $-0.04, 0.20$, $P=0.20$). Every variable in the final model except age at baseline was significantly associated with depressive symptoms burden.

Analyses with three childhood SES groups

Replication of the analyses using the original three childhood SES groups showed results similar to analyses when Classes 2 and 3 were combined. Specifically, when compared to having low educated and non-poor parents (estimate = -0.26 , CI: $-0.39, -0.12$, $P=0.0002$), or high educated and non-poor parents (estimate = 0.23 , CI: $-0.40, -0.07$, $P=0.005$), having low educated and poor parents was significantly associated with depressive burden in the basic (i.e., no mediators) multivariable model (overall $P=0.0007$). In the multiple

mediation model that included the five mediators together, compared to having low educated and non-poor parents (estimate = -0.08 , CI: $-0.20, 0.04$, $P=0.21$) or high educated and non-poor parents (estimate = -0.02 , CI: $-0.17, 0.13$, $P=0.81$) having low educated and poor parents was not associated with depressive symptom burden (overall $P=0.37$).

DISCUSSION

In this longitudinal analysis of midlife black and white U.S. women, we found that the cumulative burden of depressive symptoms over 15 years was greater for women who reported that their parents had lower educational attainment and were poor during their childhood compared to women whose families were not poor irrespective of parents' educational attainment. This association was independent of race, age, SWAN site, and menopausal status at study entry, and childhood health. The finding suggests that childhood financial circumstances but not parental educational attainment is an important predictor of high depressive symptoms burden among women during midlife.

Importantly, we found that five adult factors each had indirect effects on the childhood SES-depressive symptoms burden: difficulty paying for basic expenses, participant's lower education, low social support, low social function, and high bodily pain. However, none of these mediators individually significantly accounted for the association between childhood SES and women's depressive symptoms burden in midlife. Only when included together in the final model did the five factors influence the association between childhood SES and depressive symptoms burden and thus, indicate that they were a pathway to depressive symptoms burden during midlife. Our findings demonstrate the links between childhood SES, multiple adult psychosocial factors and depressive symptoms burden. The results regarding the absence of a mediating effect for each factor are consistent with what is theoretically stated and empirically shown about the myriad and complex risk factors for depression. Depression is influenced by multiple factors making it highly unlikely that any single factor alone would explain an association of an independent variable such as childhood disadvantage and adult depression burden. Likewise, childhood SES is a multidimensional construct and thus would not be associated in a linear fashion with one pathway (34).

Previous studies, using varied indicators of childhood economic circumstances such as, self-reported childhood financial condition (very good to ... very difficult (4) or social economic position based on the British Registrar's classification of father's occupational class (35) also reported that these were associated with a greater risk of depressive symptoms in adulthood. Results were independent of adult education.

The most commonly postulated pathway between childhood SES and adult health (27) has been adult SES. This is reasonable given that childhood SES is a primary determinant of adult SES, which is a predictor of later health status (27; 28). However, to our knowledge, only two large studies, the Tromso Study in Norway noted above (4) and the New Zealand Survey of Family, Income and Employment (31) statistically tested adult SES as a mediator and reported contradictory results. Sheikh et al (4) found that adult education was not a mediator between childhood SES and depressive symptoms whereas McKenzie et al (31)

found that it was a mediator. Differences may be related to wide variations in cohorts' ages and differences in depressive symptom measures, statistical analyses used, and in the importance of adult SES for health in their countries.

In contrast, the results of the current study support a partial mediating role for adult SES, but only as one of a cluster of five factors. The New Zealand results, although adjusted for sex and age, were based on combining men and women making it difficult to compare their results with our study of middle aged women. Importantly, despite being longitudinal surveys, the Norway study and the New Zealand study measured the depressive symptom outcome at only one point in time which may reflect a potentially transient symptom. No study has assessed multiple adult characteristics as potential mediators of the association as we did.

Our study suggests that early adversity may affect not only the economic and educational circumstances in midlife but symptoms of pain and the impact of physical health on social functioning. Early life family social and economic disadvantages may lead to deficits in social, emotional, and physical health factors in adulthood through multiple psychosocial and biological pathways. For example, early SES adversity may reduce the quality of interpersonal relationships and self-esteem during childhood and beyond and increase the risk of exposure to life stressors by influencing later life experiences that are associated with additional stressors during adolescence and early adulthood. As a consequence, the accumulation of social and economic resources that are important for adult growth and development may be limited (28). Thus, early adversity may increase exposure and susceptibility to stressors and, at the same time, negatively influence the development of financial and psychosocial resources, such as good interpersonal relationships and coping skills, to manage these stressors and decrease the risk of depression (4;6;10;14;27).

Biological pathways (36) may also link early SES disadvantage and later depressive symptoms. For example, there is evidence that childhood adversity may alter brain structure, activity, and functioning (34). The neurobiological alterations associated with the stress of early adversity may affect the biological stress regulatory system including the hypothalamic pituitary-adrenal (HPA) axis and thereby future responses to stress (36). Such neurobiologic effects may result in difficulties with emotional regulation of arousing situations, behavioral development, executive functions, and delay of learning (36; 37).

Strengths and limitations

The findings of this study should be considered in the context of its limitations. We did not have longitudinal data on the period of time that the family SES was disadvantaged. Childhood SES at a single point does not capture cumulative exposure (29), which is likely more potent. We had no data on exposures between childhood and midlife, such as trauma, health problems, and especially depression, which influence vulnerability to depressive disorder as well as depressive symptoms in mid and late life. Nor did we have information on potential confounders such as family history of depression and childhood maltreatment, both of which could be associated with childhood SES and depression in midlife. Retrospective reporting of childhood experiences and circumstances raises the issue of the reliability of recall. As noted above, there was high concordance between childhood SES

questions administered two times five years apart in Pittsburgh. Despite the longitudinal follow-ups, it is possible that earlier depressive symptoms led to low socioeconomic circumstances in midlife or to compromised social support and functioning. Finally, cumulative values of depression over time as reflected in the AUC values (depressive symptoms burden), could appear similar for women with steady symptom levels and those who participated in only one visit at which the CES-D score was high. However, we were able to capture an extensive time period of depressive symptoms because the vast majority (91%) of participants had 10 or more study visits over the course of 15 years and 99% of women had at least 5 visits. Furthermore, analyses described above indicate that use of AUC as the outcome captured overall depressive burden across midlife, whereas a repeated measures methodology would have reflected average depression at each visit.

The study has numerous strengths, which greatly outweigh its limitations. To our knowledge, this study is the first to examine the association between early socioeconomic adversity and its multiple sequelae on not just an episode of elevated depressive symptoms but on the persistence of such symptoms over 15 years in midlife. To our knowledge, this is also the first study to examine multiple adult psychosocial factors as potential mediators. Importantly, our data indicate that the pathway between childhood SES and depression symptom burden includes multiple factors with no sole factor fully explaining the relationship, suggesting that childhood SES influences later depression burden largely through a cluster of economic stress, limited social resources and physical symptoms (specifically, pain). We used established statistical methods to evaluate the mediators.

In conclusion, the results add to a growing body of literature indicating the importance of life course and adult cumulative exposures in the genesis of depressive symptoms burden, especially in women with histories of childhood socioeconomic disadvantage. Results reinforce the importance of considering early family life experiences and multiple socioeconomic determinants of depression in the treatment of midlife women to minimize the depression burden over time. Information about a patient's early family circumstances and a woman's own socioeconomic and social resources may be particularly salient for women at this stage of life given that there is no single pathway that emerged as being uniquely important. Such knowledge may help guide health care providers to develop treatment plans that focus on reducing stress, improving coping skills, and social skills. Interventions focused on coping may enhance skills, such as emotional regulation and social skills that were lacking in early life and enable individuals to deal with various stresses such as economic difficulties or health perceptions that are risk factors for depressive symptoms (1).

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Table 1

Baseline demographic and clinical characteristics among overall sample and within latent classes of childhood socioeconomic conditions defined by parental financial resources and education. Boston, Massachusetts; Chicago, Illinois; Detroit Michigan; Pittsburgh, Pennsylvania, 1996 – 2013 (N=1109)^a

Baseline Characteristic	Full Sample	Low Childhood SES	High Childhood SES	p-value
N	1109	294	815	
Age (years), mean (SD)	46.3 (2.6)	46.3 (2.6)	46.4 (2.7)	.64
Race, n (%)				<.0001
Black	506 (46)	206 (70)	300 (37)	
White	603 (54)	88 (30)	515 (63)	
SWAN site, n (%)				.004
Boston	293 (26)	80 (27)	213 (26)	
Chicago	154 (14)	38 (13)	116 (14)	
Detroit	356 (32)	115 (39)	241 (30)	
Pittsburgh	306 (28)	61 (21)	245 (30)	
Paying for basics, n (%)				<.0001
Very/somewhat hard	389 (35)	141 (48)	248 (31)	
Not very hard	715 (65)	150 (52)	565 (70)	
Education, n (%)				<.0001
Less than high school	30 (3)	16 (6)	14 (2)	
High school credential	202 (18)	76 (26)	126 (16)	
Technical/vocational/some college	392 (36)	122 (42)	270 (34)	
Bachelor's degree	200 (18)	42 (14)	158 (20)	
Postgraduate degree	272 (25)	35 (12)	237 (29)	
Childhood health, n (%)				<.0001
Good/fair/poor	281 (25)	102 (35)	179 (22)	
Excellent/very good	827 (75)	191 (65)	636 (78)	
Menopausal status, n (%)				.0004
Premenopause	584 (54)	130 (45)	454 (57)	
Early perimenopause	506 (46)	161 (55)	345 (43)	
CES-D depression scale, mean (SD)	10.3 (9.2)	12.5 (9.9)	9.6 (8.9)	<.0001
Number medical conditions ever, n (%) ^b				.04
None - One	585 (53)	140 (48)	445 (55)	
Two or more	524 (47)	154 (52)	370 (45)	
Stressful life events, n (%)				.01
None - One	745 (68)	181 (62)	564 (70)	
Two or more	355 (32)	111 (38)	244 (30)	
Social support, mean (SD)	12.4 (3.1)	11.7 (3.4)	12.7 (2.9)	<.0001
Low role physical, n (%)	223 (20)	72 (25)	151 (19)	.03
Low role emotional, n (%)	241 (22)	80 (27)	161 (20)	.007
Low social function, n (%)	295 (27)	111 (38)	184 (23)	<.0001

Baseline Characteristic	Full Sample	Low Childhood SES	High Childhood SES	p-value
N	1109	294	815	
High bodily pain, n (%)	284 (26)	100 (34)	184 (23)	.0001
Cumulative depressive symptoms burden, mean (SD)	128.3 (99.5)	159.6 (113.6)	117.1 (91.4)	<.0001
Natural log of cumulative depressive burden, mean (SD)	4.5 (0.9)	4.8 (0.9)	4.4 (0.9)	<.0001
Total number visits, mean (SD)	12.7 (2.0)	12.6 (2.1)	12.7 (2.0)	.22
Total years in study, mean (SD)	15.3 (0.9)	15.2 (0.9)	15.3 (0.9)	.11

Abbreviations: CES-D, Center for Epidemiologic Studies Depression scale; SD, standard deviation; SES, socioeconomic status.

^aColumn percentages may not sum to 100% due to rounding. The percentages are calculated after excluding participants with missing data.

^bMedical conditions included anemia, diabetes, high blood pressure, arthritis/osteoarthritis, thyroid disease, heart attack, angina, fibroids, cancer (other than skin cancer), migraines, hypercholesterolemia, osteoporosis, and stroke.

Table 2

Coefficients (estimates) from individual linear regression models assessing childhood socioeconomic conditions and potential mediators in association with AUC_{log} value of cumulative depressive symptoms burden ^a. Boston, Massachusetts; Chicago, Illinois; Detroit Michigan; Pittsburgh, Pennsylvania, 1996 – 2013

Characteristic	Basic			Model 1			Model 2			Model 3			Model 4			Model 5		
	Est	95% CI	p	Est	95% CI	p	Est	95% CI	p	Est	95% CI	p	Est	95% CI	p	Est	95% CI	p
Low childhood SES vs. high childhood SES	0.25	0.12 0.38	.0002	0.20	0.07 0.33	.002	0.20	0.07 0.33	0.002	0.20	0.07 0.32	.002	0.16	0.04 0.28	0.01	0.20	0.08 0.33	.002
Age	-0.03	-0.05 -0.01	0.01	-0.03	-0.05 -0.01	0.007	-0.02	-0.04 -0.00	0.03	-0.03	-0.05 -0.01	0.01	-0.02	-0.04 -0.00	0.11	-0.03	-0.05 -0.01	0.01
Black vs. White	-0.02	0.14 0.10	0.73	-0.05	-0.17 0.07	0.41	-0.07	-0.18 0.05	0.27	-0.05	-0.16 0.06	0.39	-0.05	-0.16 0.06	0.39	-0.04	-0.15 0.07	0.51
Early perimenopause vs. premenopause	0.18	0.07 0.29	0.002	0.18	0.07 0.29	0.002	0.16	0.05 0.27	0.004	0.13	0.02 0.23	0.02	0.15	0.04 0.25	0.006	0.16	0.05 0.26	0.004
Childhood health good/fair/poor vs. excellent/very good	0.36	0.24 0.49	<0.0001	0.35	0.23 0.48	<0.0001	0.34	0.21 0.46	<0.0001	0.31	0.43	<0.0001	0.31	0.19 0.43	<0.0001	0.32	0.19 0.44	<0.0001
Potential Mediator:																		
Education (continuous, 1< high school – 5; post graduate)	-	-	-	-0.11	-0.16 -0.06	<0.0001	-	-	-	-	-	-	-	-	-	-	-	-
Paying for basics very/somewhat hard vs. not very hard	-	-	-	-	-	-	0.37	0.25 0.48	<0.0001	-	-	-	-	-	-	-	-	-
Social support (continuous, 0; low – 16; high)	-	-	-	-	-	-	-	-	-	-0.08	-0.10 -0.06	<0.0001	-	-	-	-	-	-
Social function low vs. normal/high	-	-	-	-	-	-	-	-	-	-	-	-	0.71	0.59 0.83	<0.0001	-	-	-
Bodily pain high vs. normal/low	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.55	0.43 0.67	<0.0001
Indirect effect on depressive symptoms burden ^b																		
	-	-	-	0.04	0.02 0.08	0.001	21.8	19.9 23.8	0.003	0.05	0.02 0.10	0.003	43.5	39.8 47.3	0.0004	22.5	19.6 25.4	0.02
Proportion of effect of childhood SES explained by the mediator ^c																		
	-	-	-	0.18	0.18			0.19			0.12			0.36			0.19	

Abbreviations: AUC, area under the curve; *P*, *P*-value; SE, standard error; SES, socioeconomic status.

^aModels also include SWAN site (*P*<0.01 in each model).

^bIndirect effect of childhood SES and each mediator on depressive symptoms burden estimated in individual models. Unstandardized indirect effects and Monte Carlo intervals for dichotomous mediators: paying for basics = 0.08 (0.02, 0.15), social function = 0.15 (0.05, 0.28), and bodily pain = 0.15 (0.06, 0.25)

^cProportion of effect of childhood socioeconomic conditions explained by each mediator calculated as (β_{unadjusted} – β_{adjusted})/β_{unadjusted}.

Table 3

Coefficients (estimates) from multiple mediation linear regression model assessing associations with AUC_{log} value of cumulative depressive symptoms burden ^a. Boston, Massachusetts; Chicago, Illinois; Detroit Michigan; Pittsburgh, Pennsylvania; 1996 – 2013

Characteristic	Multiple Mediation Model		
	Estimate	95% CI	p-value
Low childhood SES vs. high childhood SES	0.08	-.04, 0.20	0.20
Age	-0.02	-0.04, 0.00	0.06
Black vs. white	-0.11	-0.22, 0.00	0.05
Early perimenopause vs. premenopause	0.11	0.01, 0.21	0.03
Childhood health good/fair/poor vs. excellent/very good	0.25	0.13, 0.36	<0.0001
Education (continuous, 1: less than high school – 5: post graduate)	-0.06	-0.11, -0.01	0.01
Paying for basics very/somewhat hard vs. not very hard	0.19	0.08, 0.30	0.001
Social support (continuous, 0: low – 16: high)	-0.06	-0.07, -0.04	<0.0001
Social function low vs. normal/high	0.44	0.31, 0.56	<0.0001
Bodily pain high vs. normal/low	0.33	0.21, 0.45	<0.0001
Proportion of effect of childhood SES explained by the five mediators ^b	0.69		

Abbreviations: AUC, area under the curve; SE, standard error; SES, socioeconomic status.

^aModel also includes SWAN site ($P = 0.003$), N=1070. Characteristics are from the baseline visit unless otherwise indicated.

^bProportion of effect of childhood socioeconomic conditions explained by the five mediators together calculated as $(\beta_{unadjusted} - \beta_{adjusted}) / \beta_{unadjusted}$.