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What is This?

Pathways to Resilience: Maternal Nurturance as a Buffer Against the Effects of Childhood Poverty on Metabolic Syndrome at Midlife

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Abstract

Children raised in families with low socioeconomic status (SES) go on to have high rates of chronic illness in adulthood. However, a sizable minority of low-SES children remain healthy across the life course, which raises questions about the factors associated with, and potentially responsible for, such resilience. Using a sample of 1,205 middle-aged Americans, we explored whether two characteristics—upward socioeconomic mobility and early parental nurturance—were associated with resilience to the health effects of childhood disadvantage. The primary outcome in our analyses was the presence of metabolic syndrome in adulthood. Results revealed that low childhood SES was associated with higher prevalence of metabolic syndrome at midlife, independently of traditional risk factors. Despite this pattern, half the participants raised in low-SES households were free of metabolic syndrome at midlife. Upward social mobility was not associated with resilience to metabolic syndrome. However, results were consistent with a buffering scenario, in which high levels of maternal nurturance offset the metabolic consequences of childhood disadvantage.

Poverty's deleterious influences on children's educational and psychosocial functioning have long been recognized (Duncan, Yeung, Brooks-Gunn, & Smith, 1998; McLoyd, 1998). But recently it has become evident that childhood poverty has even further-reaching consequences, in the form of heightened vulnerability to common medical illnesses across the life course (Shonkoff, Boyce, & McEwen, 2009). Indeed, children raised in families of low socioeconomic status (SES) go on to have elevated rates of infectious, respiratory, metabolic, and cardiovascular diseases in adulthood, independent of traditional risk factors for those conditions (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Galobardes, Lynch, & Smith, 2008).

However, not all children who grow up poor go on to develop medical problems as adults. In fact, a sizable minority of them stay in good health (Chen & Miller, 2011). This resilience was vividly illustrated in a study in which adults were exposed to a rhinovirus and then monitored in quarantine for emergence of the common cold (Cohen, Doyle, Turner, Alper, & Skoner, 2004). Participants who had been reared in low-SES families were more likely to become infected with the virus and to develop colds than participants who had been reared in high-SES families. But even among participants from the lowest SES category, fewer than 50% actually became sick. These findings show that some individuals are resilient to the health effects of low childhood SES; despite having been exposed to childhood socioeconomic adversity, they managed to resist an infectious challenge in adulthood.

Presently, little is known about the pathways associated with resilience to health problems in persons from low-SES backgrounds. Two broad routes to good health have been hypothesized. The first involves upward mobility offsetting the influence of childhood disadvantage (Lynch & Smith, 2005). The idea underlying this hypothesis is that children

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who grow up poor can have varying socioeconomic trajectories. Some will become low-SES adults, in whom the continuing and cumulative effects of disadvantage will have deleterious health effects. Other children will follow an upwardly mobile path and experience the health benefits it confers. The assumption is that favorable socioeconomic conditions in adulthood mitigate whatever disease processes were instigated by the childhood environment (Cohen et al., 2010).

The other scenario highlights the role of parental nurturance in buffering children against poverty's sequelae. Research shows that nurturant parenting can offset many of the educational and psychosocial disadvantages that beset poor children (Luthar, 2006; Masten, 2001). There is also mounting evidence that aspects of nurturant parenting—particularly warmth and sensitivity—can favorably mold the stress-response tendencies of vulnerable children (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2007). In fact, parental nurturing may help mitigate the wear and tear that low SES places on children's physiology (Chen, Miller, Kobor, & Cole, 2011; Evans, Kim, Ting, Tesher, & Shannis, 2007). That said, it remains unclear whether the benefits of nurturant parenting extend to health problems, particularly ones that do not manifest until the later years of life.

The goal of the study reported here was to begin evaluating the plausibility of these scenarios. Using a sample of middle-aged Americans, we examined links between childhood SES and later presence of metabolic syndrome, a cluster of signs that includes high blood pressure, impaired glucose control, abdominal adiposity, and lipid dysregulation. Metabolic syndrome is a precursor and contributor to a number of chronic diseases of aging, including diabetes, heart disease, and stroke. It is highly prevalent in the United States, with rates estimated at 25% to 39%, and has become even more prevalent in recent decades (Cornier et al., 2008). We examined three hypotheses about childhood SES and metabolic syndrome. The first was that low childhood SES would be associated with a higher prevalence of metabolic syndrome at midlife. The second hypothesis was that despite this general trend, there would be a sizable subgroup of individuals who came from low-SES backgrounds but did not show metabolic syndrome. Finally, we predicted that in individuals from low-SES backgrounds, both upward mobility and parental nurturance would be associated with resilience to metabolic syndrome at midlife.

Method

Participants

Participants were from the Midlife in the United States (MIDUS) study, which commenced between 1995 and 1996 with 7,108 noninstitutionalized adults selected via random-digit phone dialing from the 48 contiguous states. To allow genetically informed analyses, MIDUS included 957 pairs of twins and 950 nontwin siblings. An average of 9 years later, 75% of surviving respondents participated in a follow-up study, known as MIDUS II. Biological data were collected from a subset of participants ($n = 1,255$), who traveled to a General Clinical Research Center (GCRC) for an overnight visit. These individuals had higher educational attainment than the overall MIDUS II sample but were comparable on other demographic factors (age, sex, race, income) and biomedical characteristics (subjective health, chronic conditions, health behaviors; Dienberg Love, Seeman, Weinstein, & Ryff, 2010). The mean age of the sample was 46.40 years and 56.80 years at the MIDUS I and II assessments, respectively. For the analyses reported here, we included 1,205 MIDUS II participants who had complete data on childhood SES and metabolic syndrome.

Childhood SES

Childhood SES was indexed with MIDUS I data on parental educational attainment. Values ranged from "some grade school" through "doctoral degree." When a respondent's parents had different levels of educational attainment, the higher value was used. For analysis, the sample was later stratified into four roughly equal groups. They corresponded to families in which the highest educational achievement consisted of (a) less than a high school diploma, (b) graduation from high school or its equivalent, (c) some college education with or without an associate's degree, and (d) a bachelor's degree from a 4-year institution (or higher level of education).

Metabolic outcomes

The outcome of interest was the presence of adult metabolic syndrome, for which the International Diabetes Federation provides a worldwide definition (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 Europid and African descent, who make up nearly all of the MIDUS sample, cutoffs are ≥ 94 cm 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 (i.e., systolic pressure ≥ 130 or diastolic pressure ≥ 85), (b) raised triglyceride levels (i.e., ≥ 1.7 mmol/L), (c) raised fasting-glucose levels (i.e., ≥ 5.6 mmol/L), and (d) low high-density lipoprotein levels (i.e., ≤ 0.9 mmol/L in men and ≤ 1.1 mmol/L in women).

Metabolic-syndrome components were assessed during GCRC visits. Waist circumference was measured at the narrowest point between the ribs and iliac crest. Blood pressure was assessed three times in a seated position, with 30-s intervals between measurements. The average of the two most similar readings was used for analysis. From fasting blood samples taken in the morning, lipid fractions and blood glucose were measured (using automated instruments from Roche Diagnostics, Indianapolis, IN).

For analyses, we constructed two outcome variables. One reflected the number of metabolic-syndrome components for

which the participant met clinical cutoffs; these could range from 0 to 5. The other was a binary variable reflecting whether the participant met the International Diabetes Federation case definition for metabolic syndrome.

Parental nurturance

Parental nurturance was assessed at MIDUS I with a validated questionnaire (Rossi, 2001), which contained seven questions regarding the quality of the parental relationships during childhood. Mothers and fathers were rated separately. Sample items included, "How much did she/he understand your problems and worries?" and "How much time and attention did she give you when you needed it?" Responses to the items were averaged. Both the maternal and parental composites showed high internal consistency (α = .91 and .92, respectively).

Potential confounds

We assessed several demographic and biobehavioral characteristics that might provide alternative explanations for any observed associations, and we modeled them as covariates. The demographic covariates included age, sex, and race, coded as dummy variables reflecting European (White) or African American (Black) descent. (In preliminary analyses, we determined that none of these covariates interacted with

childhood SES in predicting metabolic outcomes, ps > .14.) We also included current SES as a covariate, using a four-level education indicator with the same categories as used to assess childhood SES. This procedure controlled for the possibility that childhood SES was a proxy for more direct health effects of current SES. The biobehavioral covariates were binary indicators that reflected current smoking, history of diabetes, and history of cardiovascular disease.

Results
Preliminary analyses

Table 1 presents descriptive statistics for the sample across four levels of childhood SES. The groups differed on all predictors, covariates, and endpoints, with disparities favoring participants from more advantaged backgrounds. The exception was the scales tapping parental nurturance, in which values were nearly identical across the childhood SES strata.

Tests of hypotheses

Our first hypothesis was that low childhood SES would be associated with metabolic-syndrome outcomes in adulthood. To examine counts of metabolic-syndrome components, we conducted an analysis of covariance (ANCOVA) with

Table 1. Characteristics of the Sample as a Function of Childhood Socioeconomic Status, Indexed by Parental Educational Attainment

	Highest educational attainment by mother or father				
Variable	Less than high school diploma (n = 289)	High school graduate or equivalent (n = 419)	Some college education or associate's degree (n = 217)	Bachelor's degree or higher (n = 280)	p[a]
Age (years)	M = 58.92, SD = 12.01	M = 53.71, SD = 11.23	M = 54.61, SD = 12.04	M = 51.06, SD = 10.58	< .001
Female (%)	58.80	51.80	62.70	55.70	.05
White (%)	68.50	81.90	82.90	89.60	< .001
Black (%)	26.60	15.00	11.50	7.10	< .001
High school graduate (%)	89.10	94.30	98.10	98.60	< .001
Bachelor's degree or higher (%)	27.80	34.10	48.60	68.60	< .001
Current smoker (%)	16.30	14.60	14.30	7.50	.01
History of cardiovascular disease (%)	22.10	13.80	11.10	12.50	< .001
History of diabetes (%)	14.20	9.30	8.80	7.50	.04
Maternal warmth (1–4)	M = 3.16, SD = 0.74	M = 3.14, SD = 0.74	M = 3.10, SD = 0.71	M = 3.10, SD = 0.65	.80
Paternal warmth (1–4)	M = 2.67, SD = 0.84	M = 2.68, SD = 0.83	M = 2.74, SD = 0.77	M = 2.74, SD = 0.75	.67
Metabolic-syndrome components (0–5)	M = 2.53, SD = 1.31	M = 2.29, SD = 1.35	M = 2.21, SD = 1.25	M = 1.93, SD = 1.38	< .001
Metabolic-syndrome diagnosis (%)	49.80	41.80	39.20	31.10	< .001

[a]The p values in this column derive from analyses comparing the four childhood-socioeconomic-status groups (one-way analyses of variance for continuous outcomes and χ^2 analyses for categorical outcomes).

childhood SES as the between-subjects variable and the same demographic and biobehavioral factors used in preliminary analyses as covariates. We found that participants with less education and a diabetes history had significantly higher counts of metabolic-syndrome components than participants with more education and no history of diabetes (see Table S1 in the Supplemental Material available online). There were also significant differences in counts of metabolic-syndrome components as a function of childhood SES, $F(3, 1183) = 3.89$, $p = .009$ (see Fig. 1a). Follow-up contrasts showed that individuals from the most disadvantaged households (i.e., from families in which neither parent had a high school diploma) had significantly higher counts relative to participants from other socioeconomic backgrounds ($p = .01$). Similarly, individuals who were raised in the most advantaged circumstances (i.e., in families in which one or both parents had a bachelor's degree) had significantly lower counts of metabolic-syndrome components than other participants ($p = .002$).

To examine whether childhood SES also predicted diagnosable metabolic syndrome, we computed a binary logistic regression with diagnostic status as the outcome and with the same predictor and covariates as in the previous analysis (see Fig. 1b; see also Table S2 in the Supplemental Material). Again, men and participants with less education and a diabetes history were more likely to have diagnosable metabolic syndrome than women and participants with more education and no diabetes history. There were also significant differences in metabolic-syndrome prevalence as a function of childhood SES (Wald = 11.29, $p = .01$). Metabolic syndrome was most common among participants from households in which neither parent completed high school. Contrasts showed that this disadvantaged population had a higher prevalence of metabolic syndrome compared with participants whose parents had earned high school diplomas ($p = .05$) and bachelor's degrees ($p = .001$). Disadvantaged participants also had a marginally higher prevalence of metabolic syndrome than participants from households in which parents had some college ($p = .09$).

Our second hypothesis was that a sizable minority of individuals from low-SES backgrounds would prove to be resilient to metabolic syndrome. This was indeed the case. As Table 1 shows, metabolic-syndrome prevalence in participants whose parents lacked a high school diploma was 50%, meaning that half of the most disadvantaged participants were free of the condition at midlife. A similar pattern was apparent in covariate-adjusted models, in which metabolic-syndrome prevalence was estimated at 56% in participants whose parents lacked a high school diploma (vs. 48%, 48%, and 39% in the high school graduate or equivalent, some college or associate's degree, and bachelor's degree and higher groups, respectively). Looking at counts of metabolic-syndrome components, we found that 4.5% of the most disadvantaged participants had a score of 0, which means that there were no metabolic-syndrome components for which they met clinical cutoffs (results for the high school graduate or equivalent, some college or associate's degree, and bachelor's degree and higher groups were 9%, 9%, and 16%, respectively).
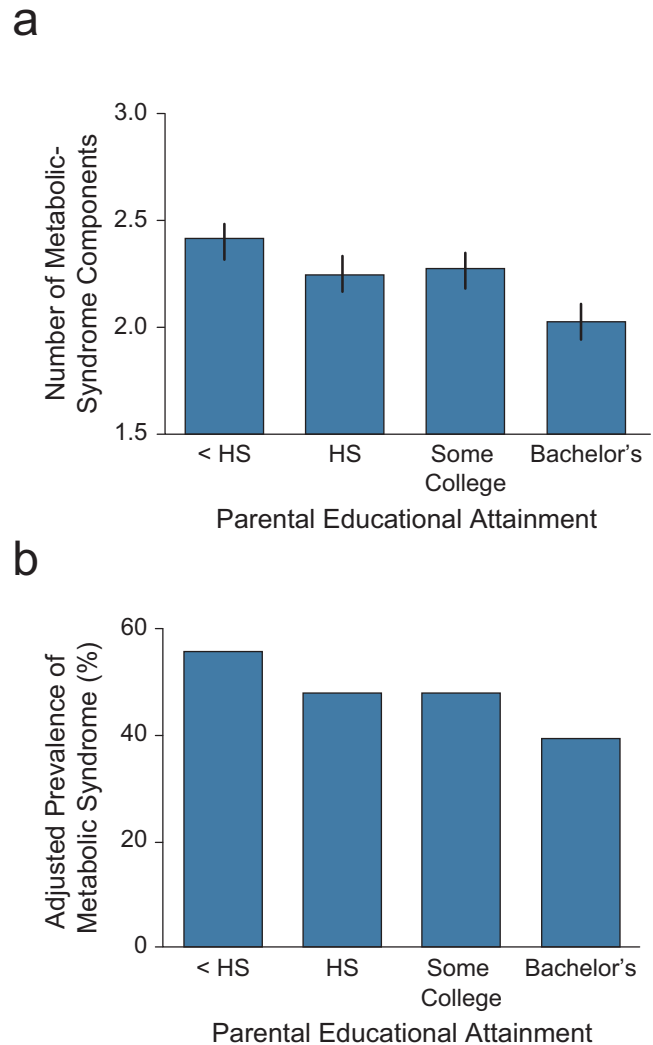
Fig. 1. Metabolic-syndrome outcomes as a function of childhood socioeconomic status (indexed by parents' highest level of education). Results are shown for (a) the mean number of metabolic-syndrome components and (b) the prevalence of diagnosable metabolic syndrome. In both cases, estimates have been adjusted for demographic and biobehavioral covariates. Parental educational attainment was divided into four categories: less than high school education (< HS), high school graduate or equivalent (HS), some college education or associate's degree (some college), and bachelor's degree or higher (bachelor's). Error bars show ±1 *SEM*.

Our third hypothesis was that upward social mobility and parental nurturance would be associated with resilience to the health effects of low childhood SES. To examine social mobility, we split the sample into four groups reflecting SES in childhood (low vs. high) and SES in adulthood (low vs. high). Categorization in these two cases was based on educational attainment, with high school diploma or less coded as relatively low and any college education or more coded as relatively high. Labels were devised for the four groups that indicated the two SES variables (e.g., "low-low" means that SES was low in childhood and low in adulthood). An ANCOVA was then computed with SES trajectory as a between-subjects variable and with the same covariates as in the previous analyses (see Fig. 2a; see also Table S1 in the Supplemental Material).
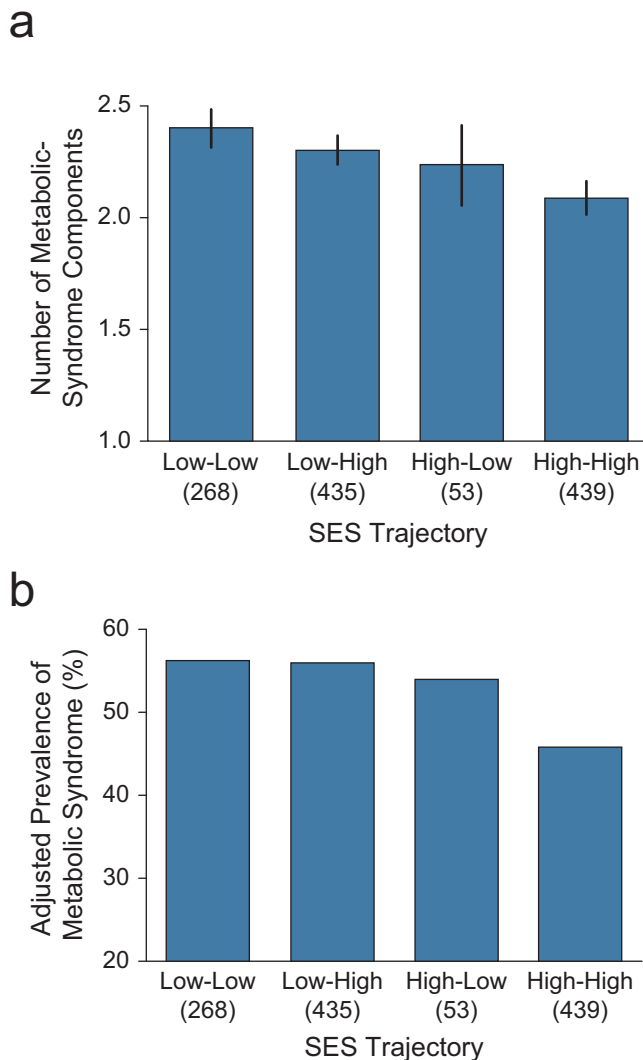
a

b

Fig. 2. Metabolic-syndrome outcomes as a function of socioeconomic status (SES) trajectory (indexed by childhood SES and adulthood SES). Results are shown for (a) the mean number of metabolic-syndrome components and (b) the prevalence of diagnosable metabolic syndrome. In both cases, estimates have been adjusted for demographic and biobehavioral covariates. In the labels along the *x*-axes, the first term refers to childhood SES (i.e., parents' education), and the second refers to adulthood SES (i.e., respondent's education); group sizes are given in parentheses. Error bars show ±1 *SEM*.

In this analysis, there were significant differences across groups, $F(3, 1183) = 3.87$, $p = .009$. Subjects with stably low SES (low-low) had the highest counts of metabolic-syndrome components, and subjects with stably high SES (high-high) had the lowest counts. The other groups fell in between, though they were closer in number to the low-low group. To more clearly examine social mobility, we performed contrast analyses comparing upwardly mobile (low-high) participants with other groups. Even though these individuals made large SES gains over their lifetimes, in terms of metabolic-syndrome components, they were statistically equivalent to low-low individuals ($p = .35$). In addition, they had significantly higher counts than high-high individuals did ($p = .01$). Supplemental

analyses showed that low-low and high-low individuals did not differ ($p = .40$), though the latter group's size ($n = 53$) limits power for this comparison.

These patterns were mirrored in diagnosable metabolic syndrome (see Fig. 2b; see also Table S2 in the Supplemental Material), whose prevalence varied across groups (Wald = 9.18, $p = .03$). Metabolic syndrome was most prevalent among groups with stably low SES, and it was least prevalent among groups with stably high SES. It is critical to note that for the social-mobility hypothesis, participants in the low-high and low-low categories were statistically indistinguishable ($p = .90$). Further, low-high participants had a higher prevalence of metabolic syndrome than high-high participants did ($p = .01$). There were no differences in prevalence of metabolic syndrome between low-low and high-low individuals ($p = .79$), but again power is an issue here.

We ran two other sets of analyses to ensure that these results were robust. In one set, we adopted a stricter definition of social mobility, which required participants to have obtained a university degree to qualify for the high-SES category. In the other set, we treated both childhood and adulthood SES as four-level indicators, using the same educational categories as previously, and looked for an interaction between them. The results of both analyses mirrored the results of the previous analyses; there was little evidence that upward mobility offset the metabolic-syndrome risks associated with low childhood SES.

To examine the role of parental nurturance, we constructed regression equations, in which metabolic-syndrome counts were predicted from three successive blocks of variables: the set of covariates, variables reflecting main effects of childhood SES and parental nurturance, and a product term representing the interaction of the latter two constructs. The results of the maternal-nurturance model revealed a significant interaction ($b = 0.09$, $SE = 0.04$, $p = .03$, $\Delta R^2 = .005$). To interpret this finding, we plotted estimated counts of metabolic-syndrome components according to standard practices (Aiken & West, 1991). As Figure 3 shows, the interaction was consistent with a buffering influence of maternal nurturance (also see Table S3 in the Supplemental Material). Childhood disadvantage was associated with higher counts of metabolic-syndrome components among participants who reported lower maternal nurturance (simple-slopes analysis, $p < .001$). But this association became progressively weaker as participants reported higher levels of maternal nurturance. In fact, among participants who reported maternal nurturance 1 standard deviation above the sample mean, the association between childhood SES and metabolic-syndrome components was nonsignificant (simple-slopes analysis, $p = .47$).

When a similar model was estimated for paternal nurturance, it did not yield evidence of a buffering influence (Childhood SES × Paternal Nurturance interaction, $b = 0.05$, $SE = 0.04$, $p = .28$; Table S5 and Fig. S1 in the Supplemental Material). This finding was not likely due to missing data, as 91% of participants completed paternal-nurturance ratings. Finally, in logistic regressions predicting metabolic-syndrome diagnosis, neither
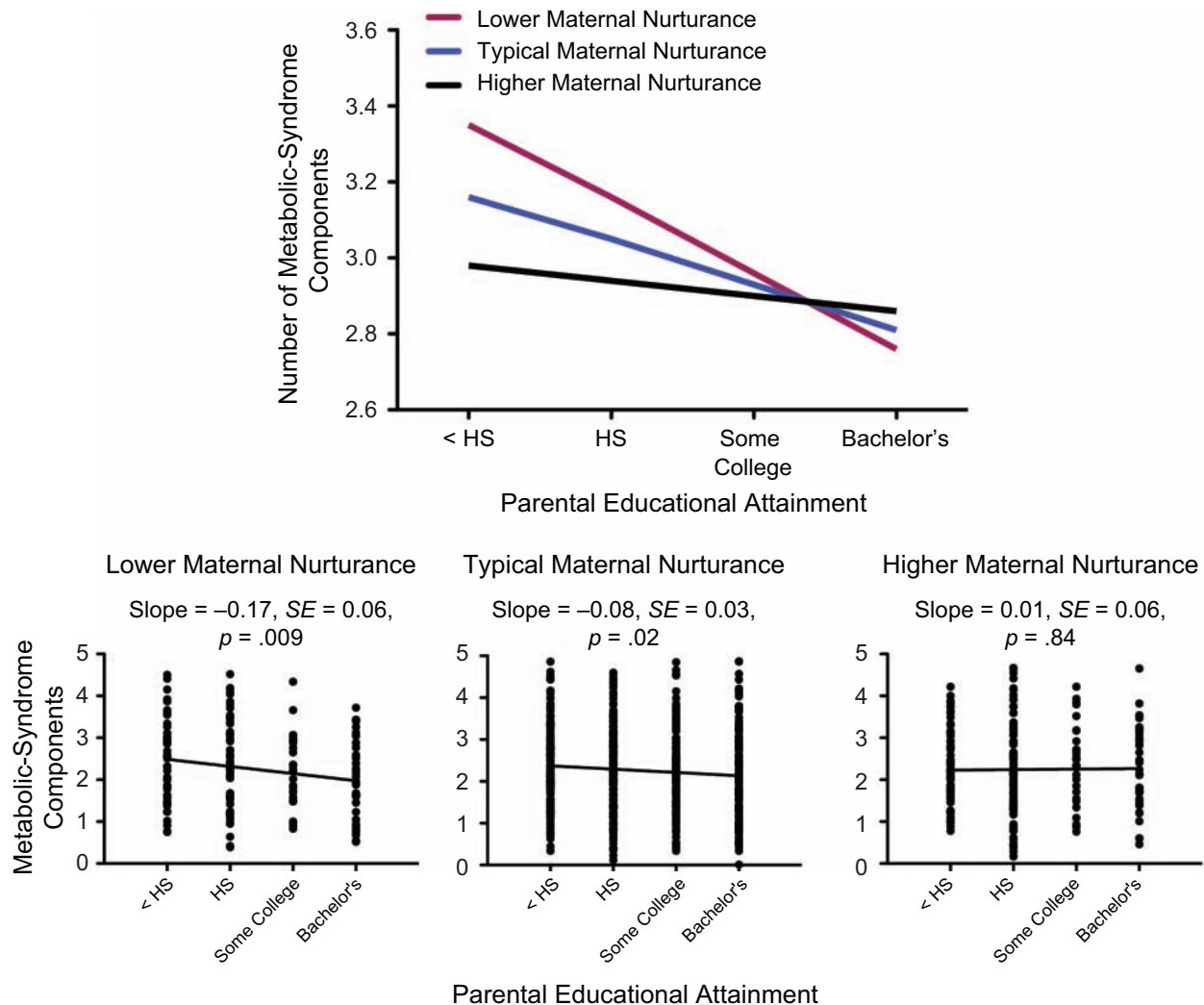
Fig. 3. Metabolic-syndrome components as a function of childhood socioeconomic status (SES; indexed by parents' level of education) and maternal nurturance. The top panel shows the estimated number of metabolic-syndrome components at three levels of maternal nurturance: low (1 *SD* below the mean), typical (mean), and high (1 *SD* above the mean). Values have been adjusted for demographic and biobehavioral covariates. The bottom panel displays results for covariate-adjusted counts of metabolic-syndrome components for three groups of participants, stratified according to whether they fell into the highest, middle, or lowest tertile of the sample distribution of maternal nurturance. Each graph shows the distributions for individual subjects, as well as a best-fitting regression line. Parental educational attainment was divided into four categories: less than high school education (< HS), high school graduate or equivalent (HS), some college education or associate's degree (some college), and bachelor's degree or higher (bachelor's).

maternal nor paternal nurturance interacted with childhood SES (*p*s > .34; Tables S4 and S6 in the Supplemental Material).

Supplemental analyses

As noted, sibling pairs are nested within the MIDUS data. Their presence violates the randomness assumption inherent in many statistical models, in which observations within a sample are assumed to be independent and identically distributed. To determine whether the siblings affected our results, we redid analyses using generalized estimating equations (Hanley, Negassa, Edwardes, & Forrester, 2003), specifying an exchangeable covariance matrix. This specification permits the independent-and-identically-distributed assumption to be relaxed. In all cases, the conclusions emanating from these

models were identical to those found in our previous analyses; this suggests that familial clustering within MIDUS did not bias the results. As a second way to evaluate this issue, we redid the analyses after randomly selecting one sibling per family for inclusion. In this subset, we continued to observe links between childhood SES and midlife metabolic syndrome, without any apparent offsetting by upward mobility. We also observed a buffering effect related to maternal nurturance. However, with 153 participants (12.70% of the sample) dropped from the analyses, the interaction term was only marginally significant (*b* = 0.07, *SE* = 0.04, *p* = .08). Nonetheless, plots of the effect revealed it to be identical to the one shown in Figure 3.

Experts disagree about how to best identify metabolic syndrome, and several groups have offered diagnostic guidelines

(Cornier et al., 2008). To ensure that our results were robust, we redid the analyses identifying metabolic syndrome by the standards of the National Cholesterol Education Program—Adult Treatment Panel III, the other commonly used guideline in North America. The analyses yielded identical conclusions.

At the time of MIDUS II, some participants already had chronic diseases to which metabolic syndrome contributes. To determine whether our findings reflected susceptibility to metabolic syndrome per se, we reconducted analyses after excluding participants with coronary heart disease and diabetes. Even though these analyses had less statistical power, they yielded identical conclusions.

Discussion

We found that lower childhood SES was associated with poorer metabolic outcomes at midlife in a large cohort from across the United States. These disparities were fairly large in magnitude. Participants raised in families in which neither parent finished high school were 1.4 times as likely to have metabolic syndrome than their peers raised in households in which parents were university educated. The prevalence of metabolic syndrome among participants whose parents earned only high school diplomas or had only some college education fell in between. This pattern of findings converges with the results of previous research on the socioeconomic origins of metabolic syndrome, which has generally reported greater prevalence of the condition among individuals reared in disadvantaged families, particularly when those individuals are women (Chichlowska et al., 2009; Langenberg, Kuh, Wadsworth, Brunner, & Hardy, 2006; Lawlor, Ebrahim, & Davey Smith, 2002; but for exceptions, see Lucove, Kaufman, & James, 2007; Parker et al., 2003).

Despite these trends, not all participants from low-SES backgrounds had metabolic syndrome at midlife. In fact, nearly half of them proved resilient to metabolic syndrome, a result consistent with findings in other disease contexts (Chichlowska et al., 2009; Cohen et al., 2004; Dong et al., 2004). (That said, some participants free of metabolic syndrome could have other medical problems, which raises questions about whether they were resilient to disease in a more general sense.) To identify characteristics associated with resilience to metabolic syndrome, we examined the influence of upward social mobility, but analyses suggested it offered little in the way of metabolic advantages. Indeed, metabolic-syndrome prevalence was equivalent in low-low and low-high participants (56% vs. 55%, respectively), despite the latter individuals having made substantial gains in SES over their lifetimes. These findings converge with results from a large study of older British women, which found that upward mobility did little to offset the metabolic risks of childhood poverty (Lawlor et al., 2002). Benefits of upward mobility have sometimes been seen in studies of other health outcomes, but these benefits are typically modest in size (Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002; Pensola & Martikainen, 2003;

Power, Hypponen, & Smith, 2005; Smith, Hart, Blane, & Hole, 1998).

Collectively, these findings suggest that socioeconomic disadvantage may get embedded in children's physiology in a manner that is not reversed by a transition into more favorable circumstances. It remains unclear what exactly this residue of childhood adversity is and how it contributes to the emergence of metabolic syndrome decades later. Biological programming of systems that regulate neuroendocrine or inflammatory signaling is one plausible mechanism. Children from low-SES families go on to have elevated cortisol output in adulthood, irrespective of their achieved social standing (Li, Power, Kelly, Kirschbaum, & Hertzman, 2007; Miller et al., 2009). Persistent exposure to high cortisol can facilitate the development of metabolic-syndrome components, such as central adiposity, insulin resistance, and high blood pressure (Bjorntorp & Rosmond, 1999). Inflammation is another candidate mechanism of embedding. Youths raised in low-SES families go on to have mild, chronic inflammation as adults (Phillips et al., 2009). This embedding is sustained by selective modulation of gene networks in white blood cells, marked by activation of proinflammatory and repression of anti-inflammatory signaling pathways (Miller et al., 2009). Inflammation plays a key pathogenic role in metabolic syndrome (Hotamisligil, 2006) and thus could be a mechanism underlying the findings seen here.

We also examined parental nurturance as a correlate of resilience to metabolic syndrome. These analyses suggested a buffering scenario, in which maternal nurturance offset the metabolic consequences of childhood disadvantage. The benefits were evident across the spectrum of maternal nurturance. As participants recalled higher levels of nurturance, the association between childhood SES and metabolic-syndrome components became progressively weaker. It is not clear why this pattern did not extend to diagnosable metabolic syndrome. Because of the binary nature of this outcome, the analysis may have lacked sufficient power. Nevertheless, the analyses focused on components suggest that maternal nurturance is an important contributor to metabolic-syndrome resilience among persons from low-SES backgrounds. In this manner, our results converge with evidence that nurturant parenting has educational, psychosocial, and biobehavioral benefits for children facing adversity (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2007; Luthar, 2006; Masten, 2001). Our findings extend this corpus of evidence to components of a health problem, metabolic syndrome, that typically manifests decades later in the middle years of adulthood.

It remains unclear how maternal nurturance buffers against the long-term health effects of childhood disadvantage. Nurturant caregivers imbue children with the sense that the world is a safe place and other people can be trusted (Cassidy & Shaver, 2008). These beliefs may enable disadvantaged youngsters to read less threat into their social worlds, with a consequent reduction in the wear and tear that such vigilance can place on bodily systems (Chen & Miller, 2011). Nurturant parents also help children to learn emotion-regulation strategies, so that

when they do encounter stress, the physiological consequences are attenuated (Repetti, Taylor, & Seeman, 2002).

Mechanistically, the benefits of nurturance could occur through increased expression of oxytocin, a peptide released when people experience warmth and security (Grewen, Girdler, Amico, & Light, 2005). Research in animals suggests that oxytocin counteracts some pathogenic mechanisms involved with metabolic syndrome (Camerino, 2009). Future research that elucidates the role of these presumptive mechanisms would be valuable. It also will be important to understand why paternal nurturance does not seem to confer lasting health benefits. Mothers could have a unique influence on their offspring's health trajectories. Alternatively, our findings could reflect a cohort effect. MIDUS participants were generally born after World War II, when cultural norms were such that fathers were not heavily engaged in childrearing. Shifting cultural norms may also explain why childhood SES and parental nurturance were unrelated in this cohort, whereas they are positively associated in today's young families (Repetti et al., 2002).

Our study has three principal limitations. First, the observational design makes it impossible to render causal inferences. By covarying out a host of demographic and biobehavior confounds, we have addressed some plausible alternative interpretations. However, this strategy is fallible when relevant covariates are overlooked or mismeasured. That said, there are a host of animal studies that have manipulated early stress experimentally and documented lasting health consequences (Avitsur, Hunzeker, & Sheridan, 2006; Kruschinski et al., 2008); results of these studies prove that effects such as those seen here can be causal in nature.

A second limitation is the study's retrospective assessments. Some participants will surely have misreported their childhood SES, and these errors would lead to misestimation of associations. With parental nurturance, misreporting seems even more likely, as participants may provide what they viewed as socially desirable responses. Even if they were not attempting to do so, their reports are likely reconstructed versions of family life rather than veridical representations of what actually transpired. That said, such reports predict the occurrence of important biomedical outcomes, both here and elsewhere (Dong et al., 2004). Thus, research is needed to identify what exactly these scales are tapping and how it confers protection against disease.

Finally, our analyses focused on a single dimension of SES (parental education) and how it relates to a single biomedical outcome (metabolic syndrome). Future research will need to determine whether similar patterns emerge when other SES indicators are considered in relation to other disease outcomes. For example, analyses of upward mobility in other SES domains, such as wealth, might yield stronger evidence of health benefits or other relevant life outcomes.

Rates of childhood poverty have been climbing steadily in the United States, fueling concerns about the long-term political, economic, and biomedical costs. Against this backdrop, our findings provide some cause for optimism, documenting an impressively high rate of resilience to childhood economic adversity and identifying maternal nurturance as a factor that promotes such resilience. Research that identifies other factors that promote resilience, especially ones that are amenable to intervention, would be an extremely valuable contribution to America's future. That said, the SES gradient arises from a complex mixture of historical, political, economic, social, and biological forces. To ameliorate it, researchers will need multifaceted interventions that operate at multiple junctures along the causal pathway.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://pss.sagepub .com/content/by/supplemental-data

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