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# Childhood Adversity, Self-Esteem, and Diurnal Cortisol Profiles across the Lifespan

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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 children, 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. Lastly, those youth who had a caregiver with high self-esteem experienced a steeper decline in cortisol throughout the day compared to those youth 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 lifespan.

### Keywords

Childhood adversity; Cortisol; Self-esteem; Physical Health

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 medical 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).

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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 elusive. 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 socio-emotional skills) of these underlying biological processes (Repetti, Taylor, & Seeman, 2002).

In this research, we propose 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, 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, whose 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 lifespan (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 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., an abusive parent) and can act as amplifiers and/or buffers of existing environmental threats (e.g., unresponsive vs. responsive parents; 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. If the proposed mechanism is functioning during adulthood, we speculate that some evidence should also exist for its presence during childhood. For this reason, we also tested this hypothesis in a sample of 645 youth from

China, who were recruited as part of a study of youth affected by parental HIV/AIDS. Further, in this sample, we also tested whether caregiver self-esteem would modulate child cortisol secretion above and beyond the effect of a child's own self-esteem.

## Method

### Study 1

**Participants and Study Timeline—**Data for Study 1 were drawn from the National Study of Daily Experiences (NSDE II, 2004 – 2009; $n$ = 2,022), a subsample of Midlife in the United States (MIDUS) II (2004–2006, average age 56.62 years) – the second wave of data collection for MIDUS I (1995–1996, average age = 47.78 years), a large panel survey of adults between the ages of 25 and 74. The NSDE II included four days of salivary cortisol collection and eight 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, demographic information (age, gender, ethnicity, education, and physical health), and potential psychological confounds (neuroticism, depressed affect, and daily positive and negative affect) during MIDUS I and/or MIDUS II, and cortisol data for NSDE II. Specifically, information about childhood adversity and parental education was collected during the first wave (i.e., MIDUS I), while information about self-esteem and psychological covariates was collected during the second wave (i.e., MIDUS II). Likewise, age, gender, ethnicity, education, and physical health as reported at MIDUS II were used. Lastly, 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, 95.5% White/Caucasian, 71.2% completed some college or more; age, $M$ = 56.62 years, $SD$ = 12.05 years).

#### Measures

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

First, we counted how many stressful events individuals underwent during their childhood (up to sixteen years of age). The list of stressful events included nineteen episodes: death of at least one of the parents, repeated a school year, sent away from home for doing something wrong, parental unemployment when wanting to work, parental alcohol problems, parental drug problems, dropped out of school, expelled from school, failed out of school, fired from a job, death of a sibling, divorced parents, lost home due to natural disaster or similar causes, physically abused, sexually abused, jail retention, receipt of welfare, moved to new neighborhood/town more than four times, and adopted. This scale, which we refer to as the Childhood Adversity Stressful Events (CA-SE) scale, was constructed by calculating the

sum of each item and was computed for cases that had at least 16 items with valid values. Scores on the CA-SE ranged from 0 to 7 ($M$ = .83, $SD$ = 1.12).

Second, we considered parental affection, which was also assessed at MIDUS I with a validated questionnaire (Rossi, 2001). Participants had to answer seven questions about their relationship with their mother and father on a 5-point Likert scale (1 = Excellent, 5 = Poor). Averaging these two scales yielded a measure of overall parental affection, which showed high internal consistency ($\alpha$ = .92). Sample items included, "How much could you confide in her/him about things that were bothering you?" and "How much effort did she/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 ranged from 1.04 to 4.04 ($M$ = 2.07, $SD$ = .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 "Often" to "Never." Items were first reverse-coded and then averaged, so that higher scores represent higher levels of abuse occurrence. We refer to this scale as the Childhood Adversity Physical/Emotional Abuse (CA-PEA) scale. Scores on the CA-PEA ranged from 1 to 4 ($M$ = 1.71, $SD$ = .54).

For each domain, measures were converted to standardized z-scores and a composite of childhood adversity, or simply CA, was computed as the sum of these z-scores, with higher scores indicating a more hostile childhood environment. CLA scores ranged from −3.66 to 10.70 ($M$ = 0.00, $SD$ = 2.19).

**Self-esteem:** In Study 1, self-esteem was assessed using the Rosenberg Self-Esteem Scale (Rosenberg, 1965), which comprised seven items answered on a 7-point Likert scale (1 = Strongly agree, 7 = Strongly disagree). Sample items included, "At times I feel that I am no good at all" and "On the whole, I am satisfied with myself." ($\alpha$ = .76). The scale was constructed by calculating the sum of the values of the items and was computed for cases that had valid values for at least four items on the scale. 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 average, saliva collection during NSDE II occurred 20.54 months ($SD$ = 13.57) after the MIDUS II questionnaire assessment. On four consecutive days of the 8-day NSDE study period, participants self-collected saliva samples at four time points each day; immediately upon waking, 30 minutes later to assess cortisol awakening response (CAR), before lunch, and at bedtime. Nightly telephone interviews and paper-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 served as a container of the participant's salivettes and was equipped with a computer chip that recorded every time it had been opened and closed. Correlations between self-reported sample times and times obtained from the "Smart Box"

were excellent, ranging from 0.75 for the evening samples to 0.95 for the morning samples (Almeida et al., 2009). As to 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 1463 individuals, of the available 5737 CAR cortisol values, 860 deviated by 15 min or more from the requested 30-min 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%. Saliva collection compliance was assessed using nightly telephone interviews and paper-and-pencil logs included in the collection kits. Cortisol values were log-transformed to correct for positive skew in the cortisol distribution (Adam & Kumari, 2009). In order to ensure that all transformed scores were positive, a constant of 1 was added 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 Study 1. Values ranged from "some grade school" to "doctoral degree" and we identified a group of clearly low childhood SES adults, which was defined as both parents having less than 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 Slopen et al.'s conceptualization (Slopen et al., 2010). Further, as reported in Table 1, while CA-SE, CA-RP, and CA-PEA 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. Specifically, for Study 1, covariates included age, gender (male = 0, female = 1), education (0 = high school or less, 1 = some college or more), and race/ethnicity (0 = white, 1 = non-white) at the person level. At the daily level we controlled for day or the week (0 = weekday, 1= weekend) and wake-up time on days of salivary cortisol sampling.

**Health Covariates:** In the analyses we further controlled for smoking status, medication use, physical health, and daily exercise. Smoking status was coded as 1 if respondents reported being a current cigarette smoker during the MIDUS II interview or reported smoking any cigarettes across the NSDE II study period. People that 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. People reporting using medication were assigned a score of 1, while people that reported no use of medication or did not answer this question were assigned a score of 0.

During MIDUS II, participants reported whether in the past twelve months they had any chronic condition (0 = no, 1 = yes). This variable was used as an index of respondents' physical health.

Lastly, as part of the NSDE II telephone interviews, participants also reported how many hours time they spent exercising on each day. This variable was winsorized at +/− three standard deviations and included as covariate at the daily level ($M$ = .6839, $SD$ = 1.37).

**Psychological Covariates:** In order to show the specificity of self-esteem in explaining the link between childhood adversity and diurnal cortisol, in our analyses we controlled for the overlapping trait of neuroticism measured at MIDUS II. Neuroticism was assessed via four personality adjectives (i.e., moody, worrying, nervous, and calm), which were rated on a 4-point Likert scale (1 = A Lot; 4 = Not At All). This scale ($\alpha$ = .74) was developed from a cluster of established Big Five trait adjectives and was constructed by calculating the mean of the values of the items, with higher scores indicating higher neuroticism. Scores ranged from 1 to 4 ($M$ = 2.04, $SD$ = .63). We controlled for neuroticism because this personality trait shows the strongest correlation with self-esteem compared to the other Big 5 personality traits (Robins, Hendin, & Trzesniewski, 2001) and, similarly to self-esteem, its development can be driven by childhood experiences (Roy, 2002). By controlling for neuroticism, we wanted to ensure that observed effects were driven by self-esteem rather than by shared variance with neuroticism. Because of the overlap between neuroticism and self-esteem, we also tested whether neuroticism would be a psychological pathway linking CLA to cortisol secretion.

We also controlled for depressed affect. Specifically, participants reported whether they experienced depressive symptoms during 2 weeks in the past 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 ($M$ = .48, $SD$ = 1.57). Previous work as suggested that depression might act as an intermediary of the CLA effects on the HPA activity (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008); thus, as in the case of neuroticism, we tested the indirect effects linking CLA to the various cortisol parameters via depressed affect.

Lastly, we also 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). For daily positive affect ($\alpha$ = 0.96), scores ranged from 0 to 4 ($M$ = 2.76, $SD$ = .76), while for daily negative affect ($\alpha$ = 0.89), scores ranged from 0 to 2.8 ($M$ = .18, $SD$ = .29).

**Data Analysis—**At the daily level, the incidence of missing data among the variables included in this study was 2.9%. In order to curtail the bias associated with pairwise or

listwise deletion of missing data (Schafer & Graham, 2002), we used the expectation maximization (EM) 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. Specifically, HLM allows regressing multiple cortisol parameters at the same time (cortisol at wakeup, CAR, and slope), on both daily-level (e.g., within-person daily wakeup time) and person-level (e.g., between-person self-esteem, age, gender) predictors. Furthermore, HLM can estimate slopes and intercepts even with missing cortisol data. Following prior diurnal cortisol research (Adam & Kumari, 2009), Time Since Waking, Time Since Waking-squared, and CAR (dummy coded 0 or 1) were modeled at Level-1 to provide estimates of each participant's diurnal cortisol rhythm. At Level-3 (person-level), we first ran models with CLA as the main predictor (Models 1 and 3), and then with CLA and self-esteem (Models 2 and 4) as the main predictors. These analyses were first conducted without controlling for covariates (Models 1 and 2) and then controlling for the covariates mentioned above (Model 3 and 4), which were included either at Level-2 (i.e., daily covariates) or at Level-3 (i.e., person covariates). Following statistical recommendations on HLM centering (Enders & Tofighi, 2007), continuous covariates at level-2 and level-3 were grand-mean centered. 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 only used as predictor for the cortisol slope. In keeping with prior studies, cortisol intercept, slope (effect of time), and CAR were all allowed to vary randomly at Level-2 and Level-3 (i.e., treated as random effects). All HLM significance tests were 2-tailed with robust standard errors.

In order to test whether individual self-esteem explained some of the covariation between CLA and cortisol parameters, we first ran a regression where self-esteem was regressed on CLA. The obtained regression coefficient and asymptotic sampling variance for the association between CLA (our IV) 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 DVs) obtained in our Model 2 (i.e., the HLM Model run without controlling for covariates) to derive 95% confidence intervals 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 where self-esteem was regressed on CLA while controlling for appropriate (i.e. non-specific to cortisol) demographic (i.e., age, gender, education, race/ethnicity, childhood SES) and psychological covariates (i.e., 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 our Model 4 (i.e., the HLM Model run controlling for covariates) to derive the 95% confidence intervals for indirect effects via the Monte Carlo method. Confidence intervals not containing 0 indicate statistically significant indirect effects.

**Supplementary analyses—**Although the main hypothesis of our study was to investigate the cumulative effect of a variety or 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 where cortisol was predicted by each facet of childhood adversity first and, next, by each facet of childhood adversities together with self-esteem. These models were run while controlling for covariates and a summary of the main results is reported in Table 3.

In a second set of supplementary analyses we tested whether neuroticism and depressed affect acted as alternative intermediaries of the effect of CLA on daily cortisol secretion. These analyses are reported in the supplementary materials in Table SI.

## Study 2

**Participants—**Data for Study 2 were drawn from a community sample of 790 children and adolescents (or simply, youth) aged 6–17 affected by parental HIV and their current primary caregivers who 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 based on power analysis for the hypothesized effect (small-to-medium hypothesized effect size) of the intervention. Of the larger sample of 790 youth, 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 youth, 645 (86.4%) provided saliva samples for cortisol analyses (48.1% female, age, M = 10.67 years, SD = 1.79 years) and were therefore used in the analyses. As reported by the field researchers in this study, 71 children had at least one dead parent due to HIV/AIDS, 554 youth had at least one HIV-positive parent, while data for this variable were not available for 12 youth.

Both youth and caregivers 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 youth—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—**Similarly to Study 1, self-report data from two sources were used to create the CLA composite. First, we used 15 items to assess youths' experience of a number of stressful life events during the past six months. Sample 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, being a witness to involuntary violence, 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: death of at least one of the parents, both parents diagnosed with HIV (as reported by the researcher), and moved to a new residence more than twice. This scale, which we refer to as the Childhood Adversity Stressful Events (CA-SE) scale, was constructed by calculating the sum of each item and was computed for cases that had at least three items with valid values (note that the

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

Second, we assessed parenting quality using both youth and caregiver reports of 1) parental responsiveness and 2) parental trust. Youth assessed parental responsiveness 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). 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 it as well (α = .66). Both scales were constructed by calculating the mean of the values of the items, with higher scores indicating higher parental responsiveness. Scores on the youth-reported parental responsiveness scale ranged from 1 to 4 ($M$ = 2.59, $SD$ = .66), while scores on the caregiver-reported parental responsiveness scale ranged from 1 to 4 ($M$ = 2.73, $SD$ = .56).

Further, youth and caregivers used the Trusting Relationship Questionnaire (TRQ; Mustillo, Dorsey, & Farmer, 2005) to asses caregiver relationship trust (α = .90, for the youth version of the questionnaire; α = .85, for the caregiver version of the questionnaire). Sample items of the caregiver version included "Does the child identify things he or she likes about you?", "Does the child talk to you about his or her problems?", with the youth version substituting "adult" for "child" (for more details, see Mustillo et al., 2005). One item (i.e., "Do you seek help from him/her when you face difficulties?", for the youth questionnaire; "Do the child seek help from you when he/she face difficulties?", for the caregiver questionnaire) was added to original list of 18 items. Scales were constructed by calculating the mean of the values of the items, with higher scores indicating higher parental trust. Scores on youth-reported parental trust ranged from 1 to 5 ($M$ = 2.66, $SD$ = .75), while scores on the caregiver-reported parental trust ranged from 1.26 to 5 ($M$ = 3.04, $SD$ = .58).

Scores on these four scales correlated among each other (average $r$ = 0.204, range $r$ = 0.102—0.467, lowest $p < 0.001$) — except for caregiver-reported parental responsiveness and youth-reported parental trust ($r$ = .025, $p$ = .535) — and were z-scored to form a composite index of parenting quality (CA-RP), similarly to Study 1. Specifically, CA-RP was constructed by calculating the sum of each subscale and was computed for cases that had valid scores on all four subscales. Final scores on the CA-RP ranged from −7.44 to 9.02 ($M$ = 1.60, $SD$ = 2.47), with higher scores representing harsher parenting.

For each childhood adversity domain available at Study 2 (CA-SE and CA-RP), measures were converted to z-scores and a composite of childhood adversity, or simply CA, was computed as the sum of these z-scores, with higher scores indicating a more hostile childhood environment. CLA scores ranged from −4.08 to 4.95 ($M$ = 0.00, SD = 1.41).

**Self-esteem:** In Study 2, youth and caregiver self-esteem were assessed using the Rosenberg Self-Esteem Scale (Rosenberg, 1965), which comprised ten items answered on a 4-point Likert scale (1 = Strongly Disagree, 4 = Strongly Agree). Sample items included, "At times I feel 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). These scales were constructed by

calculating the average of the values, with higher scores indicating higher self-esteem. Scores for the youth self-esteem scale ranged from 1.6 to 4 ($M$ = 2.76, $SD$ = .44), while scores for the caregiver self-esteem scale ranged from 1.6 to 3.9 ($M$ = 2.88, $SD$ = .41).

**Cortisol—**Participants self-collected saliva samples at four time points each day for three days: immediately upon waking (prior to any eating, drinking, or exercise), thirty minutes later to assess cortisol awakening response (CAR), one hour before dinnertime, and then at bedtime. Prior to saliva collection, the investigators showed youth the correct procedure to collect saliva samples using Salivettes (Sarstedt, Rommelsdorft, Germany) 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, Inc, Fullerton, CA). In Study 2, 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% of participants providing at least 8 of the 12 possible saliva samples across the 3 days. As to CAR compliance, in our sample of 645 individuals, of the available 1810 CAR cortisol values, 453 deviated by 15 min or more from the requested 30-min interval. Similarly to Study 1, these cortisol values were dropped from the analyses. Lastly, cortisol values were log-transformed to correct for positive skew in the cortisol distribution (Adam & Kumari, 2009). In order to ensure that all transformed scores were positive, a constant of 1 was added before the transformation.

**Demographic, Psychological, and Health Covariates:** As in the case of Study 1, demographic covariates included 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 or the week (0 = weekday, 1= weekend), and daily wake up time. At the momentary level (i.e., at the time collection of each saliva sample) youth reported whether they smoked and/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 adjectives (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). This scale was constructed by calculating the mean of the values of the items after the second item was recoded such that with higher scores indicated higher neuroticism. Scores ranged from 1 to 4 ($M$ = 2.39, $SD$ = .73).

Depression was measured using a short version (10-item) of the Center for Epidemiologic Studies Depression Scale for children (CES-D, Fendrich, Weissman, & Warner, 1990). Children were instructed to respond how much they might felt or acted as indicated in ten items in the past week on 4-point Likert scale (1 = not at all, 3 = a lot). Sample items included, "I was bothered by things that usually don't bother me", "I felt like I was too tired to do things this past week" (α = .62). This scale was constructed by calculating the sum of

the values and was computed for cases that had valid values on all ten items of the scale. Higher scores indicate higher depression ($M$ = 20.16, $SD$ = 4.29).

Daily negative affect (α = 0.76) was assessed via six adjectives (i.e., sad, upset, fear, lonely, angry, worried) rated on 3-point Likert scale) while daily positive affect (α = 0.73) was assessed via six adjectives (i.e., happy, excited, energetic, confident, curious, calm) rated on 3-point Likert scale (1 = Not At All; 3 = Almost All Day). For daily positive affect, scores ranged from 1 to 3 ($M$ = 2.19, $SD$ = .50), while for daily negative affect, scores ranged from 1 to 3 ($M$ = 1.34, $SD$ = .41).

Lastly, at the person level we also controlled for perceived health status, which was self-reported by each youth and by their caregiver on a 5-point Likert-type scale ranging from 1 (very poor) to 5 (very good). This scale was constructed by calculating the mean of the two items and was computed for cases that had valid values on both items. Higher scores indicate higher perceived health status ($M$ = 4.1, $SD$ = .75).

**Analyses—**At the daily level, the incidence of missing data among the variables included in this study was 9.8%, while at the at the person level, the incidence of missing data was 3%. In order to curtail the bias associated with pairwise or listwise deletion of missing data (Schafer & Graham, 2002), we used the expectation maximization (EM) 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, Hierarchical Linear Modeling (HLM) was used for data analyses in Study 2. Models were run with (Models 4 and 5) and without (Models 1 and 2) covariates and in a hierarchical fashion, introducing CLA first (Models 1 and 4) and, CLA and self-esteem next (Models 2 and 5). Continuous covariates at Level-2 and Level-3 were grand-mean centered. Confidence intervals for indirect effects were estimated using the Monte Carlo method. These analyses were run with and without covariates.

Lastly, in Study 2 we also tested the hypothesis that the caregiver self-esteem would modulate youth cortisol. For this reason, caregiver self-esteem was added as an additional predictor (Model 3). These analyses were repeated controlling for additional covariates, including caregiver age and caregiver gender (Model 6).

**Supplementary analyses—**Following the approach adopted in Study 1, we ran additional analyses where cortisol was predicted by each facet of childhood adversity and by each facet of childhood adversity together with self-esteem. These models were run while controlling for covariates and a summary of the main results can be found in Table 6.

As in the case of Study 1, we independently tested whether neuroticism and depression acted as intermediaries of the effect of CLA on daily cortisol secretion. These analyses are reported in the supplementary materials in Table SII.

## Results

### Study 1

Bivariate correlations among Study 1 person-level predictors are reported in Table 1, whereas the relationships between CA, self-esteem, and cortisol parameters with and without controlling for covariates are reported in Table 2.

Four models—one with CA as the only predictor (Model 1), one with CA and self-esteem as predictors (Model 2), one with CA and covariates as predictors (Model 3), and one with CA, self-esteem, and covariates as predictors (Model 4)—were run. As shown in Model 1 and Model 3, CA was a significant predictor of morning cortisol, such that individuals who reported more adverse childhood conditions had lower levels of cortisol at wakeup ($\gamma_{001}$ = −.021, $p$ < .001; $\gamma_{001}$ = −.013, $p$ = .020, after controlling for covariates). CA was also associated with a flatter cortisol slope ($\gamma_{201}$ = .001, $p$ = .032); however, this association disappeared after controlling for covariates ($\gamma_{201}$ = .000, $p$ = .395). CA was not a significant predictor of CAR ($\gamma_{101}$ = .000, $p$ = .996; $\gamma_{101}$ = −.001, $p$ = .782, after controlling for covariates). Next, self-esteem was introduced in the analyses. As shown in Model 2, individuals reporting higher self-esteem had higher morning cortisol ($\gamma_{002}$ = .005, $p$ = .006; $\gamma_{002}$ = .004, $p$ = .044, after controlling for covariates) and experienced a steeper cortisol slope ($\gamma_{202}$ = −.001, $p$ = .001; $\gamma_{202}$ = −.001, $p$ = .010, after controlling for covariates). Self-esteem was not a significant predictor of CAR ($\gamma_{102}$ = −.000, $p$ = .910; $\gamma_{102}$ = .000, $p$ = .937, after controlling for covariates). Notably, when self-esteem was introduced in the analyses, the effect of CA was reduced both for morning cortisol ($\gamma_{002}$ = −.018, $p$ = .001; $\gamma_{002}$ = −.012, $p$ = .030, after controlling for covariates) and cortisol slope ($\gamma_{201}$ = .001, $p$ = .157; $\gamma_{201}$ = .000, $p$ = .524, after controlling for covariates).

Next, we tested whether the association between CA and morning cortisol and between CA and the cortisol slope was partially explained by self-esteem. Regression analyses showed that CA negatively predicted self-esteem (β = −.696, *SE* = .083, $p$ < .001; β = −.278, *SE* = .073, $p$ < .001 after controlling for covariates). Monte Carlo analyses showed a significant indirect effect of CA on morning cortisol via self-esteem (95% CI: −0.006699, −0.001061; 95% CI: −0.002716, −0.000027, after controlling for covariates) as well as a significant indirect effect of CA on diurnal cortisol slope via self-esteem (95% CI: 0.000140, 0.000633; 95% CI: 0.000029, 0.000295, after controlling for covariates), indicating 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 one-tenth of the total effect of childhood adversity on morning cortisol and about one-third of the total effect of childhood adversity on the cortisol slope.

In a series of supplementary analyses we tested the same models reported above using CA-SE, CA-RP, and CA-PEA separately. As shown in Table 3, we found an indirect effect between CA-SE and the cortisol slope [0.000012, 0.000397] via self-esteem. Further, self-esteem partially explained the link between CA-RP and morning cortisol [−0.010140, −0.000132] as well as CA-RP and the cortisol slope [0.000145, 0.001149]. In contrast, no evidence for an indirect effect was found between CA-PEA and any of the cortisol

parameters. Lastly, no evidence was found for significant indirect effects linking CA to diurnal cortisol parameters via neuroticism or depressed affect (see Table SI).

## Study 2

Bivariate correlations among Study 2 predictors are reported in Table 4, whereas the relationship between CA, self-esteem, and cortisol parameters with and without controlling for covariates are reported in Table 5.

In addition to the four models run in Study 1, two more models were run in Study 2, one with CA, self-esteem, and caregiver self-esteem as predictors (Model 3) and one with CA, self-esteem, caregiver self-esteem, and covariates as predictors. As shown in Model 1 and Model 4, CA was a significant predictor of morning cortisol, such that individuals who reported more adverse childhood conditions had lower levels of cortisol at wakeup ($\gamma_{001}$ = −.011, $p$ = .009; $\gamma_{001}$ = −.012, $p$ = .017, after controlling for covariates). However, CLA was not associated with the cortisol slope ($\gamma_{201}$ = .000, $p$ = .551, $\gamma_{201}$ = .000, $p$ = .714 after controlling for covariates) or CAR ($\gamma_{101}$ = .006, $p$ = .192; $\gamma_{101}$ = .007, $p$ = .178, after controlling for covariates). Next, self-esteem was introduced as a predictor in the analyses. Corroborating the findings from Study 1, individuals reporting higher self-esteem had higher morning cortisol ($\gamma_{002}$ = .044, $p$ = .003; $\gamma_{002}$ = .041, $p$ = .010, after controlling for covariates) and a steeper cortisol slope ($\gamma_{202}$ = −.003, $p$ = .025; $\gamma_{202}$ = −.003, $p$ = .038, after controlling for covariates). In other words, individuals who reported higher self-esteem had higher cortisol at wakeup and a steeper cortisol decline through the day. Self-esteem was not a significant predictor of CAR ($\gamma_{102}$ = −.018, $p$ = .254; $\gamma_{102}$ = −.024, $p$ = .162, after controlling for covariates). Effect sizes in Study 2 were comparable to the effect sizes obtained in Study 1**.

We next tested whether the associations between CA 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 CA and cortisol slope through self-esteem. Regression analyses showed that CA negatively predicted self-esteem (β = −.067, *SE* = .012, $p$ < .001; β = −.034, *SE* = .011, $p$ = .003 after controlling for covariates). Monte Carlo analyses showed a significant indirect effect of CA on morning cortisol via self-esteem (95% CI: −0.005316, −0.000973; 95% CI: −0.003037, −0.000203, after controlling for covariates) as well as a significant indirect effect of CA on diurnal cortisol slope via self-esteem (95% CI: 0.000026, 0.000416; 95% CI: 0.000004, 0.000242, after controlling for covariates), indicating that high childhood adversity was linked to low morning cortisol and a flatter cortisol slope via low

**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 model, one can estimate a pseudo $R^2$ statistic using the formula (*var* unconditional − *var* conditional)/*var* unconditional, where var can represent any level variance. This formula provides an estimate of the proportion reduction in variance for any random parameter (e.g., morning cortisol and cortisol slope at Level-3) in an HLM model when adding one predictor variable (e.g., CLA 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 (i.e., morning cortisol) when adding CLA was 1%, while it was 1% when adding self-esteem. The proportional variance reduction in the Level-3 slope (i.e., cortisol slope) variance when adding CLA was 1%, while it was 2% when adding self-esteem. In Study 2, the proportional variance reduction in morning cortisol when adding CLA was 2%, while it was 4% when adding self-esteem. The proportional variance reduction in the cortisol slope variance when adding self-esteem was 2%.

self-esteem. Next, we calculated the ratio of the indirect effect to the total effect (i.e., indirect effect + direct effect) and found that self-esteem mediated approximately one-eighth of the total effect of childhood adversity on morning cortisol and three-fourths of the total effect of childhood adversity on the cortisol slope.

In Study 2, we tested the hypothesis that caregiver self-esteem would be—above and beyond the effects of youth self-esteem—associated with youth cortisol parameters. Although no association emerged with CAR ($\gamma_{103} = -.026$, $p = .104$; $\gamma_{103} = -.024$, $p = .143$, after controlling for covariates) or morning cortisol ($\gamma_{003} = .029$, $p = .082$; $\gamma_{003} = .031$, $p = .061$, after controlling for covariates), higher caregiver self-esteem predicted a steeper diurnal cortisol slope ($\gamma_{203} = -.003$, $p = .035$; $\gamma_{203} = -.003$, $p = .043$, after controlling for covariates). In other words, individuals whose caregiver reported higher self-esteem had a steeper cortisol decline throughout the day.

Lastly, in a series of supplementary analyses we tested the same models reported above using CA-SE and CA-RP separately. As shown in Table 6, self-esteem we found an indirect effect between CA-RP and morning cortisol [−0.002504, −0.000260] as well as CARP and the cortisol slope [0.000012, 0.000208] via self-esteem. In contrast, no evidence for an indirect effect was found between CA-SE and any of the cortisol parameters. Lastly, no evidence was found for significant indirect effects linking CA to diurnal cortisol parameters via neuroticism or depression (see Table SII).

## Discussion

Across two large and diverse samples of adults and youth, we found that childhood adversity was directly associated with lower levels of cortisol at wakeup, but not with CAR or cortisol slope. Childhood adversity was a strong predictor of lower self-esteem in both childhood (Study 2) and adulthood (Study 1), which in turn partially explained the effect of the former on lower morning cortisol. Further, although childhood adversity was not directly associated with a flatter cortisol slope, indirect effect 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 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 CA to cortisol secretion and were found not to be significant, suggesting that the indirect effect was specific to self-esteem. Nevertheless, it is plausible that other psychological pathways exist that might carry the effects of CA across the lifespan. 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 youth self-esteem on diurnal cortisol slope closely mirrored the effect of caregiver self-esteem on cortisol secretion. In other words, those youth 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 to those youth 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 towards 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, youth raised by neglectful/maltreating parents (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010) or exposed to childhood stressors (Koss, Hostinar, Donzella, & Gunnar, 2014) show disturbances in the normative diurnal cortisol output, with lower cortisol at wakeup and a flatter slope across the day, which have been respectively associated with depression and externalizing behavior. Interestingly, these CLA-related diurnal cortisol disturbances are found as childhood as preschool years (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 youth. However, the impact of childhood experience on one's representation of the self is not confined to childhood but can persist across the lifespan and set the stage for physical health problems during adulthood. For example, low self-esteem has been found to be a good predictor of poorer 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: 1) self-esteem calibrates sensitivity to social feedback, in particular social evaluative threats, which crucially activate stress physiology (Dickerson & Kemeny, 2004; Miller et al., 2007); and, 2) low and high self-esteem individuals 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 to high self-esteem people, 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 the 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).

Lastly, 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. 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 the child, 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 caregiver psychological functioning and child's HPA activity (Lupien et al., 2000).

The present work is not without limitations. First, all CLA measures were self-reported. Future longitudinal studies are needed wherein individuals are followed across the lifespan and indicators of childhood stressful experiences are examined at a more refined level (e.g., via naturalistic observation). Next, our study 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 CLA on HPA activity via psychological pathway. A third limitation concerns the 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. youth affected by parental HIV), and the nature of the CLA measures, which might limit the generalizability of our findings. Lastly, the effect sizes for the associations between self-esteem and cortisol parameters, despite being small in magnitude, are potentially meaningful. They are comparable to the effect sizes of associations between psychological factors (e.g., marital quality; Robles, Slatcher, Trombello, & McGinn, 2014) and physical health and health behaviors (e.g., consumption of fruits and vegetables; He, Nowson, Lucas, & MacGregor, 2007) and health.

To summarize, these studies investigated whether self-esteem is a viable mechanism through which childhood adversity impacts 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 children, we found support for the hypothesis that caregiver's self-esteem is also implicated in the regulation of children cortisol secretion throughout the day. Although future longitudinal research measuring childhood adversity, self-esteem, and cortisol multiple time across time 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 psychological residue of childhood adversity—may be explained, at least in part, by dysregulation of the HPA axis.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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**Table 1**

Bivariate Correlations Between Study 1 Person-Level Variables

| Descriptive variables | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Female | 1.000 | 0.010 | −0.030 | −0.061* | 0.009 | −0.010 | 0.194** | 0.068** | −0.009 | 0.116** | −0.043† | 0.029 | 0.135** | 0.111** | −0.103** |
| 2. Non-White | | 1.000 | −0.013 | 0.014 | 0.033 | −0.039 | −0.050† | −0.006 | 0.082* | 0.011 | 0.024 | 0.054* | −0.058* | −0.028 | 0.005 |
| 3. Age Wave 2 | | | 1.000 | −0.120** | 0.241** | −0.134** | −0.038 | 0.161** | −0.058* | −0.065* | −0.157** | −0.128** | −0.206** | −0.110** | 0.139** |
| 4. Some College Wave 2 | | | | 1.000 | −0.231** | −0.120** | 0.019 | −0.001 | −0.106** | −0.050 | −0.076** | −0.106** | −0.093** | −0.048† | 0.133** |
| 5. Childhood SES Wave 1 | | | | | 1.000 | 0.019 | −0.002 | 0.011 | 0.004 | 0.034 | −0.012 | 0.012 | −0.006 | −0.039 | −0.030 |
| 6. Smoking Status | | | | | | 1.000 | −0.017 | 0.001 | 0.074** | 0.086** | 0.122** | 0.129** | 0.092** | 0.109** | −0.090** |
| 7. Medication | | | | | | | 1.000 | 0.191** | 0.032 | 0.072** | 0.012 | 0.053* | 0.079** | 0.087** | −0.050† |
| 8. Chronic Condition | | | | | | | | 1.000 | 0.062* | 0.039 | 0.031 | 0.060* | 0.122** | 0.095** | −0.127** |
| 9. CA-SE Wave 1 | | | | | | | | | 1.000 | 0.255** | 0.218** | 0.673** | 0.117** | 0.099** | −0.134** |
| 10. CA-RP Wave 1 | | | | | | | | | | 1.000 | 0.422** | 0.766** | 0.170** | 0.115** | −0.201** |
| 11. CA-PEA Wave 1 | | | | | | | | | | | 1.000 | 0.749** | 0.153** | 0.109** | −0.134** |
| 12. CA Wave 1 | | | | | | | | | | | | 1.000 | 0.201** | 0.148** | −0.214** |
| 13. Neuroticism Wave 2 | | | | | | | | | | | | | 1.000 | 0.276** | −0.527** |
| 14. Depressed Affect Wave 2 | | | | | | | | | | | | | | 1.000 | −0.311** |
| 15. Self-Esteem Wave 2 | | | | | | | | | | | | | | | 1.000 |

*Note:* SES: Socioeconomic Status; CA-SE: Childhood Adversity Stressful Events; CA-RP: Childhood Adversity Relationship with Parents; CA-PEA: Childhood Adversity Physical/Emotional Abuse; CA: Childhood Adversity.

† $p < .10$,

* $p < .05$,

** $p < .01$

*Psychol Sci*. Author manuscript; available in PMC 2017 September 01.

**Table 2**

HLM Models of Diurnal Cortisol Parameters in Study 1

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Morning cortisol, π0** | | | | | | | | | | | | |
| Average Morning Cortisol, β00, γ000 | 2.6552 | 0.0132 | <.001 | 2.6552 | 0.0132 | <.001 | 2.7267 | 0.0369 | <.001 | 2.7263 | 0.0369 | <.001 |
| CA Wave 1, γ001 | −0.0214 | 0.0053 | <.001 | −0.0176 | 0.0055 | 0.001 | −0.0130 | 0.0056 | 0.020 | −0.0121 | 0.0056 | 0.030 |
| Self-Esteem Wave 2, γ002 | -- | -- | -- | 0.0054 | 0.0019 | 0.01 | -- | -- | -- | 0.0043 | 0.0021 | 0.044 |
| Female, γ003 | -- | -- | -- | -- | -- | -- | −0.1127 | 0.0247 | <.001 | −0.1112 | 0.0247 | <.001 |
| Non-White, γ004 | -- | -- | -- | -- | -- | -- | −0.1645 | 0.0581 | 0.005 | −0.1615 | 0.0577 | <.001 |
| Age Wave 2, γ005 | -- | -- | -- | -- | -- | -- | 0.0043 | 0.0012 | <.001 | 0.0042 | 0.0012 | <.001 |
| Some College Wave 2, γ006 | -- | -- | -- | -- | -- | -- | 0.1006 | 0.0275 | <.001 | 0.0952 | 0.0275 | 0.001 |
| Childhood SES Wave 2, γ007 | -- | -- | -- | -- | -- | -- | −0.0279 | 0.0335 | 0.406 | −0.0257 | 0.0336 | 0.444 |
| Smoking Status, γ008 | -- | -- | -- | -- | -- | -- | −0.0465 | 0.0358 | 0.194 | −0.0459 | 0.0356 | 0.197 |
| Medication, γ009 | -- | -- | -- | -- | -- | -- | −0.0442 | 0.0258 | 0.088 | −0.0454 | 0.0258 | 0.078 |
| Chronic Condition, γ0010 | -- | -- | -- | -- | -- | -- | −0.0404 | 0.0275 | 0.142 | −0.0364 | 0.0273 | 0.182 |
| Neuroticism Wave 2, γ0011 | -- | -- | -- | -- | -- | -- | 0.0125 | 0.0210 | 0.550 | 0.0324 | 0.0218 | 0.138 |
| Depressed Affect Wave 2, γ0012 | -- | -- | -- | -- | -- | -- | −0.0085 | 0.0097 | 0.382 | −0.0055 | 0.0099 | 0.576 |
| Weekend, β01, γ010 | -- | -- | -- | -- | -- | -- | −0.0504 | 0.0149 | 0.001 | −0.0499 | 0.0148 | 0.001 |
| Wakeup time, β02, γ020 | -- | -- | -- | -- | -- | -- | −0.0012 | 0.0074 | 0.867 | −0.0014 | 0.0074 | 0.854 |
| Daily Negative Affect, β03, γ030 | -- | -- | -- | -- | -- | -- | 0.0413 | 0.0286 | 0.149 | 0.0414 | 0.0287 | 0.150 |
| Daily Positive Affect, β04, γ040 | -- | -- | -- | -- | -- | -- | −0.0039 | 0.0132 | 0.770 | −0.0090 | 0.0136 | 0.506 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Cortisol Awakening Response, π1** | | | | | | | | | | | | |
| Average CAR, β10, γ100 | 0.4200 | 0.0099 | <.001 | 0.4199 | 0.0099 | 0.000 | 0.3585 | 0.0293 | <.001 | 0.3584 | 0.0293 | <.001 |
| CA Wave 1, γ101 | 0.0000 | 0.0046 | 0.996 | −0.0001 | 0.0047 | 0.99 | −0.0013 | 0.0048 | 0.78 | −0.0013 | 0.0048 | 0.789 |
| Self-Esteem Wave 2, γ102 | -- | -- | -- | −0.0002 | 0.0014 | 0.910 | -- | -- | -- | 0.0001 | 0.0017 | 0.937 |
| Female, γ103 | -- | -- | -- | -- | -- | -- | 0.0873 | 0.0203 | <.001 | 0.0872 | 0.0203 | <.001 |
| Non-White, γ104 | -- | -- | -- | -- | -- | -- | 0.0836 | 0.0486 | 0.086 | 0.0837 | 0.0486 | 0.086 |
| Age Wave 2, γ105 | -- | -- | -- | -- | -- | -- | 0.0017 | 0.0009 | 0.058 | 0.0017 | 0.0009 | 0.060 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| Some College Wave 2, γ106 | -- | -- | -- | -- | -- | -- | −0.0246 | 0.0212 | 0.246 | −0.0246 | 0.0214 | 0.251 |
| Childhood SES Wave 2, γ107 | -- | -- | -- | -- | -- | -- | 0.0054 | 0.0246 | 0.827 | 0.0054 | 0.0246 | 0.828 |
| Smoking Status, γ108 | -- | -- | -- | -- | -- | -- | 0.0786 | 0.0267 | 0.003 | 0.0786 | 0.0267 | 0.003 |
| Medication, γ109 | -- | -- | -- | -- | -- | -- | 0.0074 | 0.0214 | 0.728 | 0.0074 | 0.0214 | 0.730 |
| Chronic Condition, γ1010 | -- | -- | -- | -- | -- | -- | 0.0264 | 0.0228 | 0.247 | 0.0267 | 0.0229 | 0.245 |
| Neuroticism Wave 2, γ1011 | -- | -- | -- | -- | -- | -- | −0.0142 | 0.0177 | 0.421 | −0.0134 | 0.0195 | 0.493 |
| Depressed Affect Wave 2, γ1012 | -- | -- | -- | -- | -- | -- | 0.0003 | 0.0062 | 0.957 | 0.0004 | 0.0063 | 0.947 |
| Weekend, β11, γ110 | -- | -- | -- | -- | -- | -- | −0.0338 | 0.0169 | 0.046 | −0.0339 | 0.0169 | 0.045 |
| Wakeup time, β12, γ120 | -- | -- | -- | -- | -- | -- | −0.0157 | 0.0071 | 0.027 | −0.0157 | 0.0071 | 0.027 |
| Daily Negative Affect, β13, γ130 | -- | -- | -- | -- | -- | -- | −0.0054 | 0.0374 | 0.885 | −0.0056 | 0.0374 | 0.882 |
| Daily Positive Affect, β14, γ140 | -- | -- | -- | -- | -- | -- | −0.0020 | 0.0144 | 0.890 | −0.0015 | 0.0148 | 0.919 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Time Since Waking, π2** | | | | | | | | | | | | |
| Average Linear Slope, β20, γ200 | −0.1291 | 0.0033 | <.001 | −0.1291 | 0.0033 | 0.000 | −0.1349 | 0.0045 | <.001 | −0.1349 | 0.0045 | <.001 |
| CA Wave 1, γ201 | 0.0011 | 0.0005 | 0.032 | 0.0007 | 0.0005 | 0.157 | 0.0004 | 0.0005 | 0.395 | 0.0003 | 0.0005 | 0.524 |
| Self-Esteem Wave 2, γ202 | -- | -- | -- | −0.0005 | 0.0002 | 0 | -- | -- | -- | −0.0005 | 0.0002 | 0.010 |
| Female, γ203 | -- | -- | -- | -- | -- | -- | 0.0019 | 0.0022 | 0.38 | 0.0017 | 0.0022 | 0.433 |
| Non-White, γ204 | -- | -- | -- | -- | -- | -- | 0.0277 | 0.0049 | <.001 | 0.0274 | 0.0048 | <.001 |
| Age Wave 2, γ205 | -- | -- | -- | -- | -- | -- | 0.0004 | 0.0001 | <.001 | 0.0005 | 0.0001 | <.001 |
| Some College Wave 2, γ206 | -- | -- | -- | -- | -- | -- | −0.0054 | 0.0024 | 0.025 | −0.0048 | 0.0024 | 0.049 |
| Childhood SES Wave 2, γ207 | -- | -- | -- | -- | -- | -- | 0.0014 | 0.0027 | 0.605 | 0.0012 | 0.0027 | 0.670 |
| Smoking Status, γ208 | -- | -- | -- | -- | -- | -- | 0.0171 | 0.0028 | <.001 | 0.0171 | 0.0027 | <.001 |
| Medication, γ209 | -- | -- | -- | -- | -- | -- | 0.0069 | 0.0022 | 0.002 | 0.0070 | 0.0022 | 0.002 |
| Chronic Condition, γ2010 | -- | -- | -- | -- | -- | -- | 0.0032 | 0.0025 | 0.207 | 0.0027 | 0.0025 | 0.287 |
| Neuroticism Wave 2, γ2011 | -- | -- | -- | -- | -- | -- | 0.0006 | 0.0019 | 0.757 | −0.0018 | 0.0021 | 0.399 |
| Depressed Affect Wave 2, γ2012 | -- | -- | -- | -- | -- | -- | 0.0010 | 0.0007 | 0.137 | 0.0007 | 0.0007 | 0.333 |
| Weekend, β21, γ210 | -- | -- | -- | -- | -- | -- | 0.0027 | 0.0017 | 0.112 | 0.0026 | 0.0017 | 0.121 |
| Wakeup time, β22, γ220 | -- | -- | -- | -- | -- | -- | −0.0030 | 0.0008 | <.001 | −0.0030 | 0.0008 | <.001 |
| Daily Negative Affect, β23, γ230 | -- | -- | -- | -- | -- | -- | 0.0034 | 0.0033 | 0.304 | 0.0034 | 0.0033 | 0.304 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| Daily Positive Affect, β24, γ240 | -- | -- | -- | -- | -- | -- | −0.0003 | 0.0014 | 0.809 | 0.0004 | 0.0014 | 0.791 |
| Daily Exercise, β25, γ250 | -- | -- | -- | -- | -- | -- | 0.0007 | 0.0007 | 0.345 | 0.0007 | 0.0007 | 0.323 |
| **Time Since Waking$^2$, π3** | | | | | | | | | | | | |
| Average Curvature, β30, γ300 | 0.0024 | 0.0002 | <.001 | 0.0024 | 0.0002 | <.001 | 0.0023 | 0.0002 | <.001 | 0.0023 | 0.0002 | <.001 |

*Note.* Intercepts indicate average cortisol values at wakeup; average slopes of time since waking indicate change in cortisol per 1-hour change in time; average slopes of time since waking$^2$ indicate change in cortisol per 1-hour change in time$^2$. CAR = Cortisol Awakening Response; CA: Childhood Adversity; SES: Socioeconomic Status. Seventy-one cortisol values were above 3SD. The main results remained significant when analyses were run after winsorizing these observations.

**Table 3**

Summary of results on the links between the different facets of CA and cortisol parameters controlling for covariates in Study 1

| Cortisol Paramenters | Morning Cortisol | CAR | Cortisol Slope |
|---|---|---|---|
| CA-SE | −0.0149 (0.0109) | −0.0030 (0.0090) | 0.0019 (0.0011)† |
| CA-RP | −0.0392 (0.0199)* | 0.0040 (0.0156) | −0.0011 (0.0017) |
| CA-PEA | −0.0346 (0.0230) | −0.0105 (0.0187) | 0.0009 (0.0019) |
| CA-SE (controlling for Self-Esteem) | −0.0138 (0.0109) | −0.0030 (0.0090) | 0.0018 (0.0011)† |
| Indirect effect 95% CI | [−0.003769, 0.000002205] | [−0.001343, 0.00123] | [0.00001236, 0.0003972] |
| CA-RP (controlling for Self-Esteem) | −0.0354 (0.0199)† | 0.0043 (0.0155) | −0.0015 (0.0017) |
| Indirect effect 95% CI | [−0.01014, −0.0001315] | [−0.004049, 0.003537] | [0.0001453, 0.001149] |
| CA-PEA (controlling for Self-Esteem) | −0.0332 (0.0229) | −0.0104 (0.0187) | 0.0008 (0.0019) |
| Indirect effect 95% CI | [−0.006221, 0.0005833] | [−0.002144, 0.001868] | [−0.002716, 0.001604] |

*Note:* CA-SE: Childhood Adversity Stressful Events; CA-RP: Childhood Adversity Relationship with Parents; CA-PEA: Childhood Adversity Physical/Emotional Abuse; CAR: Cortisol Awakening Response. Regression coefficients for each CA domain are reported in the table followed by robust standard errors in brackets. 95% Confidence Intervals (CI) for indirect effects are provided for models that controlled for Self-Esteem.

† $p < .10$,

* $p < .05$,

** $p < .01$.

**Table 4**

Bivariate Correlations Between Study 2 Person-Level Variables

| Descriptive variables | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Female | 1.000 | −0.087 * | 0.004 | 0.023 | −0.038 | −0.037 | −0.054 | −0.037 | −0.110 * | 0.100 * | 0.066 † |
| 2. Age | | 1.000 | −0.025 | −0.073 † | 0.086 * | 0.058 | 0.102 ** | −0.005 | −0.025 | 0.051 | 0.048 |
| 3. Caregiver education (highschool) | | | 1.000 | −0.020 | 0.059 | 0.100 * | 0.113 ** | 0.009 | 0.051 | −0.022 | −0.121 ** |
| 4. Health Status | | | | 1.000 | −0.182 ** | −0.106 ** | −0.204 ** | −0.063 | −0.065 | 0.006 | 0.131 ** |
| 5. CA-SE | | | | | 1.000 | −0.010 | 0.703 ** | −0.044 | 0.212 ** | −0.057 | −0.037 |
| 6. CA-RP | | | | | | 1.000 | 0.703 ** | 0.121 ** | 0.152 ** | −0.247 ** | −0.218 ** |
| 7. CA | | | | | | | 1.000 | 0.055 | 0.259 ** | −0.216 ** | −0.181 ** |
| 8. Neuroticism | | | | | | | | 1.000 | −0.118 ** | −0.162 ** | −0.050 |
| 9. Depression | | | | | | | | | 1.000 | −0.399 ** | −0.139 ** |
| 10. Child Self-Esteem | | | | | | | | | | 1.000 | 0.100 * |
| 11. Caregiver Self-Esteem | | | | | | | | | | | 1.000 |

*Note:* CA-SE: Childhood Adversity Stressful Events; CA-RP: Childhood Adversity Relationship with Parents; CA: Childhood Adversity.

† $p < .10$,

* $p < .05$,

** $p < .01$.

**Table 5**

HLM Models of Diurnal Cortisol Parameters in Study 2

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Morning cortisol, π0** | | | | | | | | | | | | | | | | | | |
| Average Morning Cortisol, β00, γ000 | 0.6624 | 0.0064 | <.001 | 0.6623 | 0.0064 | <.001 | 0.6623 | 0.0063 | <.001 | 0.6934 | 0.0121 | <.001 | 0.6943 | 0.0121 | <.001 | 0.6991 | 0.0129 | 0.000 |
| CA, γ001 | −0.0113 | 0.0043 | 0.009 | −0.0083 | 0.0043 | 0.052 | −0.0069 | 0.0044 | 0.114 | −0.0109 | 0.0045 | 0.017 | −0.0093 | 0.0045 | 0.038 | −0.0079 | 0.0045 | 0.079 |
| Child Self-Esteem, γ002 | -- | -- | -- | 0.0440 | 0.0146 | 0.003 | 0.0423 | 0.0145 | 0.004 | -- | -- | -- | 0.0409 | 0.0158 | 0.010 | 0.0387 | 0.0158 | 0.015 |
| Caregiver Self-Esteem, γ003 | -- | -- | -- | -- | -- | -- | 0.0285 | 0.0164 | 0.082 | -- | -- | -- | -- | -- | -- | 0.0307 | 0.0164 | 0.061 |
| Female, γ004 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0042 | 0.0131 | 0.747 | −0.0058 | 0.0130 | 0.653 | −0.0057 | 0.0130 | 0.661 |
| Age, γ005 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0066 | 0.0037 | 0.076 | 0.0061 | 0.0037 | 0.099 | 0.0052 | 0.0037 | 0.163 |
| Caregiver Education, γ006 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0031 | 0.0132 | 0.817 | 0.0027 | 0.0131 | 0.837 | 0.0080 | 0.0135 | 0.555 |
| Health Status, γ007 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0032 | 0.0087 | 0.711 | −0.0019 | 0.0086 | 0.829 | −0.0035 | 0.0088 | 0.693 |
| Neuroticism, γ008 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0068 | 0.0093 | 0.463 | −0.0019 | 0.0095 | 0.842 | −0.0026 | 0.0096 | 0.789 |
| Depression, γ009 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0020 | 0.0015 | 0.192 | −0.0004 | 0.0017 | 0.826 | −0.0003 | 0.0017 | 0.864 |
| Caregiver Female, γ0010 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0145 | 0.0132 | 0.275 |
| Caregiver Age, γ0011 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0001 | 0.0007 | 0.844 |
| Weekend, β01, γ010 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.1018 | 0.0103 | <.001 | −0.1022 | 0.0103 | <.001 | −0.1020 | 0.0103 | 0.000 |
| Wakeup time, β02, γ020 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0132 | 0.0066 | 0.045 | −0.0127 | 0.0066 | 0.056 | −0.0130 | 0.0066 | 0.051 |
| Daily Negative Affect, β03, γ030 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0199 | 0.0147 | 0.177 | −0.0203 | 0.0147 | 0.166 | −0.0207 | 0.0147 | 0.158 |
| Daily Positive Affect, β04, γ040 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0073 | 0.0142 | 0.610 | −0.0072 | 0.0141 | 0.612 | −0.0101 | 0.0141 | 0.473 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Cortisol Awakening Response, π1** | | | | | | | | | | | | | | | | | | |
| Average CAR, β10, γ100 | 0.0007 | 0.0066 | 0.913 | 0.0008 | 0.0066 | 0.903 | 0.0009 | 0.0066 | 0.892 | 0.0072 | 0.0118 | 0.545 | 0.0066 | 0.0118 | 0.573 | 0.0030 | 0.0135 | 0.825 |
| CA, γ101 | 0.0060 | 0.0046 | 0.192 | 0.0047 | 0.0046 | 0.303 | 0.0033 | 0.0047 | 0.480 | 0.0065 | 0.0048 | 0.178 | 0.0056 | 0.0048 | 0.247 | 0.0048 | 0.0049 | 0.326 |
| Child Self-Esteem, γ102 | -- | -- | -- | −0.0183 | 0.0161 | 0.254 | −0.0164 | 0.0161 | 0.308 | -- | -- | -- | −0.0238 | 0.0170 | 0.162 | −0.0229 | 0.0173 | 0.185 |
| Caregiver Self-Esteem, γ103 | -- | -- | -- | -- | -- | -- | −0.0264 | 0.0162 | 0.104 | -- | -- | -- | -- | -- | -- | −0.0240 | 0.0163 | 0.143 |
| Female, γ104 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0319 | 0.0128 | 0.013 | −0.0310 | 0.0128 | 0.016 | −0.0309 | 0.0129 | 0.017 |
| Age, γ105 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0060 | 0.0037 | 0.106 | 0.0062 | 0.0037 | 0.092 | 0.0068 | 0.0037 | 0.066 |
| Caregiver Education, γ106 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0220 | 0.0133 | 0.098 | 0.0220 | 0.0133 | 0.098 | 0.0195 | 0.0139 | 0.159 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| Health Status, γ107 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0072 | 0.0095 | 0.451 | 0.0063 | 0.0095 | 0.509 | 0.0070 | 0.0096 | 0.463 |
| Neuroticism, γ108 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0049 | 0.0092 | 0.596 | −0.0077 | 0.0093 | 0.405 | −0.0074 | 0.0093 | 0.432 |
| Depression, γ109 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0005 | 0.0015 | 0.746 | −0.0015 | 0.0016 | 0.368 | −0.0016 | 0.0017 | 0.326 |
| Caregiver Female, γ1010 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0095 | 0.0142 | 0.503 |
| Caregiver Age, γ1011 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0004 | 0.0006 | 0.502 |
| Weekend, β11, γ110 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0293 | 0.0132 | 0.026 | −0.0291 | 0.0132 | 0.028 | −0.0292 | 0.0132 | 0.027 |
| Wakeup time, β12, γ120 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0192 | 0.0075 | 0.010 | −0.0195 | 0.0075 | 0.009 | −0.0193 | 0.0075 | 0.010 |
| Daily Negative Affect, β13, γ130 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0006 | 0.0175 | 0.971 | 0.0007 | 0.0175 | 0.968 | 0.0014 | 0.0175 | 0.934 |
| Daily Positive Affect, β14, γ140 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0105 | 0.0127 | 0.409 | −0.0107 | 0.0126 | 0.397 | −0.0080 | 0.0129 | 0.535 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Time Since Waking, π2** | | | | | | | | | | | | | | | | | | |
| Average Linear Slope, β20, γ200 | −0.0386 | 0.0019 | <.001 | −0.0386 | 0.0019 | <.001 | −0.0386 | 0.0019 | <.001 | −0.0385 | 0.0022 | <.001 | −0.0386 | 0.0022 | <.001 | −0.0390 | 0.0023 | 0.000 |
| CA, γ201 | 0.0002 | 0.0004 | 0.551 | 0.0000 | 0.0004 | 0.957 | −0.0001 | 0.0004 | 0.736 | 0.0001 | 0.0004 | 0.714 | 0.0000 | 0.0004 | 0.935 | −0.0001 | 0.0004 | 0.835 |
| Child Self-Esteem, γ202 | -- | -- | -- | −0.0031 | 0.0014 | 0.025 | −0.0029 | 0.0014 | 0.035 | -- | -- | -- | −0.0030 | 0.0015 | 0.038 | −0.0029 | 0.0015 | 0.049 |
| Caregiver Self-Esteem, γ203 | -- | -- | -- | -- | -- | -- | −0.0031 | 0.0014 | 0.028 | -- | -- | -- | -- | -- | -- | −0.0029 | 0.0014 | 0.043 |
| Female, γ204 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0012 | 0.0011 | 0.293 | −0.0011 | 0.0011 | 0.341 | −0.0011 | 0.0011 | 0.349 |
| Age, γ205 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0006 | 0.0003 | 0.088 | −0.0005 | 0.0003 | 0.111 | −0.0004 | 0.0003 | 0.18 |
| Caregiver Education, γ206 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0001 | 0.0011 | 0.954 | 0.0000 | 0.0011 | 0.971 | −0.0004 | 0.0012 | 0.705 |
| Health Status, γ207 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0002 | 0.0007 | 0.791 | −0.0003 | 0.0007 | 0.687 | −0.0002 | 0.0007 | 0.830 |
| Neuroticism, γ208 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0005 | 0.0008 | 0.560 | −0.0009 | 0.0008 | 0.312 | −0.0008 | 0.0008 | 0.345 |
| Depression, γ209 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0001 | 0.0001 | 0.311 | 0.0000 | 0.0001 | 0.905 | 0.0000 | 0.0001 | 0.970 |
| Caregiver Female, γ2010 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0012 | 0.0012 | 0.299 |
| Caregiver Age, γ2011 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0000 | 0.0001 | 0.876 |
| Weekend, β21, γ210 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0076 | 0.0010 | <.001 | 0.0076 | 0.0010 | <.001 | 0.0076 | 0.0010 | 0.000 |
| Wakeup time, β22, γ220 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0007 | 0.0006 | 0.215 | −0.0008 | 0.0006 | 0.196 | −0.0008 | 0.0006 | 0.208 |
| Daily Negative Affect, β23, γ230 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0024 | 0.0013 | 0.06 | 0.0024 | 0.0013 | 0.056 | 0.0025 | 0.0013 | 0.051 |
| Daily Positive Affect, β24, γ240 | -- | -- | -- | -- | -- | -- | -- | -- | -- | −0.0002 | 0.0012 | 0.88 | −0.0002 | 0.0012 | 0.874 | 0.0001 | 0.0012 | 0.955 |
| **Time Since Waking$^2$, π3** | | | | | | | | | | | | | | | | | | |
| Average Curvature, β30, γ300 | 0.001 | 0.000 | <.001 | 0.0010 | 0.0001 | <.001 | 0.001 | 0.000 | <.001 | 0.0008 | 0.0001 | <.001 | 0.0008 | 0.0001 | <.001 | 0.0008 | 0.0001 | <.001 |

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Fixed effect (independent variable)** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** | **Estimate** | **SE** | ***P*** |
| **Smoke, π4** | | | | | | | | | | | | | | | | | | |
| Intercept, β40, γ400 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.1472 | 0.0472 | 0.002 | 0.1471 | 0.0473 | 0.002 | 0.1469 | 0.0477 | 0.002 |
| **Exercise, π5** | | | | | | | | | | | | | | | | | | |
| Intercept, β50, γ500 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 0.0188 | 0.0083 | 0.023 | 0.0192 | 0.0082 | 0.019 | 0.0191 | 0.0082 | 0.020 |

*Note.* CA: Childhood Adversity. Intercepts indicate average cortisol values at wakeup; average slopes of time since waking indicate change in cortisol per 1-hour change in time; average slopes of time since waking$^2$ indicate change in cortisol per 1-hour change in time$^2$. CAR = Cortisol Awakening Response; CA: Childhood Adversity. Thirty-three cortisol values were above 3SD. The main results remained significant when analyses were run after winsorizing these observations.

**Table 6**

Summary of results on the links between the different facets of CA and cortisol parameters controlling for covariates in Study 2

| Cortisol Paramenters | Morning Cortisol | CAR | Cortisol Slope |
|---|---|---|---|
| CA-SE | −0.0044 (0.0026) † | 0.0008 (0.0030) | 0.0003 (0.0002) |
| CA-RP | −0.0043 (0.0027) | 0.0042 (0.0027) | −0.0001 (0.0002) |
| CA-SE (controlling for Self-Esteem) | −0.0046 (0.0025) † | 0.0009 (0.0030) | 0.0003 (0.0002) |
| Indirect effect 95% CI | [−0.0004614, 0.0009475] | [−0.0006664, 0.000305] | [−0.00006944, 0.0000315] |
| CA-RP (controlling for Self-Esteem) | −0.0030 (0.0027) | 0.0035 (0.0028) | −0.0002 (0.0002) |
| Indirect effect 95% CI | [−0.002504, −0.0002603] | [−0.000354, 0.001847] | [0.00001248, 0.0002075] |

*Note:* CA-SE: Childhood Adversity Stressful Events; CA-RP: Childhood Adversity Relationship with Parents; CAR: Cortisol Awakening Response. Regression coefficients for each CA domain are reported in the table followed by robust standard errors in brackets. 95% Confidence Intervals (CI) for indirect effects are provided for models that controlled for Self-Esteem.

† p < .10,

* p < .05,

** p < .01.