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How Low Socioeconomic Status Affects 2-Year Hormonal Trajectories in Children

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Abstract

Disparities by socioeconomic status (SES) are seen for numerous mental and physical illnesses, and yet understanding of the pathways to health disparities is limited. We tested whether SES alters longitudinal trajectories of cortisol output and what types of psychosocial factors could account for these links. Fifty healthy children collected saliva samples (four times per day for 2 days) at 6-month intervals for 2 years. At baseline, families were interviewed about SES and psychosocial factors. Lower-SES children displayed greater 2-year increases in daily cortisol output compared with higher-SES children. These effects were partially mediated by children's perceptions of threat and by family chaos. These findings may help explain, and provide some first steps toward ameliorating, low-SES children's vulnerability to health problems later in life by identifying the tendency to perceive threat in ambiguous situations and experiences of chaos as factors that link low SES to 2-year hormonal trajectories.

Keywords

socioeconomic status, hormones, family, appraisal

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Low socioeconomic status (SES) has a profound effect on a wide variety of mental and physical health outcomes. For example, individuals from a lower SES background are at increased risk for depression, anxiety, and substance use (Dohrenwend et al., 1992; Johnson, Cohen, Dohrenwend, Link, & Brook, 1999), as well as more physical health problems, including obesity, injuries, and subclinical cardiovascular disease (Adler et al., 1994; Chen, Matthews, & Boyce, 2002; Goodman, 1999).

Researchers have speculated that one reason for these associations is that living in low-SES environments takes a toll on certain physiological systems that are sensitive to social environments, which in turn can affect mental and physical health. A substantial body of literature documents that social factors such as low SES and stress are able to activate the hypothalamic-pituitary-adrenal (HPA) axis, which produces the hormone cortisol, and to alter daily-life cortisol profiles in both healthy adults and healthy children (Cohen, Doyle, & Baum, 2006; Evans & English, 2002; Kunz-Ebrecht, Kirschbaum, & Steptoe, 2004; Miller, Chen, & Zhou, 2007). In turn, HPA disturbances have been implicated in a number of psychiatric disorders and physical ailments, including depression, posttraumatic stress disorder, diabetes, and obesity (Bjorntorp & Rosmond, 1999; Epel et al., 2000; Parker, Schatzberg, & Lyons, 2003; Yehuda, 2002).

However, studies of SES and cortisol to date have largely used cross-sectional designs. Rarely have studies tested whether SES can predict trajectories of cortisol over time, yet it is these longer-term associations that would have implications for health, as they would indicate persistent biological effects of low SES. Longitudinal designs also provide more convincing evidence about the directionality of effects. Hence, in the present study, we tested associations between SES and 2-year cortisol trajectories, hypothesizing that living in a low-SES environment would increase cortisol trajectories over time. Furthermore, given the importance of identifying risk factors earlier in life, we focused on a sample of children.

A second goal of the present study was to better understand why SES would be linked to biological profiles. We evaluated two possible psychosocial explanations. First, there is evidence that lower-SES children have different ways of perceiving their social world. Lower-SES children are more likely to grow up in environments where negative life events can happen in an unpredictable fashion. As a result, lower-SES children may be more likely to develop a heightened sense of vigilance for threat. In earlier work, we hypothesized that this vigilance would differentiate children during ambiguous life situations; that is, lower-SES children would be more likely to interpret ambiguous situations as threatening. In keeping with

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Table 1. Descriptive Information About the Sample

Age: *M* = 13.18 years, *SD* = 2.20
Gender: 55% male, 45% female
Ethnicity: 67% Caucasian, 19% Asian, 14% other
Family savings[a]: *M* = 4.84, *SD* = 2.46
Family owns a home: 69%
Smoker: 6%
Pubertal status[b]: *M* = 3.24, *SD* = 1.27
Body mass index: *M* = 20.79 kg/m^2, *SD* = 3.74
Cortisol[c]: *M* = 11.51 nmol/L, *SD* = 3.20

[a]Family savings was coded from 1 to 9. The mean value for this sample corresponded to the category from $10,000 to $19,999. [b]Pubertal status was assessed with the Petersen Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). Scores ranged from 1, *prepubertal*, to 5, *postpubertal.* The mean value for this sample corresponded to the midpuberty category. [c]Mean cortisol value refers to the log-transformed average area under the curve across the five visits (see the text for more information).

this hypothesis, we found that lower SES was associated with greater threat interpretations during ambiguous, but not negative, social scenarios (Chen & Matthews, 2003). In the present study, we hypothesized that one effect of perceiving ambiguous situations in a more threatening manner would be heightened physiological responses, that is, increased cortisol output during daily life.

Second, we hypothesized that low SES would also affect the experiences children have in their home environment. Lower-SES families experience greater chaos in their day-to-day lives, in terms of greater crowding, greater noise exposure, and less predictable routines (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005). We hypothesized that this experience of more chaotic environments would also take a physiological toll and increase cortisol trajectories over time in low-SES children.

Method

Participants

Fifty-four healthy children were recruited for a 2-year longitudinal study in Vancouver, British Columbia, from newspaper advertisements and school and community center postings. Children ranged in age from 9 to 18 years, were fluent in English, had no chronic illnesses, and were free of acute respiratory illness at the time of their visit (see Table 1). Four children dropped out after completing only the baseline visit and were excluded from study analyses, leaving a total of 50 children in this study.

Measures

Family SES. SES resources were measured by two questions to the parent, which involved the amount of assets that families could easily convert to cash in an emergency (family savings) and whether the family owned their own home. These measures are used by the MacArthur Research Network on Socioeconomic Status and Health (www.macses.ucsf.edu), are standard measures in SES research (Krieger, Williams, & Moss, 1997), and parallel measures we have used in previous articles (Chen et al., 2006; Cohen, Doyle, Turner, Alper, & Skoner, 2004).

Psychosocial measures. We used two psychosocial measures. (a) Children's perceptions of threat were measured with the Cognitive Appraisal and Understanding of Social Events videos (Chen & Matthews, 2003). These videos depict ambiguous life situations (e.g., being closely attended to by a store clerk). Children were probed about their interpretations, which were then coded by raters on a 5-point scale, with higher numbers reflecting greater perceived threat, given an identical situation. Reliability and validity of this measure have been documented (Chen & Matthews, 2003). (b) Parents completed the Confusion, Hubbub, and Order Scale (Matheny, Wachs, Ludwig, & Phillips, 1995), a 15-item questionnaire that probes the degree of orderliness and routines in the household. Questions are answered using a true/false format. Higher scores indicate more chaotic, disorganized households. Reliability and validity have previously been established (Matheny et al., 1995).

Salivary cortisol. Saliva samples were collected at home (see Procedure section); 88.2% of all samples were collected within 1 hr of the instructed time. Samples were centrifuged at 800 g for 5 min, transferred to deep-well plates, and stored at −30 °C until assayed. Free cortisol levels in saliva were measured in duplicate using a commercially available chemiluminescence assay (IBL; Hamburg, Germany). Inter- and intra-assay variation was less than 10%.

Covariates

Factors that might influence cortisol were assessed and included as covariates, including pubertal status, assessed by the Petersen Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988); body mass index (BMI), calculated from height and weight taken from a standard medical balance beam scale; and smoking status, defined by whether a child had smoked in the last 6 months (no child had smoked more than 10 cigarettes during this period).

Procedure

Children and parents completed psychosocial and SES measures in the laboratory at baseline. Subsequently, children collected saliva samples for 2 days using Salivette devices (Sarstedt; Nuembrecht, Germany). Each day, saliva was collected 1, 4, 9, and 11 hr after awakening, as recommended by the MacArthur Research Network on Socioeconomic Status and Health (www.maces.ucsf.edu). MEMS 6 TrackCap

monitors (Aardex Ltd., Zug, Switzerland) were used to track actual saliva-collection times. Every 6 months, children repeated the saliva-collection procedures, for a total of five assessments across a 2-year period (0, 6, 12, 18, and 24 months).

Statistical analyses

Cortisol data were first log-transformed to reduce skew. Daily cortisol output was calculated via an area-under-the-curve (AUC) statistic using the trapezoidal rule. Cortisol values were modeled as a function of hours since awakening for each participant, based on actual sample collection times from the MEMS-Cap. AUC values were averaged across the 2 days ($r = .65$).[1]

Data were analyzed using hierarchical linear modeling (HLM). In the within-person (Level 1) model, we estimated cortisol AUC as a function of months since study enrollment, which resulted in a series of person-specific slopes and intercepts. In the between-person (Level 2) models, we estimated these person-specific slopes and intercepts as a function of factors that vary across people and random error terms. Covariates were entered as Level 2 variables before entering SES. Potential mediators were tested by simultaneously entering mediator variables (threat perceptions, chaos), together with SES and the covariates at Level 2. HLM can accommodate missing data points, and, hence, participants were included even if they had missing data; 80% of children completed data collection for three or more time points.

Results

Preliminary analyses

Children who dropped out after the first visit did not differ from those who stayed in the study on the basis of age, gender, or family home ownership, although they were lower on family savings, $t(51) = 2.88$, $p < .01$.

Over the 2-year study period, children's cortisol levels increased ($b = 1.937$, $SE = 0.2306$, $p < .001$). There was variability around the slope (variance = 0.1271, $p = .08$), and intercept (variance = 1.929, $p < .05$). BMI, smoking status, and pubertal status did not predict cortisol trajectories or intercepts (see Table 2).[2]

Table 2. Hierarchical Linear Modeling Analysis Predicting Cortisol Area-Under-the-Curve Trajectories Over the 2-Year Study Period

Level, model, and predictor	*b*	*SE*	*p*
Level 1 (within person)			
Model 1			
Time	1.9375	0.2306	< .001
Level 2 (between persons)			
Model 2			
Body mass index	0.0317	0.0611	.61
Smoking status	−0.4374	1.7384	.80
Pubertal status	0.1489	0.1664	.38
Model 3			
Body mass index	0.0217	0.0530	.68
Smoking status	−0.8999	1.5818	.57
Pubertal status	0.1805	0.1658	.28
Family savings	−0.2020	0.0944	.04
Model 4			
Body mass index	0.0193	0.0617	.76
Smoking status	−0.7122	1.5462	.65
Pubertal status	0.1070	0.1746	.54
Home ownership	1.1301	0.4622	.02
Model 5			
Body mass index	0.0381	0.0558	.50
Smoking status	−0.8972	1.7118	.60
Pubertal status	0.1544	0.1860	.41
Child threat interpretations	0.1551	0.2465	.53
Family savings	−0.1668	0.1298	.21
Model 6			
Body mass index	0.0232	0.0514	.65
Smoking status	−0.9800	1.6530	.56
Pubertal status	0.1466	0.1636	.38
Family chaos	0.1121	0.0600	.07
Family savings	−0.1557	0.1100	.16
Model 7			
Body mass index	0.0342	0.0616	.58
Smoking status	−0.7416	1.6450	.65
Pubertal status	0.0808	0.1770	.65
Child threat interpretations	0.1917	0.2377	.42
Home ownership	0.9281	0.6260	.14
Model 8			
Body mass index	0.0203	0.0581	.73
Smoking status	−0.8296	1.6061	.61
Pubertal status	0.0893	0.1717	.60
Family chaos	0.1143	0.0596	.06
Home ownership	0.8953	0.4938	.08

Note: For Models 2 through 8, the table presents the coefficients for the prediction of cortisol slopes over time. Model 2 includes only covariates. Models 3 and 4 include socioeconomic status variables. Models 5 through 8 include psychological mediators together with socioeconomic status variables.

SES and cortisol trajectories

We next tested whether family SES explained variation in cortisol slopes or intercepts over time (see Table 2). Family savings significantly predicted changes in cortisol AUC slopes over 2 years ($b = –0.2020$, $SE = 0.0944$, $p < .05$), such that children from lower-SES households showed increasing daily cortisol output over time. The between-person variance in cortisol trajectories dropped from 0.28 to 0.07, or 75%, when family savings was included, relative to when only covariates were included. To illustrate the association, we plotted cortisol trajectories for children low (at the 25th percentile) versus high (at the 75th percentile) in family savings (see Fig. 1). Cortisol output rose 40% more over 2 years in low-SES children compared with high-SES children. In contrast, family savings was less strongly related to cortisol intercepts (i.e., values at study entry; $b = 0.3391$, $SE = 0.1738$, $p < .1$).

In a separate model testing home ownership rather than family savings, children from families who rented homes showed increasing daily cortisol output over time compared

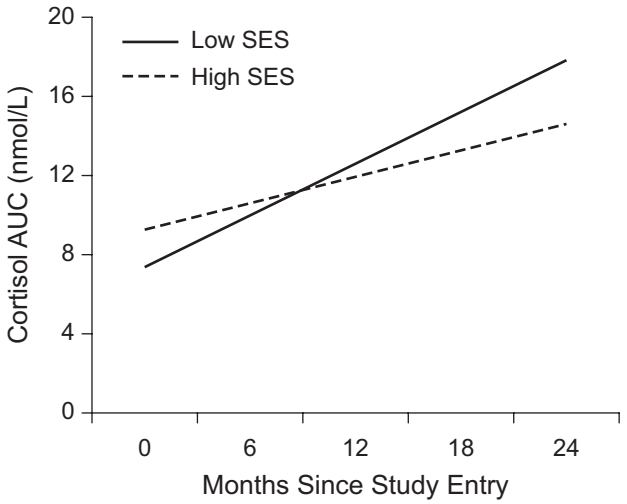

Fig. 1. Cortisol trajectories across the 2-year study period for children at the 25th percentile for socioeconomic status (low SES) and children at the 75th percentile for socioeconomic status (high SES). AUC = area-under-the-curve statistic.

with children from families who owned homes (b = 1.1301, SE = 0.4622, $p < .05$; Table 2). Cortisol output rose 55% more in low-SES children compared with high-SES children. The between-person variance in cortisol trajectories dropped from 0.28 to 0.08, or 71%, when home ownership was included. Home ownership also predicted cortisol intercepts (b = –2.3765, SE = 0.9950, $p < .05$), accounting for 35% of the between-person variance in values at study entry.

Perceptions of threat as a mediator

Less family savings was associated with greater child threat interpretations, $r(49) = –.47$, $p < .01$. Similarly, children living in rented homes made greater threat interpretations, $t(47)$ = 2.88, $p < .01$ (see Fig. 2). In turn, greater threat interpretations predicted larger increases in cortisol AUC over time (b = 0.3698, SE = 0.1853, p = .05).

After we added threat interpretations to the model, the relationship between family savings and children's cortisol trajectories became nonsignificant (b = –0.1668, SE = 0.1298, $p > .2$; see Table 2). To determine the magnitude of the effect that was accounted for by this mediator, we first computed effect sizes for the relationship between family savings and cortisol trajectories by converting the t statistic for this effect into a Cohen's d effect size. We then compared the effect size of the relationship between family savings and cortisol trajectories, controlling for the proposed mediator, with the effect size of the relationship between family savings and cortisol trajectories with no mediator in the equation. When threat interpretations were included, the effect size for the relationship between family savings and cortisol trajectories dropped from 0.62 (with no mediator) to 0.37 (with threat interpretations included). This represented a reduction of 40% [(0.62 – 0.37)/0.62]. Similarly, with threat interpretations in the model, the relationship between home ownership and cortisol trajectories became nonsignificant (b = 0.9281, SE = 0.6260, p = .14), and the effect size dropped from 0.71 to 0.43, or 39%.

Chaos as a mediator

Fewer family savings were associated with greater chaos, $r(49) = −.29$, $p < .05$. Similarly, families in rented homes had greater chaos, $t(47) = 2.32$, $p < .05$ (see Fig. 2). In turn, greater family chaos predicted greater increases in children's cortisol AUC over time (b = 0.1651, SE = 0.0514, $p < .01$).

With chaos and family savings in the model, the relationship between family savings and children's cortisol trajectories became nonsignificant (b = −0.1557, SE = 0.1100, $p > .15$; see Table 2), and the effect size dropped from 0.62 to 0.41, or 34%. Similarly, the relationship between home ownership and cortisol was marginal when chaos was included (b = 0.8953, SE = 0.4938, $p < .10$), and the effect size dropped from 0.71 to 0.53, or 25%.

With both family chaos and child threat interpretations (r = .19) in the model, the SES-cortisol relationship was not

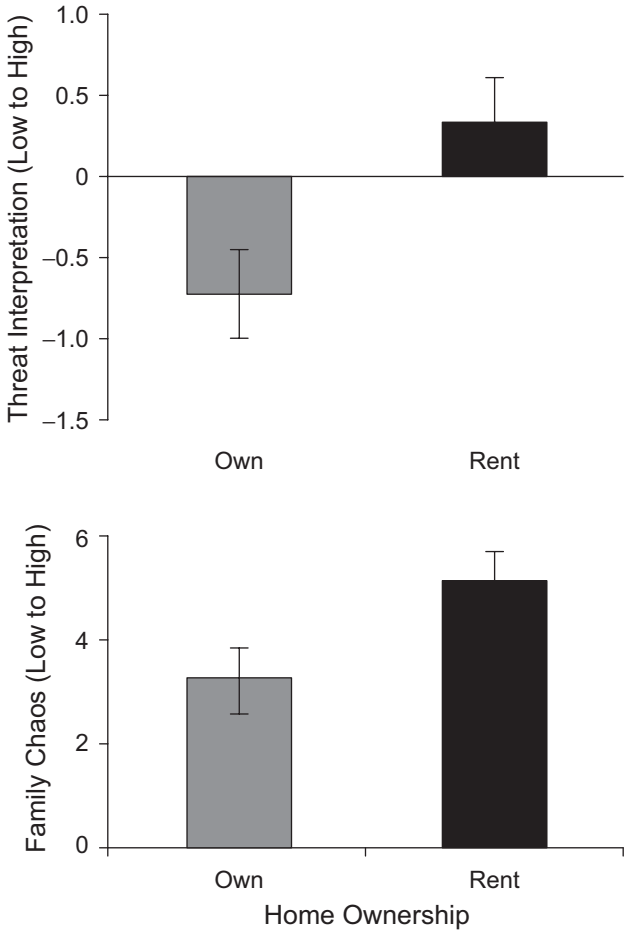

Fig. 2. Threat interpretation (top panel) and family chaos (bottom panel) as a function of home ownership (own vs. rent) for children in the study. Error bars represent ±1 *SE*.

significant ($b = 0.7471$, $SE = 0.6253$, $p > .2$, for savings; $b = -1.293$, $SE = 0.1385$, $p > .3$, for home ownership), and effect sizes dropped by 56% and 51%, respectively. We also tested whether family chaos or child threat interpretations were a stronger predictor of cortisol trajectories over time. When both were included simultaneously, chaos remained a significant predictor of cortisol trajectories ($b = 0.14$, $SE = 0.06$, $p < .05$), whereas child threat perceptions did not ($b = 0.28$, $SE = 0.20$, $p > .15$).

Interactions with pubertal status and gender

Finally, we tested whether SES might interact with either child pubertal stage or child gender in predicting cortisol trajectories over time. We found significant interactions of SES with pubertal stage ($b = –2.56$, $SE = 0.64$, $p < .001$, for the Pubertal Stage × Home Ownership interaction; $b = –0.43$, $SE = 0.16$, $p = .01$, for the Pubertal Stage × Family Savings interaction in a separate model). These interactions indicated that associations between SES and children's cortisol trajectories over time were stronger in postpubertal youth.

We also tested for interactions between SES and gender and found significant effects ($b = 2.92$, $SE = 0.83$, $p < .01$, for the Gender × Home Ownership interaction; $b = 0.58$, $SE = 0.16$, $p < .01$, for the Gender × Family Savings interaction). These interactions indicated that relationships between SES and cortisol trajectories were stronger in girls than in boys.

Discussion

This study is one of the first that we are aware of to document that lower family SES is associated with increasing daily cortisol output over time in children. Cortisol rose almost twice as much in low-SES compared with high-SES children over 2 years. To the extent that cortisol plays a role in psychiatric and physical illnesses (Bjorntorp & Rosmond, 1999; Parker et al., 2003), these findings suggest a biological explanation for why low-SES children may be more vulnerable to developing these conditions later in life. Furthermore, psychosocial factors such as perceptions of threat and family chaos played an important role in explaining the relationship between SES and cortisol trajectories, as including them in the models reduced the effect size of SES-cortisol relationships by 25% to 40%.

Why would a child's socioeconomic background affect his or her cortisol profile over time? We hypothesized that one reason might be that SES shapes the way in which children interpret their social world. In particular, children who come from low-SES backgrounds are more likely to interpret ambiguous situations as threatening (Chen & Matthews, 2003). In previous research, perceptions of threat mediated relationships between low SES and cardiovascular reactivity in healthy adolescents, as well as between low SES and allergic inflammatory responses cross-sectionally in children with asthma (Chen et al., 2006; Chen, Langer, Raphaelson, & Matthews, 2004). The present findings indicate that perceptions of threat can help explain how low-SES environments affect long-term biological profiles by making healthy children more vulnerable to increasing secretion of cortisol over time.

In addition, our findings suggest that another way in which SES is linked to biological profiles is through effects on family environments. In children, low-SES environments result in more chaotic day-to-day home life experiences, which may add to low-SES children's stress burden, promoting increased secretion of cortisol over time. In fact, family chaos was a stronger predictor of cortisol output over time than was children's perceptions of threat, suggesting that the family environment may have stronger effects on hormonal trajectories in children over time. Our study results are consistent with previous research that found that family chaos and lack of routines were major factors in the relationship between poverty and children's psychological well-being (Brody, Flor, & Gibson, 1999; Evans et al., 2005), and extends this work into the physical health domain.

Our SES-cortisol findings are consistent with previous cross-sectional research showing that low SES is associated with greater cortisol levels in healthy adults (Cohen, Doyle, & Baum, 2006; Cohen, Schwartz, et al., 2006; Steptoe et al., 2003), and in healthy children (Evans, 2003; Evans & English, 2002; Evans & Marcynyszyn, 2004; Lupien, King, Meaney, & McEwen, 2000, 2001). Research is just beginning to emerge that tests the nature of SES and cortisol relationships over longer periods of time. For example, one study of the effects of deprivation on physiological outcomes documented that a longer duration of lifetime poverty predicted greater increases in urinary cortisol across two time points in children (Evans & Kim, 2007). Differences between this study and ours include the measurement of SES (the focus on poverty in the Evans & Kim, 2007, study vs. a range of SES in our study) and the method of sampling (urine vs. saliva). In addition, our study assessed cortisol during five time periods, allowing us to model individual trajectories of cortisol over time. Despite these differences, the two studies converge on the same conclusion that SES, both currently and across the lifetime, shapes subsequent cortisol profiles.

Interestingly, when we tested for interactions with child pubertal status and gender, we found that the associations of SES with cortisol trajectories were most pronounced in postpubertal children as well as in girls. The fact that SES shows stronger associations with cortisol trajectories among older, postpubertal children suggests that the effects of SES may take some time to emerge during childhood or possibly that older children are displaying effects of longer duration of exposure to low SES. The finding among girls suggests the possibility that hormonal systems in girls are more responsive to daily life social influences and is consistent with previous research that has documented that women show a greater cortisol awakening response on work days than do men (Kunz-Ebrecht, Kirschbaum, Marmot, & Steptoe, 2004).

Strengths of this study include the 2-year longitudinal design, allowing us to draw more definitive conclusions about directionality, a sampling protocol that included five assessments over a 2-year period, and the identification of threat interpretations and family chaos as potential mediators of the effects of SES. Limitations include the sample size and the assessment of cortisol for only 2 days at each time point. The sample also included a wide age range, although pubertal status was controlled in all analyses, so this is not likely to be a confounding factor. In addition, the 4 children who dropped out after their baseline visit had less family savings than the rest of the sample; however, note that the sample of children who remained in the study had a wide range of family savings (from $0 to >$500,000) and had rates of family home ownership (69%) identical to rates in both the United States and Canada (www.census.gov and www.statcan.gc.ca). Finally, we note that family savings in this study was not related to cortisol intercepts. It is possible that this is because the effects of low SES take time to manifest biologically in children, and, hence, SES predicted slopes over time, but not baseline intercepts of cortisol.

Health disparities are a pressing reality of our society. To begin to attempt to reduce SES disparities in health, we need to better understand the reasons why they exist. The present study provides some of the first longitudinal evidence demonstrating that low SES is able to alter biological profiles in a persistent fashion—in this study by increasing daily cortisol trajectories over time. Moreover, this study identified two psychosocial factors that account for SES-biology links, specifically, the greater tendency among lower-SES children to perceive threat in ambiguous life situations and the greater day-to-day chaos that lower-SES children experience. Taken together, these findings may help explain, and provide some first steps toward ameliorating, low-SES children's vulnerability to mental and physical illnesses later in life.

Declaration of Conflicting Interests

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

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Notes

1. We tested associations with trajectories in the diurnal rhythm of cortisol (daily cortisol slopes) over time and found no significant associations, meaning that SES did not predict changes in the shape of cortisol slopes across the day over a 2-year period.

2. We tested whether controlling for child age, rather than child pubertal status, would affect any of the results. All patterns remained the same when age was included as a covariate. Because pubertal status is recognized to be a more accurate indicator of developmental stage than chronological age, findings are presented in the Results section using pubertal status as a covariate.

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