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Cortisol Secretion and Change in Sleep Problems in Early Childhood: Moderation by Maternal Overcontrol

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Abstract

Childhood sleep problems are prevalent and relate to a wide range of negative psychological outcomes. However, it remains unclear how biological processes, such as HPA activity, may predict sleep problems over time in childhood in the context of certain parenting environments. Fifty-one mothers and their 18–20 month-old toddlers participated in a short-term longitudinal study assessing how shared variance among morning levels, diurnal change, and nocturnal change in toddlers' cortisol secretion predicted change in sleep problems in the context of maternal overprotection and critical control. A composite characterized by low variability in, and, to a lesser extent, high morning values of cortisol, predicted increasing sleep problems from age 2 to age 3 when mothers reported high critical control. Results suggest value in assessing shared variance among different indices of cortisol secretion patterns and the interaction between cortisol and the environment in predicting sleep problems in early childhood.

Keywords

HPA axis; sleep/wake; parental care

Childhood sleep problems are prevalent (Halbower & Marcus, 2003) and relate to a wide range of negative psychological outcomes both in childhood and extending into adulthood (e.g., Alfano, Zakem, Costa, Taylor, & Weems, 2009; Forbes, Bertocci, Gregory, Ryan, Axelson, Birmaher, & Dahl, 2008; Gregroy, Caspi, Eley, Moffitt, O'Connor, & Poulton, 2005; Hartzinger, Brand, Perren, Stadelmann, von Wyl, von Klitzing, & Holsboer-Traschler, 2008; Johnson, Chilcoat, & Breslau, 2000). To work towards the prevention of such problems, it is important to further study both biological and environmental contributions to sleep difficulties in young children. Given that sleep problems are thought to reflect, in addition to other influences, biological dysregulation (Buxton, Spiegel, & Van Cauter, 2002), they may be predicted by other biological processes relevant to stress. In particular, aspects of HPA activity (i.e., cortisol levels), which are thought to reflect regulation of the

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stress response system, have been found to be related to sleep problems in children and adults (Garde, Karlson, Hansen, Persson, & Akerstedt, 2012; Kumari, Badrick, Ferrie, Perski, Marmot, & Chandola 2009; Scher, Hall, Zaidman-Zait, & Weinburg, 2010; White, Gunnar, Larson, Donzella, & Barr, 2000). Given that cortisol secretion may reflect dysregulated stress response and that stress may impair sleep, it seems likely that these processes are related. However, whether aspects of cortisol functioning *predict* sleep problems over time in early childhood, a salient period for prevention, remains unclear and requires a longitudinal design. Moreover, little research has addressed contexts that may affect this association. Theoretical and empirical work suggests the parenting environment often determines whether or the extent to which biological processes relate to maladjustment. Parental overcontrol, in particular, has been found to shape a variety of child outcomes (McLeod, Wood, & Weisz, 2007; McShane & Hastings, 2009; Rothbaum & Weisz, 1994; Van Leeuwen, Mervielde, Braet, & Bosmans, 2004) and is thought to create a stressful family environment (Chorpita & Barlow, 1998) that could serve as a context under which biological stress dysregulation influences toddlers' sleep quality. Studying sleep problems in the context of parenting seems particularly important in toddlerhood because, during this period, parents play an important role in helping children navigate important developmental issues (Calkins & Hill, 2007). Whether parenting affects how cortisol secretion predicts sleep problems remains unknown. Thus, the goal of the current study was to determine how multiple indices of toddlers' cortisol secretion (morning levels, diurnal change, and nocturnal change) predict change in sleep problems in the context of overcontrolling parenting. Below, we describe why sleep problems are so disruptive for child development, how cortisol and sleep have been associated in past studies, and why overcontrolling parenting might affect this association.

Sleep Problems in Childhood

Sleep plays an essential role in children's brain development and the emergence of adaptive emotional, cognitive, and behavioral regulation (Dahl, 1996). Unfortunately, sleep difficulties are prevalent in young children, with estimates ranging from 25% to 46% in the infancy and preschool years (Halbower & Marcus, 2003). Common sleep problems in childhood include short sleep duration, delayed sleep onset, poor sleep quality, and nighttime awakenings (Carter & Briggs-Gowan, 1993; Hartzinger et al., 2008).

Previous research suggests that sleep problems in childhood relate to a wide range of negative socioemotional outcomes. For example, decreased sleep efficacy has been associated with increased levels of impulsivity, peer victimization, and social inhibition in kindergarten children (Hartzinger et al., 2008). Other research has found significant associations between youths' sleep problems and their concurrent depressive and anxious symptoms (Alfano et al., 2009; Forbes et al., 2008; Johnson et al., 2000), and sleep problems in childhood have also shown prospective relations to anxiety and depressive disorders in adulthood (Gregory et al., 2005). Additionally, infant sleep disruptions have also been related to decreased psychological well-being in mothers (Brand, Furlano, Sidler, Schulz, & Holsboer-Trachsler, 2014), which could have negative implications for the parent-child relationship. Investigation into predictors of the development of sleep problems would

therefore assist in the early identification of children at risk for these problems and inform intervention and prevention efforts.

Cortisol Secretion and Sleep

An intermediary step in understanding the development of sleep problems that may confer risk for negative socioemotional outcomes is to investigate potential biological mechanisms that may underlie sleep problems. In general, sleep patterns are associated with the functioning of the neuroendocrine system, and the hypothalamic-pituitary-adrenal (HPA) axis has received particular attention. The HPA axis is one of the body's stress response and regulation systems, and the secretion of the hormone cortisol is an end-product of a chain of events signifying the activation of this system (Gunnar & Quevedo, 2007). Dysregulation of the stress response system is thought to contribute to sleep problems and disorders, such as insomnia (Buckley & Schatzburg, 2005). Thus, biological indices of stress appear to be related to sleep quality. Additionally, cortisol secretion may be a significant process in relation to sleep problems because the HPA axis follows a normal circadian rhythm related to the sleep-wake cycle. Typically, the sleep-wake cycle is characterized by a nadir in cortisol during the first few hours of nighttime sleep, a steady rise in cortisol into the waking hours, a peak after waking, and then a gradual decline in cortisol throughout the day (Buckley & Schatzberg, 2005). In typically developing children, significant decreases in cortisol have been observed between wake-up and mid-morning and between mid-afternoon and bedtime, and these patterns of cortisol secretion throughout the day are thought to be adaptive and relate to the emergence of children's self-regulation abilities (Watamura, Donzella, Kertes, & Gunnar, 2004). Therefore, a secretion pattern characterized by higher morning levels, a more negative diurnal slope, and a higher nocturnal increase seem to be shared features of an adaptive cortisol secretion pattern. In other words, steeper diurnal decline and nocturnal incline are positive.

Sleep difficulties have been related to HPA dysfunction in adults (Garde et al., 2012; Kumari et al., 2009), and a small number of studies have examined this association in children. These studies with adults suggest that, whereas sleep characterized by shorter duration and more disruption appears to be related to higher evening cortisol and a flatter daytime slope (Kumari et al., 2009), longer sleep duration is related to more dynamic cortisol secretion, with low evening cortisol levels, steep diurnal deviation of cortisol, and high area under the curve (Garde et al., 2012). Studies with infants and children have found that fragmented or otherwise poorer quality sleep has been related to higher awakening/morning and afternoon cortisol levels (Brand, Furlano, Sidler, Schulz, & Holsboer-Trachsler, 2011; El-Sheik et al., 2008; Hatzinger et al., 2008; Mueller, Kalak, Schwenzer-Zimmerer, Holsboer-Trachsler, & Brand, 2014; Scher et al., 2010). The extent to which diurnal decline and nocturnal incline in cortisol secretion relate to sleep is less clear. Less sleep has been related to blunted or flattened rhythm of cortisol secretion in infants with colic (White et al., 2000), but others have found that a sharper increase from evening to morning relates to sleep problems (Scher et al., 2010). Taken together, these studies suggest that high values at a single time point relate to sleep problems but do not yield a consistent answer about whether flatter or steeper slopes across the day and night relate to sleep problems. One approach that might clarify these discrepancies is assessing multiple indices

of cortisol secretion simultaneously and determining how shared variance among them (or patterns) relates to sleep problems. To this end, the current study included measures of morning cortisol, diurnal slope, and nocturnal slope.

Foundational work on the concurrent relation between cortisol secretion and sleep problems has stimulated interest in understanding the directionality of this association. Research focused primarily on adults suggests that the relation between sleep and HPA activity may be bidirectional, such that HPA dysfunction may be both a cause and a consequence of sleep problems (Buckley & Schatzberg, 2005; Garde et al., 2012). Prospective research with young children may be particularly suited to answering questions about the development of sleep problems. Few of these studies exist, although there are notable exceptions. Hatzinger and colleagues (2013) found that sleep problems did not predict cortisol secretion from 5.4 to 6.4 years despite being concurrently related at the latter time point, but cortisol was not assessed as a predictor of sleep. El-Sheikh and colleagues (2008) found that children with higher afternoon cortisol levels exhibited increased sleep disruptions as measured by self-report and actigraphy. However, it is unclear whether these children started out with higher cortisol levels in the morning or did not decrease as much during the day, which could be informed by studying multiple aspects of cortisol secretion patterns. Together, these studies warrant the investigation of the predictive relation between more holistic patterns of cortisol secretion and sleep problems. Inconsistency of results across studies also suggests that moderators may determine the extent to which cortisol secretion predicts sleep problems. We suggest that overcontrolling parenting, which may evince stress that also relates to cortisol secretion and sleep, may play such a role.

The Context of Overcontrolling Parenting

Overcontrolling parenting behavior likely influences children's experience of stress, which would be reflected in both their cortisol secretion patterns and their sleep quality. Two aspects of overcontrolling parenting, overprotection and critical control, may be particularly important to examine in relation to sleep problems, as they have been associated with a range of maladaptive developmental outcomes (McLeod, Wood, & Weisz, 2007; Mills & Rubin, 1998; Rothbaum & Weisz, 1994; Van Leeuwen et al., 2004). Overprotective parenting involves excessive comforting and the facilitation of avoidance of stressors, which limits children's independent coping and impedes autonomy (Rubin, Burgess, & Hastings, 2002). Parents may also control their children through derisive or critical responses, rejection, and negative labeling, a constellation of behaviors characterized as critical control (McShane & Hastings, 2009). These parenting behaviors are thought to create a stressful environment for the child in that they foster a cognitive style characterized by interpretations of events as out of one's control, thereby creating stress and psychological vulnerabilities for problems like anxiety (Chorpita & Barlow, 1998).

The association between parental overcontrol and increased stress in children is supported by evidence that overcontrol is related to children's cortisol secretion. For example, Hutt, Buss, and Kiel (2013) found that caregivers' protective behavior related to toddlers' increased cortisol reactivity and accounted for the association between toddlers' sadness and their cortisol reactivity. Additionally, Zalewski and colleagues (2012) found a significant

indirect effect of low income on low morning cortisol levels and flatter diurnal slope through maternal negativity.

Overcontrolling parenting has also been linked to sleep disruption. Much of the research investigating the parenting context in relation to children's sleep has focused on parenting behavior at nighttime (e.g. Teti & Crosby, 2012; Teti, Kim, Mayer, & Countermine, 2010), finding that maternal proximity and mother-child contact at night relate to greater infant awakenings throughout the night. However, there is also evidence that the quality of the parent-child relationship more broadly relates to children's sleep quality. For example, Bell and Belsky (2008) found that maternal insensitivity and conflicted mother-child relationships in the third grade predicted increases in children's sleep problems between the third and sixth grade. Similarly, the difficulty of the family environment relates to shorter sleep duration and higher cortisol secretion (Hanson & Chen, 2010). Brand and colleagues (2009) also found that negative parenting style, defined as reproach, restriction, and inconsistency, related to lower sleep quality and increased daytime sleepiness in a sample of adolescents. Conversely, a more sensitive and supportive caregiving environment related positively to children's percentage of nighttime sleep (Bordeleau, Bernier, & Carrier, 2012) and to adolescents' perceived sleep quality (Tynjala, Kannas, Levalahti, & Valimaa, 1999). Thus, overcontrol has been linked to children's cortisol secretion as well as sleep problems.

Recent developmental theories have suggested that, in addition to studying direct effects between either parenting or children's biology and children's outcomes, it is important to study biological processes in the context of environmental influences (e.g., Calkins, Propper, & Mills-Koonce, 2013). Examining how parental overcontrol moderates the association between children's biology and an important outcome like sleep quality acknowledges the complexity of development as unfolding among multiple levels of influence. In our study, specifically, overcontrol may make the environment more stressful for young children, and the presence of increased environmental stress may increase the extent to which biological indices of stress dysregulation such as cortisol secretion affect toddlers' sleep quality.

Current Study

Although the literature supports associations among HPA activity, sleep, and parenting, several gaps still remain. First, previous studies tend to focus on specific aspects of HPA activity in isolation or examine them additively rather than examining shared variance among pieces of an overall pattern. Investigating multiple facets of cortisol secretion simultaneously would allow better understanding of broader patterns of HPA activity and associated outcomes, and may help identify the salient features of cortisol secretion that increase children's risk for negative outcomes. Thus, the current study aimed to examine three features of cortisol: morning levels, diurnal change, and nocturnal change. Additionally, although the literature supports a relation between HPA activity and children's sleep, few studies have prospectively examined how cortisol predicts children's sleep problems, and virtually no studies have done this in the context of the caregiving environment. Thus, the goal of the current study was to understand how biological and environmental factors work together in order to create a more comprehensive picture of children's risk for sleep problems.

Given previous research on cortisol secretion patterns, we hypothesized that blunted diurnal and nocturnal cortisol change would coalesce and, together, predict increasing sleep problems across the toddler years when mothers reported more overcontrol (i.e., higher overprotection and critical control). It was unclear from the extant literature whether morning levels would be closely tied to low variability. We did not expect these features to be associated with a high morning level, but whether morning levels would be uncharacteristically low or more various was uncertain. Conversely, we hypothesized that features indicating a high morning level, sharper decline across the day, and sharper increases overnight would coalesce and either have no relation or a negative relation to changes in sleep problems across levels of maternal overcontrol.

Methods

Participants

Eighty-eight 18–20 month-old toddlers and their mothers participated at Time 1 of the short-term longitudinal study from which these data were derived. The current study includes the 51 of these toddlers ($M_{\text{age}} = 18.96$ mo, $SD_{\text{age}} = 0.81$ mo; 26 female) who provided at least one interpretable cortisol value and were not excluded for other reasons (see Missing Data section below). This sample of mothers and toddlers, respectively, comprised 45 (88.2%) and 42 (82.4%) European Americans, 1 (2.0%) and 1 (2.0%) identified as Latina/Latino Americans and no other race/ethnicity, 4 (7.8%) and 2 (3.9%) Asian Americans, 1 (2.0%) and 0 (0.0%) American Indians, and 0 (0.0%) and 6 (11.8%) biracial individuals. Three additional mothers (5.9%) identified Hispanic/Latino in their child's racial/ethnic background in addition to one of the designations above. Mothers were asked to indicate which of several ranges of incomes most accurately described them. Two mothers (3.9%) indicated an income less than \$15K/year, zero (0.0%) indicated \$16–20K, 5 (9.8%) indicated \$21–30K, five (9.8%) indicated \$31–40K, eight (15.7%) indicated \$41–50K, four (7.8%) indicated \$51–60K, 22 (43.1%) indicated an income of greater than \$60K, and five (9.8%) declined to answer. Mothers' number of years of education represented, on average, a college education ($Mean = 16.88$ years, $SD = 2.34$ years) but ranged from 11 to 20+ years. Of these 51 participants, 46 (90%) completed an assessment at Time 2 when toddlers were approximately 24-months-old, and 38 (75%) completed an assessment at Time 3 when toddlers were approximately 36-months-old.

Procedure

At Time 1, each mother-toddler dyad came to the laboratory for procedures not discussed further in the current study. At the beginning of the visit, mothers completed informed consent and a demographics questionnaire. At the end of the procedures, mothers were given supplies to collect cortisol from their infants and instructed to collect samples at standard times in the morning (8:00 – 9:00 am), afternoon (3:00 – 4:00 pm), and evening (7:00 – 8:00 pm) on two consecutive days. They refrigerated samples until all were taken and then mailed them to the laboratory in a pre-addressed, pre-stamped mailer. Samples were stored at the laboratory at -50°C until they were mailed for analysis. At Time 2, when toddlers were approximately 24 months old, mothers were invited to complete a packet of questionnaires assessing parenting and their toddlers' sleep problems. At Time 3, when toddlers were

approximately 36-months-old, mothers were asked to complete the measure assessing sleep problems again so that change could be examined.

Measures

Salivary cortisol—Mothers were trained by laboratory staff to gather saliva from their toddlers using a cotton dental roll and asked to complete a form each day about the times of the samples and the toddler's sleep/wake times, symptoms (i.e., runny nose/cough, warm/flushed, cranky/irritable even when rested, diaper rash, ear infection, teething), and medication use (i.e., antibiotics, Tylenol, other medication). Laboratory staff instructed mothers to place the cotton roll inside the toddler's mouth and gather saliva by moving it under the tongue and inside the cheek for approximately one minute or until the roll was saturated. Mothers were instructed to gather samples prior to meals and snacks, but if this was not possible, to wait 30 minutes after the toddler finished eating. The roll was then placed in a Salivette tube (Sartstedt, NC) and sealed. Once all samples had been collected, they were shipped to Biochemisches Labor in Trier, Germany on dry ice, where they were stored at -20°C until assayed for cortisol. Samples were centrifuged at 2000 g for 10 minutes and assayed using a competitive solid phase time-resolved fluorescence immunoassay with fluoroimetric end point detection (DELFI). The test used 100 μl of saliva, and all samples were analyzed in duplicate. Testing had a range of sensitivity from 0.30–100 nmol/l. Average intra- and inter-assay coefficients of variation ranged from 4.0–6.7% and 7.1–9.0% respectively. Outliers (> 3 SD above the mean) were truncated to a value equal to 3 SD above the mean value for the particular sample. Thus, no values were excluded as outliers. Cortisol values were subjected to a $\log_{10}$ transformation prior to analysis.

Maternal parenting behaviors—Mothers completed the New Friends Vignettes (NFV; McShane & Hastings, 2009), which provides mothers with two hypothetical vignettes in which they are asked to imagine their toddlers displaying shy/reticent behavior with unfamiliar peers. Mothers were asked whether they would display particular responses and could respond “No,” “Maybe,” or “Yes” (scored 0, 1, and 2, respectively). The current study focused on items indicating overprotective behaviors (12 items; $\alpha = .80$; e.g., “I would say, ‘Do you want to go home and play with Mommy?’”) and critical control (12 items; $\alpha = .76$; e.g., “I would say: ‘You don’t need to act like this’”). The NFV has demonstrated good psychometric properties, including convergent validity with mothers’ observed behaviors (McShane & Hastings, 2009).

Toddler sleep problems—Mothers completed the Infant-Toddler Social and Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 1993), a 168-item questionnaire measure designed to assess social-emotional and behavioral problems in 12–36-month-old children. Mothers complete items using a 3-point scale ranging from 0 (*not true/rarely*) to 2 (*very true/often*). Previous research has evaluated test-retest reliability of this measure, with coefficients ranging from .82 to .90 for the different subscales, and validity has been supported by associations with similar measures of child behavior problems (Carter, Briggs-Gowan, Jones, & Little, 2003). Of relevance to the current study was the Sleep scale (5 items; $\alpha = .76$; e.g., “Has trouble falling or staying asleep”).

Results

Missing Data

Of the 88 toddlers who participated at Time 1, 54 provided at least one interpretable cortisol value (one mother refused saliva collection and other samples did not have enough saliva for assaying. Of the 54, one toddler was excluded from analyses due to having an ear infection, and two toddlers' data could not be used because time of day was not recorded for the home samples in one case or time of waking in the other. This left 51 toddlers with at least one interpretable cortisol value (38 had Day 1 morning, 34 had Day 1 afternoon, 40 had Day 1 evening, 36 had Day 2 morning, 36 had Day 2 afternoon, and 35 had Day 2 evening). Of these toddlers, 15 (29.41%) had all six samples, 12 (24.5%) had five samples, 6 (11.76%) had four samples, 6 (11.76%) had three samples, 5 (9.80%) had two samples, and 7 (13.73%) had one sample. The majority of toddlers ($n = 42$; 82%) had at least 2 samples covering more than one time period (morning, afternoon, evening). Of these 51 participants, five did not have the questionnaires from the Time 2 assessment, and 13 were missing maternal perceptions of sleep problems at Time 3. Participants missing Time 2 measures did not differ from the others in terms of demographic or cortisol variables ($ts < 1.72$, $ps > .05$), and those missing Time 3 sleep problems did not differ from other participants in terms of demographics, cortisol variables, or Time 2 measures ($ts < 1.69$, $ps > .05$). Moreover, a non-significant Little's MCAR test ($\chi^2[234] = 237.652$, $p = .421$) suggested that missing data did not deviate from the pattern of missing completely at random.

Missing values within these 51 toddlers were imputed using multiple imputation. Recent research has found that children's noncompliance with saliva sampling relates to both cortisol levels and adjustment outcomes, suggesting that missing cortisol values may occur non-randomly (Kaitz, Sabato, Shalev, Ebstein, & Mankuta, 2012). Multiple imputation is the recommended approach for preventing biased results when data are not missing completely at random (Graham, 2009). Further, recommendations suggest completing statistical transformations prior to imputation (Graham, 2009), so available cortisol values were first investigated for deviations from normality. Skew of cortisol values ranged from 2.38 to 4.18 across the six samples (Day 1 morning, Day 1 afternoon, Day 1 evening, Day 2 morning, Day 2 afternoon, Day 2 evening), and some raw cortisol values were below 1, so a constant of 1 was added to all values, which were then subjected to a log transformation (resultant skew ranged from 0.41 to 1.47). It has also been recommended that auxiliary variables be included in the imputation algorithm, so the symptoms and medication usage variables listed above were investigated in relation to cortisol values. The only relation that emerged was that Day 1 runny nose/cough was related to Day 1 evening cortisol ($t = 2.15$, $p = .038$). Using available cortisol values (log transformed), number of hours since waking for each sample, Day 1 runny nose/cough, and Time 2 and Time 3 survey measures, 20 imputations were computed. Given the differing types of intended analyses (principal components analysis, multilevel modeling, multiple regression), and because precise values were required for the computation of several variables, values across these 20 imputed data sets were averaged so that each toddler had one value for each variable. This approach has the limitation of not being able to weight parameter estimates by standard errors in deriving a

pooled estimate, but it was judged to provide more reliable estimates than a single imputation.

Derivation of Cortisol Variables

Examination of toddlers' cortisol secretion patterns occurred by several means. First, multilevel modeling (MLM) was used to estimate initial levels and linear change across the day. MLM accounts for the nesting of the six samples within each toddler and allows Time to be included as a variable with different precise values for each toddler. Samples were not averaged across the two days in order to preserve within-toddler variability, which can be modeled in MLM. Cortisol values served as the dependent variable, and the predictor, Time, was derived by calculating the number of hours since waking for each sample. In this manner, the intercept reflects the estimated cortisol value at waking. We acknowledge that this value does not reflect a precise measure of children's wake-up level or cortisol awakening response. So as not to create confusion with "basal levels" or the "waking response," which were not measured, we refer to this estimated value as "morning level." Day 1 runny/nose cough was included as a covariate in all analyses. One toddler did not have this information, so variables derived from MLM have an n of 50. In an empty model with no predictors, the intraclass correlation coefficient for cortisol values was found to be .21, suggesting that 21% of the variance in cortisol values was between toddlers, warranting the multilevel approach. When Time was added to the model, we tested whether a random component improved model fit above and beyond the fixed effect. Improvement in model fit was significant ($\chi^2_{[2]} = 9.80, p = .007$), suggesting that linear change in cortisol values across the day varied across toddlers. Within this model, we found the expected decrease in cortisol values across the day ("diurnal change") across the sample ($b = -0.024, SE = 0.004, t[236] = -6.76, p < .001$). MLM also allowed us to extract an Empirical Bayes estimate of each toddler's estimated intercept (morning level) and slope (diurnal change) to use as a variable in further analyses. Higher intercept values indicated higher estimated morning levels, and lower, more negative slopes indicated sharper declines throughout the day (so higher values indicated flatter slope).

We were also interested in the nocturnal change in cortisol values for each toddler. We subtracted the Day 1 evening sample value from the Day 2 morning sample value (AM – PM). As such, higher numbers reflect greater change overnight. Thus, the variables available for each toddler were morning level (i.e., intercept value), diurnal change (i.e., slope value), and nocturnal change (i.e., difference score). Morning level and diurnal change were positively related ($r = .50, p < .001$). Diurnal and nocturnal change were negatively related ($r = -.60, p < .001$). Morning level and nocturnal change were not related ($r = .16, p = .267$).

To understand how shared variance among these variables related to other primary constructs of the current study, we subjected these three variables to a principal components analysis (PCA). PCA reduces observed variables into a smaller number of components that account for shared variance among them without assuming an underlying latent construct or modeling unique variance, as in the case of exploratory factor analysis. This analysis yielded two components that together accounted for 95.45% of the variance in variables. The first component (accounting for 56.88% of the variance) comprised diurnal change (.97),

nocturnal change, which loaded negatively ($-.71$), and, to a lesser extent, morning level ($.52$). Thus, toddlers scoring highly on this component showed flatter diurnal change (less of a decrease) and less change between evening and morning. Having a higher morning level was somewhat, but not strongly, characteristic of this component. We therefore refer to this component as “blunted.” The second component (accounting for 38.57% of the variance) comprised nocturnal change ($.68$) and morning level ($.83$) but not diurnal change ($.05$). Toddlers scoring highly on this component display higher morning levels and steeper cortisol increase overnight. Although diurnal change was not included in this component, to be able to have a high morning value and steep increase overnight, a steeper decrease across the day would also seem necessary (and would be consistent with the strong negative correlation between diurnal and nocturnal change). We refer to this component as “steep.”

Preliminary Statistics

Descriptive statistics for primary study variables are included in Table 1. All variables demonstrated reasonable adherence to a normal distribution ($\text{skew} < |2.00|$). No variables differed according to toddler gender (all t s < -1.38 , p s $> .15$), so it is not considered further. Bivariate relations among variables are also presented in Table 1. Notably, overprotection and critical control were positively correlated. To understand the unique role of these parenting behaviors, each was included as a covariate in subsequent analyses examining the other. The relations between demographic variables and sleep were investigated for identification of other potential covariates, but no significant relations were found (r s $< .18$, p s $> .20$).

Moderation Analyses

We used multiple regression moderation analyses to determine how the cortisol secretion patterns predicted change in sleep problems in the context of maladaptive parenting. Each model contained both parenting variables to isolate effects due to the specific type of parenting and not shared by maladaptive parenting in general. To maintain adequate power, each model contained one cortisol variable (blunted or steep pattern) and its interaction with one parenting variable. Each analysis controlled for age 2 sleep problems while predicting age 3 sleep problems to assess change over time. A Type I error correction was not employed because it would limit power in the current sample size, so we report effect sizes in addition to statistical tests of parameter estimates. Variables were centered at their means prior to analyses. Significant interactions were probed by examining the simple slope of the cortisol variable at low (-1 SD), mean, and high ($+1$ SD) values of the parenting variable involved in the interaction. Regression models with mean-centered variables are summarized in Tables 2 and 3. Probing of significant interactions is reported in text.

Overprotection—Models focusing on overprotection are presented in Table 2. In Model 1, the interaction between the blunted pattern and overprotective parenting was not significant in predicting sleep problems. When the interaction term was dropped from the model, only age 2 sleep problems significantly predicted age 3 sleep problems; blunted cortisol and overprotective parenting did not predict sleep problems as main effects.

In Model 2, the interaction between the steep pattern and overprotective parenting was not significant in predicting sleep problems. When it was dropped from the model, a similar pattern of main effects emerged, such that only age 2 sleep problems predicted age 3 sleep problems.

Critical control—Models focusing on critical control are presented in Table 3. In Model 3, the interaction between the blunted pattern and critical control was significant in predicting sleep problems. Probing of this interaction (Figure 1) revealed that blunted cortisol secretion did not predict sleep problems at low ($\beta = -0.21$, $t = -1.17$, $p = .270$, $sr^2 = .018$) or mean ($\beta = 0.14$, $t = 1.10$, $p = .273$, $sr^2 = .018$) critical control, but it predicted increasing sleep problems at high levels of critical control ($\beta = 0.4$, $t = 2.23$, $p = .031$, $sr^2 = .073$). Thus, above and beyond overprotective parenting and initial sleep problems at age 2, the confluence of blunted cortisol secretion and critically controlling parenting predicted an increase in sleep problems.

To understand whether particular aspects of the cortisol pattern were driving these results, morning level, diurnal change, and nocturnal change were examined individually in similar regression analyses, with the morning level included as a covariate in models examining each of the two change patterns (allowing for a test of the influence of change regardless of the “starting” level for change). Morning level did not interact with critical control to predict sleep problems ($b = 2.68$, $SE = 3.03$, $\beta = 0.12$, $t = 0.88$, $p = .382$, $sr^2 = .012$). Each of the two change variables showed a trend towards interacting with critical control in predicting sleep problems (diurnal change: $b = 34.73$, $SE = 17.96$, $\beta = 0.24$, $t = 1.93$, $p = .060$, $sr^2 = .055$; nocturnal change: $b = -1.66$, $SE = 0.96$, $\beta = -0.26$, $t = -1.73$, $p = .091$, $sr^2 = .047$). These post-hoc analyses suggest that shared variability, rather than a single aspect of cortisol functioning, was the most powerful predictor of later sleep problems in the context of critically controlling parenting.

In Model 4, the interaction between the steep pattern and critical control was not significant. When it was dropped from the model, only age 2 sleep problems predicted age 3 sleep problems.

Discussion

The current study aimed to understand how shared variability among aspects of toddlers’ cortisol secretion patterns predicted mother-perceived sleep problems in the context of two types of overcontrolling parenting (overprotection and critical control). This longitudinal investigation spanned several years, with cortisol measured at 18–20 months, parenting assessed at age 2, and change in sleep problems assessed from age 2 to age 3.

Although cortisol values have previously been linked to toddlers’ sleep problems, few studies have assessed multiple aspects of cortisol functioning simultaneously and examined shared variability among them specifically. In the current study, two variables emerged when assessing shared variance among diurnal change, nocturnal change, and morning values of cortisol: one indicating a more blunted pattern (flatter negative slope across the day, less increase from evening to morning) and a second indicating steeper change (high

morning level, steeper nocturnal increase). These aspects of shared variability map onto previous literature demonstrating that the typical pattern of cortisol is to begin high in the morning, decrease throughout the day, and increase overnight (Buckley & Schatzberg, 2005). Toddlers scoring higher on our first variable may be considered to be demonstrating more atypical and potentially maladaptive cortisol secretion, while toddlers scoring higher on the second of our variables show aspects of cortisol secretion more closely aligned with the typical pattern.

The developmental and clinical psychology literatures have increasingly recognized that children's outcomes are multiply-determined, and that individuals and their environments may make both additive and interactive contributions in predicting these outcomes (Cicchetti & Toth, 2009; Sroufe & Rutter, 1984). Although recent research on children's sleep problems largely acknowledges these complexities, few studies have specifically investigated how biological and environmental factors influence these outcomes together. To this end, we investigated whether overcontrolling (i.e., overprotective, critically controlling) parenting, which tends to be associated with maladaptive outcomes in other domains (e.g., Mills & Rubin, 1998; Rothbaum & Weisz, 1994; Van Leeuwen et al., 2004), moderated the extent to which shared aspects of cortisol secretion predicted change in toddlers' sleep problems. We found that a component indicating blunted change in cortisol secretion across the day and night, in the context of critical control, predicted an increase in mothers' perceptions of toddlers' sleep problems from age 2 to age 3. Thus, toddlers' biology was most relevant to sleep when mothers reported being outwardly negative in what could be a stressful situation (e.g., joining new children to play). This is consistent with recent developmental models suggesting that children who are both biologically reactive and experiencing an in-optimal caregiving environment may be most at risk for adverse outcomes, such as internalizing and externalizing problems (e.g., Belsky & Pluess, 2009; Zuckerman, 1999). The current study suggests that this model also extends to young children's sleep problems. Moving forward, it is therefore important to consider parenting as a significant context in which cortisol secretion, stress reactivity, and biological functioning, more broadly, predict sleep outcomes. Moreover, the current study examined toddlers' biology and caregiving experiences in a diathesis-stress framework, focusing on negative parenting practices and maladaptive outcomes. Consistent with contemporary theories of biology-environment interactions such as the differential susceptibility hypothesis and biological sensitivity to context (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2011), it would be informative to also understand whether these shared aspects of cortisol secretion interact with an enriched and supportive parenting environment to predict healthy sleep patterns.

Critical control, in particular, may be an important moderator of outcomes for children exhibiting dysregulated cortisol patterns because these children may require more external regulation of daily stressors for the development of more regulated biological patterns, and critical control, conversely, undermines regulation. Critical control has been theorized to damage the security of the parent-child relationship (McShane & Hastings, 2009), which has negative implications for a broad array of regulation outcomes, including biological regulation (Calkins & Hill, 2007). An important avenue for future research may be explicitly testing parent-child relationship quality and other possible mechanisms of this moderated relation.

Notably, it was blunted, not steep cortisol secretion that predicted sleep problems in the context of critical control. These results are consistent with previous research with adults showing that flatter or blunted diurnal change in cortisol relates to disturbed and restless sleep (Garde et al., 2012). However, another study (Scher et al., 2010) found that toddlers with fragmented sleep exhibited a steeper, rather than flatter, increase overnight. It could be that discrepant results occurred between our study and previous studies because shared variability among different aspects of cortisol functioning offers more information than a single index. Indeed, when we analyzed the three aspects of cortisol secretion separately, diurnal and nocturnal change interacted more weakly with critical control to relate to sleep problems, and the interaction with morning level was not significant. This seems to suggest that a more holistic measure of cortisol secretion offers more than the sum of its parts. It is especially important that this result occurred specifically within the context of parenting characterized by high levels of critical control, which was not considered in the aforementioned studies.

Previous studies have established cortisol secretion to be a correlate or outcome of sleep problems (e.g., El-Sheik et al., 2008; Hatzinger et al., 2008; Scher et al., 2010), but the current study is one of the few to show that cortisol is important in predicting sleep problems. Our longitudinal design, and particularly the repeated assessment of sleep problems, allowed us to more confidently address cortisol as a predictor of sleep problems. Although we did not explicitly test bi-directional effects between cortisol secretion and sleep problems, our results, in the context of extant literature, suggest that these effects may be transactional in nature. Future research could be geared toward more purposely examining such bi-directional models for a more comprehensive understanding of the relation between cortisol secretion and sleep.

The results of the current study should certainly be considered in light of several methodological considerations and limitations. The current study used principal components analysis to assess shared variance among the different aspects of cortisol secretion indicating blunted versus steep patterns of change. Neither area under the curve (AUC) nor a single aspect or measure of cortisol secretion can provide this information. Further, the current study used a parent-report measure of sleep problems. Other methods of assessing sleep problems in youth include physiological measures, such as electroencephalography (e.g., Hartzinger et al., 2008) and actigraphy (e.g., Hartzinger, Brand, Perren, Stadelmann, von Wyl, von Klitzing, & Holsboer-Traschler, 2010) and sleep diaries (e.g., Bordeleau, Bernier, & Carrier, 2012). Although physiological measures of sleep may provide more precise measures of children's sleep behavior, parental report of children's sleep problems have been shown to relate to EEG sleep patterns (Hartzinger et al., 2008). For example, one study found that children of parents who viewed their child's sleep as impaired exhibited decreased sleep efficiency and more disturbed sleep measured by EEG (Hartzinger et al., 2008). Thus, parental report of sleep problems is still largely considered a valuable source of information and is commonly used in the pediatric sleep literature (Sadeh, 2008), although replicating results with other assessments of sleep problems is an important future direction.

Our sample was relatively small and limited our ability to analyze all variables in the same model. The sample was also fairly homogenous, comprised mainly of European American,

middle class mother-toddler dyads, and focused solely on mothers to the exclusion of fathers. Fathers certainly make important contributions to children's sleep patterns, and future work should delineate the relative, and perhaps interactive, contributions from mothers and fathers in this area. Diurnal cortisol patterns were estimated using standard collection times, rather than measuring children's true wake-up level or cortisol awakening response. We cannot make conclusions about how these variables, rather than the "morning level" we estimated, may interrelate with other cortisol measures and then relate to sleep problems. Moreover, we measured cortisol secretion, parenting behaviors, and sleep problems across different time points. Although this design was necessary to assess change in sleep problems over time, it may be prudent for future research to assess cortisol secretion and parenting at the same time point to determine whether timing of these assessments affects the moderation found here. Relatedly, sleep problems were not assessed at the first time point, so it unclear whether cortisol measures were related to sleep problems at this point, which could affect interpretation of results.

In conclusion, our results suggest that blunted cortisol secretion patterns may predict risk for early-emerging sleep problems, but only when children receive parenting characterized by above-average critical control. These results suggest future avenues for understanding psychobiological indices of risk while accounting for salient features of the early caregiving environment.

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Highlights

- Cortisol secretion (morning level, diurnal change, nocturnal change) was measured when toddlers were 18–20 months old.
- Principle components analyses yielded steep and blunted components
- Sleep problems were measured longitudinally, at 24 and 36 months of age
- Parenting was considered as a moderator of the relation between cortisol secretion and sleep
- Blunted cortisol secretion predicted increases in sleep problems in the context of maternal critical control.

Figure 1.

Interaction between the blunted cortisol secretion principal component and maternal critical control (CC) in predicting age 3 sleep problems. Age 2 sleep problems were included as a covariate, so outcome represents change in sleep problems.

* $p < .05$

Table 1

Descriptive Statistics and Bivariate Relations for Primary Study Variables

Variable	1	2	3	4	5	6	7	8	9
1. Cortisol morning level	--	.49***	.16	.52***	.83***	.04	.04	-.18	-.13
2. Cortisol diurnal		--	-.60***	.97***	.05	-.03	.09	-.26 [†]	.01
3. Cortisol nocturnal			--	-.71***	.68***	-.09	-.04	.09	-.04
4. Blunted pattern PC				--	.00	.03	.07	-.24 [†]	-.02
5. Steep pattern PC					--	-.02	.02	-.09	-.11
6. Overprotection						--	.31*	.16	.31*
7. Critical Control							--	.05	.10
8. Time 2 Sleep Problems								--	.49***
9. Time 3 Sleep Problems									--
<i>Mean (SD)</i>	0.83 (0.09)	-0.02 (0.01)	0.32 (0.31)	0.00 (1.00)	0.00 - 1.00	1.12 (0.30)	0.29 (0.26)	0.43 (0.48)	0.43 (0.36)
<i>Range</i>	0.66 - 1.21	-0.04 - 0.01	-0.37 - 1.05	-1.73 - 2.65	-1.91 - 3.47	0.33 - 1.83	0.00 - 1.17	0.00 - 2.00	0.00 - 1.40

Note. Descriptive and bivariate statistics were computed after multiple imputation of missing values. Cortisol morning level and cortisol diurnal represent Empirical Bayes estimates of intercepts and slopes, respectively, from multilevel modeling analyses. Cortisol nocturnal represents the difference between Day 2 morning and Day 1 evening values (Day 2 morning - Day 1 evening). PC = principal component. $n = 51$ except for statistics involving cortisol morning level, diurnal change, blunted pattern, and steep pattern ($n = 50$).

[†] $p < .10$,

* $p < .05$,

*** $p < .001$.

Table 2

Multiple Regression Models Predicting Time 3 Sleep Problems from Cortisol Variables and Overprotection

Variable	<i>b</i> (SE)	β	<i>t</i> -test	<i>sr</i> ²
<u>Model 1 ($R^2 = .29$, $F[5,44] = 3.58^{**}$)</u>				
Constant	0.30 (0.08)		3.71 ^{**}	
Time 2 Sleep Problems	0.34 (0.10)	0.47	3.49 ^{**}	.197
Critical control	−0.08 (0.19)	−0.05	−0.40	.003
Overprotection	0.23 (0.16)	0.19	1.44	.033
Blunted pattern PC	0.03 (0.05)	0.09	0.69	.008
Blunted PC X Overprotection	−0.11 (0.17)	−0.09	−0.69	.008
<u>Model 2 ($R^2 = .28$, $F[5,44] = 3.45^*$)</u>				
Constant	0.30 (0.08)		3.75 ^{**}	
Time 2 Sleep Problems	0.33 (0.10)	0.45	3.46 ^{**}	.195
Critical control	−0.07 (0.19)	−0.05	−0.34	.002
Overprotection	0.24 (0.16)	0.20	1.51	.037
Steep pattern PC	−0.02 (0.05)	−0.06	−0.50	.004
Steep pattern X Overprotection	0.07 (0.13)	0.07	0.50	.004

Note. PC = principal component. sr^2 = squared semi-partial correlation. Missing values of variables were imputed prior to regression analyses. Predictors were centered at their means prior to calculating interaction terms.

* $p < .05$,

** $p < .01$.

Table 3

Multiple Regression Models Predicting Time 3 Sleep Problems from Cortisol Variables and Critical Control

Variable	<i>b</i> (<i>SE</i>)	β	<i>t</i> -test	<i>sr</i> ²
<u>Model 1 ($R^2 = .35$, $F[5,44] = 4.79^{**}$)</u>				
Constant	0.27 (0.06)		4.58 ^{***}	
Time 2 Sleep Problems	0.34 (0.09)	0.47	3.72 ^{**}	.203
Overprotection	0.29 (0.15)	0.24	1.87 [†]	.051
Critical control	−0.10 (0.18)	−0.07	−0.54	.004
Blunted pattern PC	0.05 (0.05)	0.14	1.11	.018
Blunted pattern PC X Critical control	0.48 (0.22)	0.28	2.20 [*]	.071
<u>Model 2 ($R^2 = .28$, $F[5,44] = 3.39^{*}$)</u>				
Constant	0.28 (0.06)		4.62 ^{***}	
Time 2 Sleep Problems	0.33 (0.10)	0.45	3.47 ^{**}	.197
Overprotection	0.24 (0.16)	0.20	1.48	.036
Critical control	−0.04 (0.20)	−0.03	−0.21	.001
Steep pattern PC	−0.03 (0.05)	−0.07	−0.52	.004
Steep pattern PC X Critical Control	−0.04 (0.29)	−0.02	−0.13	.000

Note. PC = principal component. sr^2 = squared semi-partial correlation. Missing values of variables were imputed prior to regression analyses. Predictors were centered at their means prior to calculating interaction terms.

[†]
 $p < .10$,

^{*}
 $p < .05$,

^{**}
 $p < .01$,

^{***}
 $p < .001$