Development and Psychopathology (2024), **36**, 1323–1355
doi:10.1017/S0954579423000561

CAMBRIDGE UNIVERSITY PRESS

Regular Article

Meta-analysis of associations between childhood adversity and diurnal cortisol regulation

Laura Perrone[1], Daneele Thorpe[1], Grace Shariat Panahi[1], Yukihiro Kitagawa[1], Oliver Lindhiem[2] and Kristin Bernard[1]

[1]Department of Psychology, Stony Brook, NY, USA and [2]Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, USA

Abstract

Childhood adversity has been associated with hypothalamic–pituitary–adrenal axis dysregulation, which is associated with mental and physical health consequences. However, associations between childhood adversity and cortisol regulation in the current literature vary in magnitude and direction. This multilevel meta-analysis examines the association between childhood adversity and diurnal cortisol measures, as well as potential moderators of these effects (adversity timing and type, study or sample characteristics). A search was conducted in online databases PsycINFO and PubMed for papers written in English. After screening for exclusion criteria (papers examining animals, pregnant women, people receiving hormonal treatment, people with endocrine disorders, cortisol before age 2 months, or cortisol after an intervention), 303 papers were identified for inclusion. In total, 441 effect sizes were extracted from 156 manuscripts representing 104 studies. A significant overall effect was found between childhood adversity and bedtime cortisol, $r = 0.047$, 95% CI [0.005, 0.089], $t = 2.231$, $p = 0.028$. All other overall and moderation effects were not significant. The lack of overall effects may reflect the importance of the timing and nature of childhood adversity to adversity's impact on cortisol regulation. Thus, we offer concrete recommendations for testing theoretical models linking early adversity and stress physiology.

Keywords: Childhood adversity; Cortisol; Hypothalamic–pituitary–adrenal axis; Meta-analysis

(Received 12 February 2022; revised 15 May 2023; accepted 15 May 2023; first published online 9 June 2023)

Introduction

Childhood adversity is associated with a wide range of negative mental and physical health outcomes (e.g., Felitti et al., 1998). Thus, understanding the mechanisms through which childhood adversity disrupts well-being is of critical importance. One potential mechanism is the impact of childhood adversity on the hypothalamic–pituitary–adrenal (HPA) axis, a major component of the neuroendocrine system. The end product of the HPA axis, the glucocorticoid hormone cortisol, not only contributes to the body's immediate response to stressors but also to the body's overall diurnal regulation. As a result, alterations to the HPA axis may impact the ability to regulate key bodily systems, making it important to understand how childhood adversity contributes to diurnal cortisol regulation. However, to date, literature in this field has provided mixed results.

The hypothalamic–pituitary–adrenal axis and cortisol

Cortisol is produced within the body when the hypothalamus releases corticotropin-releasing factor and arginine vasopressin. Corticotropin-releasing factor and arginine vasopressin then prompt the pituitary gland to release ACTH, which binds to the adrenal cortex and triggers the release of cortisol (Lupien et al., 2009). Cortisol may then bond with either mineralocorticoid receptors, which are involved in the maintenance of the HPA axis's circadian rhythm and regulation of key bodily functions, or glucocorticoid receptors, which are activated during stress responses and often work in opposition to the effects of mineralocorticoid receptors (Gunnar & Quevedo, 2007). The HPA axis's circadian rhythm typically produces a diurnal cortisol pattern that involves a sharp peak in the morning approximately 30 minutes after awakening, known as the cortisol awakening response (CAR), followed by a decline in cortisol throughout the day (Fries et al., 2009; Van Cauter, 1990). The CAR constitutes a unique effect of the HPA axis that includes aspects of both reactivity and diurnal regulation (Stadler et al., 2022). When faced with a stressor, the body may mount an additional cortisol stress response that appears to enhance cardiovascular activation, mediate metabolic responses, and suppress immune responses, memory formation, and reproduction (Sapolsky et al., 2000).

Although activation of the HPA axis in response to adversity may have immediate benefits in the face of a stressor, repeated activation of the HPA axis over time can lead to alterations in its overall functioning. When initially faced with stressors, individuals often experience increases in cortisol, or hypercortisolism; however, over time, responsiveness to stressors may decrease, resulting in hypocortisolism (Loman & Gunnar, 2010). Furthermore, differences in the nature of the stressor (e.g., acute vs. chronic, physically vs. socially threatening) may also contribute

Corresponding author: Laura Perrone; Email: laura.perrone@stonybrook.edu

Cite this article: Perrone, L., Thorpe, D., Shariat Panahi, G., Kitagawa, Y., Lindhiem, O., & Bernard, K. (2024). Meta-analysis of associations between childhood adversity and diurnal cortisol regulation. *Development and Psychopathology* **36**: 1323–1355, https://doi.org/10.1017/S0954579423000561

to variability in patterns of cortisol dysregulation. For example, one meta-analysis found that although chronic stress broadly was associated with lower morning cortisol levels, higher afternoon/evening cortisol levels and a more blunted diurnal slope, stressors that involved social threat or that were potentially controllable were associated with higher morning cortisol levels (Miller et al., 2007). Given that disruptions in the HPA axis are associated with various mental health and physical health outcomes (e.g., Felitti et al., 1998), HPA axis functioning may provide an important link between adversity and well-being (e.g., Doom & Gunnar, 2013).

Childhood adversity and diurnal cortisol

Adversity in childhood may be particularly impactful to HPA axis functioning. Childhood adversity has been defined as "negative environmental experiences that are likely to require significant adaptation by an average child and that represent a deviation from the expectable environment" (McLaughlin et al., 2019). Although there are a wide range of possible experiences that can be considered childhood adversities, many involve threat to one's safety and well-being (e.g., abuse, exposure to violence) and/or deprivation of expected resources and stimulation (e.g., neglect, poverty, institutionalization; McLaughlin & Sheridan, 2016). Because children are dependent on external sources for survival and regulation, children may be unable to manage adversity on their own and are reliant on a caregiver to buffer against adversity's potential impact (e.g., Gunnar & Donzella, 2002). As a result, adversity that children face without support from a caregiver and adversity that disrupts sensitive caregiving (e.g., maltreatment, parental psychopathology) may have an especially strong impact on child development and later outcomes. Early social deprivation, in particular, has been identified as a key contributor to early HPA axis development (e.g., Koss et al., 2014). It has been proposed that early adversity involving caregiving stress may accelerate emotional development including self-regulation (Callaghan & Tottenham, 2016).

Thus, a key aspect of childhood adversity is that it has the potential to alter normative developmental processes, which in turn can contribute to negative outcomes later in life, including psychopathology (McLaughlin, 2016). Children facing adversity are often exposed to high levels of stress, which can have a direct impact on the functioning of their HPA axis. Multiple theories aim to explain physiological changes that provide adaptation to adversity, such as the general adaptation syndrome model (Selye, 1946), the biological sensitivity to context model (Boyce & Ellis, 2005), the biological embedding model (Hertzman, 1999; Miller et al., 2011), the three-hit hypothesis (Daskalakis et al., 2013) and the adaptive calibration model (Del Giudice et al., 2011). These models are often based at least in part in the concept of adaptive calibration, which proposes that individuals respond to current stressors with biological adaptations that assist them in achieving allostasis amid adversity (Del Guidice et al., 2011). These biological adaptations may differ (e.g., hyper- vs. hypocortisolism) based on the nature of the adversity faced. Importantly, both hyper- and hypocortisolism result in deviations from the typical diurnal cortisol pattern described above, which in turn have been associated with a wide range of physical and mental health problems (Adam et al., 2017; Shirtcliff & Essex, 2008). As a result, understanding early contributors to diurnal cortisol dysregulation, defined as either hyper- or hypocortisolism, may provide critical avenues of intervention to prevent significant downstream consequences.

Consistent with these theories, many studies have provided support for a link between childhood adversity and dysregulation of diurnal cortisol patterns. For example, among preschool-age children, higher cumulative risk comprised of indicators including adolescent parent status, single parent status and low education has been associated with lower morning cortisol levels and more blunted diurnal slopes (Zalewski et al., 2016). In addition, moderate amounts of cumulative adversity comprised of socioeconomic disadvantage, negative life events and traumatic events have been associated with a higher CAR and less steep diurnal slope among children (Gustafsson et al., 2010). However, results are often inconsistent across studies.

Previous studies examining adversity have included numerous indicators of adversity, such as socioeconomic status, parental mental health, parental marital status, parental criminal conviction and parental education (e.g., Atkinson et al., 2015; Evans, 2003). Many of these indicators are not direct measures of threat or deprivation but rather serve as proxies for adversities such as socioeconomic hardship or a lack of sensitive caregiving. For example, parental mental illness or having a single parent may not be an adversity if the parent is able to care for their child consistently and sensitively but rather becomes an adversity when it interferes with sensitive caregiving or results in a lack of necessary resources in the family. In addition, the impact of adversities may vary based on the extent to which the child is protected by other factors, such as sensitive caregiving. Given that a child's experience of whether an event is an adversity may vary based on the child's broader context, experiences and level of support from caregivers, measuring adversity directly can prove challenging. As a result, studies examining multiple adversities have often relied on indirect proxies for threat and deprivation (e.g., parental mental health, single parent status) as well as more direct measures of threat and deprivation (e.g., community-level stressors, discrimination, financial strain, maltreatment, difficulties in parenting and parent–child relationships, parental substance use, surrogate care), with the overarching goal of capturing experiences that frequently convey deviations from the expectable environment producing significant stress for the child (either directly or indirectly) and therefore requiring physiological adaptation within the HPA axis.

Characteristics of adversity

Studies examining childhood adversity and diurnal cortisol regulation are numerous and cover a wide range of childhood adversity. However, some studies have begun to identify aspects of childhood adversity that may be particularly important in adversity's association with HPA axis functioning and diurnal cortisol patterns. For example, McLaughlin and Sheridan's (2016) dimensional model of childhood adversity suggests that categorizing adversities along dimensions of deprivation and threat may add additional insight beyond cumulative adversity models that simply tally the number of adversities. Although not yet examined with diurnal cortisol, this framework has revealed that childhood violence exposure, but not social deprivation, was associated with a blunted cortisol response to stress in urban adolescents, providing evidence that threat and deprivation may have different impacts on the HPA axis (Peckins et al., 2020). Thus, examining specific characteristics of adversity is critical to understanding the impact of childhood adversity on diurnal cortisol regulation.

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Timing of adversity

Many studies provide evidence that the timing of adversity exposure may be critical in associations between childhood adversity and diurnal cortisol. For example, age at which adversity occurs, time since adversity onset and length of adversity have all been associated with diurnal cortisol pattern differences (e.g., Doom et al., 2014; Flannery et al., 2017; Isenhour et al., 2020; Leneman et al., 2018; Lupien et al., 2001; Quevedo et al., 2012). Time since onset and duration of adversity may play important roles in shifts from hypercortisolism to hypocortisolism. Sensitive periods and critical windows may also be important to understanding the impact of childhood adversity on diurnal cortisol regulation.

Previous research indicates the possibility of multiple such sensitive periods. Both animal and human models provide evidence of a potential hyporesponsive period early in life in which cortisol responses to stressors are lessened with parental care playing a critical role (Gunnar & Donzella, 2002), suggesting that the impact of adversity in the first few years of life may depend at least in part on the availability of sensitive caregiving for external stress regulation. In addition, exposure to adversity between ages 3 and 7 has been identified as an important period for CAR dysregulation in adulthood, which may be related to early amygdala development during this period (Raymond et al., 2021). Puberty may also be an important developmental period given evidence for recalibration of cortisol reactivity during puberty (Gunnar et al., 2019), which suggests continued development of the HPA axis in adolescence.

Although much remains to be explored, these studies provide initial evidence that the timing and chronicity of childhood adversity may play a critical role in the impact of adversities on diurnal cortisol regulation. This may in part result from the critical role that caregivers play in helping their children to regulate stress, especially early in life, as well as the continued development and plasticity of biological structures and pathways related to stress regulation throughout childhood and adolescence.

Previous meta-analytic and systematic review evidence

Given the inconsistent literature, it is critical to examine previous meta-analytic findings to understand the current state of the field before planning future studies further examining associations between childhood adversity and diurnal cortisol regulation. Fogelman and Canli (2018) found no significant overall associations between early life stress and the CAR. However, their results indicated significant heterogeneity among effects such that sexually, emotionally, or physically abusive forms of early life stress were associated with a heightened CAR. Similarly, Bernard et al. (2017) found no significant overall associations between maltreatment and wake levels, the CAR, or diurnal slope; however, associations between maltreatment and lower wake levels were significant specifically for agency-referred samples (e.g., following child welfare involvement, rather than via self-report). Additionally, Hackman et al. (2018) found no overall association between parenting and morning cortisol but significant heterogeneity among effects. Specifically, there were significant associations between warm/sensitive parenting and higher levels of morning cortisol within intervention (as opposed to observational) studies and among samples that experienced maltreatment. Furthermore, associations became more positive as the interval between the measurement of parenting and cortisol increased. Taken together, these meta-analyses indicate potential associations between childhood adversity and diurnal cortisol patterns, although the overall associations remain unclear.

Additional insight can be gleaned from meta-analyses and systematic reviews examining childhood adversity and other aspects of cortisol. For example, hair cortisol serves as a proxy for cumulative levels of HPA axis activity indicating chronic stress over previous months (Gow et al., 2010). Previous meta-analyses and systematic reviews have provided mixed results for the association between adversity and hair cortisol levels, including significant associations only for adversity that occurs in adulthood (Khoury et al., 2019), limited associations for social adversity in childhood (Bryson et al., 2021) and associations with childhood adversity that vary with the age at which hair levels are measured (Grant & Meyer, 2021). An additional cortisol measure, reactivity to stressors, reflects the HPA axis's short-term response to more immediate stressors rather than the daily regulation captured in diurnal cortisol measures. Meta-analyses and systematic reviews examining childhood adversity and cortisol reactivity have also indicated mixed results, with some indicating that childhood adversity is associated with blunted cortisol reactivity (e.g., Brindle et al., 2022; Bunea et al., 2017; Hakamata et al., 2022), others indicating mixed directions of effects (e.g., Hosseini-Kamkar et al., 2021; Hunter et al., 2011) and one indicating a possible lack of significant associations (Lai et al., 2021). Although examination of both hair cortisol concentration and cortisol reactivity were beyond the scope of the present meta-analysis, findings in previous studies present a consistent picture of complex potential associations between childhood adversity and HPA axis functioning.

The present study

Given the substantial literature examining childhood adversity and diurnal cortisol, quantitative meta-analytic methods provide an opportunity to examine overall associations between childhood adversity, broadly defined, and indicators of diurnal cortisol regulation. To our knowledge, no meta-analysis has yet examined associations between childhood adversity broadly and all aspects of the diurnal cortisol pattern included herein. The present study sought to address this gap by examining associations between childhood adversity and concurrent or subsequent diurnal cortisol measures (i.e., wake levels, the CAR, diurnal cortisol change and bedtime levels) in nonintervention studies. Categories of adversity within this meta-analysis include community-level stressors, cumulative adversity, difficulties in parenting and parent–child relationships, discrimination, financial strain, maltreatment, parental status, parental mental health, parental substance use, surrogate care and other family/parenting stress. (See Supplemental Materials for additional details.) The primary aim of the present study was to estimate the magnitude of these associations. As exposure to adversity has been associated with both hypercortisolism and hypocortisolism and both are considered forms of dysregulation, we did not specify directional hypotheses. A secondary aim was to explore potential moderators of these associations, including type of adversity, timing of adversity, age at which diurnal cortisol regulation was assessed, study-level sociodemographic indicators, methodological approaches and publication year.

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Method

Procedure

This meta-analysis was reported in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines (Moher et al., 2010).

Search strategy

A systematic search of peer-reviewed journal articles and dissertations was conducted in PsycINFO and PubMed through February 2020. The search term "cortisol" was combined with terms related to a range of adverse childhood experiences (see Supplemental Materials for Boolean phrases).

Eligibility criteria

Papers were included if they were written in English and met the following criteria: (a) included at least one diurnal measure of salivary cortisol, (b) included at least one measure of childhood adversity experienced before the age of 18 years and (c) included human participants. Additionally, papers were excluded if: (a) cortisol was measured exclusively after an intervention; (b) cortisol was measured in the first two months after birth; (c) the sample was composed of pregnant women, people receiving hormonal treatment, or people with endocrine disorders; or (d) the paper was a meta-analysis, literature review, case study, or editorial.

Study selection

A total of 23,536 papers were initially screened for eligibility based on titles and abstracts by the first author. The first and second authors conducted a full-text eligibility assessment on all manuscripts initially screened for inclusion, yielding an inter-rater reliability Kappa of 0.63. All discrepancies were discussed to reach consensus for inclusion or exclusion, with the final author being consulted for any discrepancies that were difficult to resolve. Full-text eligibility assessment yielded 303 papers representing 209 unique studies for inclusion (see Figure 1 for PRISMA diagram).

Of these papers, 156 papers from 104 studies were included in the quantitative synthesis based on the availability of relevant effect sizes either reported in the paper or provided by contacted authors (see additional details below). Given the magnitude of the present meta-analysis, we included several overlapping studies from previous meta-analyses investigating the association between early life stress and cortisol regulation. However, the studies represented in this meta-analysis differed from those included in previous similar meta-analyses. Of the studies included in the present meta-analysis, 11.43% were included in the meta-analysis by Bernard et al. (2017), 4.76% were included in the meta-analysis by Fogelman and Canli (2018) and 4.76% were included in the meta-analysis by Hackman et al. (2018).

Decision rules and coding

The first and second authors coded all eligible studies for effect sizes and moderators, with the exception of the quality and threat/deprivation moderators, which were double-coded by the first, third and fourth authors. Discrepancies were discussed and resolved through conference for use in analyses. When the same sample was reported across multiple papers, authors first prioritized effect sizes from the largest sample and then effect sizes with the greatest time elapsed between assessment of childhood adversity and assessment of cortisol to prioritize longitudinal effects. Only samples that did not include individuals from the same family (e.g., siblings) were included to ensure independence of data. If siblings were included in a sample, authors were contacted to see if an effect size for a sample with one randomly selected individual in each family was available. One effect size was coded for each combination of childhood adversity and diurnal cortisol measure to avoid issues related to non-independence of multiple effect sizes from the same study. However, following completion of coding, we modified our analysis plan to conduct multi-level meta-analyses following the guidelines of Assink and Wibbelink (2016).

Childhood adversity. In order to capture a wide range of potential childhood adversities, childhood adversity was broadly defined as adverse experiences external to the child (i.e., not characteristics of the child such as psychopathology or illness). Studies examining cumulative adversity (e.g., Atkinson et al., 2015; Evans, 2003) were used as a basis for generating the categories included. Categories included cumulative adversity, community-level stressors (e.g., crime, low neighborhood socioeconomic status), discrimination, financial strain, maltreatment, difficulties in parenting and parent–child relationships, parental mental health, parental substance use, single parent status, surrogate care, other family stress and other (any adversity external to the child not encompassed in previous categories). See the Supplemental Materials for definitions from the coding guidelines used to identify variables included in each category. When multiple childhood adversity measures were available in the same category, decision rules prioritized, in order, measures that: (1) spanned the longest window of time in childhood, (2) were continuous, (3) were observed (rather than self-report), (4) included a greater proportion of individuals exposed to the adversity and (5) related to the primary caregiver (or mother when primary caregiver not noted). In addition, decision rules specific to each category were considered (see Supplemental Materials). When these rules did not yield a decision, the measure was selected randomly. When the same adversity measure was assessed at multiple time points, effect sizes were averaged when possible.

Diurnal cortisol. Although there are a wide range of measures for HPA axis cortisol production (e.g., reactivity in response to stressors, area under the curve, diurnal pattern), the present meta-analysis focused on measures that reflect the diurnal pattern of cortisol: wake levels (within 30 minutes of awakening), bedtime levels (within 30 minutes of bedtime), CAR and diurnal cortisol change. Of note, these measures are not independent since wake levels contribute to the CAR, and both wake and bedtime levels contribute to the diurnal cortisol change. A diurnal cortisol change effect size was included if the study included at least two samples that either were related to the participant's wake-up time and bedtime or that spanned a range from morning to afternoon/evening on the same day. For studies in which samples were taken at specific times of the day rather than in relation to participants' sleep patterns, diurnal cortisol change was included if at least one sample was planned to be collected at or before 10:00 AM and at least one same-day sample was planned to be collected at or after 3:00 PM. This approach was selected to include a wide range of the effect sizes available in the current literature while ensuring that effect sizes captured variability across the day. If cortisol was measured at multiple waves, the decision rules were to prioritize, in order: (1) the wave that included the greater number of days of cortisol samples and (2) the most recent wave to prioritize longitudinal effects. Only continuous cortisol measures were included. For the CAR and diurnal cortisol change, only measures representing the difference between earlier and later samples (e.g., simple/residualized difference scores, HLM slopes, regression

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

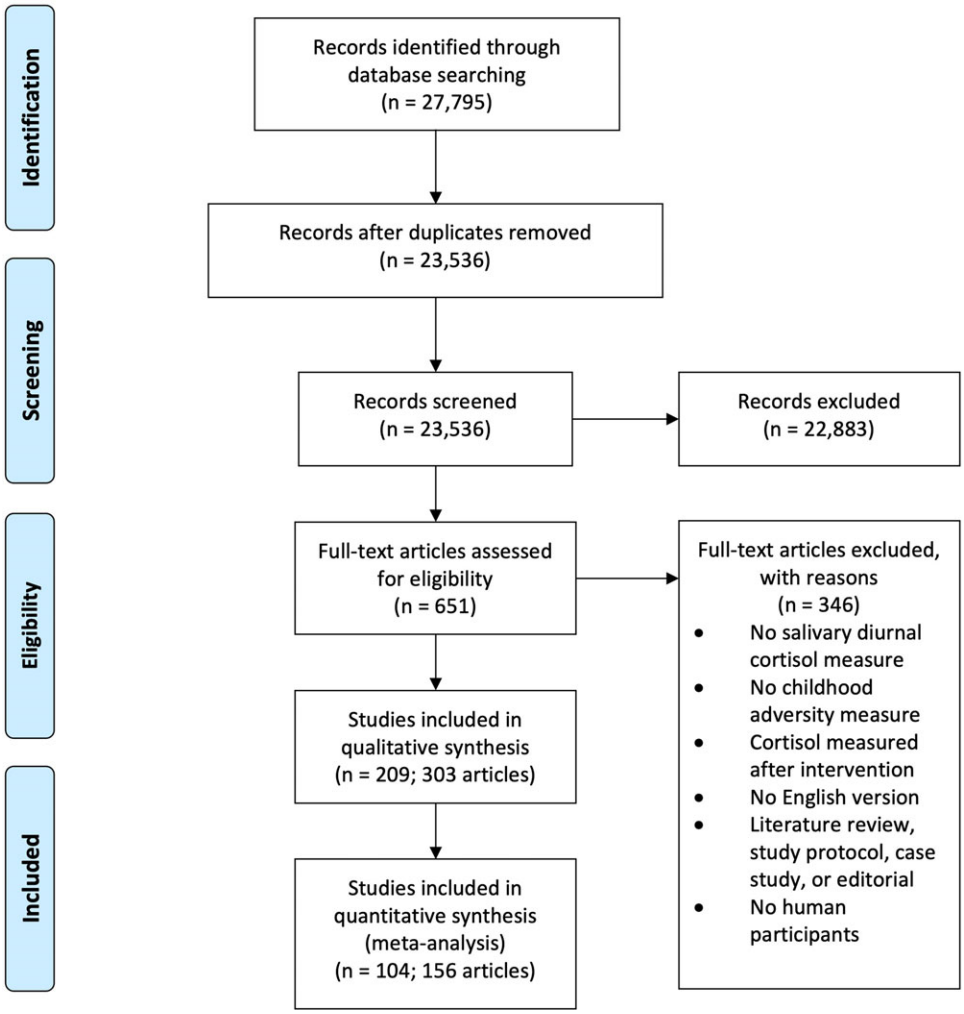

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) Flowchart. Note. The broad and inclusive search terms likely led to an inflated number of initial records for screening, resulting in the large number of records excluded at the screening level.

lines, mean percentage increase) and not measures reflecting total cortisol production (area under the curve) were included.

Moderators. Several within-study moderators were coded to explore theoretical questions about the potential associations between childhood adversity and diurnal cortisol patterns. Given that different types of childhood adversity may have different associations with diurnal cortisol patterns, category of childhood adversity was included as a within-study moderator. Each effect size was categorized as one of the childhood adversity categories described above, and this categorical moderator was included in moderation analyses. Category was selected as opposed to specific characteristics of adversity (e.g., controllability, threat to physical vs. social self) because information on categories of childhood adversity was more consistently available in manuscripts and therefore consistently codable.

At the same time, differences in the nature of childhood adversity may impact associated outcomes. One example is McLaughlin and Sheridan's (2016) threat and deprivation model of adversity. To examine threat vs. deprivation (which was added after an initial round of reviewer feedback), three coders independently reviewed all included studies to identify any coded effect sizes that reflected threat (i.e., physical abuse, sexual abuse, emotional abuse, witnessing domestic violence, exposure to violence outside the home, or bullying victimization) or deprivation (i.e., physical neglect, emotional neglect, food insecurity, low cognitive stimulation, institutional care, foster care, or poverty). Measures coded as threat or deprivation were based on guidelines from previous meta-analyses examining these dimensions (e.g., Johnson et al., 2021). Threat versus deprivation was then examined as a post hoc exploratory moderator within the subset of studies that included a measure of threat or deprivation.

Furthermore, given that timing may influence adversity's impact on diurnal cortisol regulation, age at which adversity occurred was included as a within-study moderator. Age at adversity was coded for each effect size as one of the following categories: infancy (birth through age 2), early childhood (ages 3 through 8), middle childhood (ages 9 through 12), adolescence (ages 13 through 17), or multiple time periods (if the adversity spanned multiple of these age groups). In addition, mean age of participants at the time at which diurnal cortisol was assessed was coded as a between-study continuous moderator variable to capture the timing of diurnal cortisol regulation. More specific timing aspects (e.g., duration, chronicity and time since onset of adversity) were not coded as this information was not available consistently across studies.

Several additional study-level moderators were coded. Because diurnal cortisol patterns have been found to vary with sex (e.g., Larsson et al., 2009; Netherton et al., 2004), percent of the sample

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

identified as female was coded as a continuous moderator variable. Additionally, race/ethnicity has been associated with differences in diurnal cortisol patterns when examining potential adversity or stressors (e.g., DeSantis, Adam et al., 2015; Zeiders et al., 2014). Thus, the percent of the sample identified as a racial/ethnic minority was coded as a continuous moderator variable. Racial/ethnic minority status was defined as any race/ethnicity other than non-Hispanic/Latin White for samples in the United States or based on the authors' definition for samples outside the United States. Furthermore, several design characteristics likely to increase the methodological rigor of diurnal cortisol collection were coded as categorical moderators to reduce potential noise from methodological variability: number of days of cortisol collection (one or multiple), whether cortisol was transformed, time of diurnal cortisol change (whether included samples at both wake and evening/bedtime defined as after 5:00 PM as opposed to sample(s) taken in the morning and/or in the afternoon), whether participants were instructed to take wake samples at awakening (as opposed to within a certain number of minutes after awakening), and a variable reflecting study quality. The study quality variable was defined as in previous meta-analyses examining diurnal cortisol (e.g., Adam et al., 2017; Chida & Steptoe, 2009) as a count of how many of the following covariates were accounted for within each study: age, sex, smoking, use of steroid-based medications, wake time, sampling day (weekday or weekend), self-reported adherence with sampling times, objective adherence with sampling times (e.g., electronic monitoring) and clear sampling instructions given to participants. Finally, two variables were coded for exploratory moderator analyses: whether data were received from authors or coded from the paper and publication year (to examine if effect sizes reported in the literature have changed over time).

Kappas or ICCs for these moderators were above 0.90 for all variables except time of diurnal cortisol change, which had a Kappa of 0.69; age at adversity, which had a Kappa of 0.60; and whether data was received from authors, which was not double-coded. Given the nature of our approach to coding threat vs. deprivation (retrospectively identifying effect sizes that met criteria), reliability could not be calculated. Any discrepancies for threat versus deprivation were resolved through consensus. To decrease the likelihood of Type I errors given the number of moderators examined, a Bonferroni correction was applied to the alpha level by dividing by the number of moderators, yielding a significance cutoff of $p < 0.004$ for analyses related to wake levels and diurnal cortisol change and $p < 0.005$ for analyses related to the CAR and bedtime levels.

Contacting authors. Authors were contacted if any relevant effect sizes could not be coded from the paper. Although at least one effect size could be estimated for 49 (23.44%) of studies screened for inclusion, authors were contacted for all 209 studies (representing the 303 papers included) since at least one effect size was not codable from each study. Authors provided additional data for 78 studies (37.32% of those requested), including additional effect sizes for some studies for which at least one effect size was already estimated from the text. Means and standard deviations were requested for categorical adversity variables and correlations for continuous adversity variables. Authors were asked to provide simple difference scores for the CAR and diurnal cortisol change whenever possible as this measure was most likely to be available across studies (compared to more complex measures such as change over time). In addition, missing moderator variables were requested if authors were contacted for effect sizes.

Calculating effect sizes. Effect sizes were calculated as correlation coefficients (r). Means and standard deviations and t-values were converted to correlation coefficients using the Practical Meta-Analysis Effect Size Calculator (Wilson). In addition, based on Peterson and Brown's (2005) findings that using beta coefficients to impute missing correlation coefficients produces relatively accurate effect size estimates, 14 standardized beta coefficients were converted to correlation coefficients using the conversion formula they recommend as producing the best approximation of the relation between correlation and beta coefficients:

$$r = \beta + .05\lambda$$

where λ equals 1 when β is nonnegative and 0 when β is negative. This formula accounts for the tendency of nonnegative beta coefficients to be somewhat smaller than corresponding correlation coefficients. Inter-rater reliability for effect sizes was acceptable as indicated by an ICC of .64. All effect sizes were coded such that positive effects indicate adversity is associated with elevated (higher) wake-up cortisol, a greater CAR, less cortisol change and elevated (higher) bedtime cortisol levels.

Data analyses

Analyses were conducted in R (Version 4.0.3). First, outlying effect sizes defined by Tabachnick and Fidell's (2013) guidelines of a standardized z-score greater than 3.29 or smaller than −3.29 were "winsorized" to three standard deviations from the mean, which included three effect sizes (two wake and one bed). All correlations were converted to Fisher's Z scores for analyses using the escalc function of the metafor package (Viechtbauer, 2010). Results were converted back to correlation coefficients (r) for ease of interpretation.

Because multiple effect sizes were often included from the same study (i.e., one for each type of adversity examined) and were therefore dependent, we utilized the multilevel approach to meta-analysis describe by Assink and Wibbelink (2016) with effect sizes nested within study samples. This approach allowed us to examine variance in effect sizes across three levels: level 1 examined variance between participants within each study (sampling variance), level 2 examined variance between effect sizes within the same study (within-study variance) and level 3 examined variance in effect sizes between studies (between-study variance). An overall effect for childhood adversity was calculated for each measure of diurnal cortisol (i.e., wake level, CAR, diurnal cortisol change, bedtime level) with a random-effects three-level meta-analytic model using Restricted Maximum Likelihood (REML) estimation method through the rma.mv function of the metafor package. The Knapp and Hartung (2003) adjustment was applied to decrease the likelihood of obtaining unjustified significant results. Next, overall inconsistency of results of studies was assessed using the I^2 statistic (Higgins et al., 2003), and heterogeneity of within-study variance (level 2) and between-study variance (level 3) was examined using one-sided log-likelihood-ratio tests.

Moderators related to the primary aims of this study (i.e., type of adversity, threat vs. deprivation, age at adversity and age at cortisol collection) were examined using omnibus tests as described by Assink and Wibbelink (2016). In addition, if one-sided log-likelihood-ratio tests indicated significant heterogeneity, additional study-level potential primary (i.e., sex, racial/ethnic

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

minority, number of days of cortisol collection, cortisol transformation, diurnal cortisol change timing, whether participants were instructed to take wake samples at awakening, study quality) and exploratory (whether data was received from authors, publication year) moderators were examined. Finally, a post hoc analysis examining the study methodological quality variable as a moderator was conducted. Additional post hoc moderation analyses examining individual indicators of methodological quality (e.g., objective monitoring of awakening, compliance monitoring, instructions about eating/drinking/brushing teeth, assessment of endocrine condition, quality of sleep, etc.) were also performed (please see Supplemental Materials for additional details and results). For categorical moderators, a reference category was chosen for each analysis. (Of note, selection of reference category does not make a statistical difference in overall moderation effect.) If an initial omnibus test for a moderator with multiple categories was significant, additional analyses were conducted examining each individual category as the reference category to determine which pairs of categories were associated with significantly different effects. For type of childhood adversity, only categories of adversity for which there were more than 5 studies were included in moderator analyses for each cortisol measure to ensure sufficient representation for each category; categories with 5 or fewer studies were excluded using listwise deletion for that moderator analysis. For threat versus deprivation, moderation was examined within the subset of effect sizes that reflect threat or deprivation. In addition, given that the sociopolitical context varies across countries, having a racial/ethnic minority status likely conveys different impacts in each country. As a result, moderation analyses for the percentage of participants identified as having a racial/ethnic minority status were repeated using only the studies conducted in the United States. The United States was selected both because enough studies were conducted in the United States to examine that location uniquely (56 studies in the United States, compared to 12 in Canada, the next highest nation) and because significant racism and systemic discrimination have been documented for individuals with a racial/ethnic minority status within the United States (e.g., Wright et al., 2020).

Finally, analyses were conducted to assess for potential publication bias and missing data. Funnel plot asymmetry was examined using Egger's regression test (Egger et al., 1997). Additionally, Rosenthal's fail-safe N was calculated for significant effects to assess the number of missing or unpublished effect sizes needed to produce a nonsignificant effect (Rosenthal, 1979). Based on Rosenthal's (1979) suggestion, an effect was considered resistant to the file drawer problem if the fail-safe N was greater than $5k + 10$, where k is the number of studies included. These analyses were included to be consistent with previous meta-analyses and to provide potential indicators of publication bias, even though these methods have not been tested for a multilevel approach to meta-analysis (Assink & Wibbelink, 2016).

Results

Wake levels

A total of 148 effect sizes from 83 distinct study samples were included in the meta-analysis examining childhood adversity and wake cortisol levels. The sample sizes ranged from 14 to 2,162 with a median of 102. See Table 1 for details about the studies included and Table 2 for the number of effect sizes in each category of childhood adversity.

Overall effect

The overall effect for childhood adversity and wake levels of cortisol was not significant, $r = -0.008$, 95% CI [−0.024, 0.008], $t = -0.949$, $p = 0.344$ (see Supplemental Figure 1). One-sided log-likelihood-ratio tests indicated that there was not significant variation within, $\sigma^2 < 0.001$, $\chi^2(1) = 1.372$, $p = 0.121$, or between, $\sigma^2 = 0.012$, $\chi^2(1) = 1.186$, $p = 0.138$, studies. The distribution of variance across levels was 81.36% at level 1 (i.e., 81.36% of the total variance can be attributed to sampling variance within studies), 10.66% at level 2 (i.e., 10.66% of the total variance can be attributed to differences between effect sizes within studies) and 7.98% at level 3 (i.e., 7.98% of the total variance could be attributed to differences in effect sizes between studies). The overall I^2 reflecting heterogeneity in effect sizes across both levels 2 (within studies) and 3 (between studies) was 18.64% (i.e., 18.64% of the variability in effect size estimates resulted from differences in effect sizes within and between studies rather than sampling error).

Moderators

See Table 3 for information on moderator variables for each study and Table 4 for descriptive statistics for moderator variables. None of the moderators related to primary aims yielded significant results: type of adversity, $F(6, 126) = 1.911$, $p = 0.084$; age at adversity, $F(4, 143) = 1.064$, $p = 0.377$; and age at cortisol collection, $F(1, 142) = 0.122$, $p = 0.727$. Given that the between-study variance was not significant, none of the additional between-study moderators were examined. Finally, neither the post hoc analysis examining threat vs. deprivation as a moderator in the subset of studies that included threat or deprivation effect sizes ($n = 18$), $F(1, 16) = 0.581$, $p = 0.457$, nor the post hoc analysis examining study methodological quality, $F(1, 146) = 0.024$, $p = 0.876$, were significant. (See Supplemental Materials for more detailed methodological quality analyses.)

Publication bias

Egger's regression test did not indicate significant funnel plot asymmetry, $z = 0.567$, $p = 0.571$ (see Figure 2).

Cortisol awakening response

A total of 76 effect sizes from 45 distinct study samples were included in the meta-analysis examining childhood adversity and the CAR. Sample sizes ranged from 15 to 2,162 with a median of 89. See Table 1 for details about the studies included and Table 2 for the number of effect sizes in each category of childhood adversity.

The overall effect of childhood adversity on the CAR was not significant, $r = 0.021$, 95% CI [−0.013, 0.054], $t = 1.223$, $p = 0.225$ (see Supplemental Figure 2). One-sided log-likelihood-ratio tests indicated that variation was significant between, $\sigma^2 = 0.006$, $\chi^2(1) = 6.019$, $p = 0.007$, but not within, $\sigma^2 < 0.001$, $\chi^2(1) < 0.001$, $p > 0.500$, studies. The distribution of variance across levels was 50.59% at level 1 (sampling variance), <0.01% at level 2 (within-study variance) and 49.41% at level 3 (between-study variance), with an overall I^2 of 49.41%.

Moderators

None of the moderators related to primary aims yielded significant results: type of adversity, $F(5, 60) = 0.674$, $p = 0.645$; age at adversity, $F(4, 71) = 1.129$, $p = 0.350$; and age at cortisol collection, $F(1, 73) = 0.174$, $p = 0.678$. Given the significant between-study variance, study-level variables were also examined as potential moderators of the overall effect. None of the primary or

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Table 1. Study characteristics

Paper(s)	Country	Sample	Sample size (N)	Age at time of cortisol collection	Percent female	Percent racial/ ethnic minority	Type of adversity	Age at time of adversity	Adversity details	Cortisol measure
Albers et al., 2016	Netherlands	Infants recruited from childcare center waitlists	60	1 year ($M = 1.00$)	45.00%	Missing	Parenting difficulties	Infancy	Observed maternal behavior (sensitivity and cooperation)	Diurnal Change
Alink et al., 2012; Cicchetti & Rogosch, 2007; Cicchetti et al., 2010, 2011; Granger et al., 2007; Murray-Close et al., 2008	United States	School-aged maltreated and demographically comparable low-income nonmaltreated children who attended a day camp research program	599–605	6–10 years ($M = 9.41$)	47.80%	91.90%	Maltreatment; single parent status	Multiple	Maltreatment status; single parent	Diurnal Change
Ashman et al., 2002	United States	Children of mothers with and without history of depression	68–69	7–8 years ($M = 7.60$)	56.76%	8.10%	Parental mental health	Multiple	Maternal history of depression based on SCID[1], CES-D[2] and adapted Longitudinal Interval Follow-up Evaluation	Wake; Bed
Badanes et al., 2011, 2012; Miles et al., 2018	United States	Preschoolers recruited from child care centers, oversampling those using subsidies	87–99	2–6 years ($M = 4.10$)	53.00%	66.25%	Financial strain; parental mental health; other family stress	Multiple	Loss of housing or at least one utility due to financial strain; CES-D[2] (mother); Multicultural Events Schedule for Adolescents	Diurnal Change
Basu et al., 2013	United States	Women with and without IPV exposure	88	18–41 years ($M = 27.00$)	100%	48.00%	Maltreatment	Multiple	CTQ-SF[3] physical abuse subscale	Wake
Beijers et al., 2020; Evans et al., 2020; Simons et al., 2015	Netherlands	Healthy mother–child dyads from a community sample in longitudinal Basal Influences on Child Development (BIBO) project	115–135	6 years ($M = 6.05$)	49.30%	Missing	Community-level stressors; parental mental health	Early childhood; multiple	Neighborhood-level SES income component; maternal trait anxiety from State-Trait Anxiety Inventory	Wake; Diurnal Change
Berger et al., 2017[a]	Australia	Domestic students from James Cook University	22	20–36 years ($M = 23.50$)	72.73%	50.00%	Maltreatment	Multiple	Maltreatment and Abuse Expose Scale	Wake; CAR; Diurnal Change
Bernard et al., 2010; Dozier et al., 2006	United States	Children with CPS involvement and control sample	313–327	0–2 years ($M = 1.08$)	44.77%	67.75%	Surrogate care	Infancy	Placed in foster care	Wake; Diurnal Change; Bed
Boyer & Nelson, 2015; Roisman et al., 2009	United States	Adolescents enrolled in the NICHD Study of Early Child Care and Youth Development (SECCYD)	821–884	15 years ($M = 15.07$)	50.50%–51.00%	15.00%–33.00%	Financial strain; parenting difficulties	Multiple; early childhood	Income-to-needs ratio; Sensitivity coded from Mother-Child Interaction Tasks	Wake
Braehler et al., 2005[a]	Canada	Medicated outpatients with schizophrenia diagnosis	14	$M = 33.07$	7.14%	Missing	Maltreatment	Multiple	At least one moderate rating on one CTQ[4] subscale	Wake
Brendgen et al., 2017; Ouellet-Morin et al., 2009	Canada	Twins recruited from greater Montreal area	211–278	Grade 8 ($M = 14.07$) or 6 months ($M = 0.50$)	51.60%–54.41%	7.29% – 13.00%	Parenting difficulties; single parent status; parental mental health (6 month sample); cumulative adversity (6 month sample)	Adolescence; missing; infancy; infancy	Network of Relationships Inventory (for mother and father); single parent; parental postnatal depression; cumulative familial adversity	Wake; CAR; Diurnal Change; Bed

Brewer-Smyth et al., 2004	United States	Female inmates	113	18–58 years ($M = 33.40$)	100%	55.75%	Maltreatment	Multiple	Revised Muenzenmaier's Childhood Physical and Sexual Abuse Scale total score	Wake; Diurnal Change
Brummelte et al., 2015	Canada	Children born prematurely	92–126	7 years ($M = 7.73$)	55.63%	19.40%	Parental mental health; single parent status; other family stress	Multiple	BDI-II[5]; single parent; Parenting Stress Index	Wake; Diurnal Change
Butler et al., 2017	United Kingdom	Healthy adult males	99–109	21–63 years ($M = 34.20$)	0%	5.50%	Maltreatment; cumulative adversity	Multiple	Physical abuse or violence from Childhood Traumatic Events Scale; Score of 6 or 7 on trauma scale of Childhood Traumatic Events Scale	Wake; CAR
Carrion et al., 2010; Taylor et al., 2009[a]	United States	Youth with and without post-traumatic stress symptoms	33–49	10–16 years ($M = 13.23$) or 7–14 years ($M = 10.60$)	31.10%–42.37%	40.33%–57.63%	Maltreatment; Other family stress	Multiple	History of interpersonal trauma; history of separation/loss traumatic event	Bed
Chen et al., 2017	United States	Community sample of early adolescent girls	117	$M = 12.39$	100%	11.00%	Cumulative adversity	Multiple	Overall severity rating score from lifetime adversity section of the Youth Life Stress Interview	Wake
Chernego et al., 2019	Russia	Institutionally reared and family-reared children	49–50	0–3 years ($M = 2.14$)	43.84%	0%	Surrogate care	Multiple	Institutional rearing	Wake; Diurnal Change; Bed
Chiang et al., 2016; Huynh et al., 2016	United States	Adolescents recruited from Los Angeles area high schools	289–308	14–20 years ($M = 16.39$)	56.96%	70.45%	Other family stress	Multiple	Daily reports of family demands and conflict	Wake; CAR; Diurnal Change; Bed
Cicchetti & Rogosch, 2001a; Cicchetti & Rogosch, 2001b	United States	Low-income maltreated and nonmaltreated children attending day camp research program	384	5–13 years ($M = 9.25$)	39.60%	80.20%	Maltreatment; single parent status	Multiple	Presence/absence of maltreatment; single parent	Diurnal Change
Cima et al., 2008	Netherlands	Undergraduate students (control group)	27	$M = 28.39$	0%	0%	Maltreatment	Multiple	CTQ[4]	Diurnal Change
Clearfield et al., 2014	United States	Infants and mothers with low and high socioeconomic status	32	0.5–1 years ($M = 0.77$)	34.37%	9.38%	Financial strain	Infancy	Low versus high socioeconomic status (based on state assistance for food/housing and maternal education)	Wake; Diurnal Change; Bed
Clowtis et al., 2016	United States	Mother–child dyads recruited from Head Start in Texas	50–52	3–5 years ($M = 4.30$)	36.20%	Missing	Parenting difficulties; parental mental health; other family stress	Early childhood	Keys to Interactive Parenting Scale; maternal CES-D[2]; Maternal Perceived Stress Scale	Bed
Cordero et al., 2017	Switzerland	Mothers with and without interpersonal violence PTSD and toddlers	26–30	1–3 years	59.26%	29.63%	Parental mental health	Multiple	Presence/absence of maternal interpersonal violence PTSD assessed by Clinician Administered PTSD Scale and Posttraumatic Symptom Checklist - short version	Wake; Bed

(*Continued*)

Table 1. (*Continued*)

Paper(s)	Country	Sample	Sample size (N)	Age at time of cortisol collection	Percent female	Percent racial/ ethnic minority	Type of adversity	Age at time of adversity	Adversity details	Cortisol measure
Cullen et al., 2014	United Kingdom	Children with or without increased risk for schizophrenia	82–109	11–14 years ($M = 13.04$)	46.32%	66.97%	Cumulative adversity; parental mental health	Multiple	Total negative life event exposure in childhood; parent has mental health diagnosis	Wake; CAR; Diurnal Change
DeCaro & Worthman, 2008	United States	Cohort of children recruited in preschool	28	5–6 years ($M = 5.58$)	Missing	29.00%	Parenting difficulties; single parent status; other family stress	Early childhood	Berkeley Puppet Interview maternal warmth; single parent; Parenting Stress Index Short Form Parent-Child Dysfunctional Interactions scale	CAR
Donoho et al., 2011	United States	Female minority youth from Los Angeles County participating in longitudinal study	23	8–11 years ($M = 9.20$)	100%	100%	Other family stress	Multiple	Checklist of negative family life events	CAR
Doom et al., 2013; Rogosch et al., 2011	United States	Low-income, racially and ethnically diverse children who attended a summer research day camp	240–242	7–11 years ($M = 9.41$)	47.80%	91.90%	Maltreatment; single parent status	Multiple	Maltreatment status; single parent	Diurnal Change
Doom et al., 2018; Elhassan et al., 2015; Lumeng et al., 2014; Miller et al., 2017	United States	Low-income preschoolers in the Midwest attending Head Start	347–364	2–5 years ($M = 4.23$)	50.40%	43.30%	Financial strain; single parent status; other family stress	Multiple	Income-to-needs ratio; single parent; Chaos, Hubbub, and Order Scale (CHAOS)	Diurnal Change
Dougherty et al., 2009, 2013	United States	Children participating in a study examining early temperament and risk for psychopathology	201–230	5–6 years ($M = 6.22$)	46.5%	14.5%	Parental mental health; single parent status	Early childhood	Maternal depression during pregnancy and/or child's lifetime; one-parent household	Wake; Diurnal Change; Bed
Ellenbogen & Hodgins, 2009; Ellenbogen et al., 2004, 2006; Ostiguy et al., 2011	Canada	Children of parents with bipolar disorder or no mental health disorder	104–132	13–28 years ($M = 19.30$)	47.54%	5.00%	Parenting difficulties; parental mental health	Multiple	Parenting Dimensions Inventory supportiveness dimension; parental mental health diagnosis during child's lifetime	Wake; Diurnal Change; CAR; Bed
Engert et al., 2011	Canada	Adult participants recruited from university website	58	19–27 years ($M = 21.29$)	67.24%	Missing	Parenting difficulties	Multiple	Low parental care based on Parental Bonding Instrument cutoff score	Wake; CAR
Epstein et al., 2019	United States	Pregnant women	63	19–37 years ($M = 28.16$)	100%	38.46%	Cumulative adversity	Multiple	Early life adversity from Stress and Adversity Inventory	Wake; CAR; Diurnal Change; Bed
Essex et al., 2011	United States	Wisconsin Study of Families and Work	273	14–16 years ($M = 15.50$)	Missing	11.00%	Parental mental health; other family stress	Multiple	CES-D^2 (parents); Family expressed anger (Anger Expression Inventory, items from Partner Role Quality,and Negative subscale of the Family Expressiveness Questionnaire)	Diurnal Change
Evans et al., 2013	Netherlands	Children of parents with and without a substance use disorder	140	11–20 years ($M = 16.09$)	40.96%	Missing	Parental substance use	Multiple	Parental diagnosis of substance use disorder	Wake

Evans et al., 2020	Netherlands	JOiN sample participants part of a longitudinal general population study in youth	286–291	7–12 years ($M = 10.64$)	53.00%	12.91%	Community-level stressors	Multiple	Neighborhood-level income	Wake; CAR; Diurnal Change
Farrell et al., 2018	Ireland	Depressed patients and healthy controls	38–40	17–45 years ($M = 28.24$)	67.16%	Missing	Maltreatment	Multiple	CTQ[3]	Wake; Diurnal Change
Fisher et al., 2007	United States	Foster preschoolers entering new placements and low-income nonmaltreated community preschoolers	120	4–7 years ($M = 5.40$)	44.17%	11.00%	Surrogate care	Missing	Foster care placement	Wake; Diurnal Change; Bed
Flom et al., 2017; St. John et al., 2017; Tarullo et al., 2017	United States	Mother-infant dyads	79–84	$M = 0.56$ years	57.10%	29.76%	Parenting difficulties; parental mental health; other family stress	Infancy	Maternal intrusiveness coded from mother-infant play interaction; maternal CES-D[2]; Parenting Stress Index	Wake; Diurnal Change; Bed
Foland-Ross et al., 2014; LeMoult, Chen, et al., 2015; LeMoult, Ordaz, et al., 2015	United States	Never-disorder daughters with and without maternal history of depression	112	9–15 years ($M = 12.03$)	100%	30.36%	Parental mental health	Multiple	Maternal history of depression during daughter's lifetime	Wake; CAR; Diurnal Change; Bed
Fuchs et al., 2017	Germany	Children of women with and without moderate to severe childhood abuse	73–81	3 years ($M = 2.84$)	50.00%	Missing	Parenting difficulties; parental mental health; single parent status	Infancy; multiple; multiple	Emotional Availability Scales total score; German Version of the Symptom Checklist 90-Revised (mother); single parent	Wake; CAR; Diurnal Change
Gartstein et al., 2018; Lengua et al., 2013; Thompson et al., 2018; Zalewski et al., 2012, 2016	United States	Community sample of preschool-aged children	114–265	3 years ($M = 3.06$)	50.30%	33.00%	Cumulative adversity; community-level stressors; parenting difficulties; parental mental health; single parent status; other family stress	Multiple; early childhood; early childhood; early childhood; early childhood; early childhood	Cumulative family risk (low maternal education, single parent status, divorce, adolescent parent, maternal depression, negative life events, residential instability and household density); Crime Proximity Index; negativity coded from mother–child interactions; maternal CES-D[2]; single parent; residential crowding	Wake; Diurnal Change; Bed
Gerritsen et al., 2017; Holleman et al., 2012; Vreeburg et al., 2010	Netherlands	Netherlands Study of Depression and Anxiety (NESDA) cohort study	2088–2126	18–65 years ($M = 43.37$)	66.00%	4.70%	Maltreatment	Multiple	Childhood trauma index from NEMESIS childhood trauma interview	Wake; CAR; Diurnal Change
Goldstein et al., 2017; Liu et al., 2016	United States	Adolescent females with at least one available biological parent	506–538	13–15 years ($M = 14.40$)	100%	18.71%	Parental mental health; parenting difficulties; single parent status	Adolescence	Parental psychopathology during child's lifetime; Parental Bonding Instrument care dimension; single parent	Wake; CAR; Diurnal Change
Gunnar et al., 2001	Canada	Children reared in Romanian orphanages in their first years of life, early adopted children, and children born in Canada	65–66	6–11 years ($M = 7.82$)	53.33%	Missing	Surrogate care	Multiple	Adoption (children in Romanian orphanages and early adopted children)	Wake; Diurnal Change; Bed
Habersaat et al., 2014	Switzerland	Preterm infants	46	14 months ($M = 1.17$ years)	50.00%	Missing	Parental mental health	Infancy	Global score from French version of Perinatal PTSD Questionnaire	Diurnal Change

(*Continued*)

Table 1. (*Continued*)

Paper(s)	Country	Sample	Sample size (N)	Age at time of cortisol collection	Percent female	Percent racial/ ethnic minority	Type of adversity	Age at time of adversity	Adversity details	Cortisol measure
Halligan et al., 2004; Murray et al., 2010	United Kingdom	Adolescents who had or had not been exposed to postnatal maternal depression	82–87	13–14 years ($M = 13.33$)	54.30%	0%	Parenting difficulties; parental mental health; other family stress	Adolescence; early childhood; multiple	Maternal sensitivity rated from mother–child interaction at age 5; maternal depression from Structured Clinical Interview for DSM-IV; parental conflict (perceived and felt criticism);	Diurnal Change
Hanson & Chen, 2010	Canada	College undergraduate students	87	19–25 years ($M = 21.51$)	67.00%	72.00%	Parenting difficulties; other family stress	Multiple	Maternal warmth from Parent Bonding Inventory; Risky Families Questionnaire	Diurnal Change
Harris et al., 2017	United Kingdom	Population sample in Scotland	145–150	$M = 76.60$ years	50.70%	Missing	Cumulative adversity; financial strain; other family stress	Multiple	Childhood high stress (lost a parent, scored highest on deprivation, or moved school more often than 95% of the sample; childhood deprivation based on paternal social class and unemployment; childhood and adolescence parental loss based on number of deaths, divorces and separations	Wake; CAR; Diurnal Change
Harvey et al., 2019	United States	Youth with asthma	95–122	10–17 years ($M = 12.90$)	42.03%	75.61%	Parenting difficulties; single parent status	Multiple	Parental warmth from Parental Behavior Inventory; single parent	Wake; CAR; Diurnal Change; Bed
Hibel et al., 2019, 2020; Valentino et al., 2015	United States	Children of maltreating and non-maltreating mothers	220–228	3–6 years ($M = 4.90$)	49.60%	74.56%	Maltreatment; parenting difficulties; parental mental health; single parent status; other family stress	Multiple; multiple; early childhood; early childhood; early childhood	CTQ[4]; maternal sensitivity coded from play interaction using Mini-Preschool Maternal Behavior Q-sort; maternal CES-D[5]; single parent; Revised Conflict Tactics Scale- Short Form	Wake; Diurnal Change; Bed
Huizink et al., 2009; Laceulle et al., 2017; Marsman et al., 2012; Zandstra et al., 2015	Netherlands	Tracking Adolescents' Individual Lives Survey (TRAILS) participants	893–2162	10–12 years ($M = 11.09$)	46.88%	10.00%	Parental mental health; parental substance use	Multiple; middle childhood	History of parental mental health problems during childhood; high maternal substance use (11–20 cigarettes per day and/or more than 11 glasses of alcohol containing beverages per week) compared to low maternal substance use (less than one cigarette per day and less than one glass of alcohol containing beverage per week)	Wake; CAR
Hustedt et al., 2017	United States	Families participating in Early Head Start Program in a Mid-Atlantic state	54–79	0–3 years ($M = 1.60$)	45.20%	57.20%	Parental mental health; single parent status; other family stress	Multiple	Parental Generalized Anxiety Disorder 7-Item Scale; single parent; Parenting Stress Index: Short Form	Wake; Diurnal Change; Bed
Johnson et al., 2011	United States	Internationally adopted children from institutional and foster care and non-adopted children	113	6–8 years ($M = 6.85$)	75.00%	69.00%	Surrogate care	Early childhood	Adoption	Wake; Diurnal Change; Bed

Johnson & Tottenham, 2015	United States	Adults with and without history of foster care	54	18–33 years ($M = 21.00$)	64.15%	79.25%	Surrogate care	Multiple	History of foster care	Wake
Keeshin et al., 2014	United States	Adolescent girls with and without a history of recent sexual abuse	36	12–17 years ($M = 14.97$)	100%	58.00%	Maltreatment	Multiple	History of recent sexual abuse	Wake; CAR; Diurnal Change
Kertes et al., 2008	United States	International Adoptees	150–155	7–11 years ($M = 9.21$)	55.00%	66.45%	Maltreatment; single parent status	Multiple	Parent-rated pre-adoption neglect/abuse; single parent	Wake; Diurnal Change
Kiel et al., 2015	United States	Toddlers and mothers in longitudinal study	34	18–20 months ($M = 1.58$ years)	50.98%	21.05%	Parenting difficulties	Infancy	New Friends Vignettes	Diurnal Change
King et al., 2017	United States	Children in longitudinal study	140–145	9–13 years ($M = 11.40$)	57.00%	41.96%	Cumulative adversity	Multiple	Severity of early life stress based on modified Traumatic Events Screening Inventory for Children	Wake; CAR; Diurnal Change
Kliewer et al., 2009; Kliewer, 2006	United States	Black youth living in neighborhoods with low socioeconomic status or moderate/high violence in Richmond, VA	59–84	8–13 years ($M = 11.30$)	51.00%	100%	Cumulative adversity; community-level stressors; parental mental health; parenting difficulties; single parent status	Multiple	Multiple risks index; Survey of Children's Exposure to Community Violence (community violence witnessed); Brief Symptom Inventory depression subscale; quality of mother–child interaction in two tasks; single parent	Wake; CAR
Kohrt et al., 2015	Nepal	Community sample of adults	118	$M = 37.00$ years	49.10%	3.20%	Maltreatment	Multiple	Above median on CTQ[4] score	Wake; CAR
Korpa et al., 2017	Greece	Children with and without an ADHD diagnosis	45	6–11 years ($M = 8.70$)	31.67%	Missing	Other family stress	Multiple	Parenting Stress Index – Short Form	Wake
Koss et al., 2014, 2016; Perry et al., 2019; Pitula et al., 2019	United States	Post-institutionalzied, post-foster care, and non-adopted children	147–165	3–5 years ($M = 4.27$)	49.82%	68.15%	Parenting difficulties; surrogate care	Multiple	Parenting quality (supportive presence and structure and limit setting) rated from parent-child interactions; surrogate care (post-institutionalized or foster care)	Wake; Diurnal Change; Bed
Kuhlman, Geiss, et al., 2015; Kuhlman, Vargas, et al., 2015	United States	Community sample of youth	78–121	9–16 years ($M = 12.80$)	49.00%	21.69%	Cumulative adversity; maltreatment; single parent status	Multiple	Early Trauma Inventory total score; Early Trauma Inventory combined physical, sexual and emotional abuse items; single parent	Wake; CAR; Diurnal Change; Bed
Kumsta et al., 2017	United Kingdom	Young adult adoptees who lived under deprivation in Romania orphanages or non-deprived in UK	43–57	22–26 years ($M = 24.04$)	61.40%	0%	Other	Multiple; adolescence	Early deprivation exposure	Wake; CAR; Diurnal Change
Kwak et al., 2017	United States	Latino mother–adolescent dyads with a fifth grade child	51–57	10–11 years ($M = 11.08$)	54.00%	100%	Cumulative adversity; discrimination; financial strain; single parent status; other family stress	Middle childhood	CRISYS total score; CRISYS prejudice domain; CRISYS financial domain; single parent status; CRISYS home issues domain	Wake; CAR; Diurnal Change

(*Continued*)

Table 1. (*Continued*)

Paper(s)	Country	Sample	Sample size (N)	Age at time of cortisol collection	Percent female	Percent racial/ ethnic minority	Type of adversity	Age at time of adversity	Adversity details	Cortisol measure
Laurent, Leve, Neiderhiser, Natsuaki, Shaw, Fisher, et al., 2013; Laurent et al., 2013, 2014; Marceau et al., 2013	United States	Adoption sample from Early Growth and Development Study	191–213	6 years ($M = 5.98$)	47.10%	43.00%	Cumulative adversity; financial strain; single parent status; other family stress	Early childhood; early childhood; early childhood; multiple	Cumulative adversity at age 6; financial need (demographic subscale assessing degree to which insufficient money to cover material needs); single parent; Home Chaos, Hubbub and Order Scale (CHAOS)	Wake; Diurnal Change; Bed
Leneman et al., 2018	United States	Children adopted between 6 and 60 months or not adopted	275–277	7–15 years ($M = 11.26$)	58.84%	32.13%	Surrogate care	Multiple	Adoption status (adopted vs. biological child)	Wake; CAR; Diurnal Change
Leppert et al., 2018; Merwin et al., 2018; Merwin, 2017; Merwin, Leppert, et al., 2017; Merwin, Smith, et al., 2017	United States	Preschool-age children and their biological parents with and without a history of depression	140–148	3–5 years ($M = 4.14$)	51.40%	51.37%	Parental mental health; single parent status	Early childhood	Presence of maternal psychopathology during pregnancy and child's lifetime; single parent	Wake; CAR; Diurnal Change; Bed
Letourneau et al., 2011	Canada	Infants of mothers affected by postpartum depression	43–53	3–12 months ($M = 0.41$ years)	47.17%	Missing	Cumulative adversity; financial strain; parenting difficulties; parental mental health	Infancy	Difficult Life Circumstances; low income determined by Low Income Cut-Off score; Nursing Child Assessment Teaching Scale; Edinburgh Postnatal Depression Scale	Wake; Diurnal Change; Bed
Lovallo et al., 2019	United States	Community sample of young adults	169–580	18–30 years ($M = 23.66$)	56.14%	19.14%	Cumulative adversity; maltreatment	Multiple	Early life adversity from computerized Diagnostic Interview Schedule-IV; CTQ[4]	Wake; Diurnal Change; Bed
Mangold et al., 2010	United States	Mexican-American adults	59	18–38 years ($M = 22.00$)	55.93%	100%	Maltreatment	Multiple	Middle or high score on Early Trauma Inventory Short Form	CAR
McLachlan et al., 2016	Canada	Children and adolescents from NeuroDevNet FASD study cohort	35	5–18 years ($M = 11.54$)	55.30%	Missing	Cumulative adversity; maltreatment; surrogate care	Multiple	Parental adversity (serious mental illness, suspected of having FASD, substance-abuse, trouble with law); physical/sexual abuse; foster care	Wake; Diurnal Change; Bed
Meinlschmidt & Heim, 2005	Germany	Healthy college students	95	19–36 years ($M = 21.70$)	69.47%	Missing	Other family stress	Multiple	Early loss experience	CAR
Messerli-Burgy et al., 2018	Switzerland	Swiss Preschooler's Health Study (SPLASHY) prospective cohort study of early childhood	238	2–6 years ($M = 3.91$)	47.27%	Missing	Parenting difficulties; other family stress	Multiple	Alabama Parenting Questionnaire inconsistent parenting; Parenting Stress Scale	Wake; CAR; Diurnal Change; Bed
Miller et al., 2018; Miller, Margolin, et al., 2017	United States	Community cohort of youth recruited for longitudinal study	92–94	14–21 years ($M = 18.05$)	46.46%	71.43%	Cumulative adversity	Multiple	Life Events Checklist	Wake; CAR; Diurnal Change

Monteleone et al., 2015	Italy	Women with and without eating disorders	44	18–49 years ($M = 28.72$)	100%	0%	Maltreatment	Multiple	Experienced maltreatment based on CTQ[4]	Wake
O'Connor et al., 2005	United Kingdom	Children from Avon Longitudinal Study of Parents and Children	71	10 years ($M = 10.52$)	49.00%	Missing	Parental mental health	Multiple	Edinburgh Postnatal Depression Scale (18 and 32 week gestation, 8 weeks postnatal	Wake
O'Loughlin et al., 2020	Canada	Women without diagnosis of clinical depression, major medical illnesses that impact sexual function, or use of medications with sexual-functioning side effects	276	19–65 years ($M = 32.41$)	100%	40.29%	Maltreatment	Multiple	CTQ[4]	Wake; CAR; Diurnal Change; Bed
Pawluski et al., 2012	Canada	Infants of SSRI-exposed and nonexposed mothers	50	3 months ($M = 0.27$ years)	53.85%	18.00%	Parental mental health	Multiple	Maternal Edinburgh Postnatal Depression Scale score	Diurnal Change
Pendry & Adam, 2007	United States	Kindergarten-aged children and adolescents	61	5–18 years ($M = 10.82$)	60.32%	Missing	Parenting difficulties; parental mental health; other family stress	Multiple	Maternal parenting (parent involvement, parent warmth); maternal emotional functioning (anxiety, depression, self-esteem); Marital functioning (marital satisfaction, conflict tactics)	Wake; Diurnal Change; Bed
Peng et al., 2014	China	Patients with depression and matched controls with and without childhood neglect	109	18–45 years ($M = 28.37$)	46.79%	Missing	Maltreatment	Multiple	Neglect measured from CTQ[4]	CAR
Philbrook & Teti, 2016	United States	Infants in Project SIESTA	82	9 months ($M = 0.77$ years)	53.00%	Missing	Parenting difficulties	Infancy	Average Emotional Availability Scale score across ages 3, 6 and 9 months	Wake; Bed
Plant et al., 2016	United Kingdom	Young adult offspring of the South London Child Development Study (SLCDS)	74	24–26 years ($M = 25.15$)	48.55%	28.38%	Maltreatment	Multiple	Maltreatment or neglect based on Childhood Experience of Care and Abuse Questionnaire and Child and Adolescent Psychiatric Assessment	Wake; CAR
Pruessner et al., 2013	Canada	Patients with first episode psychosis and controls	93–94	15–36 years ($M = 23.00$)	40.66%	26.09%	Parenting difficulties	Multiple	Maternal Parental Bonding Inventory care score	Wake; CAR
Puetz et al., 2016	Germany	Children placed out of home before age 3 because of maltreatment and controls	36–40	8–14 years ($M = 10.49$)	50.98%	29.41%	Surrogate care	Multiple	Placement in long-term foster care or adoptive families	Wake; Diurnal Change; Bed
Quevedo et al., 2017	United States	Maltreated and non-maltreated depressed youth	55	$M = 14.91$ years	63.64%	50.91%	Maltreatment	Multiple	Maltreatment as established by the PTSD section of the K-SADS[6]	Wake; CAR
Raffington, Prindle, et al., 2018	Germany	Healthy, sociodemographically diverse children	97–98	6–7 years ($M = 7.16$)	46.00%	15.00%	Single parent	Early childhood	Single parent	Wake; CAR
Raffington, Schmiedek, et al., 2018	Germany	Children in longitudinal HippoKID study	49–52	6–7 years ($M = 6.68$)	54.27%	Missing	Cumulative adversity; other family stress	Multiple; early childhood	Children's Stress Questionnaire; Parenting Stress Index	Wake; CAR; Diurnal Change

(*Continued*)

Table 1. (*Continued*)

Paper(s)	Country	Sample	Sample size (N)	Age at time of cortisol collection	Percent female	Percent racial/ ethnic minority	Type of adversity	Age at time of adversity	Adversity details	Cortisol measure
Reichl et al., 2016	Germany	Adolescents engaging in NSSI and matched healthy controls	25–26	14–18 years ($M = 16.22$)	92.31%	Missing	Cumulative adversity; maltreatment; parenting difficulties	Multiple	Childhood Experiences of Care and Abuse Interview (CECA); neglect, physical abuse, psychological abuse, and sexual abuse subscales of CECA; antipathy from CECA	Wake; CAR; Diurnal Change; Bed
Russ et al., 2012	United Kingdom	Children of mothers with social phobia, GAD, or no history of anxiety	65–88	Missing	56.25%	Missing	Parental mental health	Missing	Maternal diagnosis of GAD or social phobia based on Structured Clinical Interview for DSM-IV	Wake; Diurnal Change; Bed
Seidenfaden et al., 2017	Netherlands	Healthy controls	37–38	20–55 years ($M = 31.69$)	51.32%	0%	Cumulative adversity; maltreatment	Multiple	Childhood Abuse and Trauma Scale (CATS) total score; CATS sexual abuse scale	Wake; CAR; Diurnal Change
Simmons et al., 2015	Australia	Early adolescents in the Orygen Adolescent Development Study (OADS)	20–42	$M = 15.56$ years	43.42%	17.00%	Maltreatment; parenting difficulties	Multiple; middle childhood; adolescence	CTQ[4]; maternal aggression frequency coded using Living in Familial Environments from Family Interaction Task	Wake
Smeets et al., 2007	Netherlands	Women with continuous memories of childhood sexual abuse (CSA) and women without CSA	15	22–53 years ($M = 40.40$)	100%	Missing	Maltreatment	Multiple	Continuous memory of childhood sexual abuse based on semi-structured memory interview	Wake; CAR; Diurnal Change
Starr et al., 2017	United States	Adolescents from midsized metropolitan area in the Northeast United States	195–200	14–17 years ($M = 15.90$)	54.00%	27.86%	Cumulative adversity; other family stress	Multiple; adolescence	Youth Life Stress Interview childhood adversity; chronic stress family domain from Life Stres Interview	Wake; Diurnal Change
Theall et al., 2017	United States	African-American children from neighborhoods in the greater New Orleans, Louisiana, area	85	5–16 years ($M = 9.80$)	58.82%	100%	Community-level stressors; other family stress	Multiple	Police-reported number of assault crimes within one meter radius of child's residence; witnessing physical or verbal fighting at home based on items adapted from Preschool Age Psychiatric Assessment	Wake; CAR; Diurnal Change; Bed
Van den Bergh et al., 2008	Belgium	First-born children of healthy Dutch-speaking mothers	58	14–15 years ($M = 15.00$)	50.00%	0%	Parental mental health	Multiple	Mean maternal State-Trait Anxiety Inventory trait score across multiple measurements in pregnancy and childhood	Wake; Diurnal Change
van der Vegt et al., 2009	Netherlands	International adoptees adopted by nonrelatives in the Netherlands	529	28–34 years ($M = 30.83$)	33.08%	Missing	Maltreatment	Multiple	Presence of neglect experienced prior to adoption	Wake; Bed
Vänskä et al., 2016	Finland	Children of mothers recruited during pregnancy	104–108	10–12 years ($M = 10.60$)	51.49%	0%	Parental mental health	Multiple	Maternal Beck's Depression Inventory (BDI-13)	Wake; CAR; Diurnal Change; Bed

Weissbecker et al., 2006	United States	Women with fibromyalgia	85	$M = 48.20$ years	100%	4.00%	Maltreatment	Multiple	CTQ[4] total score	CAR; Diurnal Change
Wielaard et al., 2018	Netherlands	Netherlands Study of Depression in Older Persons (NESDO)	402	$M = 70.80$	62.92%	Missing	Maltreatment	Multiple	Presence of childhood abuse assessed using Childhood Abuse Inventory	Wake
Yehuda et al., 2005	United States	Infants of mothers directly exposed to World Trade Center collapse during pregnancy	31	1 year ($M = 0.75$)	Missing	Missing	Parental mental health	Multiple	Maternal Beck Depression Inventory	Wake; Bed
Zalewski, Lengua, Fisher et al., 2012	United States	Preschool-age children	76–80	3–4 years ($M = 3.50$)	47.00%	27.63%	Parenting difficulties; single parent status	Multiple	Parental negative affect rated during 5-minute mother–child interaction; single parent	Wake; Diurnal Change; Bed
Zeiders et al., 2012	United States	Mexican-American 10th graders in longitudinal study	100	14–16 years ($M = 15.29$)	51.00%	100%	Cumulative adversity; discrimination	Adolescence	Multicultural Events Scale for Adolescents; Brief Perceived Ethnic Discrimination Questionnaire-Community Version	Wake; CAR; Diurnal Change; Bed
Zhu et al., 2019	China	Children and parents	197–200	6–15 years ($M = 9.46$)	46.96%	Missing	Parental mental health	Multiple	Parental trait anxiety from State-Trait Anxiety Inventory	Wake; Bed

Note. Papers are grouped together based on study. If effect sizes were pulled from a specific paper, that paper is included. If authors provided data for a study, all papers related to that study are included. Papers are listed in alphabetical order.
[1]The Structured Clinical Interview for DSM-5; [2]Center for Epidemiologic Studies Depression Scale; [3]Childhood Trauma Questionnaire – Short Form; [4]Childhood Trauma Questionnaire; [5]Beck Depression Inventory-II; [6]Kiddie Schedule for Affective Disorders and Schizophrenia

Table 2. Number of effect sizes in each category of childhood adversity by diurnal cortisol measure

	Diurnal Cortisol Measure			
Childhood Adversity	Wake	CAR	Diurnal Change	Bed
Community-Level Stressors	5	3	4	2
Cumulative Adversity	20	12	17	8
Discrimination	2	2	2	1
Financial Strain	5	2	7	2
Maltreatment	24	15	19	8
Parenting Difficulties	22	12	18	14
Parental Mental Health	26	9	23	17
Parental Substance Use	2	1	0	0
Single Parent	16	9	19	9
Surrogate Care	10	1	9	8
Other Family Stress	15	9	18	11
Other	1	1	1	0
Total	148	76	137	80

exploratory moderator variables were significant (see Supplemental Table 1). Finally, neither the post hoc analysis examining threat vs. deprivation as a moderator in the subset of studies that included threat or deprivation effect sizes ($n = 9$), $F(1, 7) = 0.279$, $p = 0.613$, nor the post hoc analysis examining study methodological quality, $F(1, 74) = 1.979$, $p = 0.164$, were significant. (See Supplemental Materials for more detailed methodological quality analyses.)

Publication bias

Egger's regression test did not indicate significant funnel plot asymmetry, $z = -0.506$, $p = 0.613$ (see Figure 2).

Diurnal cortisol change

A total of 137 effect sizes from 71 distinct study samples were included in the meta-analysis examining childhood adversity and diurnal cortisol change levels. The sample sizes ranged from 15 to 2,088 with a median of 100. See Table 1 for details about the studies included and Table 2 for the number of effect sizes in each category of childhood adversity.

The overall effect of childhood adversity on diurnal cortisol change was not significant, $r = 0.017$, 95% CI [−0.010, 0.043], $t = 1.264$, $p = 0.215$ (see Supplemental Figure 3). One-sided log-likelihood-ratio tests indicated significant variation between, $\sigma^2 = 0.006$, $\chi^2(1) = 9.960$, $p = 0.001$, but not within, $\sigma^2 = 0.001$, $\chi^2(1) = 0.879$, $p = 0.174$, studies. The distribution of variance across levels was 47.77% at level 1 (sampling variance), 6.01% at level 2 (within-study variance) and 46.21% at level 3 (between-study variance), with an overall I^2 of 52.23%.

Moderators

None of the moderators related to primary aims yielded significant results: type of adversity, $F(7, 122) = 0.321$, $p = 0.943$; age at adversity, $F(4, 132) = 2.402$, $p = 0.053$; and age at cortisol collection, $F(1, 134) = 1.351$, $p = 0.247$. Because there was significant between-study variance, study-level variables were also examined as potential moderators. None of the primary or exploratory moderator variables were significant (see Supplemental Table 1). Finally, neither the post hoc analysis examining threat vs. deprivation as a moderator in the subset of studies that included threat or deprivation effect sizes ($n = 15$), $F(1, 13) = 0.072$, $p = 0.793$, nor the post hoc analysis examining study methodological quality, $F(1, 135) = 1.711$, $p = 0.362$, were significant. (See Supplemental Materials for more detailed methodological quality analyses.)

Publication bias

Egger's regression test did not indicate significant funnel plot asymmetry, $z = -0.021$, $p = 0.983$ (see Figure 2).

Bedtime levels

A total of 80 effect sizes from 42 distinct study samples were included in the meta-analysis examining childhood adversity and bedtime levels of cortisol. The sample sizes ranged from 26 to 580 with a median of 103.5. See Table 2 for the number of effect sizes in each category of childhood adversity and Table 1 for details about the studies included.

A significant effect was found for childhood adversity and bedtime levels of cortisol, $r = 0.047$, 95% CI [0.005, 0.089], $t = 2.231$, $p = 0.028$, indicating that children exposed to higher levels of adversity had higher bedtime cortisol levels than children exposed to lower levels of adversity (see Supplemental Figure 4). One-sided log-likelihood-ratio tests indicated significant variation between, $\sigma^2 = 0.012$, $\chi^2(1) = 20.727$, $p < .001$, but not within, $\sigma^2 < 0.001$, $\chi^2(1) < 0.001$, $p > .500$, studies. The distribution of variances was 39.09% at level 1 (sampling variance), <0.01% at level 2 (within-study variance) and 60.91% at level 3 (between-study variance), with an overall I^2 of 60.91%.

Moderators

None of the moderators related to primary aims yielded significant results: type of adversity, $F(6, 68) = 0.877$, $p = 0.516$; age at adversity, $F(3, 76) = 1.175$, $p = 0.325$; and age at cortisol collection, $F(1, 76) = 0.513$, $p = 0.476$. Given the significant between-study variance, study-level variables were also examined as potential moderators of the overall effect. None of the primary or exploratory moderator variables were significant (see Supplemental Table 1). Finally, neither the post hoc analysis examining threat vs. deprivation as a moderator in the subset of studies that included threat or deprivation effect sizes ($n = 9$), $F(1, 7) = 0.355$, $p = 0.570$, nor the post hoc analysis examining study methodological quality, $F(1, 78) = 0.090$, $p = 0.765$, were significant. (See Supplemental Materials for more detailed methodological quality analyses.)

Publication bias

Egger's regression test did not indicate significant funnel plot asymmetry, $z = 0.902$, $p = 0.367$ (see Figure 2). In addition, Rosenthal's (1979) fail-safe N indicated that 299 unpublished or yet-to-be-conducted studies with nonsignificant findings would be necessary to produce a null overall effect. Since this is above the cutoff of 220, the fail-safe N indicates that the effect is not likely to be due to publication bias alone.

Discussion

Meta-analytic findings

Within this meta-analysis, the only significant overall effect was the association between childhood adversity and higher bedtime

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Table 3. Additional information on moderator variables by study

Paper(s)	Data provided by authors	Multiple days of cortisol	Cortisol values transformed	Instructed to take sample at wake-up	Diurnal change samples at wake and bedtime
Albers et al., 2016	Yes	Yes	Yes	N/A	No
Alink et al., 2012; Cicchetti & Rogosch, 2007; Cicchetti et al., 2010, 2011; Granger et al., 2007; Murray-Close et al., 2008	Yes	Yes	Yes	N/A	No
Ashman et al., 2002	No	No	Yes	No	N/A
Badanes et al., 2011; Miles et al., 2018	Yes	Yes	No	N/A	No
Basu et al., 2013	No	No	Yes	Yes	N/A
Beijers et al., 2020; Evans et al., 2020; Simons et al., 2015	Yes	Yes	No	Yes	Yes
Berger et al., 2017	Yes	Yes	No	Yes	Yes
Bernard et al., 2010; Dozier et al., 2006	Yes	Yes	Yes	Yes	Yes
Boyer & Nelson, 2015; Roisman et al., 2009	No	Yes	No	Yes	N/A
Braehler et al., 2005[a]	No	No	No	No	N/A
Brendgen et al., 2017; Ouellet-Morin et al., 2009	Yes	Yes	Yes	Yes	Yes
Brewer-Smyth et al., 2004	No	No	No	Yes	No
Brummelte et al., 2015	Yes	Yes	Yes	No	Yes
Butler et al., 2017	No	Yes	No	Yes	N/A
Carrion et al., 2010; Taylor et al., 2009	No	Yes	No	N/A	N/A
Chen et al., 2017	No	Yes	No	Yes	N/A
Chernego et al., 2019	Yes	Yes	Yes	No	Yes
Chiang et al., 2016; Huynh et al., 2016	Yes	Yes	Yes	Yes	Yes
Cicchetti & Rogosch, 2001a; Cicchetti & Rogosch, 2001b	Yes	Yes	Yes	N/A	No
Cima et al., 2008	No	No	Yes	N/A	No
Clearfield et al., 2014	Yes	No	No	No	Yes
Clowtis et al., 2016	No	No	Yes	N/A	N/A
Cordero et al., 2017	No	No	No	No	N/A
Cullen et al., 2014	Yes	No	No	Yes	Yes
DeCaro, & Worthman, 2008	No	Yes	No	N/A	N/A
Donoho et al., 2011	No	No	No	N/A	N/A
Doom et al., 2013; Rogosch et al., 2011	Yes	Yes	Yes	N/A	No
Doom et al., 2018; Elhassan et al., 2015; Lumeng et al., 2014; Miller et al., 2017	Yes	Yes	Yes	N/A	No
Dougherty et al., 2009, 2013	Yes	Yes	Yes	No	Yes
Ellenbogen & Hodgins, 2009; Ellenbogen et al., 2004, 2006; Ostiguy et al., 2011	Yes	Yes	No	Yes	Yes
Engert et al., 2011	No	Yes	Yes	Yes	N/A
Epstein et al., 2019	Yes	Yes	No	Yes	Yes
Essex et al., 2011	No	Yes	Yes	N/A	Yes
Evans et al., 2013	No	No	Yes	Yes	N/A
Evans et al., 2020	Yes	No	No	Yes	Yes
Farrell et al., 2018	Yes	No	Yes	Yes	Yes
Fisher et al., 2007	No	Yes	Yes	No	Yes
Flom et al., 2017; St. John et al., 2017; Tarullo et al., 2017	Yes	Yes	Yes	Yes	Yes
Foland-Ross et al., 2014; LeMoult, Chen, et al., 2015; LeMoult, Ordaz, et al., 2015	Yes	Yes	No	Yes	Yes
Fuchs et al., 2017	Yes	Yes	No	Yes	Yes
Gartstein et al., 2018; Lengua et al., 2013; Thompson et al., 2018; Zalewski et al., 2012, 2016	Yes	Yes	Yes	No	Yes

(*Continued*)

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Table 3. (*Continued*)

Paper(s)	Data provided by authors	Multiple days of cortisol	Cortisol values transformed	Instructed to take sample at wake-up	Diurnal change samples at wake and bedtime
Gerritsen et al., 2017; Holleman et al., 2012; Vreeburg et al., 2010	Yes	No	No	Yes	Yes
Goldstein et al., 2017; Liu et al., 2016	Yes	Yes	No	Yes	Yes
Gunnar et al., 2001	Yes	Yes	Yes	No	Yes
Habersaat et al., 2014	No	Yes	Yes	N/A	Yes
Halligan et al., 2004; Murray et al., 2010	Yes	Yes	No	N/A	No
Hanson & Chen, 2010	Yes	Yes	Yes	N/A	No
Harris et al., 2017	No	No	Yes	Yes	Yes
Harvey et al., 2018	Yes	Yes	Yes	Yes	Yes
Hibel et al., 2019, 2020; Valentino et al., 2015	Yes	Yes	Yes	Yes	No
Huizink et al., 2009; Laceulle et al., 2017; Marsman et al., 2012; Zandstra et al., 2015	Yes	No	Some	Yes	N/A
Hustedt et al., 2017	Yes	Yes	No	No	Yes
Johnson et al., 2011	Yes	Yes	No	No	Yes
Johnson & Tottenham, 2015	Yes	Yes	Yes	Yes	N/A
Keeshin et al., 2014	No	Yes	Some	Yes	No
Kertes et al., 2008	Yes	Yes	No	No	Yes
Kiel et al., 2015	Yes	Yes	Yes	N/A	No
King et al., 2017	Yes	Yes	No	Yes	Yes
Kliewer et al., 2009; Kliewer, 2006	Yes	No	No	Yes	No
Kohrt et al., 2015	No	Yes	Yes	Yes	No
Korpa et al., 2017	No	No	No	Yes	
Koss et al., 2014, 2016; Perry et al., 2019; Pitula et al., 2019	Yes	Yes	No	No	Yes
Kuhlman, Geiss, et al., 2015; Kuhlman, Vargas, et al., 2015	Yes	Yes	Yes	Yes	Yes
Kumsta et al., 2017	Yes	Yes	Yes	Yes	Yes
Kwak et al., 2017	Yes	Yes	No	Yes	No
Laurent, Leve, Neiderhiser, Natsuaki, Shaw, Fisher, et al., 2013; Laurent et al., 2013, 2014; Marceau et al., 2013	Yes	Yes	No	No	Yes
Leneman et al., 2018	Yes	Yes	No	Yes	Yes
Leppert et al., 2018; Merwin et al., 2018; Merwin, 2017; Merwin, Leppert, et al., 2017; Merwin, Smith, et al., 2017	Yes	Yes	Yes	Yes	Yes
Letourneau et al., 2011	Yes	No	No	No	Yes
Lovallo et al., 2019	Yes	Yes	Yes	Yes	Yes
Mangold et al., 2010	No	Yes	Yes	N/A	N/A
McLachlan et al., 2016	No	Yes	Some	No	Yes
Meinlschmidt & Heim, 2005	No	No	No	N/A	N/A
Messerli-Burgy et al., 2018	Yes	Yes	Yes	Yes	Yes
Miller et al., 2018; Miller, Margolin, et al., 2017	Yes	Yes	No	Yes	Yes
Monteleone et al., 2015	Yes	No	No	Yes	N/A
O'Connor et al., 2005	No	Yes	No	Yes	N/A
O'Loughlin et al., 2020	Yes	Yes	No	Yes	No
Pawluski et al., 2012	Yes	No	Yes	N/A	Yes
Pendry & Adam, 2007	No	Yes	Yes	Yes	Yes
Peng et al., 2014	No	Yes	No	N/A	N/A
Philbrook & Teti, 2016	No	No	Yes	Yes	N/A

(*Continued*)

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Table 3. (*Continued*)

Paper(s)	Data provided by authors	Multiple days of cortisol	Cortisol values transformed	Instructed to take sample at wake-up	Diurnal change samples at wake and bedtime
Plant et al., 2016	Yes	No	No	Yes	N/A
Pruessner et al., 2013	Yes	No	No	Yes	N/A
Puetz et al., 2016	No	Yes	No	No	Yes
Quevedo et al., 2017	No	Yes	Yes	Yes	No
Raffington, Prindle, et al., 2018	Yes	Yes	Yes	Yes	N/A
Raffington, Schmiedek, et al., 2018	Yes	Yes	Yes	Yes	Yes
Reichl et al., 2016	Yes	Yes	No	Yes	Yes
Russ et al., 2012	No	Yes	Some	No	Yes
Seidenfaden et al., 2017	Yes	No	No	Yes	Yes
Simmons et al., 2015	No	Yes	Some	Yes	No
Smeets et al., 2007	Yes	Yes	No	Yes	Yes
Starr et al., 2017	Yes	Yes	No	Yes	Yes
Theall et al., 2017	Yes	Yes	Yes	Yes	Yes
Van den Bergh et al., 2008	Yes	No	Yes	Yes	Yes
van der Vegt et al., 2009	No	No	No	Yes	No
Vänskä et al., 2016	Yes	No	No	Yes	Yes
Weissbecker et al., 2006	No	Yes	Yes	N/A	Yes
Wielaard et al., 2018	No	Yes	No	Yes	No
Yehuda et al., 2005	No	No	Yes	Yes	N/A
Zalewski, Lengua, Fisher et al., 2012	Yes	Yes	No	No	Yes
Zeiders et al., 2012	No	Yes	Yes	Yes	Yes
Zhu et al., 2019	Yes	No	Yes	Yes	N/A

Note. This table includes information on moderator variables not already provided in Table 1. Papers are grouped together based on study. If effect sizes were pulled from a specific paper, that paper is included. If authors provided data for a study, all papers related to that study are included. Papers are listed in alphabetical order.

cortisol levels, which could not be accounted for by publication bias alone. The lack of significant overall effects for wake, the CAR and diurnal cortisol change is consistent with previous meta-analyses that have not yielded significant overall associations between childhood adversities of early life stress, maltreatment and parenting and components of the diurnal cortisol pattern (Bernard et al., 2017; Fogelman & Canli, 2018; Hackman et al., 2018). However, the significant association between childhood adversity and bedtime cortisol levels is a novel meta-analytic contribution that suggests those who have experienced childhood adversity have more difficulty downregulating cortisol production as the day ends.

Understanding the magnitude of this effect is challenging as the current literature does not provide clear guidelines as to what magnitude of change in diurnal cortisol regulation is associated with clinically significant outcomes. Although this effect would be categorized as "small" by Cohen's benchmarks, Cohen also emphasized the necessity of considering the research area and methodology when examining the magnitude of effect sizes (Cohen, 1988). This meta-analysis examines bedtime cortisol values on a small sample of days. Though the effect of childhood adversity on cortisol levels measured at a single bedtime (or small sample of bedtimes) is clearly small, the cumulative effect over thousands of bedtimes (365 each year) may be consequential (see Funder & Ozer, 2019). Future studies examining the relationship between diurnal cortisol effect sizes and the magnitude of corresponding clinical impacts, especially cumulatively, will enhance our understanding of the significance of the overall effect presented here.

Despite significant heterogeneity within and between studies, no moderation effects were identified. This heterogeneity may be explained by substantial variability in methods utilized to assess diurnal cortisol, including timing of samples collected, number of collection days, use of adherence protocols, methods for data cleaning/preparation and calculation of outcome measures, which may contribute to mixed results. Heterogeneity in diurnal cortisol methodology has been found within randomized controlled trials that include diurnal cortisol as an outcome, highlighting the need for more consistent methods (Ryan et al., 2016). We attempted to capture this methodological heterogeneity by examining multiple moderators related to methods. As can be seen in Table 4, there was substantial variability across most methodological moderators examined herein; however, these moderators do not provide an indicator as to which methodological characteristics may be of particular importance as none were significant.

Findings in the context of current theoretical models

Interpreting these results within the context of the current literature on diurnal cortisol regulation may add insight into these

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

Table 4. Moderator variable descriptive statistics

	Diurnal Cortisol Measure			
Moderator Variable	Wake	CAR	Diurnal Change	Bed
Mean age at time of cortisol collection	*M* = 13.59 *SD* = 13.88	*M* = 18.00 *SD* = 15.47	*M* = 12.26 *SD* = 13.22	*M* = 8.38 *SD* = 7.18
Age at time of adversity	12 Infant 21 Early childhood 8 Middle childhood 7 Adolescent 97 Multiple ages	1 Infant 8 Early childhood 6 Middle childhood 6 Adolescent 54 Multiple ages	13 Infant 19 Early childhood 5 Middle childhood 8 Adolescent 89 Multiple ages	9 Infant 19 Early childhood 0 Middle childhood 3 Adolescent 46 Multiple ages
Percent of sample identified as female	*M* = 55.23 *SD* = 16.59	*M* = 59.85 *SD* = 20.06	*M* = 55.85 *SD* = 15.72	*M* = 52.69 *SD* = 13.47
Percent of sample with racial/ethnic minority status (all studies)	*M* = 42.02 *SD* = 31.37	*M* = 49.04 *SD* = 37.68	*M* = 43.62 *SD* = 31.15	*M* = 42.87 *SD* = 27.37
Percent of sample with racial/ethnic minority status (U.S. only)	*M* = 53.38 *SD* = 29.51	*M* = 65.52 *SD* = 33.69	*M* = 52.00 *SD* = 28.62	*M* = 48.21 *SD* = 25.77
Cortisol collected over multiple days	73.6%	72.4%	85.4%	82.5%
Cortisol data transformed	46.6%	36.8%	53.3%	63.7%
Instructed to take wake immediately upon awakening	73.0%	N/A	N/A	N/A
Diurnal change calculated using wake and bedtime samples	N/A	N/A	75.9%	N/A
Study quality	*M* = 5.03 *SD* = 1.88	*M* = 5.25 *SD* = 1.77	*M* = 5.09 *SD* = 1.72	*M* = 4.91 *SD* = 1.86
Included data received from authors	74.3%	76.3%	84.7%	73.8%
Publication year	*M* = 2014.34 *SD* = 4.05	*M* = 2014.70 *SD* = 3.79	*M* = 2014.11 *SD* = 4.21	*M* = 2014.35 *SD* = 4.07
Threat versus deprivation	9 Threat 9 Deprivation	7 Threat 2 Deprivation	7 Threat 8 Deprivation	3 Threat 6 Deprivation

meta-analytic results. Many theoretical models, including the cumulative adversity, biological embedding and three-hit models, suggest that the impact of childhood adversity on diurnal cortisol regulation may change over time. This is consistent with the concept of adaptive calibration (Del Giudice et al., 2011), in which the body adapts in response to stressors and changes in the environment to maintain stability, such as by producing an initial increase in cortisol production (hypercortisolism) in response to an adversity at onset but shifting to a blunted response (hypocortisolism) with prolonged adversity duration. Thus, any attempt to capture overall associations between childhood adversity and diurnal cortisol regulation may include effects that differ in magnitude and direction based on the adversity's timing of onset, duration, chronicity, current presence or absence and time since initial onset.

One implication of this is that the associations between childhood adversity and diurnal cortisol regulation may not be linear. Previous studies have provided evidence for such potential nonlinear relationships. For example, the study by Zalewski et al. (2016) examining associations between cumulative risk and diurnal cortisol among preschool-age children found that both high and low levels of cumulative risk were associated with lower morning levels of cortisol and more blunted cortisol slopes compared to moderate levels of cumulative risk, suggesting that the association between amount of adversity and diurnal cortisol response may not be linear. Similarly, studies have indicated that the association between childhood adversity and diurnal cortisol regulation may not be linear across development, such as a study by VanTieghem et al. (2021) that found morning cortisol levels in previously institutionalized children shift across development from blunted during childhood to heightened in adolescence. As a result, understanding the associations between childhood adversity and diurnal cortisol may require analytic methods that examine possible nonlinear relationships that could not be captured in the present meta-analysis.

An additional implication is that including elements of adversity timing is crucial to understanding childhood adversity's impact on diurnal cortisol regulation. Although the current meta-analysis aimed to capture aspects of childhood adversity timing, these attempts were limited by information available in the current literature. Studies do not consistently include information on the onset, duration, chronicity and current presence (except when childhood adversity and diurnal cortisol are measured concurrently) of childhood adversities, and few studies examine these associations longitudinally. As a result, we were restricted to coding timing of adversity as the developmental period for which the adversity was being assessed (e.g., infancy, adolescence, multiple periods). This methodological approach carried significant limitations, as it only provides information on whether the adversity was present during a particular developmental window and does not provide information on the adversity's duration or presence before or after that developmental period. In addition, most studies examined childhood adversity across multiple developmental periods (e.g., at any point in childhood). Similarly, time elapsed between experiencing childhood adversity and assessing diurnal cortisol is not consistently reported across studies. This meta-analysis examined mean age at time of cortisol assessment as a potential moderator since it is plausible that older

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

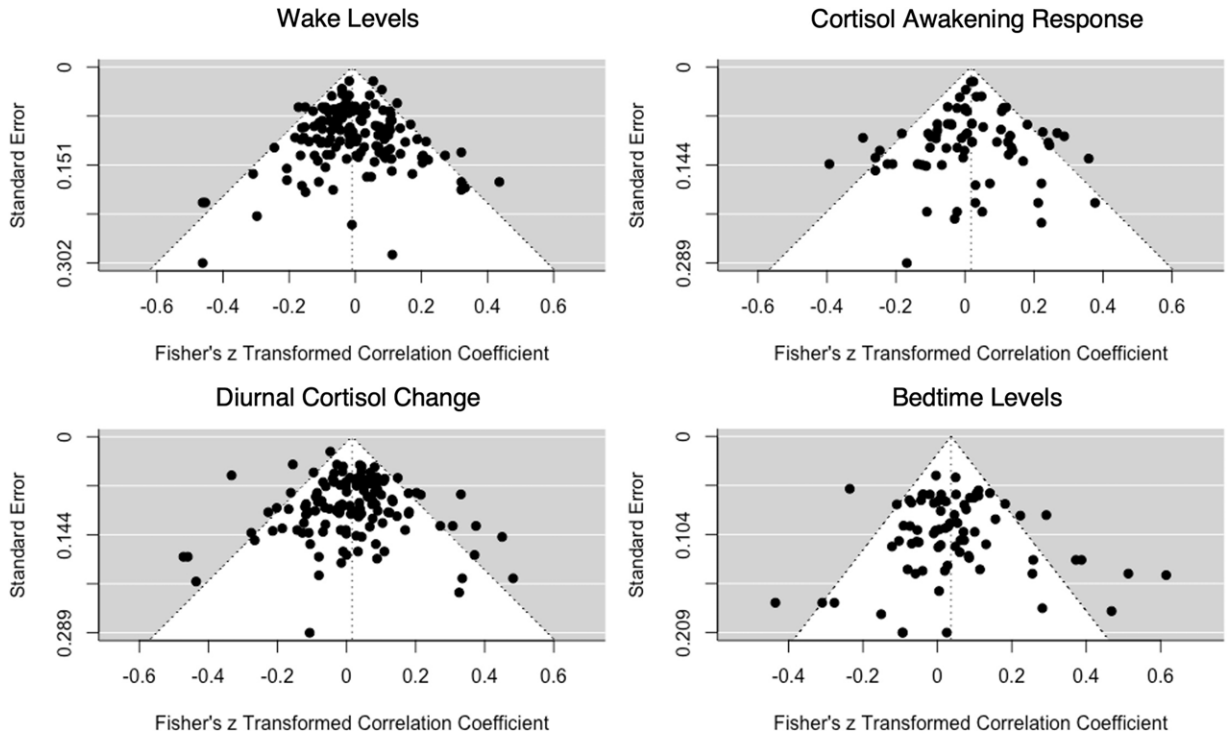

Figure 2. Funnel Plots with Trim and Fill. Note. In each figure, the shaded area represents significant effect sizes ($p < .05$). Black circles represent coded effect sizes. White circles represent effect sizes added by trim and fill analysis.

samples, especially those including adults, have more time elapsed since childhood adversity onset than younger samples. However, this measure is neither consistent nor precise and therefore provides limited insight. Of note, the feasibility of examining early adversity within the context of sensitive period models has been questioned given the complexity of identifying the nature, timing and duration of early adversities (which are often overlapping) and the possibility that some adversities may impact neurobiology through experience-dependent mechanisms (Gabard-Durnam & McLaughlin, 2019). Thus, the potential impact of adversity's timing on HPA axis regulation remains a complex question requiring further investigation.

Additionally, a variety of adversity characteristics have been proposed to impact associations between adversity and HPA axis regulation, including traumatic nature, threat to physical self, uncontrollability, elicitation of emotions such as shame or loss, and whether the impact is threat or deprivation (McLaughlin & Sheridan, 2016; Miller et al., 2007). These characteristics are not consistently reported across studies. Although the present study examined category of adversity as a moderator, these categories were broad and likely contained significant heterogeneity in type, intensity and duration of stressor, which may have obscured effects. Furthermore, measures of adversity sometimes fit into multiple categories. For example, low levels of conflict at home is an example of family stress, but high levels of conflict at home may take the form of domestic violence, which is both a family stressor and a form of maltreatment. Because many measures of family conflict within this meta-analysis included a range of behaviors that could not be considered exclusively domestic violence, we categorized family conflict under other family stress. However, two studies categorized as other family stress did focus specifically on measures of domestic violence (i.e., Hibel et al., 2020; Theall et al., 2017) and therefore would also have been a good fit for the maltreatment category. Similarly, the category of surrogate care includes both foster care or institutionalization and adoption, which may convey different experiences of caregiving and different levels of adversity. (Of note, all children included in the surrogate care category experienced foster care, institutional care and/or maltreatment resulting in separation from biological parents with the possible exception of a subset of Gunnar et al. (2001) sample who were adopted so early they had not yet been placed in orphanages.) We attempted to place adversities in the categories with which they would match most consistently, but these instances of overlap mean that categories of adversity are not fully independent.

A further limitation of this approach is the likelihood that some of these categories serve as proxies for childhood adversity but do not capture true "deviation from the expectable environment" (McLaughlin et al., 2019). A better way to assess childhood adversity, as suggested by McLaughlin (2016), may be to include only events that result in deviations from expected caregiving or other significant adversity for the child. Although we could not make this distinction within the present meta-analysis based on the information consistently available in manuscripts, future studies that consider whether potential adversities constitute significant deviations from the expectable environment will likely enhance our understanding of the impact of early adversity. In addition, it is important to note that intervention efforts should be aimed at addressing sources of adversity directly (e.g., increasing financial and social support for overburdened parents) rather than the proxies for adversity (e.g., incentivizing single parents to be married).

Although we attempted to include a broad range of childhood adversity, other forms of adversity are likely missing. In particular,

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

there was a lack of studies directly examining the impact of systemic racism and discrimination experienced during childhood on diurnal cortisol regulation. Racial and ethnic health disparities have been theorized to stem from increased allostatic load, to which systemic racism and discrimination are likely contributors (Carlson & Chamberlain, 2005). Understanding associations between experiences of discrimination and diurnal cortisol regulation could help explain racial/ethnic differences in diurnal cortisol patterns (e.g., DeSantis et al., 2007; Martin et al., 2012) and provide insight into pathways through which discrimination may impact health and well-being. Some studies have already provided preliminary evidence in identifying associations between experiences of discrimination and blunted diurnal slopes among individuals with a racial/ethnic minority status (Adam et al., 2015; Zeiders et al., 2014). In addition, it is essential to examine the impact of discrimination on minoritized youth more broadly, such as those with sexual and gender minority identities (Williams & Mann, 2017). In one example, experience of greater LGBT stressors throughout the week was associated with elevated cortisol at awakening and 45 minutes after awakening in young adults (Figueroa et al., 2021). Future studies further examining the impact of discrimination on diurnal cortisol patterns among youth with minoritized identities may increase our understanding of pathways contributing to alteration in the HPA axis and possible health disparities.

Finally, the random selection of a single measure of adversity for each category of childhood adversity from each study in the present meta-analysis meant that some effect sizes were excluded due to the study design; however, the use of random selection and the large number of effect sizes included likely provide a representative sample. To supplement our analyses utilizing broad adversity categories, we also attempted to examine whether categorization of adversity as threat or deprivation was a significant moderator of overall effect sizes in post hoc analyses. However, these analyses were limited by the small number of effect sizes included as a result of our retrospective approach to identifying threat and deprivation and of the difficulty of distinguishing between threat and deprivation given that many measures of adversity assessed both threat and deprivation together. As a result, we were likely underpowered to detect significant moderation effects related to threat vs. deprivation. Overall, a more nuanced examination of the characteristics of adversity that was beyond the scope of this meta-analysis may be required to understand complex associations between childhood adversity and diurnal cortisol regulation in future studies.

Strengths and limitations

Several methodological decisions strengthened our ability to capture a broad overall picture of the association between childhood adversity and diurnal cortisol. First, we defined childhood adversity broadly, including a wide range of adversity categories. Further, we utilized multilevel meta-analysis techniques, allowing the inclusion of multiple effects from the same study while accounting for interdependence of effects.

This meta-analysis also included several limitations in addition to those related to assessment of childhood adversity timing and nature discussed above. First, although the use of simple difference scores to calculate diurnal cortisol change and the CAR allowed us to include findings across a wide range of studies, these measures did not reflect change over time. Given the importance of timing to diurnal cortisol patterns, this may have resulted in inconsistencies in the included cortisol measures. In particular, timing is of critical importance to the accurate measurement of the CAR, and current recommendations include repeated sampling across the period after awakening, particularly at wake-up and from 30 to 45 minutes after awakening, as well as objective monitoring of participant adherence (Stalder et al., 2016). This meta-analysis relied on authors' determinations of timing for their measure of the CAR and therefore did not restrict inclusion of measures of the CAR based on sample timing. As a result, variability in the timing of the CAR measurements included herein may be creating additional noise in the data that could obscure true effects.

Similarly, diurnal cortisol change was included if cortisol samples from at least two time points (one at awakening or in the morning and one in the afternoon, evening, or at bedtime) were available to include the wide range of the effect sizes available in the present literature while also capturing variability across the waking day. However, it is important to note that there are numerous approaches for measuring changes in cortisol across the day, including wake to bedtime, peak to bedtime and morning to afternoon among others. The true effect for diurnal cortisol change may be obscured by the range of methodologies of the studies included in this meta-analysis. In a preliminary exploration of this possibility, whether diurnal cortisol change samples were collected at awakening and bedtime was included as a moderator and was not significant; however, the design of this meta-analysis precluded more nuanced examination of differences in diurnal cortisol change measurement. Furthermore, more comprehensive measures of change in cortisol throughout the day such as diurnal cortisol slope may provide a clearer picture of the association between childhood adversity and diurnal cortisol patterns. Examination of cortisol collection methodology suggests the diurnal cortisol slope is well approximated by methods that include fixed samples at awakening and bedtime as well as at three additional points in the day, providing guidance for future studies (Hoyt et al., 2016). In addition, we did not examine other biomarkers or genetic factors that may contribute to an individual's response to stress and interact with the diurnal cortisol pattern.

Another limitation is that our examination of racial/ethnic minority status as a study-level moderator may be impacted by differences in the experiences of those with racial/ethnic minority statuses that likely vary within and across countries. Although we attempted to explore this by examining this moderator in the subset of studies specific to United States, this moderator may be capturing diverse experiences even within a single country.

Finally, our measure of study quality was limited by the information reported on methodology within each manuscript. It is possible that some studies did account for covariates included within this measure that were not reported in their manuscripts, resulting in a lower quality score and adding noise to this measure of study quality. In addition, this measure weights every covariate equally and does not account for variability in the rigor of methods used to account for these covariates. Furthermore, although our post hoc analyses examined whether biobehavioral variables were accounted for by authors in the original study (i.e., included as a covariate, used as an exclusion criterion, or examined in follow-up analyses), they do not reflect whether these variables were accounted for as covariates in the effect sizes reported in this meta-analysis. As a result, although the majority of the post hoc moderation analyses examining study methodological quality and individual biobehavioral variables presented here were not significant, the potential noise within these variables in

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

combination with the extensive previous literature indicating the importance of these variables prevents us from concluding that they are not relevant to cortisol analyses related to early adversity. Both consistent use of rigorous and high-quality methodological approaches and consistent reporting of such measures taken will be important for future studies.

Implications and future directions

In the present meta-analysis, the association between childhood adversity and bedtime cortisol levels emerged as the only significant association. This finding indicates that measuring bedtime cortisol levels may provide an important opportunity to capture the impact of early adversity on diurnal cortisol regulation in future studies. Clinically, developing interventions that ameliorate the negative impact of childhood adversity on bedtime cortisol levels is also important.

A consistent result throughout this meta-analysis was the lack of significant overall and moderation effects. Given that current theoretical models emphasize the importance of the timing and nature of childhood adversity, the lack of significant findings may in part result from the nuanced and complex nature of associations between adversity and HPA axis regulation. Many of these nuances were unable to be examined on a meta-analytic level, in part because they are not consistently examined or reported in the current literature. As a result, this meta-analysis highlights the importance of capturing specificity in the timing and nature of childhood adversity when examining associations with diurnal cortisol. When possible and relevant, future studies should make efforts to assess and report:

1. Age of onset of adversity
2. Ages at which adversity occurred
3. Duration/chronicity of adversity
4. Whether the adversity is concurrent
5. Time between adversity onset/termination and diurnal cortisol measurement
6. Characteristics of adversity, including whether it was traumatic, threatened physical integrity and involved deprivation or threat
7. Participants' perceptions of adversity, including whether it was uncontrollable, whether it elicited emotions such as shame or loss, and its intensity

Furthermore, when considering age it may be important not only to consider chronological age but also to consider pubertal status since puberty may be an important developmental window for HPA axis functioning. Although it likely will not be feasible to assess each of these characteristics in every study, moving toward greater inclusion of these factors and considering the implications when they are unknown will likely strengthen the literature.

In addition to these recommendations specific to examining childhood adversity, it is of critical importance that studies continue to follow methodological guidelines for the accurate assessment of diurnal cortisol (e.g., Hoyt et al., 2016; Stalder et al., 2016). Given the importance of timing to the accurate assessment of diurnal cortisol, collecting wake samples immediately upon awakening and utilizing objective monitoring of sample timing (e.g., with MEMS caps) are necessary for consistent and accurate measurements of the diurnal cortisol pattern. Of note, instructions to take samples immediately upon awakening were included in the study description for only 72.79% of wake level effect sizes in the present meta-analysis, and objective monitoring of adherence to sample timing was included in 18.37% of studies included in this meta-analysis, suggesting that inconsistencies in diurnal cortisol measurement may be a methodological limitation of the literature broadly. Furthermore, it is important to account for covariates related to the day of sampling (e.g., sleep quality, weekday vs. weekend) and traits of the individual (e.g., age, sex, contraception use) that may impact diurnal cortisol production, as well as to consider possible exclusion criteria for factors that cannot sufficiently be controlled (Stalder et al., 2016). Consistent adherence to methodological recommendations in future diurnal cortisol studies will reduce potential noise that may interfere with identifying true effects.

In addition, it is important to note that diurnal cortisol patterns also reflect individual differences and day-to-day variability. As a result, person-centered approaches to examining individual diurnal cortisol profiles (e.g., Hoyt et al., 2021), particularly longitudinally, may provide additional insight. Furthermore, longitudinal examinations of associations between childhood adversity and diurnal cortisol will be crucial to our ability to understand the impact of timing, intensity, chronicity and type of adversity on diurnal cortisol regulation.

Finally, this meta-analysis only examined diurnal cortisol as defined as cortisol levels at awakening or bedtime, the CAR, or diurnal cortisol change and did not capture measures of overall cortisol output (e.g., area under the curve), cumulative measures of cortisol (e.g., hair cortisol), or cortisol reactivity, which may provide additional insight into the impact of childhood adversity on HPA axis functioning. As discussed earlier, nonsignificant, significant positive and significant negative effects have all been found for associations between childhood adversity and hair cortisol levels (Bryson et al., 2021; Grant & Meyer, 2021; Khoury et al., 2019) or cortisol reactivity (Bunea et al., 2017; Hakamata et al., 2022; Hosseini-Kamkar et al., 2021; Hunter et al., 2011; Lai et al., 2021), suggesting many complexities remain to be untangled related to childhood adversity's impact on HPA axis functioning. As a result, future studies examining these additional cortisol measures may be important to our understanding of childhood adversity and stress regulation.

Conclusions

In summary, the present meta-analysis found a significant association between childhood adversity and higher bedtime cortisol levels, with no significant moderation effects. These findings highlight that childhood adversity may particularly impact the ability to downregulate cortisol levels throughout the day, resulting in higher bedtime levels. In contrast, the overall effects for childhood adversity and other measures of diurnal cortisol (e.g., morning levels, the CAR and diurnal cortisol changes) were not significant. Given the limitations discussed above, it is not yet clear whether these null effects result from the complexity of these relationships or are a true reflection of overall associations. Associations between childhood adversity and diurnal cortisol may be too complex to capture with studies, including the present meta-analysis, that examine adversity broadly defined and diurnal cortisol regulation measured across developmental stages at varying lengths from the onset of adversity. As it is possible that the lack of stronger effects resulted from variability in the timing and characteristics of childhood adversity assessed, future longitudinal studies utilizing consistent rigorous methods and adversity assessment that allow for the possibility of nonlinear relationships will be necessary to clarify

https://doi.org/10.1017/S0954579423000561 Published online by Cambridge University Press

possible nuances in the relation between childhood adversity and diurnal cortisol. Such studies have great potential for increasing our understanding of the likely complex impact of childhood adversity on diurnal cortisol regulation.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579423000561.

Acknowledgements. We would like to thank Christopher Facompré for providing consultation, resources and advice in preparing a meta-analysis.

Funding statement. This work was supported by the National Science Foundation Graduate Research Fellowship (LP and DT, grant number 1315232).

Competing interests. None.

References

Adam, E. K., Heissel, J. A., Zeiders, K. H., Richeson, J. A., Ross, E. C., Ehrlich, K. B., Levy, D. J., Kemeny, M., Brodish, A. B., Malanchuk, O., Peck, S. C., Fuller-Rowell, T. E., Eccles, J. S. (2015). Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: A 20-year prospective study. *Psychoneuroendocrinology*, *62*, 279–291, https://doi.org/10.1016/j.psyneuen.2015.08.018,

Adam, E. K., Quinn, M. E., Tavernier, R., McQuillan, M. T., Dahlke, K. A., & Gilbert, K. E. (2017). Diurnal cortisol slopes and mental and physical health outcomes: A systematic review and meta-analysis. *Psychoneuroendocrinology*, *83*, 25–41. https://doi.org/10.1016/j.psyneuen.2017.05.018

Albers, E. M., Beijers, R., Riksen-Walraven, J. M., Sweep, F. C., & de Weerth, C. (2016). Cortisol levels of infants in center care across the first year of life: Links with quality of care and infant temperament. *Stress-the International Journal on The Biology of Stress*, *19*(1), 8–17. https://doi.org/10.3109/10253890.2015.1089230

Alink, L. R., Cicchetti, D., Kim, J., & Rogosch, F. A. (2012). Longitudinal associations among child maltreatment, social functioning, and cortisol regulation. *Developmental Psychology*, *48*(1), 224–236. https://doi.org/10.1037/a0024892

Ashman, S. B., Dawson, G., Panagiotides, H., Yamada, E., & Wilkinson, C. W. (2002). Stress hormone levels of children of depressed mothers. *Development and Psychopathology*, *14*(2), 333–349. https://doi.org/10.1017/s0954579402002080

Assink, M., & Wibbelink, C. J. (2016). Fitting three-level meta-analytic models in R: A step-by-step tutorial. *The Quantitative Methods for Psychology*, *12*(3), 154–174. https://doi.org/10.20982/tqmp.12.3.p154

Atkinson, L., Beitchman, J., Gonzalez, A., Young, A., Wilson, B., Escobar, M., Chisholm, V., Brownlie, E., Khoury, J. E., Ludmer, J., Villani, V., Eapen, V. (2015). Cumulative risk, cumulative outcome: A 20-year longitudinal study. *PloS One*, *10*(6), e0127650, https://doi.org/10.1371/journal.pone.0127650,

Badanes, L. S., Dmitrieva, J., & Watamura, S. E. (2012). Understanding cortisol reactivity across the day at child care: The potential buffering role of secure attachments to caregivers. *Early Childhood Research Quarterly*, *27*(1), 156–165. https://doi.org/10.1016/j.ecresq.2011.05.005

Badanes, L. S., Watamura, S. E., & Hankin, B. L. (2011). Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders. *Development and Psychopathology*, *23*(3), 881–896. https://doi.org/10.1017/S095457941100037X

Basu, A., Levendosky, A. A., & Lonstein, J. S. (2013). Trauma sequelae and cortisol levels in women exposed to intimate partner violence. *Psychodynamic Psychiatry*, *41*(2), 247–275. https://doi.org/10.1521/pdps.2013.41.2.247

Beijers, R., Daehn, D., Shalev, I., Belsky, J., & de Weerth, C. (2020). Biological embedding of maternal postpartum depressive symptoms: The potential role of cortisol and telomere length. *Biological Psychology*, *150*, 107809. https://doi.org/10.1016/j.biopsycho.2019.107809

Berger, M., Leicht, A., Slatcher, A., Kraeuter, A. K., Ketheesan, S., Larkins, S., & Sarnyai, Z. (2017). Cortisol awakening response and acute stress reactivity in first nations people. *Scientific Reports*, *7*(1https://doi.org/10.1038/srep41760

Bernard, K., Butzin-Dozier, Z., Rittenhouse, J., & Dozier, M. (2010). Cortisol production patterns in young children living with birth parents vs children placed in foster care following involvement of Child Protective Services. *Archives of Pediatrics & Adolescent Medicine*, *164*(5), 438–443. https://doi.org/10.1001/archpediatrics.2010.54

Bernard, K., Frost, A., Bennett, C. B., & Lindhiem, O. (2017). Maltreatment and diurnal cortisol regulation: A meta-analysis. *Psychoneuroendocrinology*, *78*, 57–67. https://doi.org/10.1016/j.psyneuen.2017.01.005

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*(2), 271–301. https://doi.org/10.1017/S0954579405050145

Boyer, B. P., & Nelson, J. A. (2015). Longitudinal associations of childhood parenting and adolescent health: The mediating influence of social competence. *Child Development*, *86*(3), 828–843. https://doi.org/10.1111/cdev.12347

Braehler, C., Holowka, D., Brunet, A., Beaulieu, S., Baptista, T., DebruilleI, B., Walker, C., & King, S. (2005). Diurnal cortisol in schizophrenia patients with childhood trauma. *Schizophrenia Research*, *79*(2-3), 353–354, https://doi.org/10.1016/j.schres.2004.07.007,

Brendgen, M., Ouellet-Morin, I., Lupien, S. J., Vitaro, F., Dionne, G., & Boivin, M. (2017). Environmental influence of problematic social relationships on adolescents' daily cortisol secretion: A monozygotic twin-difference study. *Psychological Medicine*, *47*(3), 460–470. https://doi.org/10.1017/S003329171600252X

Brewer-Smyth, K., Burgess, A. W., & Shults, J. (2004). Physical and sexual abuse, salivary cortisol, and neurologic correlates of violent criminal behavior in female prison inmates. *Biological Psychiatry*, *55*(1), 21–31. https://doi.org/10.1016/S0006-3223(03)00705-4

Brindle, R. C., Pearson, A., & Ginty, A. T. (2022). Adverse childhood experiences (ACEs) relate to blunted cardiovascular and cortisol reactivity to acute laboratory stress: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *134*, 104530. https://doi.org/10.1016/j.neubiorev.2022.104530

Brummelte, S., Chau, C. M., Cepeda, I. L., Degenhardt, A., Weinberg, J., Synnes, A. R., & Grunau, R. E. (2015). Cortisol levels in former preterm children at school age are predicted by neonatal procedural pain-related stress. *Psychoneuroendocrinology*, *51*, 151–163. https://doi.org/10.1016/j.psyneuen.2014.09.018Get

Bryson, H. E., Price, A. M., Goldfeld, S., & Mensah, F. (2021). Associations between social adversity and young children's hair cortisol: A systematic review. *Psychoneuroendocrinology*, *127*, 105176. https://doi.org/10.1016/j.psyneuen.2021.105176

Bunea, I. M., Szentágotai-Tătar, A., & Miu, A. C. (2017). Early-life adversity and cortisol response to social stress: A meta-analysis. *Translational Psychiatry*, *7*(12https://doi.org/10.1038/s41398-017-0032-3

Butler, K., Klaus, K., Edwards, L., & Pennington, K. (2017). Elevated cortisol awakening response associated with early life stress and impaired executive function in healthy adult males. *Hormones and Behavior*, *95*, 13–21. https://doi.org/10.1016/j.yhbeh.2017.07.013

Callaghan, B. L., & Tottenham, N. (2016). The stress acceleration hypothesis: Effects of early-life adversity on emotion circuits and behavior. *Current Opinion in Behavioral Sciences*, *7*, 76–81. https://doi.org/10.1016/j.cobeha.2015.11.018

Carlson, E. D., & Chamberlain, R. M. (2005). Allostatic load and health disparities: A theoretical orientation. *Research in Nursing & Health*, *28*(4), 306–315. https://doi.org/10.1002/nur.20084

Carrion, V. G., Weems, C. F., Richert, K., Hoffman, B. C., & Reiss, A. L. (2010). Decreased prefrontal cortical volume associated with increased bedtime cortisol in traumatized youth. *Biological Psychiatry*, *68*(5), 491–493. https://doi.org/10.1016/j.biopsych.2010.05.010

Chen, F. R., Stroud, C. B., Vrshek-Schallhorn, S., Doane, L. D., & Granger, D. A. (2017). Individual differences in early adolescents' latent trait cortisol:

Interaction of early adversity and 5-HTTLPR. *Biological Psychology*, *129*, 8–15. https://doi.org/10.1016/j.biopsycho.2017.07.017

Chernego, D., Martin, C., Bernard, K., Muhamedrahimov, R., Gordon, M. K., & Dozier, M. (2019). Effects of institutional rearing on children's diurnal cortisol production. *Psychoneuroendocrinology*, *106*, 161–164. https://doi.org/10.1016/j.psyneuen.2019.04.010

Chiang, J. J., Tsai, K. M., Park, H., Bower, J. E., Almeida, D. M., Dahl, R. E., Irwin, M. R., Seeman, T. E., Fuligni, A. J. (2016). Daily family stress and HPA axis functioning during adolescence: The moderating role of sleep. *Psychoneuroendocrinology*, *71*, 43–53, https://doi.org/10.1016/j.psyneuen.2016.05.009,

Chida, Y., & Steptoe, A. (2009). Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. *Biological Psychology*, *80*(3), 265–278. https://doi.org/10.1016/j.biopsycho.2008.10.004

Cicchetti, D., & Rogosch, F. A. (2001a). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, *13*(3), 677–693. https://doi.org/10.1017/S0954579401003145

Cicchetti, D., & Rogosch, F. A. (2001b). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Development and Psychopathology*, *13*(4), 783–804. https://doi.org/10.1017/S0954579401004035

Cicchetti, D., & Rogosch, F. A. (2007). Personality, adrenal steroid hormones, and resilience in maltreated children: A multi-level perspective. *Development and Psychopathology*, *19*(3), 787–809. https://doi.org/10.1017/S0954579407000399

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81*(1), 252–269. https://doi.org/10.1111/j.1467-8624.2009.01393.x

Cicchetti, D., Rogosch, F. A., & Oshri, A. (2011). Interactive effects of corticotropin releasing hormone receptor 1, serotonin transporter linked polymorphic region, and child maltreatment on diurnal cortisol regulation and internalizing symptomatology. *Development and Psychopathology*, *23*(4), 1125–1138. https://doi.org/10.1017/S0954579411000599

Cima, M., Smeets, T., & Jelicic, M. (2008). Self-reported trauma, cortisol levels, and aggression in psychopathic and non-psychopathic prison inmates. *Biological Psychology*, *78*(1), 75–86. https://doi.org/10.1016/j.biopsycho.2007.12.011

Clearfield, M. W., Carter-Rodriguez, A., Merali, A. R., & Shober, R. (2014). The effects of SES on infant and maternal diurnal salivary cortisol output. *Infant Behavior and Development*, *37*(3), 298–304. https://doi.org/10.1016/j.infbeh.2014.04.008

Clowtis, L. M., Kang, D. H., Padhye, N. S., Rozmus, C., & Barratt, M. S. (2016). Biobehavioral factors in child health outcomes: The roles of maternal stress, maternal-child engagement, salivary cortisol, and salivary testosterone. *Nursing Research*, *65*(5), 340–351. https://doi.org/10.1097/NNR.0000000000000172

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd edn. Erlbaum.

Cordero, M. I., Moser, D. A., Manini, A., Suardi, F., Sancho-Rossignol, A., Torrisi, R., Rossier, M. F., Ansermet, Fçois, Dayer, A. G., Rusconi-Serpa, S., Schechter, D. S. (2017). Effects of interpersonal violence-related post-traumatic stress disorder (PTSD) on mother and child diurnal cortisol rhythm and cortisol reactivity to a laboratory stressor involving separation. *Hormones and Behavior*, *90*, 15–24, https://doi.org/10.1016/j.yhbeh.2017.02.007

Cullen, A. E., Zunszain, P. A., Dickson, H., Roberts, R. E., Fisher, H. L., Pariante, C. M., & Laurens, K. R. (2014). Cortisol awakening response and diurnal cortisol among children at elevated risk for schizophrenia: Relationship to psychosocial stress and cognition. *Psychoneuroendocrinology*, *46*, 1–13. https://doi.org/10.1016/j.psyneuen.2014.03.010

Daskalakis, N. P., Bagot, R. C., Parker, K. J., Vinkers, C. H., & de Kloet, E. R. (2013). The three-hit concept of vulnerability and resilience: Toward understanding adaptation to early-life adversity outcome. *Psychoneuroendocrinology*, *38*(9), 1858–1873. https://doi.org/10.1016/j.psyneuen.2013.06.008

DeCaro, J. A., & Worthman, C. M. (2008). Return to school accompanied by changing associations between family ecology and cortisol. *Developmental Psychobiology*, *50*(2), 183–195. https://doi.org/10.1002/dev.20255

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*(7), 1562–1592. https://doi.org/10.1016/j.neubiorev.2010.11.007

DeSantis, A. S., Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2015). Racial and ethnic differences in diurnal cortisol rhythms: Are they consistent over time? *Psychosomatic Medicine*, *77*(1), 6–15. https://doi.org/10.1097/PSY.0000000000000131

Donoho, C. J., Weigensberg, M. J., Emken, B. A., Hsu, J. W., & Spruijt-Metz, D. (2011). Stress and abdominal fat: Preliminary evidence of moderation by the cortisol awakening response in Hispanic peripubertal girls. *Obesity*, *19*(5), 946–952. https://doi.org/10.1038/oby.2010.287

Doom, J. R., Cicchetti, D., & Rogosch, F. A. (2014). Longitudinal patterns of cortisol regulation differ in maltreated and nonmaltreated children. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(11), 1206–1215. https://doi.org/10.1016/j.jaac.2014.08.006

Doom, J. R., Cicchetti, D., Rogosch, F. A., & Dackis, M. N. (2013). Child maltreatment and gender interactions as predictors of differential neuroendocrine profiles. *Psychoneuroendocrinology*, *38*(8), 1442–1454. https://doi.org/10.1016/j.psyneuen.2012.12.019

Doom, J. R., Cook, S. H., Sturza, J., Kaciroti, N., Gearhardt, A. N., Vazquez, D. M., Lumeng, J. C., Miller, A. L. (2018). Family conflict, chaos, and negative life events predict cortisol activity in low-income children. *Developmental Psychobiology*, *60*(4), 364–379, https://doi.org/10.1002/dev.21602

Doom, J. R., & Gunnar, M. R. (2013). Stress physiology and developmental psychopathology: Past, present, and future. *Development and Psychopathology*, *25*(4pt2), 1359–1373. https://doi.org/10.1017/S0954579413000667

Dougherty, L. R., Klein, D. N., Olino, T. M., Dyson, M., & Rose, S. (2009). Increased waking salivary cortisol and depression risk in preschoolers: The role of maternal history of melancholic depression and early child temperament. *Journal of Child Psychology and Psychiatry*, *50*(12), 1495–1503. https://doi.org/10.1111/j.1469-7610.2009.02116.x

Dougherty, L. R., Smith, V. C., Olino, T. M., Dyson, M. W., Bufferd, S. J., Rose, S. A., & Klein, D. N. (2013). Maternal psychopathology and early child temperament predict young children's salivary cortisol 3 years later. *Journal of Abnormal Child Psychology*, *41*(4), 531–542. https://doi.org/10.1007/s10802-012-9703-y

Dozier, M., Manni, M., Gordon, M. K., Peloso, E., Gunnar, M. R., Stovall-McClough, K. C., Eldreth, D., Levine, S. (2006). Foster children's diurnal production of cortisol: An exploratory study. *Child Maltreatment*, *11*(2), 189–197, https://doi.org/10.1177/1077559505285779,

Egger, M., Smith, G. D., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *British Medical Journal*, *315*(7109), 629–634. https://doi.org/10.1136/bmj.315.7109.629

Elhassan, M. E., Miller, A. L., Vazquez, D. M., & Lumeng, J. C. (2015). Associations of prenatal and perinatal factors with cortisol diurnal pattern and reactivity to stress at preschool age among children living in poverty. *Journal of Clinical Research in Pediatric Endocrinology*, *7*(2), 114–120. https://doi.org/10.4274/jcrpe.1685

Ellenbogen, M. A., & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology*, *34*(5), 773–785. https://doi.org/10.1016/j.psyneuen.2008.12.011

Ellenbogen, M. A., Hodgins, S., & Walker, C. D. (2004). High levels of cortisol among adolescent offspring of parents with bipolar disorder: A pilot study. *Psychoneuroendocrinology*, *29*(1), 99–106. https://doi.org/10.1016/S0306-4530(02)00135-X

Ellenbogen, M. A., Hodgins, S., Walker, C. D., Couture, S., & Adam, S. (2006). Daytime cortisol and stress reactivity in the offspring of parents with bipolar disorder. *Psychoneuroendocrinology*, *31*(10), 1164–1180. https://doi.org/10.1016/j.psyneuen.2006.08.004

Engert, V., Efanov, S. I., Dedovic, K., Dagher, A., & Pruessner, J. C. (2011). Increased cortisol awakening response and afternoon/evening cortisol

output in healthy young adults with low early life parental care. *Psychopharmacology*, *214*(1), 261–268. https://doi.org/10.1007/s00213-010-1918-4

Epstein, C. M., Houfek, J. F., Rice, M. J., Weiss, S. J., French, J. A., Kupzyk, K. A., Hammer, S. J., Pullen, C. H. (2019). Early life adversity and depressive symptoms predict cortisol in p'egnancy. *Archives of Women's Mental Health*, *23*(3), 379–389, https://doi.org/10.1007/s00737-019-00983-3

Essex, M. J., Shirtcliff, E. A., Burk, L. R., Ruttle, P. L., Klein, M. H., Slattery, M. J., Kalin, N. H., Armstrong, J. M. (2011). Influence of early life stress on later hypothalamic-pituitary–adrenal axis functioning and its covariation with mental health symptoms: A study of the allostatic process from childhood into adolescence. *Development and Psychopathology*, *23*(4), 1039–1058, https://doi.org/10.1017/S0954579411000484,

Evans, B. E., Greaves-Lord, K., Euser, A. S., Franken, I. H., & Huizink, A. C. (2013). Cortisol levels in children of parents with a substance use disorder. *Psychoneuroendocrinology*, *38*(10), 2109–2120. https://doi.org/10.1016/j.psyneuen.2013.03.021

Evans, B. E., van der Ende, J., Greaves-Lord, K., Huizink, A. C., Beijers, R., & de Weerth, C. (2020). Urbanicity, hypothalamic-pituitary-adrenal axis functioning, and behavioral and emotional problems in children: A path analysis. *BMC Psychology*, *8*(12https://doi.org/10.1186/s40359-019-0364-2

Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*, *39*(5), 924–933. https://doi.org/10.1037/0012-1649.39.5.924

Farrell, Cë, Doolin, K., O' Leary, N., Jairaj, C., Roddy, D., Tozzi, L., Morris, D., Harkin, A., Frodl, T., Nemoda, Zófia, Szyf, M., Booij, L., O'Keane, V. (2018). DNA methylation differences at the glucocorticoid receptor gene in depression are related to functional alterations in hypothalamic-pituitary–adrenal axis activity and to early life emotional abuse. *Psychiatry Research*, *265*, 341–348, https://doi.org/10.1016/j.psychres.2018.04.064

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*(4), 245–258. https://doi.org/10.1016/S0749-3797(98)00017-8

Figueroa, W. S., Zoccola, P. M., Manigault, A. W., Hamilton, K. R., Scanlin, M. C., & Johnson, R. C. (2021). Daily stressors and diurnal cortisol among sexual and gender minority young adults. *Health Psychology*, *40*(2), 145–154. https://doi.org/10.1037/hea0001054

Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology*, *32*(8-10), 892–905. https://doi.org/10.1016/j.psyneuen.2007.06.008

Flannery, J. E., Gabard-Durnam, L. J., Shapiro, M., Goff, B., Caldera, C., Louie, J., Gee, D. G., Telzer, E. H., Humphreys, K. L., Lumian, D. S., Tottenham, N. (2017). Diurnal cort-sol after early institutional care - Age matters. *Developmental Cognitive Neuroscience*, *25*, 160–166, https://doi.org/10.1016/j.dcn.2017.03.006,

Flom, M., St. John, A. M., Meyer, J. S., & Tarullo, A. R. (2017). Infant hair cortisol: Associations with salivary cortisol and environmental context. *Developmental Psychobiology*, *59*(1), 26–38. https://doi.org/10.1002/dev.21449

Fogelman, N., & Canli, T. (2018). Early life stress and cortisol: A meta-analysis. *Hormones and Behavior*, *98*, 63–76. https://doi.org/10.1016/j.yhbeh.2017.12.014

Foland-Ross, L. C., Kircanski, K., & Gotlib, I. H. (2014). Coping with having a depressed mother: The role of stress and coping in hypothalamic-pituitary–adrenal axis dysfunction in girls at familial risk for major depression. *Development and Psychopathology*, *26*(4pt2), 1401–1409. https://doi.org/10.1017/S0954579414001102

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology*, *72*(1), 67–73. https://doi.org/10.1016/j.ijpsycho.2008.03.014

Fuchs, A., Moehler, E., Resch, F., & Kaess, M. (2017). The effect of a maternal history of childhood abuse on adrenocortical attunement in mothers and their toddlers. *Developmental Psychobiology*, *59*(5), 639–652. https://doi.org/10.1002/dev.21531

Funder, D. C., & Ozer, D. J. (2019). Evaluating effect size in psychological research: Sense and nonsense. *Advances in Methods and Practices in Psychological Science*, *2*(2), 156–168. https://doi.org/10.1177/2515245919847202

Gabard-Durnam, L. J., & McLaughlin, K. A. (2019). Do sensitive periods exist for exposure to adversity? *Biological Psychiatry*, *85*(10), 789–791. https://doi.org/10.1016/j.biopsych.2019.03.975

Gartstein, M. A., Seamon, E., Thompson, S. F., & Lengua, L. J. (2018). Parenting matters: Moderation of biological and community risk for obesity. *Journal of Applied Developmental Psychology*, *56*, 21–34. https://doi.org/10.1016/j.appdev.2018.01.004

Gerritsen, L., Milaneschi, Y., Vinkers, C. H., van Hemert, A. M., van Velzen, L., Schmaal, L., Penninx, B. W. J. H. (2017). HPA axis genes, and their interaction with childhood maltreatment, are related to cortisol levels and stress-related phenotypes. *Neuropsychopharmacology*, *42*(12), 2446–2455, https://doi.org/10.1038/npp.2017.118,

Goldstein, B. L., Perlman, G., Kotov, R., Broderick, J. E., Liu, K., Ruggero, C., & Klein, D. N. (2017). Etiologic specificity of waking cortisol: Links with maternal history of depression and anxiety in adolescent girls. *Journal of Affective Disorders*, *208*, 103–109. https://doi.org/10.1016/j.jad.2016.08.079

Gow, R., Thomson, S., Rieder, M., Van Uum, S., & Koren, G. (2010). An assessment of cortisol analysis in hair and its clinical applications. *Forensic Science International*, *196*(1-3), 32–37. https://doi.org/10.1016/j.forsciint.2009.12.040

Granger, D. A., Cicchetti, D., Rogosch, F. A., Hibel, L. C., Teisl, M., & Flores, E. (2007). Blood contamination in children's saliva: Prevalence, stability, and impact on the measurement of salivary cortisol, testosterone, and dehydroepiandrosterone. *Psychoneuroendocrinology*, *32*(6), 724–733. https://doi.org/10.1016/j.psyneuen.2007.05.003

Grant, B., & Meyer, D. (2021). Childhood adversity and hair cortisol concentration: A systematic review. *Psychoneuroendocrinology*, *131*, 105536. https://doi.org/10.1016/j.psyneuen.2021.105536

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*(1), 145–173. https://doi.org/10.1146/annurev.psych.58.110405.085605

Gunnar, M. R., DePasquale, C. E., Reid, B. M., Donzella, B., & Miller, B. S. (2019). Pubertal stress recalibration reverses the effects of early life stress in postinstitutionalized children. *Proceedings of The National Academy of Sciences of The United States of America*, *116*(48), 23984–23988. https://doi.org/10.1073/pnas.1909699116

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, *27*(1-2), 199–220. https://doi.org/10.1016/S0306-4530(01)00045-2

Gunnar, M. R., Morison, S. J., Chisholm, K. I. M., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology*, *13*(3), 611–628. https://doi.org/10.1017/s095457940100311x

Gustafsson, P. E., Anckarsäter, H., Lichtenstein, P., Nelson, N., & Gustafsson, P. A. (2010). Does quantity have a quality all its own? Cumulative adversity and up-and down-regulation of circadian salivary cortisol levels in healthy children. *Psychoneuroendocrinology*, *35*(9), 1410–1415. https://doi.org/10.1016/j.psyneuen.2010.04.004

Habersaat, S., Borghini, A., Nessi, J., Forcada-Guex, M., Müller-Nix, C., Pierrehumbert, B., & Ansermet, F. (2014). Effects of perinatal stress and maternal traumatic stress on the cortisol regulation of preterm infants. *Journal of Traumatic Stress*, *27*(4), 488–491. https://doi.org/10.1002/jts.21939

Hackman, D. A., O'Brien, J. R., & Zalewski, M. (2018). Enduring association between parenting and cortisol: A meta-analysis. *Child Development*, *89*(5), 1485–1503. https://doi.org/10.1111/cdev.13077

Hakamata, Y., Suzuki, Y., Kobashikawa, H., & Hori, H. (2022). Neurobiology of early life adversity: A systematic review of meta-analyses towards an integrative account of its neurobiological trajectories to mental disorders. *Frontiers in Neuroendocrinology*, *65*, 100994. https://doi.org/10.1016/j.yfrne.2022.100994

Halligan, S. L., Herbert, J., Goodyer, I. M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biological Psychiatry*, *55*(4), 376–381. https://doi.org/10.1016/j.biopsych.2003.09.013

Hanson, M. D., & Chen, E. (2010). Daily stress, cortisol, and sleep: The moderating role of childhood psychosocial environments. *Health Psychology*, *29*(4), 394–402. https://doi.org/10.1037/a0019879

Harris, M. A., Cox, S. R., Brett, C. E., Deary, I. J., & MacLullich, A. M. (2017). Stress in childhood, adolescence and early adulthood, and cortisol levels in older age. *Stress - the International Journal on The Biology of Stress*, *20*(2), 140–148. https://doi.org/10.1080/10253890.2017.1289168

Harvey, M. W., Farrell, A. K., Imami, L., Carré, J. M., & Slatcher, R. B. (2019). Maternal attachment avoidance is linked to youth diurnal cortisol slopes in children with asthma. *Attachment & Human Development*, *21*(1), 23–37. https://doi.org/10.1080/14616734.2018.1541514

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. *Annals of the New York Academy of Sciences*, *896*(1), 85–95. https://doi.org/10.1111/j.1749-6632.1999.tb08107.x

Hibel, L. C., Mercado, E., & Valentino, K. (2019). Child maltreatment and mother-child transmission of stress physiology. *Child Maltreatment*, *24*(4), 340–352. https://doi.org/10.1177/1077559519826295

Hibel, L. C., Nuttall, A. K., & Valentino, K. (2020). Intimate partner violence indirectly dysregulates child diurnal adrenocortical functioning through positive parenting. *International Journal of Developmental Neuroscience*, *80*(1), 28–41. https://doi.org/10.1002/jdn.10002

Higgins, J. P., Thompson, S. G., Deeks, J. J., & Altman, D. G. (2003). Measuring inconsistency in meta-analyses. *British Medical Journal*, *327*(7414), 557–560. https://doi.org/10.1136/bmj.327.7414.557

Holleman, M., Vreeburg, S. A., Dekker, J. J., & Penninx, B. W. (2012). The relationships of working conditions, recent stressors and childhood trauma with salivary cortisol levels. *Psychoneuroendocrinology*, *37*(6), 801–809. https://doi.org/10.1016/j.psyneuen.2011.09.012

Hosseini-Kamkar, N., Lowe, C., & Morton, J. B. (2021). The differential calibration of the HPA axis as a function of trauma versus adversity: A systematic review and p-curve meta-analyses. *Neuroscience & Biobehavioral Reviews*, *127*, 54–135. https://doi.org/10.1016/j.neubiorev.2021.04.006

Hoyt, L. T., Ehrlich, K. B., Cham, H., & Adam, E. K. (2016). Balancing scientific accuracy and participant burden: Testing the impact of sampling intensity on diurnal cortisol indices. *Stress - the International Journal on The Biology of Stress*, *19*(5), 476–485. https://doi.org/10.1080/10253890.2016.1206884

Hoyt, L. T., Zeiders, K. H., Chaku, N., Niu, L., & Cook, S. H. (2021). Identifying diurnal cortisol profiles among young adults: Physiological signatures of mental health trajectories. *Psychoneuroendocrinology*, *128*, 105204. https://doi.org/10.1016/j.psyneuen.2021.105204

Huizink, A. C., Greaves-Lord, K., Oldehinkel, A. J., Ormel, J., & Verhulst, F. C. (2009). Hypothalamic-pituitary–adrenal axis and smoking and drinking onset among adolescents: The longitudinal cohort TRacking Adolescents' Individual Lives Survey (TRAILS). *Addiction*, *104*(11), 1927–1936. https://doi.org/10.1111/j.1360-0443.2009.02685.x

Hunter, A. L., Minnis, H., & Wilson, P. (2011). Altered stress responses in children exposed to early adversity: A systematic review of salivary cortisol studies. *Stress - the International Journal on The Biology of Stress*, *14*(6), 614–626. https://doi.org/10.3109/10253890.2011.577848

Hustedt, J. T., Vu, J. A., Bargreen, K. N., Hallam, R. A., & Han, M. (2017). Early Head Start families' experiences with stress: Understanding variations within a high-risk, low-income sample. *Infant Mental Health Journal*, *38*(5), 602–616. https://doi.org/10.1002/imhj.21667

Huynh, V. W., Guan, S. S. A., Almeida, D. M., McCreath, H., & Fuligni, A. J. (2016). Everyday discrimination and diurnal cortisol during adolescence. *Hormones and Behavior*, *80*, 76–81. https://doi.org/10.1016/j.yhbeh.2016.01.009

Isenhour, J., Raby, K. L., & Dozier, M. (2020). The persistent associations between early institutional care and diurnal cortisol outcomes among children adopted internationally. *Developmental Psychobiology*, *63*(5), 1156–1166. https://doi.org/10.1002/dev.22069

Johnson, A. E., Bruce, J., Tarullo, A. R., & Gunnar, M. R. (2011). Growth delay as an index of allostatic load in young children: Predictions to disinhibited social approach and diurnal cortisol activity. *Development and Psychopathology*, *23*(3), 859–871. https://doi.org/10.1017/S0954579411000356

Johnson, A. J., & Tottenham, N. (2015). Regulatory skill as a resilience factor for adults with a history of foster care: A pilot study. *Developmental Psychobiology*, *57*(1), 1–16. https://doi.org/10.1002/dev.2122

Johnson, D., Policelli, J., Li, M., Dharamsi, A., Hu, Q., Sheridan, M. A., & Wade, M. (2021). Associations of early-life threat and deprivation with executive functioning in childhood and adolescence: A systematic review and meta-analysis. *JAMA Pediatrics*, *175*(11), e212511. https://doi.org/10.1001/jamapediatrics.2021.2511

Keeshin, B. R., Strawn, J. R., Out, D., Granger, D. A., & Putnam, F. W. (2014). Cortisol awakening response in adolescents with acute sexual abuse related posttraumatic stress disorder. *Depression and Anxiety*, *31*(2), 107–114. https://doi.org/10.1002/da.22154

Kertes, D. A., Gunnar, M. R., Madsen, N. J., & Long, J. D. (2008). Early deprivation and home basal cortisol levels: A study of internationally adopted children. *Development and Psychopathology*, *20*(2), 473–491. https://doi.org/10.1017/S0954579408000230

Khoury, J. E., Enlow, M. B., Plamondon, A., & Lyons-Ruth, K. (2019). The association between adversity and hair cortisol levels in humans: A meta-analysis. *Psychoneuroendocrinology*, *103*, 104–117. https://doi.org/10.1016/j.psyneuen.2019.01.009

Kiel, E. J., Hummel, A. C., & Luebbe, A. M. (2015). Cortisol secretion and change in sleep problems in early childhood: Moderation by maternal overcontrol. *Biological Psychology*, *107*, 52–60. https://doi.org/10.1016/j.biopsycho.2015.03.001

King, L. S., Colich, N. L., LeMoult, J., Humphreys, K. L., Ordaz, S. J., Price, A. N., & Gotlib, I. H. (2017). The impact of the severity of early life stress on diurnal cortisol: The role of puberty. *Psychoneuroendocrinology*, *77*, 68–74. https://doi.org/10.1016/j.psyneuen.2016.11.024Get

Kliewer, W. (2006). Violence exposure and cortisol responses in urban youth. *International Journal of Behavioral Medicine*, *13*(2), 109–120. https://doi.org/10.1207/s15327558ijbm1302_2

Kliewer, W., Reid-Quiñones, K., Shields, B. J., & Foutz, L. (2009). Multiple risks, emotion regulation skill, and cortisol in low-income African American youth: A prospective study. *Journal of Black Psychology*, *35*(1), 24–43. https://doi.org/10.1177/0095798408323355

Knapp, G., & Hartung, J. (2003). Improved tests for a random effects meta-regression with a single covariate. *Statistics in Medicine*, *22*(17), 2693–2710. https://doi.org/10.1002/sim.1482

Kohrt, B. A., Worthman, C. M., Ressler, K. J., Mercer, K. B., Upadhaya, N., Koirala, S., & Binder, E. B. (2015). Cross-cultural gene– environment interactions in depression, post-traumatic stress disorder, and the cortisol awakening response: FKBP5 polymorphisms and childhood trauma in South Asia: GxE interactions in South Asia. *International Review of Psychiatry*, *27*(3), 180–196. https://doi.org/10.3109/09540261.2015.1020052

Korpa, T., Pervanidou, P., Angeli, E., Apostolakou, F., Papanikolaou, K., Papassotiriou, I., & Kolaitis, G. (2017). Mothers' parenting stress is associated with salivary cortisol profiles in children with attention deficit hyperactivity disorder. *Stress - the International Journal on The Biology of Stress*, *20*(2), 149–158. https://doi.org/10.1080/10253890.2017.1303472

Koss, K. J., Hostinar, C. E., Donzella, B., & Gunnar, M. R. (2014). Social deprivation and the HPA axis in early development. *Psychoneuroendocrinology*, *50*, 1–13. https://doi.org/10.1016/j.psyneuen.2014.07.028

Koss, K. J., Mliner, S. B., Donzella, B., & Gunnar, M. R. (2016). Early adversity, hypocortisolism, and behavior problems at school entry: A study of internationally adopted children. *Psychoneuroendocrinology*, *66*, 31–38. https://doi.org/10.1016/j.psyneuen.2015.12.018

Kuhlman, K. R., Geiss, E. G., Vargas, I., & Lopez-Duran, N. L. (2015). Differential associations between childhood trauma subtypes and adolescent HPA-axis functioning. *Psychoneuroendocrinology*, *54*, 103–114. https://doi.org/10.1016/j.psyneuen.2015.01.020

Kuhlman, K. R., Vargas, I., Geiss, E. G., & Lopez-Duran, N. L. (2015). Age of trauma onset and HPA axis dysregulation among trauma-exposed youth. *Journal of Traumatic Stress*, *28*(6), 572–579. https://doi.org/10.1002/jts.22054

Kumsta, R., Schlotz, W., Golm, D., Moser, D., Kennedy, M., Knights, N., Kreppner, J., Maughan, B., Rutter, M., Sonuga-Barke, E. (2017). HPA axis dysregulation in adult adoptees twenty years after severe institutional deprivation in childhood. *Psychoneuroendocrinology*, *86*, 196–202, https://doi.org/10.1016/j.psyneuen.2017.09.021,

Kwak, Y., Taylor, Z. E., Anaya, L. Y., Feng, Y., Evich, C. D., & Jones, B. L. (2017). Cumulative family stress and diurnal cortisol responses in Midwest Latino families. *Hispanic Journal of Behavioral Sciences*, *39*(1), 82–97. https://doi.org/10.1177/0739986316684130

Laceulle, O. M., Nederhof, E., van Aken, M. A., & Ormel, J. (2017). Adversity-driven changes in hypothalamic-pituitary-adrenal axis functioning during adolescence. The trails study. *Psychoneuroendocrinology*, *85*, 49–55. https://doi.org/10.1016/j.psyneuen.2017.08.002

Lai, C. L. J., Lee, D. Y. H., & Leung, M. O. Y. (2021). Childhood adversities and salivary cortisol responses to the Trier Social Stress Test: A systematic review of studies using the Children Trauma Questionnaire (CTQ). *International Journal of Environmental Research and Public Health*, *18*(1), 29. https://doi.org/10.3390/ijerph18010029

Larsson, C. A., Gullberg, B., Råstam, L., & Lindblad, U. (2009). Salivary cortisol differs with age and sex and shows inverse associations with WHR in Swedish women: A cross-sectional study. *BMC Endocrine Disorders*, *9*(1https://doi.org/10.1186/1472-6823-9-16

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Fisher, P. A., & Reiss, D. (2013). Effects of parental depressive symptoms on child adjustment moderated by hypothalamic pituitary adrenal activity: Within-and between-family risk. *Child Development*, *84*(2), 528–542. https://doi.org/10.1111/j.1467-8624.2012.01859.x

Laurent, H. K., Leve, L. D., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Harold, G. T., & Reiss, D. (2013). Effects of prenatal and postnatal parent depressive symptoms on adopted child HPA regulation: Independent and moderated influences. *Developmental Psychology*, *49*(5), 876–886. https://doi.org/10.1037/a0028800

Laurent, H. K., Neiderhiser, J. M., Natsuaki, M. N., Shaw, D. S., Fisher, P. A., Reiss, D., & Leve, L. D. (2014). Stress system development from age 4.5 to 6: Family environment predictors and adjustment implications of HPA activity stability versus change. *Developmental Psychobiology*, *56*(3), 340–354. https://doi.org/10.1002/dev.21103

LeMoult, J., Chen, M. C., Foland-Ross, L. C., Burley, H. W., & Gotlib, I. H. (2015). Concordance of mother-daughter diurnal cortisol production: Understanding the intergenerational transmission of risk for depression. *Biological Psychology*, *108*, 98–104. https://doi.org/10.1016/j.biopsycho.2015.03.019

LeMoult, J., Ordaz, S. J., Kircanski, K., Singh, M. K., & Gotlib, I. H. (2015). Predicting first onset of depression in young girls: Interaction of diurnal cortisol and negative life events. *Journal of Abnormal Psychology*, *124*(4), 850–859. https://doi.org/10.1037/abn0000087

Leneman, K. B., Donzella, B., Desjardins, C. D., Miller, B. S., & Gunnar, M. R. (2018). The slope of cortisol from awakening to 30 min post-wake in post-institutionalized children and early adolescents. *Psychoneuroendocrinology*, *96*, 93–99. https://doi.org/10.1016/j.psyneuen.2018.06.011

Lengua, L. J., Zalewski, M., Fisher, P., & Moran, L. (2013). Does HPA-Axis dysregulation account for the effects of income on effortful control and adjustment in preschool children? *Infant and Child Development*, *22*(5), 439–458. https://doi.org/10.1002/icd.1805

Leppert, K. A., Smith, V. C., Merwin, S. M., Kushner, M., & Dougherty, L. R. (2018). Cortisol rhythm in preschoolers: Relations with maternal depression and child temperament. *Journal of Psychopathology and Behavioral Assessment*, *40*(3), 386–401. https://doi.org/10.1007/s10862-018-9650-1

Letourneau, N., Watson, B., Duffett-Leger, L., Hegadoren, K., & Tryphonopoulos, P. (2011). Cortisol patterns of depressed mothers and their infants are related to maternal-infant interactive behaviours. *Journal of Reproductive and Infant Psychology*, *29*(5), 439–459. https://doi.org/10.1080/02646838.2011.649474

Liu, K., Ruggero, C. J., Goldstein, B., Klein, D. N., Perlman, G., Broderick, J., & Kotov, R. (2016). Elevated cortisol in healthy female adolescent offspring of mothers with posttraumatic stress disorder. *Journal of Anxiety Disorders*, *40*, 37–43. https://doi.org/10.1016/j.janxdis.2016.04.003

Loman, M. M., & Gunnar, M. R. (2010). Early experience and the development of stress reactivity and regulation in children. *Neuroscience & Biobehavioral Reviews*, *34*(6), 867–876. https://doi.org/10.1016/j.neubiorev.2009.05.007

Lovallo, W. R., Cohoon, A. J., Acheson, A., Sorocco, K. H., & Vincent, A. S. (2019). Blunted stress reactivity reveals vulnerability to early life adversity in young adults with a family history of alcoholism. *Addiction*, *114*(5), 798–806. https://doi.org/10.1111/add.14501

Lumeng, J. C., Miller, A., Peterson, K. E., Kaciroti, N., Sturza, J., Rosenblum, K., & Vazquez, D. M. (2014). Diurnal cortisol pattern, eating behaviors and overweight in low-income preschool-aged children. *Appetite*, *73*, 65–72. https://doi.org/10.1016/j.appet.2013.10.016

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology*, *13*(3), 653–676. https://doi.org/10.1017/S0954579401003133

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445. https://doi.org/10.1038/nrn2639

Mangold, D., Wand, G., Javors, M., & Mintz, J. (2010). Acculturation, childhood trauma and the cortisol awakening response in Mexican-American adults. *Hormones and Behavior*, *58*(4), 637–646. https://doi.org/10.1016/j.yhbeh.2010.06.010

Marceau, K., Ram, N., Neiderhiser, J. M., Laurent, H. K., Shaw, D. S., Fisher, P., & Leve, L. D. (2013). Disentangling the effects of genetic, prenatal and parenting influences on children's cortisol variability. *Stress - the International Journal on The Biology of Stress*, *16*(6), 607–615. https://doi.org/10.3109/10253890.2013.825766

Marsman, R., Nederhof, E., Rosmalen, J. G., Oldehinkel, A. J., Ormel, J., & Buitelaar, J. K. (2012). Family environment is associated with HPA-axis activity in adolescents. The TRAILS study. *Biological Psychology*, *89*(2), 460–466. https://doi.org/10.1016/j.biopsycho.2011.12.013

Martin, C. G., Bruce, J., & Fisher, P. A. (2012). Racial and ethnic differences in diurnal cortisol rhythms in preadolescents: The role of parental psychosocial risk and monitoring. *Hormones and Behavior*, *61*(5), 661–668. https://doi.org/10.1016/j.yhbeh.2012.02.025

McLachlan, K., Rasmussen, C., Oberlander, T. F., Loock, C., Pei, J., Andrew, G., Reynolds, J., Weinberg, J. (2016). Dysregulation of the cortisol diurnal rhythm following prenatal alcohol exposure and early life adversity. *Alcohol*, *53*, 9–18, https://doi.org/10.1016/j.alcohol.2016.03.003,

McLaughlin, K. A. (2016). Future directions in childhood adversity and youth psychopathology. *Journal of Clinical Child & Adolescent Psychology*, *45*(3), 361–382. https://doi.org/10.1080/15374416.2015.1110823

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Psychological Science*, *25*(4), 239–245. https://doi.org/10.1177/0963721416655883

McLaughlin, K. A., Weissman, D., & Bitrán, D. (2019). Childhood adversity and neural development: A systematic review. *Annual Review of Developmental Psychology*, *1*(1), 277–312. https://doi.org/10.1146/annurev-devpsych-121318-084950

Meinlschmidt, G., & Heim, C. (2005). Decreased cortisol awakening response after early loss experience. *Psychoneuroendocrinology*, *30*(6), 568–576. https://doi.org/10.1016/j.psyneuen.2005.01.006

Merwin, S. Psychological and neurobiological outcomes of parent-child adrenocortical concordance, 2017, (Doctoral dissertation). Retrieved from Digital Repository at the University of Maryland, https://doi.org/10.13016/M2P84402R

Merwin, S. M., Barrios, C., Smith, V. C., Lemay, E. P. Jr., & Dougherty, L. R. (2018). Outcomes of early parent-child adrenocortical attunement in the high-risk offspring of depressed parents. *Developmental Psychobiology*, *60*(4), 468–482. https://doi.org/10.1002/dev.21623

Merwin, S. M., Leppert, K. A., Smith, V. C., & Dougherty, L. R. (2017). Parental depression and parent and child stress physiology: Moderation by

parental hostility. *Developmental Psychobiology*, *59*(8), 997–1009. https://doi.org/10.1002/dev.21556

Merwin, S. M., Smith, V. C., Kushner, M., Lemay, E. P. Jr, & Dougherty, L. R. (2017). Parent-child adrenocortical concordance in early childhood: The moderating role of parental depression and child temperament. *Biological Psychology*, *124*, 100–110, https://doi.org/10.1016/j.biopsycho.2017.01.013,

Messerli-Bürgy, N., Stülb, K., Kakebeeke, T. H., Arhab, A., Zysset, A. E., Leeger-Aschmann, C. S., Schmutz, E. A., Meyer, A. H., Ehlert, U., Garcia-Burgos, D., Kriemler, S., Jenni, O. G., Puder, J. J., & Munsch, S. (2018). Emotional eating is related with temperament but not with stress biomarkers in preschool children. *Appetite*, *120*, 256–264. https://doi.org/10.1016/j.appet.2017.08.032Get,

Miles, E. M., Dmitrieva, J., Hurwich-Reiss, E., Badanes, L., Mendoza, M. M., Perreira, K. M., & Watamura, S. E. (2018). Evidence for a physiologic home-school gap in children of Latina immigrants. *Early Childhood Research Quarterly*, *52*, 86–100. https://doi.org/10.1016/j.ecresq.2018.03.010

Miller, A. L., Song, J. H., Sturza, J., Lumeng, J. C., Rosenblum, K., Kaciroti, N., & Vazquez, D. M. (2017). Child cortisol moderates the association between family routines and emotion regulation in low-income children. *Developmental Psychobiology*, *59*(1), 99–110. https://doi.org/10.1002/dev.21471

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*(6), 959–997. https://doi.org/10.1037/a0024768

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*(1), 25–45. https://doi.org/10.1037/0033-2909.133.1.25

Miller, K. F., Arbel, R., Shapiro, L. S., Han, S. C., & Margolin, G. (2018). Does the cortisol awakening response link childhood adversity to adult BMI? *Health Psychology*, *37*(6), 526–529. https://doi.org/10.1037/hea0000601

Miller, K. F., Margolin, G., Shapiro, L. S., & Timmons, A. C. (2017). Adolescent life stress and the cortisol awakening response: The moderating roles of attachment and sex. *Journal of Research on Adolescence*, *27*(1), 34–48. https://doi.org/10.1111/jora.12250

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D. G. (2010). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *International Journal of Surgery*, *8*(5), 336–341. https://doi.org/10.1016/j.ijsu.2010.02.007

Monteleone, A. M., Monteleone, P., Serino, I., Scognamiglio, P., Di Genio, M., & Maj, M. (2015). Childhood trauma and cortisol awakening response in symptomatic patients with anorexia nervosa and bulimia nervosa. *International Journal of Eating Disorders*, *48*(6), 615–621. https://doi.org/10.1002/eat.22375

Murray, L., Halligan, S. L., Goodyer, I., & Herbert, J. (2010). Disturbances in early parenting of depressed mothers and cortisol secretion in offspring: A preliminary study. *Journal of Affective Disorders*, *122*(3), 218–223. https://doi.org/10.1016/j.jad.2009.06.034

Murray-Close, D., Han, G., Cicchetti, D., Crick, N. R., & Rogosch, F. A. (2008). Neuroendocrine regulation and physical and relational aggression: The moderating roles of child maltreatment and gender. *Developmental Psychology*, *44*(4), 1160–1176. https://doi.org/10.1037/a0012564

Netherton, C., Goodyer, I., Tamplin, A., & Herbert, J. (2004). Salivary cortisol and dehydroepiandrosterone in relation to puberty and gender. *Psychoneuroendocrinology*, *29*(2), 125–140. https://doi.org/10.1016/S0306-4530(02)00150-6

O'Connor, T. G., Ben-Shlomo, Y., Heron, J., Golding, J., Adams, D., & Glover, V. (2005). Prenatal anxiety predicts individual differences in cortisol in pre-adolescent children. *Biological Psychiatry*, *58*(3), 211–217. https://doi.org/10.1016/j.biopsych.2005.03.032

O'Loughlin, J. I., Rellini, A. H., & Brotto, L. A. (2020). How does childhood trauma impact women's sexual desire? Role of depression, stress, and cortisol. *The Journal of Sex Research*, *57*(7), 836–847. https://doi.org/10.1080/00224499.2019.1693490

Ostiguy, C. S., Ellenbogen, M. A., Walker, C. D., Walker, E. F., & Hodgins, S. (2011). Sensitivity to stress among the offspring of parents with bipolar disorder: A study of daytime cortisol levels. *Psychological Medicine*, *41*(11), 2447–2457. https://doi.org/10.1017/S0033291711000523

Ouellet-Morin, I., Dionne, G., Pérusse, D., Lupien, S. J., Arseneault, L., Barr, R. G., & Boivin, M. (2009). Daytime cortisol secretion in 6-month-old twins: Genetic and environmental contributions as a function of early familial adversity. *Biological Psychiatry*, *65*(5), 409–416. https://doi.org/10.1016/j.biopsych.2008.10.003

Pawluski, J. L., Brain, U. M., Underhill, C. M., Hammond, G. L., & Oberlander, T. F. (2012). Prenatal SSRI exposure alters neonatal corticosteroid binding globulin, infant cortisol levels, and emerging HPA function. *Psychoneuroendocrinology*, *37*(7), 1019–1028. https://doi.org/10.1016/j.psyneuen.2011.11.011

Peckins, M. K., Roberts, A. G., Hein, T. C., Hyde, L. W., Mitchell, C., Brooks-Gunn, J., McLanahan, S. S., Monk, C. S., Lopez-Duran, N. L. (2020). Violence exposure and social deprivation is associated with cortisol reactivity in urban adolescents. *Psychoneuroendocrinology*, *111*, 104426, https://doi.org/10.1016/j.psyneuen.2019.104426,

Pendry, P., & Adam, E. K. (2007). Associations between parents' marital functioning, maternal parenting quality, maternal emotion and child cortisol levels. *International Journal of Behavioral Development*, *31*(3), 218–231. https://doi.org/10.1177/0165025407074634

Peng, H., Long, Y., Li, J., Guo, Y., Wu, H., Yang, Y. L., Ding, Y., He, J., Ning, Y. (2014). Hypothalamic-pituitary-adrenal axis functioning and dysfunctional attitude in depressed patients with and without childhood neglect. *BMC Psychiatry*, *14*(45, https://doi.org/10.1186/1471-244X-14-45

Perry, N. B., DePasquale, C. E., Fisher, P. H., & Gunnar, M. R. (2019). Comparison of institutionally reared and maltreated children on socioemotional and biological functioning. *Child Maltreatment*, *24*(3), 235–243. https://doi.org/10.1177/1077559518823074

Peterson, R. A., & Brown, S. P. (2005). On the use of beta coefficients in meta-analysis. *Journal of Applied Psychology*, *90*(1), 175–181. https://doi.org/10.1037/0021-9010.90.1.175

Philbrook, L. E., & Teti, D. M. (2016). Associations between bedtime and nighttime parenting and infant cortisol in the first year. *Developmental Psychobiology*, *58*(8), 1087–1100. https://doi.org/10.1002/dev.21442

Pitula, C. E., DePasquale, C. E., Mliner, S. B., & Gunnar, M. R. (2019). Peer problems among postinstitutionalized, internationally adopted children: Relations to hypocortisolism, parenting quality, and ADHD symptoms. *Child Development*, *90*(3), e339–e355. https://doi.org/10.1111/cdev.12986

Plant, D. T., Pawlby, S., Sharp, D., Zunszain, P. A., & Pariante, C. M. (2016). Prenatal maternal depression is associated with offspring inflammation at 25 years: A prospective longitudinal cohort study. *Translational Psychiatry*, *6*(11), e936–e936. https://doi.org/10.1038/tp.2015.155

Pruessner, M., Vracotas, N., Joober, R., Pruessner, J. C., & Malla, A. K. (2013). Blunted cortisol awakening response in men with first episode psychosis: Relationship to parental bonding. *Psychoneuroendocrinology*, *38*(2), 229–240. https://doi.org/10.1016/j.psyneuen.2012.06.002

Puetz, V. B., Zweerings, J., Dahmen, B., Ruf, C., Scharke, W., Herpertz-Dahlmann, B., & Konrad, K. (2016). Multidimensional assessment of neuroendocrine and psychopathological profiles in maltreated youth. *Journal of Neural Transmission*, *123*(9), 1095–1106. https://doi.org/10.1007/s00702-016-1509-6

Quevedo, K., Doty, J., Roos, L., & Anker, J. J. (2017). The cortisol awakening response and anterior cingulate cortex function in maltreated depressed versus non-maltreated depressed youth. *Psychoneuroendocrinology*, *86*, 87–95. https://doi.org/10.1016/j.psyneuen.2017.09.001Get

Quevedo, K., Johnson, A. E., Loman, M. L., LaFavor, T. L., & Gunnar, M. (2012). The confluence of adverse early experience and puberty on the cortisol awakening response. *International Journal of Behavioral Development*, *36*(1), 19–28. https://doi.org/10.1177/0165025411406860

Raffington, L., Prindle, J., Keresztes, A., Binder, J., Heim, C., & Shing, Y. L. (2018). Blunted cortisol stress reactivity in low-income children relates to lower memory function. *Psychoneuroendocrinology*, *90*, 110–121. https://doi.org/10.1016/j.psyneuen.2018.02.002

Raffington, L., Schmiedek, F., Heim, C., & Shing, Y. L. (2018). Cognitive control moderates parenting stress effects on children's diurnal cortisol. *PLoS One*, *13*(1), e0191215. https://doi.org/10.1371/journal.pone.0191215

Raymond, C., Marin, M. F., Wolosianski, V., Journault, A. A., Longpré, C., Leclaire, S., & Lupien, S. J. (2021). Early childhood adversity and HPA axis activity in adulthood: The importance of considering minimal age at exposure. *Psychoneuroendocrinology*, *124*, 105042. https://doi.org/10.1016/j.psyneuen.2020.105042

Reichl, C., Heyer, A., Brunner, R., Parzer, P., Völker, J. M., Resch, F., & Kaess, M. (2016). Hypothalamic-pituitary-adrenal axis, childhood adversity and adolescent nonsuicidal self-injury. *Psychoneuroendocrinology*, *74*, 203–211. https://doi.org/10.1016/j.psyneuen.2016.09.011

Rogosch, F. A., Dackis, M. N., & Cicchetti, D. (2011). Child maltreatment and allostatic load: Consequences for physical and mental health in children from low-income families. *Development and Psychopathology*, *23*(4), 1107–1124. https://doi.org/10.1017/S0954579411000587

Roisman, G. I., Susman, E., Barnett-Walker, K., Booth-LaForce, C., Owen, M. T., Belsky, J., Bradley, R. H., Houts, R., Steinberg, L., The NICHD Early Child Care Research Network (2009). Early family and child-care antecedents of awakening cortisol levels in adolescence. *Child Development*, *80*(3), 907–920, https://doi.org/10.1111/j.1467-8624.2009.01305.x,

Rosenthal, R. (1979). The file drawer problem and tolerance for null results. *Psychological Bulletin*, *86*(3), 638–641. https://doi.org/10.1037/0033-2909.86.3.638

Russ, S. J., Herbert, J., Cooper, P., Gunnar, M. R., Goodyer, I., Croudace, T., & Murray, L. (2012). Cortisol levels in response to starting school in children at increased risk for social phobia. *Psychoneuroendocrinology*, *37*(4), 462–474. https://doi.org/10.1016/j.psyneuen.2011.07.014

Ryan, R., Booth, S., Spathis, A., Mollart, S., & Clow, A. (2016). Use of salivary diurnal cortisol as an outcome measure in randomised controlled trials: A systematic review. *Annals of Behavioral Medicine*, *50*(2), 210–236. https://doi.org/10.1007/s12160-015-9753-9

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*(1), 55–89. https://doi.org/10.1210/edrv.21.1.0389

Seidenfaden, D., Knorr, U., Soendergaard, M. G., Poulsen, H. E., Fink-Jensen, A., Jorgensen, M. B., & Jorgensen, A. (2017). The relationship between self-reported childhood adversities, adulthood psychopathology and psychological stress markers in patients with schizophrenia. *Comprehensive Psychiatry*, *72*, 48–55. https://doi.org/10.1016/j.comppsych.2016.09.009

Selye, H. (1946). The general adaptation syndrome and the diseases of adaptation. *The Journal of Clinical Endocrinology*, *6*(2), 117–230. https://doi.org/10.1210/jcem-6-2-117

Shirtcliff, E. A., & Essex, M. J. (2008). Concurrent and longitudinal associations of basal and diurnal cortisol with mental health symptoms in early adolescence. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *50*(7), 690–703. https://doi.org/10.1002/dev.20336

Simmons, J. G., Byrne, M. L., Schwartz, O. S., Whittle, S. L., Sheeber, L., Kaess, M., & Allen, N. B. (2015). Dual-axis hormonal covariation in adolescence and the moderating influence of prior trauma and aversive maternal parenting. *Developmental Psychobiology*, *57*(6), 670–687. https://doi.org/10.1002/dev.21275

Simons, S. S., Beijers, R., Cillessen, A. H., & de Weerth, C. (2015). Development of the cortisol circadian rhythm in the light of stress early in life. *Psychoneuroendocrinology*, *62*, 292–300. https://doi.org/10.1016/j.psyneuen.2015.08.024

Smeets, T., Geraerts, E., Jelicic, M., & Merckelbach, H. (2007). Delayed recall of childhood sexual abuse memories and the awakening rise and diurnal pattern of cortisol. *Psychiatry Research*, *152*(2-3), 197–204. https://doi.org/10.1016/j.psychres.2006.07.008

St. John, A. M., Kao, K., Liederman, J., Grieve, P. G., & Tarullo, A. R. (2017). Maternal cortisol slope at 6 months predicts infant cortisol slope and EEG power at 12 months. *Developmental Psychobiology*, *59*(6), 787–801. https://doi.org/10.1002/dev.21540

Stalder, T., Kirschbaum, C., Kudielka, B. M., Adam, E. K., Pruessner, J. C., Wüst, S., Dockray, S., Smyth, N., Evans, P., Hellhammer, D. H., Miller, R., Wetherell, M. A., Lupien, S. J., Clow, A. (2016). Assessment of the cortisol awakening response: Expert consensus guidelines. *Psychoneuroendocrinology*, *63*, 414–432, https://doi.org/10.1016/j.psyneuen.2015.10.010

Stalder, T., Lupien, S. J., Kudielka, B. M., Adam, E. K., Pruessner, J. C., Wüst, S., Dockray, S., Smyth, N., Evans, P., Kirschbaum, C., Miller, R., Wetherell, M. A., Finke, J. B., Klucken, T., & Clow, A. (2022). Evaluation and update of the expert consensus guidelines for the assessment of the cortisol awakening response (CAR). *Psychoneuroendocrinology*, *146*, 105946, https://doi.org/doi:1016/j.psyneuen.2022.105946

Starr, L. R., Dienes, K., Stroud, C. B., Shaw, Z. A., Li, Y. I., Mlawer, F., & Huang, M. (2017). Childhood adversity moderates the influence of proximal episodic stress on the cortisol awakening response and depressive symptoms in adolescents. *Development and Psychopathology*, *29*(5), 1877–1893. https://doi.org/10.1017/S0954579417001468

Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (6th edn. Pearson.

Tarullo, A. R., St. John, A. M., & Meyer, J. S. (2017). Chronic stress in the mother-infant dyad: Maternal hair cortisol, infant salivary cortisol and interactional synchrony. *Infant Behavior and Development*, *47*, 92–102. https://doi.org/10.1016/j.infbeh.2017.03.007

Taylor, L. K., Weems, C. F., Costa, N. M., & Carrión, V. G. (2009). Loss and the experience of emotional distress in childhood. *Journal of Loss and Trauma*, *14*(1), 1–16. https://doi.org/10.1080/15325020802173843

Theall, K. P., Shirtcliff, E. A., Dismukes, A. R., Wallace, M., & Drury, S. S. (2017). Association between neighborhood violence and biological stress in children. *JAMA Pediatrics*, *171*(1), 53–60. https://doi.org/10.1001/jamapediatrics.2016.2321

Thompson, S. F., Zalewski, M., Kiff, C. J., & Lengua, L. J. (2018). A state-trait model of cortisol in early childhood: Contextual and parental predictors of stable and time-varying effects. *Hormones and Behavior*, *98*, 198–209. https://doi.org/10.1016/j.yhbeh.2017.12.009

Valentino, K., Hibel, L. C., Cummings, E. M., Nuttall, A. K., Comas, M., & McDonnell, C. G. (2015). Maternal elaborative reminiscing mediates the effect of child maltreatment on behavioral and physiological functioning. *Development and Psychopathology*, *27*(4 Pt 2), 1515–1526. https://doi.org/10.1017/S0954579415000917

Van Cauter, E. (1990). Diurnal and ultradian rhythms in human endocrine function: A minireview. *Hormone Research in Paediatrics*, *34*(2), 45–53. https://doi.org/10.1159/000181794

Van den Bergh, B. R., Van Calster, B., Smits, T., Van Huffel, S., & Lagae, L. (2008). Antenatal maternal anxiety is related to HPA-axis dysregulation and self-reported depressive symptoms in adolescence: A prospective study on the fetal origins of depressed mood. *Neuropsychopharmacology*, *33*(3), 536–545. https://doi.org/10.1038/sj.npp.1301450

van der Vegt, E. J., van der Ende, J., Kirschbaum, C., Verhulst, F. C., & Tiemeier, H. (2009). Early neglect and abuse predict diurnal cortisol patterns in adults: A study of international adoptees. *Psychoneuroendocrinology*, *34*(5), 660–669. https://doi.org/10.1016/j.psyneuen.2008.11.004

Vänskä, M., Punamäki, R. L., Lindblom, J., Tolvanen, A., Flykt, M., Unkila-Kallio, L., & Tiitinen, A. (2016). Timing of early maternal mental health and child cortisol regulation. *Infant and Child Development*, *25*(6), 461–483. https://doi.org/10.1002/icd.1948

VanTieghem, M., Korom, M., Flannery, J., Choy, T., Caldera, C., Humphreys, K. L., Gabard-Durnam, L., Goff, B., Gee, D. G., Telzer, E. H., Shapiro, M., Louie, J. Y., Fareri, D. S., Bolger, N., Tottenham, N. (2021). Longitudinal changes in amygdala, hippocampus and cortisol development following early caregiving adversity. *Developmental Cognitive Neuroscience*, *48*, 100916, https://doi.org/10.1016/j.dcn.2021.100916

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software*, *36*(3), 1–48. https://doi.org/10.18637/jss.v036.i03

Vreeburg, S. A., Hartman, C. A., Hoogendijk, W. J., van Dyck, R., Zitman, F. G., Ormel, J., & Penninx, B. W. (2010). Parental history of depression or anxiety and the cortisol awakening response. *The British Journal of Psychiatry*, *197*(3), 180–185. https://doi.org/10.1192/bjp.bp.109.076869

Weissbecker, I., Floyd, A., Dedert, E., Salmon, P., & Sephton, S. (2006). Childhood trauma and diurnal cortisol disruption in fibromyalgia syndrome. *Psychoneuroendocrinology*, *31*(3), 312–324. https://doi.org/10.1016/j.psyneuen.2005.08.009

Wielaard, I., Schaakxs, R., Comijs, H. C., Stek, M. L., & Rhebergen, D. (2018). The influence of childhood abuse on cortisol levels and the cortisol awakening response in depressed and nondepressed older adults. *The World Journal of Biological Psychiatry*, *19*(6), 440–449. https://doi.org/10.1080/15622975.2016.1274829

Williams, S. L., & Mann, A. K. (2017). Sexual and gender minority health disparities as a social issue: How stigma and intergroup relations can explain and reduce health disparities. *Journal of Social Issues*, *73*(3), 450–461. https://doi.org/10.1111/josi.12225

Wilson, D. B., Practical meta-analysis effect size calculator, n.d., Campbell Collaboration. https://www.campbellcollaboration.org/escalc/html/EffectSizeCalculator-R-main.php

Wright, J. L., Jarvis, J. N., Pachter, L. M., & Walker-Harding, L. R. (2020). Racism as a public health issue, APS racism series: At the intersection of equity, science, and social justice. *Pediatric Research*, *88*(5), 696–698. https://doi.org/10.1038/s41390-020-01141-7

Yehuda, R., Engel, S. M., Brand, S. R., Seckl, J., Marcus, S. M., & Berkowitz, G. S. (2005). Transgenerational effects of posttraumatic stress disorder in babies of mothers exposed to the World Trade Center attacks during pregnancy. *The Journal of Clinical Endocrinology & Metabolism*, *90*(7), 4115–4118. https://doi.org/10.1210/jc.2005-0550

Zalewski, M., Lengua, L. J., Fisher, P. A., Trancik, A., Bush, N. R., & Meltzoff, A. N. (2012). Poverty and single parenting: Relations with preschoolers' cortisol and effortful control. *Infant and Child Development*, *21*(5), 537–554. https://doi.org/10.1002/icd.1759

Zalewski, M., Lengua, L. J., Kiff, C. J., & Fisher, P. A. (2012). Understanding the relation of low income to HPA-axis functioning in preschool children: Cumulative family risk and parenting as pathways to disruptions in cortisol. *Child Psychiatry & Human Development*, *43*(6), 924–942. https://doi.org/10.1007/s10578-012-0304-3

Zalewski, M., Lengua, L. J., Thompson, S. F., & Kiff, C. J. (2016). Income, cumulative risk, and longitudinal profiles of hypothalamic-pituitary–adrenal axis activity in preschool-age children. *Development and Psychopathology*, *28*(2), 341–353. https://doi.org/10.1017/S0954579415000474

Zandstra, A. R. E., Hartman, C. A., Nederhof, E., van den Heuvel, E. R., Dietrich, A., Hoekstra, P. J., & Ormel, J. (2015). Chronic stress and adolescents' mental health: Modifying effects of basal cortisol and parental psychiatric history. The TRAILS study. *Journal of Abnormal Child Psychology*, *43*(6), 1119–1130. https://doi.org/10.1007/s10802-014-9970-x

Zeiders, K. H., Doane, L. D., & Roosa, M. W. (2012). Perceived discrimination and diurnal cortisol: Examining relations among Mexican American adolescents. *Hormones and Behavior*, *61*(4), 541–548. https://doi.org/10.1016/j.yhbeh.2012.01.018

Zeiders, K. H., Hoyt, L. T., & Adam, E. K. (2014). Associations between self-reported discrimination and diurnal cortisol rhythms among young adults: The moderating role of racial-ethnic minority status. *Psychoneuroendocrinology*, *50*, 280–288. https://doi.org/10.1016/j.psyneuen.2014.08.023

Zhu, Y., Chen, X., Zhao, H., Chen, M., Tian, Y., Liu, C., & Qin, S. (2019). Socioeconomic status disparities affect children's anxiety and stress-sensitive cortisol awakening response through parental anxiety. *Psychoneuroendocrinology*, *103*, 96–103. https://doi.org/10.1016/j.psyneuen.2019.01.008