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Childhood Adversity, Self-Esteem, and Diurnal Cortisol Profiles Across the Life Span

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Abstract

Childhood adversity is associated with poor health outcomes in adulthood; the hypothalamic-pituitary-adrenal (HPA) axis has been proposed as a crucial biological intermediary of these long-term effects. Here, we tested whether childhood adversity was associated with diurnal cortisol parameters and whether this link was partially explained by self-esteem. In both adults and youths, childhood adversity was associated with lower levels of cortisol at awakening, and this association was partially driven by low self-esteem. Further, we found a significant indirect pathway through which greater adversity during childhood was linked to a flatter cortisol slope via self-esteem. Finally, youths who had a caregiver with high self-esteem experienced a steeper decline in cortisol throughout the day compared with youths whose caregiver reported low self-esteem. We conclude that self-esteem is a plausible psychological mechanism through which childhood adversity may get embedded in the activity of the HPA axis across the life span.

Keywords

childhood adversity, cortisol, self-esteem, physical health

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A large body of empirical evidence has demonstrated that harsh social and physical environments early in life are associated with a substantial increase in the risk of chronic illnesses, such as heart disease, diabetes, and some forms of cancer (Galobardes, Lynch, & Smith, 2004). The hypothalamic-pituitary-adrenal (HPA) axis has been proposed as an essential biological intermediary of the long-term effects of childhood adversity on poor health outcomes in adulthood (Gunnar & Vazquez, 2001).

Although the links between adversity early in life and HPA dysregulation in childhood and adulthood are now well established, identifying psychological mechanisms through which distal environmental factors, such as childhood adversity, affect mental and physical health has remained difficult. In other words, although the modulation of HPA axis activity by childhood experiences is known to be mediated by neural mechanisms (e.g., heightened amygdala activation), little is known about the psychological manifestations (e.g., reduced socioemotional skills) of these underlying biological processes (Repetti, Taylor, & Seeman, 2002).

In this research, we proposed that one of the most likely intermediaries of the effects of childhood adversity on diurnal cortisol patterns in adulthood is self-esteem, the overall perception of one's self worth. According to the *sociometer hypothesis* (Leary, Tambor, Terdal, & Downs,

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1995), the self-esteem system acts as an affective preconscious barometer that responds to threats to affiliation and social status, so that when social threat cues are detected, the system triggers unpleasant emotions and, consequently, behaviors necessary to maintain or restore the potential loss of status. Notably, threats to the social self as well as negative emotions—shame in particular—are strong modulators of the tonic activity of the HPA axis (Miller, Chen, & Zhou, 2007). Therefore, it is possible that self-esteem is a psychological antecedent of these effects, similar to what is observed in laboratory experiments modeling social-evaluative stress (Dickerson & Kemeny, 2004). Self-esteem has been shown to modulate acute cortisol changes in response to a variety of stressors (Ford & Collins, 2010)—likely through neural pathways involving the hippocampus, in which reduced volume has been associated with low self-esteem (Pruessner et al., 2005) as well as chronic stress, including childhood adversity (Rao et al., 2010). Thus, it is not surprising that virtually all theories that model the association between childhood adversity and adult health include self-esteem as a plausible mechanism through which childhood experiences are carried across the life span (Taylor, 2010). These models also build on a large literature showing how the family environment during childhood has enduring and long-lasting influences on personal worth and self-acceptance (Ryan, Stiller, & Lynch, 1994).

Further, especially during development, it is possible that the self-esteem of significant others (e.g., caregivers) functions as an additional modulator of offspring's cortisol. How information about environmental challenges is encoded and filtered by the young is critically affected by how adults surrounding them react to and cope with the same challenges. Caregivers can be a source of threats (e.g., abusive parents) and can act as amplifiers or buffers of existing environmental threats (e.g., unresponsive and responsive parents, respectively; Repetti et al., 2002). Additionally, caregivers serve as examples for how to handle stressors, so their ways of interpreting and coping with social and physical threats can easily spread to their children, influencing children's coping style and, potentially, their stress physiology. Support for this hypothesis comes from prior work showing that caregiver psychological functioning can affect children's cortisol (Lupien, King, Meaney, & McEwen, 2000).

In the current project, we first investigated whether self-esteem acts as a modulator of the tonic activity of the HPA axis in adulthood by analyzing diurnal cortisol profiles in a sample of 1,463 adults varying in childhood adversity (Study 1). If the proposed mechanism is functioning during adulthood, we speculated that some evidence should also exist for its presence during childhood. For this reason, we also tested this hypothesis in a sample of 645 youths from China (Study 2), who were recruited as part of a study of youths affected by parental HIV/AIDS. Further, in this sample, we also tested whether caregivers' self-esteem would modulate youths' cortisol secretion above and beyond the effect of youths' own self-esteem.

Method

Study 1

Participants and study timeline. Data for Study 1 were drawn from Wave 2 of the National Study of Daily Experiences (NSDE II, 2004–2009; n = 2,022), a subsample of Wave 2 of the Midlife in the United States study (MIDUS II, 2004–2006; average participant age = 56.62 years). The first wave of data collection for MIDUS (MIDUS I), a large panel survey of adults between the ages of 25 and 74 years (average age = 47.78 years), occurred from 1995 to 1996. The NSDE II included 4 days of salivary cortisol collection and 8 days of daily phone interviews (see Almeida, McGonagle, & King, 2009, for a more detailed description of the sample and assessment protocols for NSDE II). For the current study, inclusion criteria required that participants provided data about parents' education, childhood adversity, self-esteem, and potential psychological confounds (neuroticism, depressed affect, and daily positive and negative affect); demographic information (age, gender, ethnicity, education, and physical health); and cortisol data. Information about childhood adversity and parental education was collected during MIDUS I, while information about self-esteem and psychological covariates was collected during MIDUS II. Age, gender, ethnicity, education, and physical health as reported at MIDUS II were used. Cortisol data were collected during NSDE II, which on average occurred 20.54 months (SD = 13.57) after MIDUS II. The final sample consisted of 1,463 adults (55.3% female, 44.7% male; 95.5% White; 71.2% completed some college or had obtained degrees; mean age = 56.62 years, SD = 12.05 years).

Measures

Childhood adversity. Following previous studies on the same sample (Slopen et al., 2010), we used self-report data collected during the first wave to derive an index of childhood adversity. This approach allowed us to address a limitation of previous studies, which often have focused on single stressors (e.g., sexual abuse) without considering the graded effect on health of interrelated adversities. We used self-report data from three sources.

First, we counted how many stressful events individuals underwent during their childhood (up to 16 years of age). The list of stressful events consisted of 19 episodes: experiencing the death of at least one of the parents, repeating a school year, being sent away from home for

doing something wrong, having an unemployed parent who wanted to work, having a parent with alcohol problems, having a parent with drug problems, dropping out of school, being expelled from school, failing out of school, getting fired from a job, experiencing the death of a sibling, experiencing parents' divorce, losing one's home because of natural disaster or similar causes, being physically abused, being sexually abused, spending time in jail, receiving welfare, moving to a new neighborhood or town more than four times, and getting adopted. Scores on this scale, which we refer to as the Childhood Adversity Stressful Events (CA-SE) scale, were calculated by summing each item. A score was computed only if the participant had valid values (0 = no, 1 = yes) for at least 16 of the items. Scores on the CA-SE scale ranged from 0 to 7 (M = 0.83, SD = 1.12).

Second, we considered parental affection, which was assessed at MIDUS I with a validated questionnaire (Rossi, 2001). Participants had to answer seven questions about their relationships with their mother and father on a 5-point Likert scale (1 = *a lot*, 5 = *not at all*). Averaging these two scales yielded a measure of overall parental affection, which showed high internal consistency (α = .92). Items included "How much could you confide in her about things that were bothering you?" and "How much effort did he put into watching over you and making sure you had a good upbringing?" We refer to this scale as the Childhood Adversity Relationship With Parents (CA-RP) scale. Scores on the CA-RP scale ranged from 1.04 to 4.04 (M = 2.07, SD = 0.64).

Third, frequency of emotional (e.g., insulting, threatening to hit, smashing something in anger), physical (e.g., pushing, slapping, throwing objects), and severe physical (e.g., hitting with a fist, biting, beating) abuse by parents and siblings was evaluated, using nine items from the Conflict Tactics Inventory (Straus, 1979). Each item was rated on a 4-point Likert scale ranging from 1, *often*, to 4, *never*. Items were first reverse-coded and then averaged, so that higher scores represented higher levels of abuse. We refer to this scale as the Childhood Adversity Physical/Emotional Abuse (CA-PEA) scale. Scores on the CA-PEA scale ranged from 1 to 4 (M = 1.71, SD = 0.54).

For each domain, measures were z-scored and a composite of childhood adversity was computed by summing these z scores, with higher scores indicating a more hostile childhood environment. Scores on the childhood-adversity composite ranged from −3.66 to 10.70 (M = 0.00, SD = 2.19).

Self-esteem. Self-esteem was assessed using a modified version of the Rosenberg Self-Esteem Scale (Rosenberg, 1965), which comprised seven items answered on a 7-point Likert scale (1 = *strongly agree*, 7 = *strongly disagree*; α = .76). For participants who answered at least four items on the scale, ratings were summed to create a self-esteem score. Higher scores indicated higher self-esteem. Scores ranged from 11 to 49 (M = 38.35, SD = 7.11).

Salivary cortisol. Salivary cortisol was collected using Salivettes (Sarstedt, Rommelsdorft, Germany). On 4 consecutive days of the 8-day NSDE study period, participants self-collected saliva samples at four time points each day: immediately on waking, 30 min later to assess cortisol awakening response (CAR), before lunch, and at bedtime. Nightly telephone interviews and paper-and-pencil logs received by the participants were used as the main sources of data on the time participants provided each saliva sample. Further, about 25% of NSDE II participants used a "smart box" to collect their saliva samples. Each box contained the participant's Salivettes and was equipped with a computer chip that recorded every time it was opened and closed. Correlations between self-reported sample times and times obtained from the smart box were excellent, ranging from .75 for the evening samples to .95 for the morning samples (Almeida et al., 2009). As for CAR compliance, on about 10% of collection days, participants deviated by 15 min or more from the requested 30-min interval (Almeida et al., 2009). Specifically, in our sample of 1,463 individuals, 5,737 CAR cortisol values were available, 860 of which deviated by at least 15 min from the requested interval. These cortisol values were dropped from the analyses (i.e., treated as missing values at Level 1 in our multilevel models).

Cortisol concentrations (nmol/L) were quantified with a commercially available luminescence immunoassay (IBL, Hamburg, Germany) with intra-assay and interassay coefficients of variability less than 5%. Cortisol values were log-transformed to correct for positive skew in the distribution (Adam & Kumari, 2009). To ensure that all transformed scores were positive, we added a constant of 1 before the transformation.

Demographic covariates. Because parents' educational attainment is recommended as a reliable index of childhood socioeconomic status (SES) in retrospective studies, this variable was also included in analyses. Values ranged from "some grade school" to "doctoral degree," and we identified a group of adults with clearly low childhood SES, which was defined as both parents having not obtained a high school diploma (21.1%). This variable (1 = less than a high school diploma, 0 = high school diploma or more) was included as a covariate and not as an additional childhood-adversity domain, in line with the conceptualization of Slopen et al. (2010). Further, while CA-SE, CA-RP, and CA-PEA scores all correlated among each other, childhood SES did not correlate with any of the childhood-adversity domains.

Several standard demographic covariates in diurnal cortisol studies (Adam & Kumari, 2009) were included in the analyses: age, gender (male = 0, female = 1), education (0 = none beyond high school, 1 = at least some college), and race/ethnicity (0 = White, 1 = non-White) at the person level. At the daily level, we controlled for day of the week (0 = weekday, 1 = weekend) and wake-up time on days of salivary-cortisol sampling.

Health covariates. We further controlled for smoking status, medication use, physical health, and daily exercise. Smoking status was coded as 1 if participants reported being a current cigarette smoker during the MIDUS II interview or reported smoking any cigarettes across the NSDE II study period. Participants who did not report smoking or had missing values were coded as 0. The use of medications relevant to cortisol was assessed on the last day of saliva collection during the NSDE II. Participants who reported using medication were assigned a score of 1, while participants who reported no use of medication or did not answer this question were assigned a score of 0. During MIDUS II, participants reported whether they had any chronic condition in the previous 12 months (0 = no, 1 = yes). This variable was used as an index of participants' physical health. Finally, as part of the NSDE II telephone interviews, participants also reported how many hours they spent exercising each day. This variable was Winsorized at ±3 standard deviations from the mean and included as a covariate at the daily level (M = 0.6839, SD = 1.37).

Psychological covariates. To show the specificity of self-esteem in explaining the link between childhood adversity and diurnal cortisol, we controlled for the overlapping trait of neuroticism. Neuroticism was assessed via four personality adjectives (moody, worrying, nervous, and calm), which were each rated separately on a 4-point Likert scale (1 = *a lot*, 4 = *not at all*). This scale (α = .74) was developed from a cluster of Big Five trait adjectives, and the mean of the four items was obtained to determine a score, with higher values indicating higher neuroticism. Scores ranged from 1 to 4 (M = 2.04, SD = 0.63). We controlled for neuroticism because this personality trait shows the strongest correlation with self-esteem compared with the other Big Five personality traits (Robins, Hendin, & Trzesniewski, 2001), and, similarly to self-esteem, its development can be driven by childhood experiences (Roy, 2002). Because of the overlap between neuroticism and self-esteem, we also tested whether neuroticism would be a psychological pathway linking childhood adversity to cortisol secretion.

We also controlled for depressed affect. Specifically, participants reported whether they experienced each of seven depressive symptoms during 2 weeks in the previous 12 months (e.g., "Did you lose interest in most things?" "Did you have more trouble falling asleep than usual?" "Did you think a lot about death?"; Wang, Berglund, & Kessler, 2000). Responses on each item (0 = no, 1 = yes) were added to derive a continuous measure of depressed affect, so that higher scores indicate higher depressed affect (M = 0.48, SD = 1.57). Previous work has suggested that depression might act as an intermediary of the effects of childhood adversity on HPA-axis activity (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008); thus, as in the case of neuroticism, we tested the indirect effects linking childhood adversity to the various cortisol parameters via depressed affect.

Finally, we controlled for two broader psychological covariates, daily negative affect and positive affect, as a stringent test of the robustness of effects of self-esteem on cortisol parameters. On each day of the NSDE II sampling period, participants were asked to rate on a 5-point Likert scale how much they felt each of 14 negative affective states (e.g., restless, lonely, sad, irritable, afraid) and 13 positive affective states (e.g., happy, cheerful, confident, attentive, peaceful). Scores for each measure were averaged. For daily positive affect (α = .96), scores ranged from 0 to 4 (M = 2.76, SD = 0.76), while for daily negative affect (α = .89), scores ranged from 0 to 2.8 (M = 0.18, SD = 0.29).

Data analysis. At the daily level, the incidence of missing data among the variables was 2.9%. To curtail the bias associated with pairwise or listwise deletion of missing data (Schafer & Graham, 2002), we used the expectation-maximization algorithm to impute missing data.

Given the longitudinal nature of our endocrine data (i.e., cortisol within days, within people), hierarchical linear modeling (HLM) was used for data analyses. HLM allowed us to regress multiple cortisol parameters at the same time (cortisol at awakening, CAR, and slope) on both daily-level predictors (e.g., within-persons daily wake-up time) and person-level predictors (e.g., between-persons self-esteem, age, gender). Furthermore, HLM can estimate slopes and intercepts even with missing cortisol data. Following prior diurnal cortisol research (Adam & Kumari, 2009), we modeled time since waking, time since waking^2, and CAR (dummy coded 0 or 1) at Level 1 to estimate each participant's diurnal cortisol rhythm. At Level 3 (person level), we ran four models. In Model 1, childhood adversity was the main predictor, and in Model 2, childhood adversity and self-esteem were the main predictors. These models did not control for covariates. Models 3 and 4 mirrored Models 1 and 2, respectively, but also they controlled for covariates. Daily covariates were included at Level 2, and person covariates were included at Level 3. Following statistical recommendations on HLM centering (Enders & Tofighi, 2007), we

grand-mean-centered continuous covariates at Level 2 and Level 3. Wake-up time, day of the week, positive affect, and negative affect were used as Level 2 predictors for all the cortisol parameters, while daily exercise was used only as a predictor for the cortisol slope. In keeping with prior studies, we allowed cortisol intercept, slope (effect of time), and CAR to vary randomly at Level 2 and Level 3 (i.e., they were treated as random effects). All HLM significance tests were two-tailed with robust standard errors.

To test whether individual self-esteem explained some of the covariation between childhood adversity and cortisol parameters, we first regressed self-esteem on childhood adversity. The obtained regression coefficient and asymptotic sampling variance for the association between childhood adversity (our independent variable) and self-esteem were then used in association with the regression coefficients and the asymptotic sampling variances for the association between self-esteem and each cortisol parameter (our dependent variables) obtained in Model 2 (i.e., the HLM run without controlling for covariates) to derive 95% confidence intervals (CIs) for indirect effects through the Monte Carlo method (20,000 repetitions; Preacher & Selig, 2012). These analyses were then repeated, controlling for covariates. Specifically, we first ran a multiple regression analysis in which self-esteem was regressed on childhood adversity while we controlled for appropriate (i.e., nonspecific to cortisol) demographic covariates (age, gender, education, race/ethnicity, childhood SES) and psychological covariates (neuroticism, depressive symptoms, and daily positive and negative affect). Then we used the obtained regression coefficient and the asymptotic sampling variance with the regression coefficients and the asymptotic sampling variances obtained in Model 4 (i.e., the HLM controlling for covariates) to derive the 95% CIs for indirect effects via the Monte Carlo method. CIs not including 0 indicate statistically significant indirect effects.

Supplementary analyses. Although the main purpose of our study was to investigate the cumulative effect of a variety of stressors considered simultaneously on self-esteem and diurnal cortisol (for a similar approach, see Slopen et al., 2010), we also explored how each facet of childhood adversity was related to self-esteem and cortisol. For this reason, we ran additional analyses in which cortisol was predicted first by each facet of childhood adversity and, next, by each facet of childhood adversity together with self-esteem. These models controlled for covariates. In a second set of supplementary analyses, we tested whether neuroticism and depressed affect acted as alternative intermediaries of the effect of childhood adversity on daily cortisol secretion. These analyses are reported in Table S1 in the Supplemental Material available online.

Study 2

Participants. Data for Study 2 were drawn from a community sample of 790 children and adolescents (or *youths*) and their current primary caregivers. Youths were between the ages of 6 and 17 years and affected by parental HIV. Participants were recruited for a psychosocial-intervention trial in a rural county in central China, where many residents have been infected with HIV through unhygienic blood-collection practices; all of these data were collected at baseline, prior to the intervention. A sample of 790 individuals was chosen on the basis of a power analysis for the hypothesized effect of the intervention (small-to-medium hypothesized effect size). Of the larger sample of 790 youths, 746 fit the inclusion criterion of 8 to 15 years of age, based on the age range for which the self-report measures used in the present analyses were normed. Of those 746 youths, 645 (86.4%) provided saliva samples for cortisol analyses (final sample: 48.1% female, 51.9% male; mean age = 10.67 years, *SD* = 1.79 years). As reported by the field researchers in this study, 79 youths had at least one parent who died from HIV/AIDS, and 554 youths had at least one HIV-positive parent (data for this variable were not available for 12 youths).

Youths and caregivers both completed confidential survey questionnaires in Chinese. The survey included detailed measures of demographic information and several psychosocial scales. Most of the surveys were self-administrated in a small group in which two interviewers were present. These adult facilitators provided assistance to the youths—especially the younger ones—by clarifying questions for them as well as by asking them to confirm their answers before reporting them on paper.

Measures

Childhood adversity and self-esteem. Similarly to Study 1, self-report data from two sources were used to create the childhood-adversity composite. First, we used 15 items to assess youths' experience of a number of stressful life events during the previous 6 months. Items from this list, which was developed for this population and successfully validated in previous research (Li et al., 2009), included being in a traffic accident; witnessing involuntary violence; and experiencing hospitalization, natural disaster, severe sickness or death of friends, relocation of the family, and death of family members. Three additional items were added to the list for the present study: experiencing the death of at least one of the parents, having both parents diagnosed with HIV (as reported by the researcher), and moving to a new residence more than twice. This was the equivalent of the CA-SE scale used in Study 1. Scores were obtained by calculating the sum of each item, and participants received a score only if they answered at least three items (note that the 15-item

list counted as a single item). Scores on the CA-SE scale ranged from 0 to 14 (M = 2.81, SD = 2.25).

Second, we assessed parenting quality using both youth and caregiver reports of (a) parental responsiveness and (b) parental trust. Youths assessed parental responsiveness of their primary caregiver with a 6-item scale adapted and back-translated from a previously validated parental-responsiveness scale (Jackson, Henriksen, & Foshee, 1998; α = .76 in the current sample). Items, which were rated on a 4-point Likert scale (1 = *never*, 4 = *always*), included "He/she wants to hear about my problems," and "He/she makes me feel better when I am upset." Items were adapted so that caregivers could complete the scale as well (α = .66). Scores on both scales were obtained by calculating the mean of the six items, with higher scores indicating higher parental responsiveness. Scores on the youth-reported parental-responsiveness scale ranged from 1 to 4 (M = 2.59, SD = 0.66), and scores on the caregiver-reported parental-responsiveness scale ranged from 1 to 4 (M = 2.73, SD = 0.56).

Further, youths and caregivers used the Trusting Relationship Questionnaire (Mustillo, Dorsey, & Farmer, 2005) to assess caregiver relationship trust (α = .90 for the youth version of the questionnaire; α = .85 for the caregiver version of the questionnaire). Items on the caregiver version included "Does the child identify things he or she likes about you?" and "Does the child talk to you about his or her problems?" (the youth version used similar questions; e.g., "Does the adult talk to you about his or her problems?"; for more details, see Mustillo et al., 2005). One item ("Do you seek help from him/her when you face difficulties?" for the youth questionnaire; "Does the child seek help from you when he/she faces difficulties?" for the caregiver questionnaire) was added to the original list of 18 items. Scores were obtained by calculating the mean of the items, with higher values indicating higher parental trust. Scores on the youth-reported scale ranged from 1 to 5 (M = 2.66, SD = 0.75), while scores on the caregiver-reported scale ranged from 1.26 to 5 (M = 3.04, SD = 0.58).

Scores on these four scales correlated among each other (average r = .204, range = .102–.467, lowest p < .001)—except for caregiver-reported parental responsiveness and youth-reported parental trust (r = .025, p = .535). All items were z-scored to form a composite index of parenting quality (CA-RP), similarly to Study 1. Specifically, scores on the CA-RP scale were obtained by summing each subscale and were computed for participants who had valid scores on all four subscales. Final scores on the CA-RP scale ranged from −7.44 to 9.02 (M = 1.60, SD = 2.47), with higher scores representing harsher parenting.

For each childhood-adversity domain (CA-SE and CA-RP), measures were converted to z scores, and a composite of childhood adversity was computed by summing these z scores. Higher scores indicated a more hostile childhood environment. Scores on the childhood-adversity composite ranged from −4.08 to 4.95 (M = 0.00, SD = 1.41).

Youths' and caregivers' self-esteem were assessed using the Rosenberg Self-Esteem Scale (Rosenberg, 1965), which comprised 10 items answered on a 4-point Likert scale (1 = *strongly disagree*, 4 = *strongly agree*). Items included "At times I think that I am no good at all" and "On the whole, I am satisfied with myself" (α = .63, for the youth scale; α = .72, for the caregiver scale). Scores on these scales were obtained by averaging the responses, with higher values indicating higher self-esteem. Scores for the youth self-esteem scale ranged from 1.6 to 4.0 (M = 2.76, SD = 0.44), while scores for the caregiver self-esteem scale ranged from 1.6 to 3.9 (M = 2.88, SD = 0.41).

Salivary cortisol. Participants self-collected saliva samples at four time points each day for 3 days: immediately upon waking (prior to any eating, drinking, or exercise), 30 min later to assess CAR, 1 hr before dinnertime, and then at bedtime. Prior to saliva collection, the investigators showed youths the correct procedure to collect saliva samples using Salivettes and emphasized the importance of compliance with the time of collection. Cortisol levels (μg/dL) were determined via chemiluminescent immunoassay (Access Cortisol kit YZB/USA 2802, Beckman Coulter, Fullerton, CA). Compliance with the saliva-collection procedures was excellent. Participants provided a total of 11.17 out of 12 samples on average, with 93% of all possible saliva samples collected. Altogether, 61.3% of participants did not miss any samples, with 90.4% providing between 10 and 12 samples and 96% providing at least 8 of the 12 possible saliva samples across the 3 days. As for CAR compliance, in our sample of 645 individuals, of the available 1,810 CAR cortisol values, 453 self-reported deviating by 15 min or more from the requested 30-min interval. As in Study 1, these cortisol values were dropped from the analyses. Finally, cortisol values were log-transformed to correct for positive skew in the cortisol distribution (Adam & Kumari, 2009). To ensure that all transformed scores were positive, we added a constant of 1 before the transformation.

Demographic, psychological, and health covariates. As in Study 1, demographic covariates consisted of age, gender (male = 0, female = 1), and caregiver's educational attainment (1 = elementary school or no school, 0 = high school or more). At the daily level, we controlled for day of the week (0 = weekday, 1 = weekend) and daily wake-up time. At the momentary level (i.e., at collection time of each saliva sample), youths reported whether they smoked or practiced any sport. These variables were

included at Level 1 as health covariates. As in the case of Study 1, missing cases were replaced by the mode.

In terms of psychological covariates, neuroticism, depression, daily positive affect, and daily negative affect were included. Neuroticism was assessed via two personality descriptors (i.e., "anxious, easily upset," and "calm, emotionally stable"; reverse-scored; $r = .202$, $p < .001$), which were rated on a 4-point Likert scale (1 = *strongly disagree*, 4 = *strongly agree*; Gosling, Rentfrow, & Swann, 2003). Responses to the two items on this scale were averaged, with higher scores indicating higher neuroticism. Scores ranged from 1 to 4 ($M = 2.39$, $SD = 0.73$).

Depression was measured using a short version of the Center for Epidemiologic Studies Depression Scale for children (Fendrich, Weissman, & Warner, 1990). Children used a 4-point Likert scale (1 = *not at all*, 4 = *a lot*) to respond to 10 items asking how they felt or acted in the previous week. Items included "I was bothered by things that usually don't bother me," and "I felt like I was too tired to do things this past week" ($\alpha = .62$). Scores on this scale were obtained by summing the responses and were computed only for participants who answered all 10 items. Higher scores indicated higher depression ($M = 20.16$, $SD = 4.29$).

A 3-point Likert scale (1 = *not at all*; 3 = *almost all day*) was used to assess both daily negative affect and daily positive affect. Daily negative affect ($\alpha = .76$) was assessed via six adjectives (i.e., sad, upset, fear, lonely, angry, worried), and daily positive affect ($\alpha = .73$) was assessed via six adjectives (i.e., happy, excited, energetic, confident, curious, calm). For daily positive affect, scores ranged from 1 to 3 ($M = 2.19$, $SD = 0.50$); for daily negative affect, scores also ranged from 1 to 3 ($M = 1.34$, $SD = 0.41$).

Finally, at the person level, we controlled for perceived health status, which was self-reported by each youth and by his or her caregiver on a 5-point Likert-type scale ranging from 1 (*very poor*) to 5 (*very good*). Scores on this scale were obtained by calculating the mean for participants who answered both items. Higher scores indicated higher perceived health status ($M = 4.1$, $SD = 0.75$).

Data analysis. At the daily level, the incidence of missing data among the variables was 9.8%, while at the person level, the incidence of missing data was 3%. To curtail the bias associated with pairwise or listwise deletion of missing data (Schafer & Graham, 2002), we used the expectation-maximization algorithm to impute missing data. Because this algorithm does not allow value replacement for categorical data, mode imputation was used to replace missing cases for two variables: caregiver educational attainment ($n = 32$) and caregiver gender ($n = 29$).

As in Study 1, HLM was used for data analyses. Six models were run. In Model 1, childhood adversity was the predictor; in Model 2, both childhood adversity and self-esteem were predictors. Models 4 and 5 mirrored Models 1 and 2, respectively, but they also controlled for covariates. Continuous covariates at Level 2 and Level 3 were grand-mean centered. CIs for indirect effects were estimated using the Monte Carlo method. These analyses were run with and without covariates.

We ran two additional models to test the hypothesis that caregiver self-esteem would modulate youth cortisol. Models 3 and 6 contained the same predictors as Models 2 and 5, respectively, but caregiver self-esteem was added alongside youth self-esteem as a further predictor. Model 3 was run without covariates, whereas Model 6 controlled for two additional covariates: caregiver age and caregiver gender (Model 6).

Supplementary analyses. Following the approach adopted in Study 1, we ran additional analyses in which cortisol was predicted by each facet of childhood adversity and by each facet of childhood adversity together with self-esteem. These models controlled for covariates. As in Study 1, we independently tested whether neuroticism and depression acted as intermediaries of the effect of childhood adversity on daily cortisol secretion. The results of these analyses are reported in Table S2 in the Supplemental Material.

Results

Study 1

Bivariate correlations between Study 1 person-level predictors are reported in Table 1, and the relationships between childhood adversity, self-esteem, and cortisol parameters in models controlling for covariates are reported in Table 2.

We created four models—one with childhood adversity as the only predictor (Model 1); one with childhood adversity and self-esteem as predictors (Model 2); one with childhood adversity and covariates as predictors (Model 3); and one with childhood adversity, self-esteem, and covariates as predictors (Model 4). In Model 1 and Model 3, childhood adversity was a significant predictor of morning cortisol: Participants who reported more adverse childhood conditions had lower levels of cortisol at awakening (Model 1: $\gamma_{001} = -0.021$, $p < .001$; Model 3: $\gamma_{001} = -0.013$, $p = .020$). Greater childhood adversity was also associated with a flatter cortisol slope (Model 1: $\gamma_{201} = 0.001$, $p = .032$); however, this association disappeared after we controlled for covariates (Model 3: $\gamma_{201} = 0.000$, $p = .395$). Childhood adversity was not a significant predictor of CAR (Model 1: $\gamma_{101} = 0.000$, $p = .996$; Model 3: $\gamma_{101} = -0.001$, $p = .782$). Next, self-esteem was introduced in the analyses (Model 2 and Model 4). Individuals

Table 1. Bivariate Correlations Between Person-Level Variables in Study 1

Variable	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Female	.010	−.030	−.061*	.009	−.010	.194**	.068**	−.009	.116**	−.043†	.029	.135**	.111**	−.103**
2. Non-White	—	−.013	.014	.033	−.039	−.050†	−.006	.082*	.011	.024	.054*	−.058*	−.028	.005
3. Age: Wave 2		—	−.120**	.241**	−.134**	−.038	.161**	−.058*	−.065*	−.157**	−.128**	−.206**	−.110**	.139**
4. Some college: Wave 2			—	−.231**	−.120**	.019	−.001	−.106**	−.050	−.076**	−.106**	−.093**	−.048†	.133**
5. Childhood SES: Wave 1				—	.019	−.002	.011	.004	.034	−.012	.012	−.006	−.039	−.030
6. Smoking status					—	−.017	.001	.074**	.086**	.122**	.129**	.092**	.109**	−.090**
7. Medication						—	.191**	.032	.072**	.012	.053*	.079**	.087**	−.050†
8. Chronic condition							—	.062*	.039	.031	.060*	.122**	.095**	−.127**
9. CA-SE: Wave 1								—	.255**	.218**	.673**	.117**	.099**	−.134**
10. CA-RP: Wave 1									—	.422**	.766**	.170**	.115**	−.201**
11. CA-PEA: Wave 1										—	.749**	.153**	.109**	−.134**
12. Childhood adversity: Wave 1											—	.201**	.148**	−.214**
13. Neuroticism: Wave 2												—	.276**	−.527**
14. Depressed affect: Wave 2													—	−.311**
15. Self-esteem: Wave 2														—

Note: SES = socioeconomic status; CA-SE = Childhood Adversity Stressful Events scale; CA-RP = Childhood Adversity Relationship With Parents scale; CA-PEA = Childhood Adversity Physical/Emotional Abuse scale.
†$p < .10$. *$p < .05$. **$p < .01$.

Table 2. Results of Selected Hierarchical Linear Models of Diurnal Cortisol Parameters in Study 1

	Model 3			Model 4		
Fixed effect	Estimate	*SE*	*p*	Estimate	*SE*	*p*
Morning cortisol, π_0						
Average morning cortisol, β_{00}, γ_{000}	2.7267	0.0369	< .001	2.7263	0.0369	< .001
Childhood adversity: Wave 1, γ_{001}	–0.0130	0.0056	.020	–0.0121	0.0056	.030
Wave 2 self-esteem, γ_{002}	—	—	—	0.0043	0.0021	.044
Female, γ_{003}	–0.1127	0.0247	< .001	–0.1112	0.0247	< .001
Non-White, γ_{004}	–0.1645	0.0581	.005	–0.1615	0.0577	< .001
Wave 2 age, γ_{005}	0.0043	0.0012	< .001	0.0042	0.0012	< .001
Wave 2: some college, γ_{006}	0.1006	0.0275	< .001	0.0952	0.0275	.001
Wave 2 childhood SES, γ_{007}	–0.0279	0.0335	.406	–0.0257	0.0336	.444
Smoking status, γ_{008}	–0.0465	0.0358	.194	–0.0459	0.0356	.197
Medication, γ_{009}	–0.0442	0.0258	.088	–0.0454	0.0258	.078
Chronic condition, γ_{0010}	–0.0404	0.0275	.142	–0.0364	0.0273	.182
Wave 2 neuroticism, γ_{0011}	0.0125	0.0210	.550	0.0324	0.0218	.138
Wave 2 depressed affect, γ_{0012}	–0.0085	0.0097	.382	–0.0055	0.0099	.576
Weekend, β_{01}, γ_{010}	–0.0504	0.0149	.001	–0.0499	0.0148	.001
Wake-up time, β_{02}, γ_{020}	–0.0012	0.0074	.867	–0.0014	0.0074	.854
Daily negative affect, β_{03}, γ_{030}	0.0413	0.0286	.149	0.0414	0.0287	.150
Daily positive affect, β_{04}, γ_{040}	–0.0039	0.0132	.770	–0.0090	0.0136	.506
Cortisol awakening response (CAR), π_1						
Average CAR, β_{10}, γ_{100}	0.3585	0.0293	< .001	0.3584	0.0293	< .001
Wave 1 childhood adversity, γ_{101}	–0.0013	0.0048	.782	–0.0013	0.0048	.789
Wave 2 self-esteem, γ_{102}	—	—	—	0.0001	0.0017	.937
Female, γ_{103}	0.0873	0.0203	< .001	0.0872	0.0203	< .001
Non-White, γ_{104}	0.0836	0.0486	.086	0.0837	0.0486	.086
Wave 2 age, γ_{105}	0.0017	0.0009	.058	0.0017	0.0009	.060
Wave 2: some college, γ_{106}	–0.0246	0.0212	.246	–0.0246	0.0214	.251
Wave 2 childhood SES, γ_{107}	0.0054	0.0246	.827	0.0054	0.0246	.828
Smoking status, γ_{108}	0.0786	0.0267	.003	0.0786	0.0267	.003
Medication, γ_{109}	0.0074	0.0214	.728	0.0074	0.0214	.730
Chronic condition, γ_{1010}	0.0264	0.0228	.247	0.0267	0.0229	.245
Wave 2 neuroticism, γ_{1011}	–0.0142	0.0177	.421	–0.0134	0.0195	.493
Wave 2 depressed affect, γ_{1012}	0.0003	0.0062	.957	0.0004	0.0063	.947
Weekend, β_{11}, γ_{110}	–0.0338	0.0169	.046	–0.0339	0.0169	.045
Wake-up time, β_{12}, γ_{120}	–0.0157	0.0071	.027	–0.0157	0.0071	.027
Daily negative affect, β_{13}, γ_{130}	–0.0054	0.0374	.885	–0.0056	0.0374	.882
Daily positive affect, β_{14}, γ_{140}	–0.0020	0.0144	.890	–0.0015	0.0148	.919
Time since waking, π_2						
Average linear slope, β_{20}, γ_{200}	–0.1349	0.0045	< .001	–0.1349	0.0045	< .001
Wave 1 childhood adversity, γ_{201}	0.0004	0.0005	.395	0.0003	0.0005	.524
Wave 2 self-esteem, γ_{202}	—	—	—	–0.0005	0.0002	.010
Female, γ_{203}	0.0019	0.0022	.383	0.0017	0.0022	.433
Non-White, γ_{204}	0.0277	0.0049	< .001	0.0274	0.0048	< .001
Wave 2 age, γ_{205}	0.0004	0.0001	< .001	0.0005	0.0001	< .001
Wave 2: some college, γ_{206}	–0.0054	0.0024	.025	–0.0048	0.0024	.049
Wave 2 childhood SES, γ_{207}	0.0014	0.0027	.605	0.0012	0.0027	.670
Smoking status, γ_{208}	0.0171	0.0028	< .001	0.0171	0.0027	< .001
Medication, γ_{209}	0.0069	0.0022	.002	0.0070	0.0022	.002
Chronic condition, γ_{2010}	0.0032	0.0025	.207	0.0027	0.0025	.287
Wave 2 neuroticism, γ_{2011}	0.0006	0.0019	.757	–0.0018	0.0021	.399
Wave 2 depressed affect, γ_{2012}	0.0010	0.0007	.137	0.0007	0.0007	.333
Weekend, β_{21}, γ_{210}	0.0027	0.0017	.112	0.0026	0.0017	.121
Wake-up time, β_{22}, γ_{220}	–0.0030	0.0008	< .001	–0.0030	0.0008	< .001

(continued)

Table 2. *(continued)*

Fixed effect	Model 3			Model 4		
	Estimate	*SE*	*p*	Estimate	*SE*	*p*
Daily negative affect, β_{23}, γ_{230}	0.0034	0.0033	.304	0.0034	0.0033	.304
Daily positive affect, β_{24}, γ_{240}	–0.0003	0.0014	.809	0.0004	0.0014	.791
Daily exercise, β_{25}, γ_{250}	0.0007	0.0007	.345	0.0007	0.0007	.323
Time since waking^2, π_3						
Average curvature, β_{30}, γ_{300}	0.0023	0.0002	< .001	0.0023	0.0002	< .001

Note: Intercepts indicate average cortisol values at awakening, average slopes of time since waking indicate change in cortisol per 1-hr change in time, and average slopes of time since waking^2 indicate change in cortisol per 1-hr change in time^2. Seventy-one cortisol values were more than 3 standard deviations above the mean. Results for the main variables of interest remained significant when these observations were Winsorized. SES = socioeconomic status.

who reported higher self-esteem had higher morning cortisol (Model 2: $\gamma_{002} = 0.005$, $p = .006$; Model 4: $\gamma_{002} = 0.004$, $p = .044$) and a steeper cortisol slope (Model 2: $\gamma_{202} = -0.001$, $p = .001$; Model 4: $\gamma_{202} = -0.001$, $p = .010$). Self-esteem was not a significant predictor of CAR regardless of whether we did not include covariates (Model 2: $\gamma_{102} = -0.000$, $p = .910$) or included covariates (Model 4: $\gamma_{102} = 0.000$, $p = .937$). However, when self-esteem was introduced in the analyses, the effect of childhood adversity was reduced both for morning cortisol (Model 2: $\gamma_{002} = -0.018$, $p = .001$; Model 4: $\gamma_{002} = -0.012$, $p = .030$) and cortisol slope (Model 2: $\gamma_{201} = 0.001$, $p = .157$; Model 4: $\gamma_{201} = 0.000$, $p = .524$).

Next, we tested whether the association between childhood adversity and morning cortisol and between childhood adversity and the cortisol slope was partially explained by self-esteem. Regression analyses showed that childhood adversity negatively predicted self-esteem (without controlling for covariates: $\beta = -0.696$, $SE = 0.083$, $p < .001$; controlling for covariates: $\beta = -0.278$, $SE = 0.073$, $p < .001$). Monte Carlo analyses showed a significant indirect effect of childhood adversity on morning cortisol via self-esteem (without controlling for covariates: 95% CI = [–0.006699, –0.001061]; controlling for covariates: 95% CI = [–0.002716, –0.000027]) as well as a significant indirect effect of childhood adversity on diurnal cortisol slope via self-esteem (without controlling for covariates: 95% CI = [0.000140, 0.000633]; controlling for covariates: 95% CI = [0.000029, 0.000295]), which indicates that high childhood adversity was linked to low morning cortisol and a flatter cortisol slope via low self-esteem. Next, we calculated the ratio of the indirect effect to the total effect (i.e., indirect effect + direct effect; Preacher & Kelley, 2011) and found that self-esteem mediated about 1/10 of the total effect of childhood adversity on morning cortisol and about 1/3 of the total effect of childhood adversity on the cortisol slope.

In a series of supplementary analyses, we tested Models 1 through 4 using the CA-SE, CA-RP, and CA-PEA scales, separately. As shown in Table 3, we found an indirect effect between CA-SE scores and the cortisol slope via self-esteem, 95% CI = [0.000012, 0.000397]. Further, self-esteem partially explained the link between CA-RP and morning cortisol, 95% CI = [–0.010140, –0.000132], as well as between CA-RP and the cortisol slope, 95% CI = [0.000145, 0.001149]. In contrast, no evidence for an indirect effect was found between CA-PEA and any of the cortisol parameters. Finally, no evidence was found for significant indirect effects linking childhood adversity to diurnal cortisol parameters via neuroticism or depressed affect (see Table S1).

Study 2

Bivariate correlations between Study 2 predictors are reported in Table 4, and the relationship between childhood adversity, self-esteem, and cortisol parameters in models controlling for covariates are reported in Table 5.

In Model 1 and Model 4, childhood adversity was a significant predictor of morning cortisol, such that individuals who reported more adverse childhood conditions had lower levels of cortisol at awakening (Model 1: $\gamma_{001} = -0.011$, $p = .009$; Model 4: $\gamma_{001} = -0.012$, $p = .017$). However, childhood adversity was not associated with the cortisol slope (Model 1: $\gamma_{201} = 0.000$, $p = .551$; Model 4: $\gamma_{201} = 0.000$, $p = .714$) or CAR (Model 1: $\gamma_{101} = 0.006$, $p = .192$; Model 4: $\gamma_{101} = 0.007$, $p = .178$). Next, self-esteem was introduced as a predictor in the analyses. Corroborating the findings from Study 1, results showed that individuals with higher self-esteem had higher morning cortisol (Model 2: $\gamma_{002} = 0.044$, $p = .003$; Model 5: $\gamma_{002} = 0.041$, $p = .010$) and a steeper cortisol slope (Model 2: $\gamma_{202} = -0.003$, $p = .025$; Model 5: $\gamma_{202} = -0.003$, $p = .038$). In other words, individuals who reported higher self-esteem had higher cortisol at awakening and a steeper cortisol decline through the day. Self-esteem was not a significant predictor of CAR (Model 2: $\gamma_{102} = -0.018$, $p = .254$; Model 5: $\gamma_{102} = -0.024$, $p = .162$). Effect sizes in Study 2 were comparable with the effect sizes in Study 1.[1]

Table 3. Results of Analyses on the Effects of Childhood-Adversity Measures on Cortisol Parameters in Study 1

Measure	Morning cortisol	Cortisol awakening response	Cortisol slope
	Without controlling for self-esteem		
Childhood Adversity Stressful Events score	–0.0149 (0.0109)	–0.0030 (0.0090)	0.0019† (0.0011)
Childhood Adversity Relationship With Parents score	–0.0392* (0.0199)	0.0040 (0.0156)	–0.0011 (0.0017)
Childhood Adversity Physical/Emotional Abuse score	–0.0346 (0.0230)	–0.0105 (0.0187)	0.0009 (0.0019)
	Controlling for self-esteem		
Childhood Adversity Stressful Events score	–0.0138 (0.0109), [–0.003769, 0.000002205]	–0.0030 (0.0090), [–0.001343, 0.00123]	0.0018† (0.0011), [0.00001236, 0.0003972]
Childhood Adversity Relationship With Parents score	–0.0354† (0.0199), [–0.01014, –0.0001315]	0.0043 (0.0155), [–0.004049, 0.003537]	–0.0015 (0.0017), [0.0001453, 0.001149]
Childhood Adversity Physical/Emotional Abuse score	–0.0332 (0.0229), [–0.006221, 0.0005833]	–0.0104 (0.0187), [–0.002144, 0.001868]	0.0008 (0.0019), [–0.002716, 0.001604]

Note: The table shows unstandardized regression coefficients, followed by robust standard errors in parentheses; 95% confidence intervals are given in brackets for indirect effects.
†$p < .10$. *$p < .05$.

We next tested whether the associations between childhood adversity and the cortisol parameters were partially explained by self-esteem. Further, because indirect effects can exist in absence of a significant total effect (Zhao, Lynch, & Chen, 2010), we also tested the significance of a potential indirect effect of childhood adversity on cortisol slope through self-esteem. Regression analyses showed that childhood adversity negatively predicted self-esteem (without controlling for covariates: $\beta = -0.067$, $SE = 0.012$, $p < .001$; controlling for covariates: $\beta = -0.034$, $SE = 0.011$, $p = .003$). Monte Carlo analyses showed a significant indirect effect of childhood adversity on morning cortisol via self-esteem (without controlling for covariates: 95% CI = [–0.005316, –0.000973]; controlling for covariates: 95% CI = [–0.003037, –0.000203]) as well as a significant indirect effect of childhood adversity on diurnal cortisol slope via self-esteem (without controlling for covariates: 95% CI = [0.000026, 0.000416]; controlling for covariates: 95% CI = [0.000004, 0.000242]), which indicates that high childhood adversity was linked to low morning cortisol and a flatter cortisol slope via low self-esteem. Next, we calculated the ratio of the indirect effect to the total effect and found that self-esteem mediated approximately 1/8 of the total effect of childhood adversity on morning cortisol and 3/4 of the total effect of childhood adversity on the cortisol slope.

We also tested the hypothesis that caregiver self-esteem would be—above and beyond the effects of youth self-esteem—associated with youths' cortisol parameters. To test this, we ran two more models in

Table 4. Bivariate Correlations Between Person-Level Variables in Study 2

Variable	2	3	4	5	6	7	8	9	10	11
1. Female	–.087*	.004	.023	–.038	–.037	–.054	–.037	–.110*	.100*	.066†
2. Age	—	–.025	–.073†	.086*	.058	.102**	–.005	–.025	.051	.048
3. Caregiver education (high school)		—	–.020	.059	.100*	.113**	.009	.051	–.022	–.121**
4. Health status			—	–.182**	–.106**	–.204**	–.063	–.065	.006	.131**
5. CA-SE				—	–.010	.703**	–.044	.212**	–.057	–.037
6. CA-RP					—	.703**	.121**	.152**	–.247**	–.218**
7. Childhood adversity						—	.055	.259**	–.216**	–.181**
8. Neuroticism							—	–.118**	–.162**	–.050
9. Depression								—	–.399**	–.139**
10. Youth self-esteem									—	.100*
11. Caregiver self-esteem										—

Note: CA-SE = Childhood Adversity Stressful Events scale; CA-RP = Childhood Adversity Relationship With Parents scale.
†$p < .10$. *$p < .05$. **$p < .01$.

Table 5. Results of Selected Hierarchical Linear Models of Diurnal Cortisol Parameters in Study 2

	Model 4			Model 5			Model 6		
Fixed effect	Estimate	*SE*	*p*	Estimate	*SE*	*p*	Estimate	*SE*	*p*
Morning cortisol, π_0									
Average morning cortisol, β_{00}, γ_{000}	0.6934	0.0121	< .001	0.6943	0.0121	< .001	0.6991	0.0129	.000
Childhood adversity, γ_{001}	–0.0109	0.0045	.017	–0.0093	0.0045	.038	–0.0079	0.0045	.079
Youth self-esteem, γ_{002}	—	—	—	0.0409	0.0158	.010	0.0387	0.0158	.015
Caregiver self-esteem, γ_{003}	—	—	—	—	—	—	0.0307	0.0164	.061
Female, γ_{004}	–0.0042	0.0131	.747	–0.0058	0.0130	.653	–0.0057	0.0130	.661
Age, γ_{005}	0.0066	0.0037	.076	0.0061	0.0037	.099	0.0052	0.0037	.163
Caregiver education, γ_{006}	0.0031	0.0132	.817	0.0027	0.0131	.837	0.0080	0.0135	.555
Health status, γ_{007}	–0.0032	0.0087	.711	–0.0019	0.0086	.829	–0.0035	0.0088	.693
Neuroticism, γ_{008}	–0.0068	0.0093	.463	–0.0019	0.0095	.842	–0.0026	0.0096	.789
Depression, γ_{009}	–0.0020	0.0015	.192	–0.0004	0.0017	.826	–0.0003	0.0017	.864
Caregiver sex: female, γ_{0010}	—	—	—	—	—	—	–0.0145	0.0132	.275
Caregiver age, γ_{0011}	—	—	—	—	—	—	–0.0001	0.0007	.844
Weekend, β_{01}, γ_{010}	–0.1018	0.0103	< .001	–0.1022	0.0103	< .001	–0.1020	0.0103	.000
Wake-up time, β_{02}, γ_{020}	–0.0132	0.0066	.045	–0.0127	0.0066	.056	–0.0130	0.0066	.051
Daily negative affect, β_{03}, γ_{030}	–0.0199	0.0147	.177	–0.0203	0.0147	.166	–0.0207	0.0147	.158
Daily positive affect, β_{04}, γ_{040}	–0.0073	0.0142	.610	–0.0072	0.0141	.612	–0.0101	0.0141	.473
Cortisol awakening response (CAR), π_1									
Average CAR, β_{10}, γ_{100}	0.0072	0.0118	.545	0.0066	0.0118	.573	0.0030	0.0135	.825
Childhood adversity, γ_{101}	0.0065	0.0048	.178	0.0056	0.0048	.247	0.0048	0.0049	.326
Youth self-esteem, γ_{102}	—	—	—	–0.0238	0.0170	.162	–0.0229	0.0173	.185
Caregiver self-esteem, γ_{103}	—	—	—	—	—	—	–0.0240	0.0163	.143
Female, γ_{104}	–0.0319	0.0128	.013	–0.0310	0.0128	.016	–0.0309	0.0129	.017
Age, γ_{105}	0.0060	0.0037	.106	0.0062	0.0037	.092	0.0068	0.0037	.066
Caregiver education, γ_{106}	0.0220	0.0133	.098	0.0220	0.0133	.098	0.0195	0.0139	.159
Health status, γ_{107}	0.0072	0.0095	.451	0.0063	0.0095	.509	0.0070	0.0096	.463
Neuroticism, γ_{108}	–0.0049	0.0092	.596	–0.0077	0.0093	.405	–0.0074	0.0093	.432
Depression, γ_{109}	–0.0005	0.0015	.746	–0.0015	0.0016	.368	–0.0016	0.0017	.326
Caregiver sex: female, γ_{1010}	—	—	—	—	—	—	0.0095	0.0142	.503
Caregiver age, γ_{1011}	—	—	—	—	—	—	–0.0004	0.0006	.502
Weekend, β_{11}, γ_{110}	–0.0293	0.0132	.026	–0.0291	0.0132	.028	–0.0292	0.0132	.027
Wake-up time, β_{12}, γ_{120}	–0.0192	0.0075	.010	–0.0195	0.0075	.009	–0.0193	0.0075	.010
Daily negative affect, β_{13}, γ_{130}	0.0006	0.0175	.971	0.0007	0.0175	.968	0.0014	0.0175	.934
Daily positive affect, β_{14}, γ_{140}	–0.0105	0.0127	.409	–0.0107	0.0126	.397	–0.0080	0.0129	.535
Time since waking, π_2									
Average linear slope, β_{20}, γ_{200}	–0.0385	0.0022	< .001	–0.0386	0.0022	< .001	–0.0390	0.0023	.000
Childhood adversity, γ_{201}	0.0001	0.0004	.714	0.0000	0.0004	.935	–0.0001	0.0004	.835
Child self-esteem, γ_{202}	—	—	—	–0.0030	0.0015	.038	–0.0029	0.0015	.049
Caregiver self-esteem, γ_{203}	—	—	—	—	—	—	–0.0029	0.0014	.043
Female, γ_{204}	–0.0012	0.0011	.293	–0.0011	0.0011	.341	–0.0011	0.0011	.349
Age, γ_{205}	–0.0006	0.0003	.088	–0.0005	0.0003	.111	–0.0004	0.0003	.180
Caregiver education, γ_{206}	–0.0001	0.0011	.954	0.0000	0.0011	.971	–0.0004	0.0012	.705
Health status, γ_{207}	–0.0002	0.0007	.791	–0.0003	0.0007	.687	–0.0002	0.0007	.830
Neuroticism, γ_{208}	–0.0005	0.0008	.560	–0.0009	0.0008	.312	–0.0008	0.0008	.345
Depression, γ_{209}	0.0001	0.0001	.311	0.0000	0.0001	.905	0.0000	0.0001	.970
Caregiver sex: female, γ_{2010}	—	—	—	—	—	—	0.0012	0.0012	.299
Caregiver age, γ_{2011}	—	—	—	—	—	—	0.0000	0.0001	.876
Weekend, β_{21}, γ_{210}	0.0076	0.0010	< .001	0.0076	0.0010	< .001	0.0076	0.0010	.000
Wake-up time, β_{22}, γ_{220}	–0.0007	0.0006	.215	–0.0008	0.0006	.196	–0.0008	0.0006	.208
Daily negative affect, β_{23}, γ_{230}	0.0024	0.0013	.060	0.0024	0.0013	.056	0.0025	0.0013	.051
Daily positive affect, β_{24}, γ_{240}	–0.0002	0.0012	.880	–0.0002	0.0012	.874	0.0001	0.0012	.955

(continued)

Table 5. *(continued)*

Fixed effect	Model 4			Model 5			Model 6		
	Estimate	*SE*	*p*	Estimate	*SE*	*p*	Estimate	*SE*	*p*
Time since waking^2, π_3									
Average curvature, β_{30}, γ_{300}	0.0008	0.0001	< .001	0.0008	0.0001	< .001	0.0008	0.0001	< .001
Smoking, π_4									
Intercept, β_{40}, γ_{400}	0.1472	0.0472	.002	0.1471	0.0473	.002	0.1469	0.0477	.002
Exercise, π_5									
Intercept, β_{50}, γ_{500}	0.0188	0.0083	.023	0.0192	0.0082	.019	0.0191	0.0082	.020

Note: Intercepts indicate average cortisol values at awakening, average slopes of time since waking indicate change in cortisol per 1-hr change in time, and average slopes of time since waking^2 indicate change in cortisol per 1-hr change in time^2. Thirty-three cortisol values were more than 3 standard deviations above the mean. Results for the main variables of interest remained significant when these observations were Winsorized.

Study 2, one with childhood adversity, youth self-esteem, and caregiver self-esteem as predictors (Model 3) and one with childhood adversity, youth self-esteem, caregiver self-esteem, and covariates as predictors (Model 6). Although no association emerged with CAR (Model 3: $\gamma_{103} = -0.026$, $p = .104$; Model 6: $\gamma_{103} = -0.024$, $p = .143$) or morning cortisol (Model 3: $\gamma_{003} = 0.029$, $p = .082$; Model 6: $\gamma_{003} = 0.031$, $p = .061$), higher caregiver self-esteem predicted a steeper diurnal cortisol slope (Model 3: $\gamma_{203} = -0.003$, $p = .035$; Model 6: $\gamma_{203} = -0.003$, $p = .043$). In other words, youths whose caregiver reported higher self-esteem had a steeper cortisol decline throughout the day.

Finally, in a series of supplementary analyses, we tested the same models using CA-SE and CA-RP separately. As shown in Table 6, we found an indirect effect between CA-RP scores and the cortisol slope via self-esteem, 95% CI = [−0.002504, −0.000260], as well as between CA-RP and the cortisol slope via self-esteem, 95% CI = [0.000012, 0.000208]. In contrast, no evidence for an indirect effect was found between CA-SE and any of the cortisol parameters. Further, no evidence was found for significant indirect effects linking childhood adversity to diurnal cortisol parameters via neuroticism or depression (see Table S2).

Discussion

Across two large and diverse samples of adults and youths, childhood adversity was directly associated with lower levels of cortisol at awakening, but not with CAR or cortisol slope. Childhood adversity was a predictor of lower self-esteem in both childhood (Study 2) and adulthood (Study 1), which in turn partially explained the effect of childhood adversity on lower morning cortisol. Further, although greater childhood adversity was not directly associated with a flatter cortisol slope, analyses revealed a significant indirect pathway through which greater adversity during development was linked to a flatter cortisol slope via self-esteem. These findings suggest that one's sense of self-worth might act as a proximal psychological mechanism through which childhood

Table 6. Results of Analyses on the Effects of Childhood-Adversity Measures on Cortisol Parameters Controlling for Covariates in Study 2

Measure	Morning cortisol	Cortisol awakening response	Cortisol slope
	Without controlling for self-esteem		
Childhood Adversity Stressful Events score	−0.0044† (0.0026)	0.0008 (0.0030)	0.0003 (0.0002)
Childhood Adversity Relationship With Parents score	−0.0043 (0.0027)	0.0042 (0.0027)	−0.0001 (0.0002)
	Controlling for self-esteem		
Childhood Adversity Stressful Events score	−0.0046† (0.0025), [−0.0004614, 0.0009475]	0.0009 (0.0030), [−0.0006664, 0.000305]	0.0003 (0.0002), [−0.00006944, 0.0000315]
Childhood Adversity Relationship With Parents score	−0.0030 (0.0027), [−0.002504, −0.0002603]	0.0035 (0.0028), [−0.000354, 0.001847]	−0.0002 (0.0002), [0.00001248, 0.0002075]

Note: The table shows unstandardized regression coefficients, followed by robust standard errors in parentheses; 95% confidence intervals are given in brackets for indirect effects.
$^\dagger p < .10$.

adversity gets embedded in human stress physiology. Specifically, higher self-esteem was associated with a steeper (i.e., healthier) cortisol decline during the day, whereas low self-esteem was associated with a flatter cortisol slope. Depression and neuroticism were tested as alterative pathways linking childhood adversity to cortisol secretion and were found not to be significant, which suggests that the indirect effect was specific to self-esteem. Nevertheless, it is plausible that other psychological pathways exist that might carry the effects of childhood adversity across the life span. For example, attachment security, a potential antecedent of self-esteem that forms during childhood (Pietromonaco & Powers, 2015), would be a strong candidate for playing such a role. Unfortunately, this construct was not assessed in our studies, but we hope that future work will test this hypothesis. Notably, in Study 2, the effect of youths' self-esteem on diurnal cortisol slope closely mirrored the effect of caregiver self-esteem on cortisol secretion. In other words, youths who had a caregiver with high self-esteem experienced a steeper decline in cortisol throughout the day—independent of the effects of youths' own self-esteem—compared with youths whose caregiver reported lower levels of self-esteem.

A harsh social environment during development (e.g., inconsistent parenting, poor sibling relationships, dysfunctional interactions within the family) can contribute to negative attitudes toward the self (Ryan et al., 1994), which can lead to adverse social, behavioral, and health consequences during childhood, such as social isolation from the peer group (Salzinger, Feldman, Ng-Mak, Mojica, & Stockhammer, 2001), antisocial behavior, and depression (Robinson, Garber, & Hilsman, 1995). Correspondingly, youths raised by neglectful or maltreating parents (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010) or exposed to childhood stressors (Koss, Hostinar, Donzella, & Gunnar, 2014) show disturbances in normative diurnal cortisol output, with lower cortisol at awakening and a flatter slope across the day, which have been respectively associated with depression and externalizing behavior. Interestingly, these childhood-adversity-related diurnal cortisol disturbances are found even at preschool age (1–5 years old; Gunnar & Vazquez, 2001; Koss et al., 2014). Whether these disturbances are driven by an earlier period of chronic cortisol elevation or other mechanisms (e.g., fetal programming by maternal stress) remains to be determined.

Regardless of the mechanism at play, the current findings provide empirical evidence that self-esteem is a pathway through which childhood adversity affects health via cortisol secretion in youths. However, the impact of childhood experience on one's representation of the self is not confined to childhood but can persist across the life span and set the stage for physical health problems during adulthood. For example, low self-esteem has been found to be a good predictor of decreased physical health (Trzesniewski et al., 2006) and death from myocardial infarction in adults at risk of heart disease. Similarly, low morning cortisol and flatter cortisol slopes are also associated with similar negative health-related outcomes in adulthood (Kumari, Shipley, Stafford, & Kivimaki, 2011). In light of this literature, our findings suggest that some of these effects might be connected to a dysregulation in the HPA axis driven by individual differences in self-esteem.

How does self-esteem influence daily cortisol fluctuation? The answer might be twofold. First, self-esteem calibrates sensitivity to social feedback, in particular social evaluative threats, which crucially activate stress physiology (Dickerson & Kemeny, 2004; Miller et al., 2007). Second, individuals with low and high self-esteem follow different pathways when dealing with cues of social threat. Individuals with low self-esteem often need and seek constant approval from others, and new potential experiences of disapproval, rejection, and failure can be agonizing for them. For this reason, their self-esteem system is overactive, with filtering of social threats being impaired (i.e., ambiguous cues of social exclusion are more likely to be perceived as social devaluations from others; Baldwin, Baccus, & Fitzsimons, 2004) and appraisal of social devaluations being more likely to be translated into negative self-evaluations (Baldwin et al., 2004). Consequently, compared with individuals who have high self-esteem, individuals with low self-esteem feel more shame (Leary et al., 1995) and engage more in rumination (Di Paula & Campbell, 2002), which, as proposed by social-self-preservation theory, strongly elicit cortisol secretion in both adults (Dickerson & Kemeny, 2004; Miller et al., 2007) and children (Lewis & Ramsay, 2002). Further, people with low self-esteem tend to rely on disengagement strategies (Ford & Collins, 2010) and social isolation (Leary et al., 1995) as ways to safeguard their weak sense of self, and the link between loneliness and HPA-atypical activation might explain not only the pattern of cortisol secretion observed in our sample during waking hours, but also the low cortisol level at awakening (Doane & Adam, 2010).

Finally, a pattern of covariation was found between caregivers' self-esteem and youths' diurnal cortisol rhythm. During development, children collect information about the social environment (i.e., presence and predictability of social threats) and physical environment (i.e., presence and predictability of physical threats) around them, and such information is used to program stress physiology, including the HPA axis. Parental figures (or caregivers) are the primary source of this information; their behavior, emotional tone, and even physiology (Papp, Pendry, & Adam, 2009) are detected by children, who adjust—to a different extent depending on the

age—their own biobehavioral responses accordingly. Thus, our findings are in line with this idea of transmission of environmental information from parent to child, especially the empirical work that showed associations between caregivers' psychological functioning and children's HPA activity (Lupien et al., 2000).

The present work is not without limitations. First, all childhood-adversity measures were self-reported. Future longitudinal studies are needed wherein individuals are followed across the life span and indicators of childhood stressful experiences are examined at a more refined level (e.g., via naturalistic observation). Next, our design did not allow us to consider any genetic effects. Obviously, the individual genetic makeup, the environment, and the interaction between genes and environment play a role in the emergence of the phenotypes under investigation. For example, genotypic variation in the serotonin transporter gene might be an interesting candidate for future studies that examine the role played by genetic effects in modulating the effect of childhood adversity on HPA activity via psychological pathways. A third limitation concerns our inability in Study 2 to assess participants' pubertal stage as well as compliance with the timing of cortisol sampling measures, which is particularly important when assessing CAR. Further, Study 1 and Study 2 differed in some important aspects, such as the research design (e.g., longitudinal vs. cross-sectional), the sample (adult community sample vs. youths affected by parental HIV), and the nature of the childhood-adversity measures, which might limit the generalizability of our findings. Finally, the effect sizes for the associations between self-esteem and cortisol parameters, despite being small in magnitude, are potentially meaningful. They are comparable with the effect sizes of associations between psychological factors (e.g., marital quality; Robles, Slatcher, Trombello, & McGinn, 2014) and physical health and between health behaviors (e.g., consumption of fruits and vegetables; He, Nowson, Lucas, & MacGregor, 2007) and health.

To summarize, in these studies, we investigated whether self-esteem is a viable mechanism through which childhood adversity affects diurnal cortisol parameters. Across two large and ethnically diverse samples, we found that, in both adults and children, childhood adversity was associated with disturbance in the typical cortisol circadian rhythm and that this association was mediated by low self-esteem. Further, in youths, we found support for the hypothesis that caregivers' self-esteem is also implicated in the regulation of youths' cortisol secretion throughout the day. Although future longitudinal research measuring childhood adversity, self-esteem, and cortisol multiple times is needed to corroborate these findings, they are consistent with the idea that some of the deleterious effects on health that are attributed to low self-esteem—which contains a psychological residue of childhood adversity—may be explained, at least in part, by dysregulation of the HPA axis.

Action Editor

Ian Gotlib served as action editor for this article.

Author Contributions

S. Zilioli, R. B. Slatcher, P. Chi, X. Li, J. Zhao, and G. Zhao conceived the studies. S. Zilioli analyzed the data. S. Zilioli, R. B. Slatcher, and P. Chi wrote the manuscript, and X. Li edited the manuscript.

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.

Supplemental Material

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

Note

1. It should be noted that there is no direct measure of the variance accounted for in HLM. However, once variables have been entered into an HLM, one can estimate a pseudo-R^2 statistic using the formula (*var* unconditional – *var* conditional)/*var* unconditional, where *var* can represent any level of variance. This formula provides an estimate of the proportion of reduction in variance for any random parameter (e.g., morning cortisol and cortisol slope at Level 3) in an HLM when one predictor variable is added (e.g., childhood adversity or self-esteem) to an unconditional growth-curve model (empty model, with no predictors at Level 2 and Level 3). In Study 1, the proportional variance reduction in the Level 3 intercept (morning cortisol) when childhood adversity was added was 1%, while it was 1% when self-esteem was added. The proportional variance reduction in the Level 3 slope (cortisol slope) variance when childhood adversity was added was 1%, while it was 2% when self-esteem was added. In Study 2, the proportional variance reduction in morning cortisol when childhood adversity was added was 2%, while it was 4% when self-esteem was added. The proportional variance reduction in the cortisol slope variance when self-esteem was added was 2%.

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