The Dual Impact of Early and Concurrent Life Stress on Adult Diurnal Cortisol Patterns:

A Prospective Study

Abstract

Major life stress often produces a flat diurnal cortisol slope, an indicator of potential long-term health problems. Exposure to stress early in childhood or the accumulation of stress across the lifespan may be responsible for this pattern. However, the relative impact of life stress during distinct life stages on diurnal cortisol is unknown. Using a longitudinal sample of adults followed from birth, we examine three stress exposure models on diurnal cortisol: the cumulative stress model (CM), the biological embedding model (BEM), and the sensitization model (SM). The CM targets cumulative life stress, the BEM implicates early childhood stress, and the SM suggests current stress interacts with early life stress to produce flat diurnal cortisol slopes. Analyses indicate that high early stress exposure and high current stress predict flat diurnal cortisol slopes, consistent with the SM. These novel findings advance our understanding of diurnal cortisol patterns and highlight avenues for intervention.

Keywords: Diurnal Cortisol, Life Stress, Cumulative Stress, Allostatic Load, Development
The Dual Impact of Early and Concurrent Life Stress on Adult Diurnal Cortisol Patterns: A Prospective Study

Much research has documented that experiencing more stress during childhood is associated with poorer long-term health outcomes (Miller, Chen, & Parker, 2011). The Hypothalamic-Pituitary-Adrenal (HPA) axis, in particular, is one of the key biological systems that is responsive to stressful life events and is responsible for mobilizing energetic resources to help confront and cope with environmental challenges (McEwen, 1998, 2008). However, major life stress can lead to dysregulated circadian patterns of cortisol secretion, such as a flatter cortisol slope across the day (Adam et al., 2017; Heim, Ehlert, & Hellhammer, 2000; Miller, Chen, & Cole, 2009), which affects the functioning of various bodily systems (e.g., nervous, immune, vascular, metabolic) and makes people vulnerable to mental and physical health problems (Adam & Kumari, 2009; Adam et al., 2017; Kumari et al., 2009; Kumari, Shipley, Stafford, & Kivimaki, 2011).

Few (if any) prospective, longitudinal studies have examined whether the amount of stress experienced at particular periods of development (e.g., early childhood, adolescence) versus over the lifespan is systematically related to HPA axis dysregulation in adults; virtually all existing research has either been cross-sectional or has relied on retrospective reports of prior stress exposure. Moreover, we do not know whether the impact of stress experienced at different time periods is additive or statistically interacts to predict HPA axis dysregulation in adults (Ross, Murphy, Adam, Chen, & Miller, 2014; Smyth et al., 1997; Stone et al., 2001). Addressing these important gaps in our knowledge could bring greater theoretical clarity to stress research, a field where understanding how, when, and why stress contributes to HPA axis dysregulation is a fundamental, unresolved question.

In most individuals, basal cortisol starts high in the morning, reaching a peak 30-45 minutes after awakening, after which it gradually declines throughout the day, with brief
increases around the midday meal (Miller, Chen, & Zhou, 2007; Smyth et al., 1997; Stone et al., 2001). A flattening of this typical pattern is associated with impaired health (Adam et al., 2017; Kumari et al., 2009, 2011). Two forms of flattened slopes have been found in relation to chronic stress: a lower early morning level with less decline over the day (Gunnar & Quevedo, 2007), and an elevated later afternoon and evening level that also results in less decline over the day (Miller et al., 2009, 2007). Deviations from the typical pattern — particularly a flattened cortisol profile in which morning levels of cortisol decline at a slower rate across the day than expected (Adam et al., 2017) — are associated with poorer long-term physical health outcomes.

Individuals who report retrospectively that they had encountered highly stressful conditions during childhood, such as maltreatment or low socioeconomic status, also tend to have flatter, more dysregulated diurnal cortisol slopes across the day (Gunnar & Quevedo, 2007; Lupien, McEwen, Gunnar, & Heim, 2009; Miller et al., 2009, 2007). Retrospective reports of stressful experiences, however, can be problematic because memory is imperfect (Rubin, Rahhal, & Poon, 1998) and current psychological states (Reuben et al., 2016) or unmeasured variables may cause people to have distorted perceptions of their earlier experiences, both of which can increase confounding and measurement error. In contrast, prospective measures, which better capture variance in life stress as it occurs (Farrell, Simpson, Carlson, Englund, & Sung, 2017), can provide more accurate insights into the impact of stressful experiences on HPA functioning at different life stages.

Three plausible models could explain how stress exposure affects diurnal HPA axis functioning in adulthood. The Cumulative Stress Model (CM) suggests that chronic activation of the HPA axis produces dysregulation of stress-mediating systems, such as the HPA axis, and eventual physical wear-and-tear on the body (Karatsoreos & McEwen, 2013; McEwen, 1998, 2008). Despite the adaptive short-term benefits of the HPA response (Karatsoreos & McEwen, 2013), continued activation of this stress system typically produces
cell damage and long-term health problems. Although the CM acknowledges the possible role of sensitive periods when stress might have a stronger impact on long-term health outcomes, it assumes that the total amount of stress experienced across life is the key variable generating HPA axis dysregulation.

The Biological Embedding Model (BEM), in contrast, claims that stress experienced during certain sensitive periods influences HPA axis development in a durable manner across the lifespan (Hertzman, 1999; Lupien et al., 2009; Miller et al., 2011; Power & Hertzman, 1997; Shonkoff, Boyce, & McEwen, 2009). The most important sensitive period is early childhood (e.g., the first few years of life), during which biological systems are developing and are most vulnerable to stress (Lupien et al., 2009). Accordingly, stress experienced during early childhood is believed to calibrate the HPA axis, which then affects how it functions throughout life (Lupien et al., 2009). The early empirical support for the BEM came from animal research (Levine, 2005; Meaney, 2001), and this model is currently being tested in humans with increasing supportive evidence (Koss, Mliner, Donzella, & Gunnar, 2016; McLaughlin et al., 2015; Roisman et al., n.d.).

The Sensitization Model (SM) also claims that early life stress calibrates HPA functioning, but extends the BEM by proposing that early life experiences shape how the HPA axis responds to stressful experiences later in life (Daskalakis, Bagot, Parker, Vinkers, & de Kloet, 2013). According to this view, HPA functioning depends on both early life stress and current stress levels, meaning that early life stress should statistically interact with current stress to predict HPA functioning, including the flattened diurnal pattern.

With regard to intervention, the CM, BEM, and SM all claim that higher stress should result in greater HPA dysregulation, indicating that effective interventions should attempt to reduce or eliminate psychosocial stressors that negatively impact most people. In contrast to the CM, however, the BEM and SM suggest that early intervention should be a critical point of entry for ameliorating the negative effects of stress on HPA dysregulation. Additionally,
unlike the BEM, the CM and SM suggest that interventions that reduce the effect of chronic stress across the lifespan, such as teaching people more adaptive coping strategies, may also improve HPA regulation. The SM, however, suggests that such interventions might be most effective for individuals who have experienced early life stress.

We examined these three models using 37 years of prospective, longitudinal data from a high-risk birth cohort, the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA) (Sroufe, Egeland, Carlson, & Collins, 2005). The MLSRA is well-positioned to test these models by leveraging 19 waves of objective life stress data collected across the lives of its participants. When participants were 37 years old, two days of diurnal cortisol were collected following standard cortisol collection techniques.

**Method**

**Participants**

In 1975-1976, 267 pregnant women were recruited to participate in the MLSRA (Mean age = 20.6 years, age range 12-34 years). At recruitment, all mothers were living below the poverty line, receiving free health care services, and expecting their first-born child. The children of these mothers are the target participants in the MLSRA. At birth, 48% of mothers were teenagers, 65% were single, and 42% had not completed high school. For the current analyses, we focused on all participants for whom we had non-missing salivary cortisol and early life stress data and who were not pregnant at the age 37 year data collection. Thus, the current analyses are based on 90 participants (51 females, 39 males) who met these criteria. This subset of participants did not differ from the original sample in terms of gender, ethnicity, or socioeconomic status.

**Measures**
**Life Stress.** When target participants were 12, 18, 30, 42, 48, 54, 64 months old, in grades 1, 2, 3, and 6, and at ages 16 and 17, each target’s mother completed the Life Events Schedule (LES) interview. When target participants were 23, 26, 28, 32, 34, and 37 years old, they completed the LES themselves. The LES interview asked mothers (and later targets) about life events that might have occurred and caused stress since the last interview (or within the past year). This included potentially stressful events associated with financial troubles (e.g., job changes, lack of money, debt), relationships (e.g. family members/partners drinking heavily, partners moving in or out, separations and break-ups), and physical danger/mortality (e.g. death of a family member, family members being ill, getting into physical fights). Mother and target participant responses to each question were audiotaped, transcribed, and then rated by trained coders for the level of disruption each event caused on a scale from 0 (*no disruption*) to 3 (*severe disruption*).

To index life stress at each assessment period, the sum of all coded responses was calculated. These scores were then grouped into four developmental periods (Farrell et al., 2017): early life stress (1-5 years, 7 assessments, $\alpha = 0.83$), middle childhood stress (grades 1, 2, 3 and 6, 4 assessments, $\alpha = 0.66$), adolescent stress (ages 16 and 17, 2 assessments, $r = 0.46$), and early adult stress (age 23 to age 34, 5 assessments, $\alpha = 0.76$). Current life stress was indexed by LES scores at age 37 years (when the diurnal cortisol assessments were taken). To examine the cumulative stress model, we summed all coded responses across all time-periods (1 to 37 years, 19 assessments, $\alpha = 0.81$). Figure 1 shows life stress trajectories and box plots for each life stress assessment.

**Diurnal Cortisol.** At age 37 years, participants provided five saliva samples across two consecutive days by passively drooling through a straw into labeled vials. Specifically, they were instructed to provide samples upon waking, 30 minutes after waking, one hour after waking, in the afternoon, and just before going to bed. MEMS® track caps were used to confirm when saliva samples were provided and to corroborate self-reported sample times.
Figure 1. The 19 total life stress scores plotted across 37 years. The black line represents a smoothed sample average of life stress trend across all assessments. 12 months through 64 months represent early childhood (seven assessments), grades 1 through 6 represent middle childhood (four assessments), 16 and 17 years represent adolescence (two assessments), 23 to 34 years represent early adulthood (five assessments), and the 37-year assessment represents current life stress. At the 37-year assessment, both life stress and diurnal cortisol was measured.

In cases where discrepancies occurred between self-reports and track cap times, track cap time stamps were used. The vast majority of participants provided samples within the designated windows, but some did not comply with the instructions (see supplemental material). Those who did not comply with instructions were not deleted from the sample. This is because our target outcome was diurnal cortisol slope and not the cortisol awakening response. Thus, because invalid early morning samples can still be used to model cortisol slopes during the day, we retained them to minimize missing data. Critically, the results of our analysis do not change as a function of removing or retaining invalid morning samples.

After providing all ten samples, participants mailed samples back to the University of
Minnesota and were stored in an industrial freezer at -20 C. The samples were then shipped to the University of Trier, Germany for assaying using time-resolved fluorescence immunoassay (dissociation-enhanced lanthanide fluorescent immunoassay [DELFIA]). All samples were assayed in duplicate and averaged.

All cortisol data were log transformed prior to conducting analyses to correct for positive skew. The log transformed measures showed the typical diurnal rhythm across each day. After transforming the data, we winsorized log transformed cortisol values three standard deviations above the mean. Five cortisol values met this threshold and were subsequently winsorized.

**Results**

**Data Analytic Approach**

The primary outcome was target participants’ pattern of diurnal cortisol release each day. We used mixed modeling given the nested structure of the data across days and participants. For all analyses, the slope variable was Time Since Awakening (TSA), which reflects the number of hours since waking associated with each participant’s cortisol samples. We analyzed cortisol release over an 18-hour period, assuming that most individuals sleep at least 6 hours per night under typical circumstances. Most participants provided their final cortisol sample much earlier than this benchmark (mean TSA was 14.6, SD = 2.78), but there were 11 cortisol samples (<2% of all samples) that were provided after 18 hours since awakening. These extreme samples were removed prior to the analyses. All 90 participants, however, were still represented in the final analyses despite removing these observations. Removing or retaining these 11 samples did not change the results (see the supplemental material for the analyses using the full range of TSA).

In order to analyze cortisol slopes, we necessarily had to assess intercept, or cortisol
levels at awakening. While our focus is on the slope, the intercept provides additional information as a flat slope with high intercept has different biological significance than a flatter slope with low intercept. As such, we fitted mixed models with random intercepts and slopes (i.e., TSA) nested with the two consecutive days of each participant’s data. All models tested the fixed effects of both a linear and a quadratic slope; that is, each model tested the effect of TSA and TSA squared on cortisol. We also entered the following covariates into all models: gender (effects coded; male = -1, female =1), ethnicity (white/non-Hispanic = -1, otherwise = 1), a variable reflecting the number of medications currently being taken by each participant that could have affected their cortisol patterns (Granger, Hibel, Fortunato, & Kapelewski, 2009), and whether or not each participant reported currently having the flu and/or cold symptoms (six participants reported having cold-like symptoms, but no fever). In all of the models reported below, there were no effects of these covariates.

Primary Analyses

**Cumulative Stress.** The CM predicts that individuals exposed to more stress across their entire lives (summed across 1-37 years) should have a relatively flatter diurnal cortisol pattern, with morning cortisol levels declining at a slower rate (i.e., a flattened diurnal cortisol slope) compared to those exposed to less total life stress. To test this possibility, we entered the fixed effect of total (accumulated) life stress and the interaction between total life stress and TSA to determine whether total stress moderated the slope of diurnal cortisol across each day. Both the linear and quadratic slope significantly predicted cortisol across the day (see Table 1). However, total life stress did not predict cortisol output across the day as a main effect, nor did it interact with the linear or quadratic slope term.

**Biological Embedding.** The BEM predicts that individuals exposed to more early life stress (during the first five years) should have a flatter diurnal cortisol pattern compared to those exposed to less early life stress. To test the BEM, we entered the main effect of
Table 1

*Linear mixed models for the CM, BEM, and SM.*

<table>
<thead>
<tr>
<th>Term</th>
<th>CM</th>
<th>BEM</th>
<th>SM</th>
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<tbody>
<tr>
<td></td>
<td>β</td>
<td>95% CI</td>
<td>β</td>
</tr>
<tr>
<td>TSA</td>
<td>-1.06***</td>
<td>[-1.21, -0.91]</td>
<td>-1.06***</td>
</tr>
<tr>
<td>TSA²</td>
<td>0.38***</td>
<td>[0.24, 0.52]</td>
<td>0.38***</td>
</tr>
<tr>
<td>Early</td>
<td>-</td>
<td>-</td>
<td>0.01</td>
</tr>
<tr>
<td>Current</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cumulative</td>
<td>-0.07</td>
<td>[-0.2, 0.07]</td>
<td>-</td>
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<tr>
<td>Early × Current</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Early × TSA</td>
<td>-</td>
<td>-</td>
<td>0.04</td>
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<tr>
<td>Current × TSA</td>
<td>-</td>
<td>-</td>
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<tr>
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<td>-</td>
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<tr>
<td>Early × TSA²</td>
<td>-</td>
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<td>-0.02</td>
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<tr>
<td>Current × TSA²</td>
<td>-</td>
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<td>-0.02</td>
</tr>
<tr>
<td>Cumulative × TSA²</td>
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<td>-0.02</td>
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<td>Early × Current × TSA²</td>
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*Note:* All estimates are standardized beta weights with corresponding 95% confidence intervals. TSA = time since awakening, Early = early life stress, Current = current life stress, Cumulative = total accumulated life stress across all life stress assessments. * p < .05, *** p < .01, ** p < .001

Early life stress and the interaction between early life stress and the linear and quadratic slope into the mixed model. The linear and quadratic TSA terms predicted cortisol output across the day (see Table 1). Similar to the CM model, however, there was no main effect of early life stress on cortisol output across the day, and early life stress did not moderate the effect of either the linear or quadratic slopes.
Sensitization. The SM predicts that individuals who experienced higher levels of stress early in life (during the first five years) and are currently experiencing higher stress in adulthood (at age 37) should have a flatter diurnal cortisol pattern. The effect of early life stress, therefore, should be moderated by current life stress, with the two variables interacting to predict a flatter diurnal cortisol pattern. To test this, we entered two three-way interactions. The first interaction included early life stress, current stress, and the linear slope; the second one included early life stress, current stress, and the quadratic slope. All lower-order two-way interactions and main effects were also entered in the model. There were no main effects or two-way interactions (see Table 1). However, both the linear and quadratic three-way interactions were significant, indicating that diurnal cortisol output was dependent on both early life stress and current life stress. As shown in Figure 2, when early life stress was low (-1 SD), diurnal cortisol patterns for individuals exposed to either higher or lower levels of current life stress did not differ. But for individuals exposed to high early life stress (+1 SD), individuals experiencing higher current life stress (+1 SD) had consistently flatter cortisol slopes across the day than those experiencing lower current life stress (-1 SD).

Exploratory Analyses

Life stress across other periods. Thus far, the main focus of our analyses has been to test the CM, BEM, and SM in the context of diurnal cortisol slopes. However, it remains possible that life stress experienced in other developmental periods, such as middle childhood or adolescence, may also be important predictors of diurnal cortisol slopes. For example, recent models propose other sensitive periods beyond early childhood (Del Giudice, Ellis, & Shirtcliff, 2011; Lupien et al., 2009). In addition, the prospective, longitudinal design of this study included 19 measurements of life stress across 37 years of life, which allowed us to test the influence of life stress during other developmental periods. As such, we tested
Figure 2. A visualization of the SM model overlaid against the raw diurnal cortisol data (gray points and lines; note that these data are the same across both panels). The y-axis represents log-transformed diurnal cortisol values; the x-axis is the time since waking in hours. Blue and red lines represent high and low current life stress at age 37 (+/- 1 SD above and below the sample mean). The left panel depicts the effects of high and low current life stress for individuals who experienced low early life stress (~1 SD); the effects of current life stress are similar under low early life stress. The right panel depicts the effects of high and low current life stress when early life stress is high (+1 SD); the effect of high early life stress is associated with a flatter diurnal cortisol slope when current stress is also high.

Three additional exploratory models using the same mixed modeling approach and covariates as our three confirmatory analyses. The first exploratory analysis examined the effect of life stress during middle childhood (from grade 1 to grade 6), the second one examined adolescent stress (from age 16 and 17), and the third one examined early adulthood (from age 23 to 37). As described in the supplemental material, there were no consistent effects of any of these exploratory analyses. In addition, none of these time periods interacted with current stress to predict diurnal cortisol patterns.
Socioeconomic Status.. Socioeconomic status (SES) has been studied widely in relation to stress and physiology, and it has been a key predictor in past research. Importantly, however, SES is most often treated as a proxy for stress because studies often lack direct measures of stress and, in the absence of direct measures, SES is used as the next best variable to quantify stress exposure. In the current research, we have direct measures of stress (e.g., the life stress interview data), which is a key construct in all of the models we test. Therefore, we did not control for SES in our primary analyses.

Nonetheless, it is important to understand how the potential effects of early and current SES compare with life stress. As such, we ran a set of exploratory analyses using both early SES and current SES. In the first five years of life, there were two assessments of socioeconomic status: one at birth and at age 42 months. At both time-points, the Duncan Socioeconomic Index (Duncan, 1961) was used to measure occupational prestige and income. The two Duncan scores were averaged to create an early life SES score. For current SES at age 37, target participants were interviewed regarding their yearly income.

To explore the effects of early and current SES, we first re-ran our main analyses controlling for the main effects of both early and current SES in the context of the sensitization model (i.e., early life stress by current life stress). This analysis did not reveal any main effects of SES. However, adding SES (early and current) as main effect covariates in a mixed model controls for the effect of SES only on cortisol intercepts. To control for potential slope effects of SES, we next ran three separate models using the same covariates as our focal analyses. The first tested for intercept and slope effects of early SES in a separate mixed model. The second tested for intercept and slope effects of current SES. The final analysis examined the interactive effect of early SES by current SES on cortisol slopes. This final analysis parallels the sensitization model using life stress by testing whether early SES influences cortisol slopes when current SES is low versus high.

Importantly, none of these analyses revealed SES effects. Specifically, there were no
effects of either early or current SES on cortisol intercepts or slopes. Furthermore, there were no interactive effects of early and current SES on cortisol intercepts or slopes (see the supplemental material for more details).

Discussion

Prolonged stress exposure affects HPA functioning negatively, but the relative impact of stress experienced at different life stages has not been definitively established in humans. We compared three theoretically-relevant models that describe how exposure to stress at distinct life stages could be related to HPA dysregulation in adulthood: the CM (Karatsoreos & McEwen, 2013; McEwen, 1998, 2008), the BEM (Hertzman, 1999; Lupien et al., 2009; Miller et al., 2011; Power & Hertzman, 1997; Shonkoff et al., 2009), and the SM (Daskalakis et al., 2013). The CM posits cumulative life stress as the key factor predicting HPA dysregulation. The BEM suggests early life stress is the critical factor because the HPA axis is still developing. The SM suggests that the influence of early life stress on adult HPA functioning should be most evident when current life stress is also high.

We did not find support for the CM or the BEM in the current study. That is, neither cumulative stress nor stress experienced during the first five years of life alone predicted flatter diurnal cortisol patterns at age 37. We did, however, find support for the SM. Specifically, individuals exposed to greater early life stress had relatively flatter diurnal cortisol patterns, but only when they were also experiencing higher current stress (at age 37). When current life conditions were not stressful, the diurnal cortisol patterns of individuals exposed to greater early life stress did not differ from those exposed to less early life stress. These findings suggest that early life stress may serve a “sensitizing” role by calibrating later responses to stressful conditions in adulthood. Cast another way, early childhood may be a sensitive period during which important biological systems are particularly responsive to external influences, such as life stress. Such calibrations, however, may influence how the
stress response system reacts to future stressful experiences, but remain latent until the system is challenged by concurrent life stress. Importantly, our exploratory analyses did not reveal any effects of other developmental periods during which life stress might impact HPA functioning.

These findings will need to be replicated in other samples. Studies of early life stress using samples of children and adults who were reared in orphanage-like institutions in infancy have reported significant impacts on the HPA axis, with regard to the cortisol awakening response, diurnal rhythm, and cortisol stress response to psychosocial challenges (Koss et al., 2016; Kumsta et al., 2017; McLaughlin et al., 2015). However, so far, none of this work has examined whether individuals institutionalized in infancy show dysregulated HPA functioning when experiencing high current stress in adulthood.

There are clear parallels between the SM and diathesis-stress models in the literature. In both models, particular psychological or biological factors are conceptualized as “risk factors.” These risk factors make certain individuals more likely to develop certain pathologies or health problems, especially in combination with environmental stressors such as exposure to major trauma or very stressful events. The SM can be framed as a special case of a diathesis-stress model in that early life stress is construed as a risk factor and current life stress as an environmental stressor. In other words, stress early in life puts individuals at risk of dysregulated HPA functioning, which is then manifested when stressful experiences occur later in life. It is important to note, however, that the SM is a developmentally informed model for two reasons. First, the SM targets exposure to early life stress during a specific developmental period that should be especially sensitive to “programming” effects by major external stressors. Second, it proposes that exposure to stressful early life experiences should shape how the HPA system functions during exposure to stressful events later in life.

The current study has some limitations. First, we measured diurnal cortisol and life stress concurrently at only one time-point (age 37), limiting our ability to make inferences
about HPA functioning at other time-points. Nonetheless, the MLSRA measured life stress at 19 prior time-points, giving us a unique opportunity to compare the CM, BEM, and SM using the same measures and same sample of participants. Second, the current sample is modest in size. Third, our findings may generalize to only initially at-risk samples who have similar demographic characteristics. In addition, though we have adjusted for obvious confounds, other unmeasured factors might have contributed to these associations (e.g., environmental pollutants, allelic vacation, perinatal complications). Finally, diurnal cortisol slope is only one measure of HPA functioning and, although cortisol slopes have been linked to health disparities, other aspects of HPA functioning, such as cortisol reactivity, and other biological systems, such as the immune, metabolic, and sympathetic nervous system, remain critically important in the study of stress and its impact on health. Despite these limitations, the MLSRA’s prospective, longitudinal design spanning over 37 years along with its in-depth interview measures of life stress is very rare, if not completely unique, in the study of developmental processes and HPA functioning.

In summary, the current research examined three theoretically-derived models of exposure to life stress and HPA dysregulation in adults. By comparing the impact of life stress at 19 time-points across 37 years, we were able to assess the relative influence of stress exposure on diurnal cortisol slopes at age 37. Consistent with the SM, our findings reveal that the interaction between exposure to more life stress during the first 5 years of life and higher current life stress in adulthood is most clearly associated with the prototypical flat diurnal cortisol slope known to predict many negative health outcomes. These findings are important because they suggest that targeted interventions should be developed to ameliorate the negative effect of early life stress or, when not feasible, to at least reduce the negative effect of current stress in adulthood, especially for individuals who were exposed to higher levels of early life stress. Theoretically, our findings suggest that measuring and modeling stress both early in life and concurrently may be essential to fully understand how biological stress response systems become dysregulated.
References


