Daily Stress, Cortisol, and Sleep: The Moderating Role of Childhood Psychosocial Environments

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Objective: The purpose of this study was to explore whether childhood family environments moderated the relation between daily stress and daily biological outcomes (sleep, cortisol output) in healthy young adults. ***Design:*** There were 87 participants, ages 19 to 25 who provided information on characteristics of their childhood family environment (conflict, parental warmth). ***Main Outcome Measures:*** For 1 week they completed a daily stress checklist via electronic diary, provided salivary cortisol samples 4 times a day, and wore an Actiwatch to measure sleep (minutes, efficiency). Data was analyzed using hierarchical linear modeling. ***Results:*** Family risk significantly moderated the relation between daily number of stressors and sleep minutes ($b = -12.10$, $p = .02$), such that the more difficult one's childhood environment, the less sleep individuals got on days in which they experienced a greater number of stressors. Parental warmth moderated the relation between stress severity and cortisol output ($b = -0.19$, $p = .04$), such that the less parental warmth individuals received during childhood, the more cortisol they secreted on days that they experienced more severe stress. ***Conclusions:*** The childhood psychosocial environment may have long-term effects on biological responses to daily stress, creating vulnerability to disease in individuals from difficult childhoods.

Keywords: stress, cortisol, sleep, childhood environment

Psychological stress has been associated with poor health in individuals across the life span and in many countries (Lin & Ensel, 1989; Marmot & Wilkinson, 1999). For example, greater psychological stress has been linked to increased risk for cardiovascular disease, autoimmune disorders, infectious disease, and mental illness (McEwen, 1998). This relationship is so robust that it has been observed across the life span, from infants to older people (Graham, Christian, & Kiecolt-Glaser, 2006).

One mechanism linking psychological stress to disease is the biological responses that are associated with stress. Under high levels of acute stress, individuals exhibit a heightened activation of certain biological systems, termed the *fight-or-flight response* (Canon, 1932). The biological fight-or-flight response involves the activation of neural, neuroendocrine, and immune mechanisms that prepare the body to overcome or to avoid danger. Over time, it is possible that repeated activation of these systems can cause wear and tear on the body, referred to as allostatic load (McEwen, 1998), eventually leading to poor health.

Researchers have long known that stress does not have the same biological effects on all individuals (Miller, Chen, & Cole, 2009). As well, differences in biological responses to stress have implications for who is at greatest risk to develop diseases (McEwen, 1998). Therefore, previous researchers have sought to determine whether types of individual difference factors might help us to understand variability in individual biological responses to stress

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(Boyce & Ellis, 2005; Miller & Chen, 2007; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). In this study we assessed whether variations in early life environments could explain differences in the relation between daily stressful experiences and daily biological patterns in healthy young adults.

Previous research has suggested that characteristics of the childhood psychosocial environment may partially explain differences in biological responses to stress in adults (Miller & Chen, 2007; Repetti, Taylor, & Seeman, 2002). For example, according to Taylor and colleagues (2004), children who experience insecure attachments to caregivers, harsh parenting, or lack of social support exhibited greater secretion of stress hormones, higher heart rates, and higher blood pressure in response to an acute laboratory stressor in adulthood than individuals raised in more nurturing environments. In addition, early experiences of social deprivation, such as a child experiencing distant or cold parental relationships (Heim et al., 2000), or spending the first years of life in a Romanian orphanage (Rutter & O'Connor, 2004), also have been shown to result in heightened biological reactivity to stress in adulthood.

These early life experiences also have implications for later-life health problems. For example, a recent review of the literature reported that children raised in "risky families" (i.e., families characterized by harsh or cold parenting) were at greater risk for heart disease, cancer, chronic lung disease, and skeletal fractures in adulthood than children whose early life parental relationships were warm and nurturing (Repetti et al., 2002). Taken together, previous findings suggest that family relationships in childhood may continue to impact both biological stress responses and health in adulthood.

In the present study, we investigate whether childhood family environments affect the relationship between daily stress and daily cortisol and sleep patterns. The majority of previous research has

been laboratory based or cross-sectional, and it is important to understand how childhood environments may affect the relationship between naturally occurring stress and daily biological rhythms (van Eck, Nicolson, Berkhol, & Sulon, 1996). We focused on cortisol and sleep because dysregulations in both have been noted as markers of allostatic load (McEwen, 2007) and are risk factors for poor health (Cohen, Janicki-Deverts, & Miller, 2007; Dickerson & Kemeny, 2004). As well, both follow daily circadian patterns (Van Cauter, Polonsky, & Scheen, 1997), enabling us to assess whether daily stress was related to altered daily biological patterns. In addition, both the patterns of cortisol secretion and sleep are established early in life (Phillips & Jones, 2006; Rivkees, 2003), and hence form plausible targets that may be influenced by childhood family environments.

Stress and Cortisol

Cortisol is a hormone released by the hypothalamic-pituitary-adrenal (HPA) axis. Previous research has reported that a variety of stressful experiences alter cortisol secretion. For example, acute psychological stressors that involve social evaluation are known to increase cortisol levels (Dickerson & Kemeny, 2004). Chronic stressors have been found to initially increase cortisol secretion, but over time to result in blunted cortisol secretion (Miller, Chen, & Zhou, 2007). The majority of daily diary studies have reported that naturally occurring stressors elicited greater daily cortisol secretion (Peeters, Nicholson, & Berkhof, 2003; Schlotz, Schulz, Hellhammer, Stone, & Hellhammer, 2006; Smyth et al., 1998; van Eck et al., 1996, for an exception see Hanson, Maas, Meijman, & Godaert, 2000).

Furthermore, previous research has demonstrated that the early life environment also affects daily cortisol secretion. Nicolson (2004) found that parental loss during childhood was related to elevated daily cortisol in adulthood. As well, Heim and colleagues (Heim, Mletzko, Purselle, Musselman, & Nemeroff, 2008) reported that adult men with childhood histories of trauma showed hyperactive HPA responses to the administration of an exogenous steroid test (indicative of dysregulated cortisol secretion), as compared to men with no history of childhood trauma.

Furthermore, some researchers have argued that childhood environments may moderate the relationship between stress and cortisol. For example, some studies have found that difficult childhood environments predict increased cortisol response in response to acute laboratory stressors in adults (Luecken, 1998; Repetti et al., 2002, see Carpenter et al., 2007, for an exception), and conversely, that parental warmth mitigates the effects of an acute lab stressor on cortisol (Evans, Kim, Ting, Tesher, & Shannis, 2007). Similarly, animal research has found that nonhuman primates raised in stressful childhood environments displayed amplified cortisol responses to laboratory stressors as adults (Gorman, Mathew, & Coplan, 2002; Hennessy, 1997; Rosenblum, Forger, Noland, Trost, & Coplan, 2001), and that rats raised in nurturing environments display more modest HPA responses to restraint stress as adults (Caldji, Diorio, & Meaney, 2000; Meaney, 2001).

However, these past studies have focused on laboratory responses to stress. In the present study, we extend this previous research by focusing on naturalistically occurring daily life stressors, and testing whether childhood family environments moderate the relationship between naturally occurring daily stress and daily secretion of cortisol in young adulthood. We hypothesize that the relation between daily stress and increased cortisol secretion will be stronger the more difficult the childhood environment an individual comes from.

Stress and Sleep

Sleep is another process that is both important for health and affected by stress. Although it is also a behavior, sleep can be considered a biological process in that it is regulated by the brain stem, thalamus, hypothalamic hormones, and external stimuli (i.e., light; Dahl & Lewin, 2002; Hall, 1998). Experimental studies in both humans and animals have documented that stressors experienced during the day result in disruptions in sleep architecture, including longer transitions into REM sleep, at night (Cheeta, Ruigt, van Proosdij, & Willner, 1997). Naturally occurring stressors such as periods of marital separation are associated with less delta sleep (Cartwright & Wood, 1991). Daily diary studies also have demonstrated that daily stress is associated with poorer sleep (Åkerstedt, 2007; Ancoli-Israel & Roth, 1999; Hall et al., 2000; Tworoger, Davis, Vitiello, Lentz, & McTiernan, 2005; Urponen, Vuori, Hasan, & Partinen, 1988). Effects of stress on sleep are sometimes apparent only in certain subgroups (Dagan, Zinger, & Lavie, 1997; Hall et al., 1997; Pillar, Malhotra, & Lavie, 2000; Sadeh, Keinan, & Daon, 2004), and occasionally, studies have reported no significant relationship between stress and sleep (Paulsen & Shaver, 1991).

Studies investigating childhood adversity also have found associations with sleep, using both human and animal models. For example, in a longitudinal study by Gregory and colleagues (Gregory, Caspi, Moffitt, & Poulton, 2006), greater family conflict in childhood predicted symptoms of insomnia at age 18, over and above the current psychosocial environment. Similarly, animal studies have shown that primates separated from their mothers displayed sleep disturbances including a greater number of arousals and decreased REM sleep (Reite & Snyder, 1982). Conversely, childhood environments characterized by parental warmth have been shown to be predictive of earlier bedtimes and longer sleep times (Adam, Snell, & Pendry, 2007).

In the present study, as with cortisol, we tested whether childhood environments might moderate the relationship between daily stress and sleep, hypothesizing that the relation between greater daily stress and poorer sleep (i.e., shorter duration, lower efficiency) will be stronger the more difficult the childhood environment an individual comes from.

Method

Participants

There were 87 healthy college undergraduate students who participated in the study. They were recruited from the University of British Columbia in Vancouver, Canada through campus postings. Students were eligible to participate in the study if they (1) were between the ages of 19 and 25 years, (2) were medically healthy, and (3) were fluent in English. The study sample was 67% women, and was 28% White, 57% Asian, and 15% other.

Measures

Childhood family psychosocial environment.

Risky Families Questionnaire.   The Risky Families Questionnaire (Taylor et al., 2006) measures the level of family conflict as well as parental coldness/lack of affection in the family environment during childhood. Response options are based on a 4-point Likert scale ranging from 1 (*none of the time*) to 4 (*most or all of the time*). Sample items include, "how often would you say there was quarreling, arguing, or shouting between a parent and you?" and, "how often would you say you were left on your own to fend for yourself?" Higher scores indicate greater family conflict/risk. Previous research has demonstrated that this measure has high reliability ($\alpha = .77$). As well, responses to the Risky Family Questionnaire are highly related to responses to interviews regarding early life family environments, demonstrating adequate validity (Taylor et al., 2004).

Parental warmth.   We also considered the effects of positive childhood family characteristics on responses to daily stress. Participants reported the degree of warmth in their parental relationships by completing the 13-item Parent Bonding Inventory (PBI; Parker, 1979). Participants reported on a 5-point Likert scale, from 0 (*not at all true*) to 4 (*very much true*). Sample items included, "my mother spoke to me in a warm and friendly voice," and, "my father frequently smiled at me." We utilized the parental warmth/care dimension in this study. The inventory has demonstrated adequate test–retest reliability, ranging from .60 to .79 and has demonstrated adequate validity when scores were compared between MZ and DZ twins, as well as with interview-based ratings (Wilhelm & Parker, 1990).

Daily diary variables.

Daily stressors.   Participants reported both the number of stressors that occurred during their day, as well as the negative impact that the most bothersome stressor of the day had on them using a web-based diary format. Once an evening for 7 days participants were asked to check off which of a list of 16 items they had experienced within the last 24 hr, such as academic deadlines, relationship problems with a friend/colleague/family member/girl or boyfriend, and financial insecurity. The number of items that participants endorsed was summed to indicate the number of stressors they experienced that day. Participants were then asked to select the most severe stressor experienced during the day and respond to the question, "How serious was this for you?" Responses ranged from 1 (*not at all*) to 5 (*very serious*). This measure was a modified version of the Hassles Scale (from the Hassles and Uplifts Scales; DeLongis, Folkman, & Lazarus, 1988). Previous research has demonstrated significant relations between the Hassles Scale and other life event scales (Holmes & Rahe, 1967), psychological symptoms scales (Derogatis, Lipman, Covi, Rickels, & Uhlenhuth, 1970), and health symptom checklists (DeLongis et al., 1988).

Sleep.   Participants were instructed to wear an ambulatory wristwatch monitor called an Actiwatch (MiniMitter Co., Boulder, CO) for 7 nights following their lab visit. Participants were instructed to wear the watch at all times. The Actiwatch measures gross motor movement via a sensor that generates a voltage when the Actiwatch senses acceleration. Watches are worn on the nondominant arm and can be worn in the shower. Sleep quantity and efficiency (e.g., percentage of the sleep interval in which the

person is motionless) for each night was calculated using the Actiwatch software. More specifically, sleep intervals were extracted from the data using the sleep algorithm provided by Actiware (http://www.learnactiware.com/).

Cortisol.   Participants collected salivary cortisol samples using Salivettes (Sarstedt, Nuembrecht, Germany). Samples were collected four times per day at 1, 4, 9, and 11 hr after waking over 5 consecutive days following the lab visit to capture total cortisol secretion across the day. To determine whether participants were compliant with the sampling schedule, Salivettes were stored in a bottle sealed by a MEMS 6 TrackCap Monitor (Medication Event Monitoring System, Aardex Ltd., Switzerland). Caps record the date and time of each opening. 86.6% of samples were completed, and 88.1% of completed saliva samples within 1 hr of the scheduled collection time. Saliva samples that were completed more than 1 hr after the scheduled time, and samples that were collected less than 1 hr after awakening, were discarded. Saliva samples were returned to the lab and then centrifuged at 1,000 g for 5 min, transferred to deep-well plates, and stored at $-30\ °C$ until assayed. Free cortisol levels in saliva were measured in duplicates using a commercially available chemiluminescence assay (IBL, Hamburg, Germany). To assess total cortisol secretion throughout the day, data was first log transformed to reduce substantial skewness. Daily cortisol output was calculated via an area under the curve (AUC) statistic using the trapezoidal rule.

Procedure

Participants came to the lab and signed consent forms. During their lab visit, participants provided background information on childhood family environment and demographic information. As well participants were given instructions on how to complete web-based surveys and how to collect salivary cortisol samples. Schedules for saliva sampling were set for the 5 days following the lab visit. Finally, Actiwatches were described and distributed to participants.

For the 7 days following the lab visit, participants completed a web-based diary entry at end of each day. Specifically, participants were asked to report on any stressful events they experienced in the past 24 hr. To increase compliance, study participants received a daily reminder email with a link to the web-based survey, and those who did not complete the entry the previous day were phoned by the study coordinator. Participants completed an average of 93.9% of daily diary entries over the 7 days. Three participants completed diaries on paper because they did not have daily access to computers. If diaries were completed more than 1 day late, data was excluded from analyses. Participants received $1.00 per diary entry completed on the appropriate day. Finally, participants received $10 dollars at the initial lab visit, and $10 for returning their Actiwatch and MEMS cap at the end of the study.

Statistical Analyses

Data were analyzed using hierarchical linear modeling techniques (HLM). This method of statistical analysis enabled us to test the within-person (Level 1) relationships between daily stress and daily biological outcomes. It also allowed us to test whether between-person (Level 2) factors moderate these day-to-day stress-health associations. Stress and biological variables were

modeled as within-person factors because they were collected daily. Childhood family environment variables were modeled as between-person factors because they were collected at one time point and reflect a factor thought to vary across people.

First, we conducted a series of Level 1 models to predict how sleep and cortisol output varied as a function of daily stress experiences. Level 1 models generate a set of slopes for each individual that reflect variations in biological markers as a function of daily stress. Level 1 predictor variables were centered around each individual's mean, allowing us to examine whether deviations from an individuals' average stress experiences, for example, impacted sleep that night.

Next, we conducted a series of Level 2 models to determine whether between-person factors (i.e., childhood family environment) explained the variance in slopes from the Level 1 models. Significant interactions were graphed at two arbitrary points at the 25th and 75th percentile along the continuous childhood environment distribution, and are not intended to represent groups per se, but rather to illustrate the nature of the interaction effect. We used full maximum likelihood and robust standard errors to estimate all models. The multilevel model was specified as follows:

Level 1: $sleep\ minutes_{ij} = b_{0i} + b_{1i}\ (severity\ of\ stressor) + r_{ij}.$

Level 2: $b_{0j} = b_{00} + b_{01}\ (risky\ families) + b_{02}\ (ethnicity) + \mu_{0i}$

$$b_{1j} = b_{10} + b_{11}\ (risky\ families) + b_{12}\ (ethnicity) + \mu_{1i}.$$

In other words, at Level 1, minutes of sleep on any given night ($sleep\ minutes_{ij}$) is a function of their minutes of sleep on an average stressor severity day (b_{0i}), the severity of the worst stressor that day (b_{1i}), and random error (r_{ij}). At Level 2, b_{0j} represents a person's intercept (i.e., the expected value of sleep minutes on average stressor severity days) as a function of intercepts across all participants (β_{00}), the participant's Risky Families score, ethnic group, and random error (μ_{0i}). As well, b_{1j} represents a person's slope (i.e., how sleep minutes vary in response to deviations from a person's average levels of stressor severity) as a function of the average slope across participants (β_{10}), the participant's Risky Families score, ethnicity, and random error (μ_{1i}). Subsequent models were computed, substituting sleep efficiency and cortisol output as Level 1 outcome variables, the number of daily stressors

as the Level 1 predictor variable, and parental warmth as the between-person Level 2 variable.

Results

Descriptive Data and Preliminary Analyses

Means, standard deviations, and ranges for all study variables are presented in Table 1. Participants slept an average of about 420 min (about 7 hr) per night, and their nightly sleep was about 84% "efficient" or motionless. The Centers for Disease Control and Prevention recommends that adults receive 7 to 9 hr of sleep per night (http://www.cdc.gov/Features/Sleep/). As well, less than 85% sleep efficiency is considered poor sleep (Javaheri, Storfer-Isser, Rosen, & Redline, 2008). In our sample, on average, about 33% of participants slept for less than 7 hr per night and about 16% slept less than 6 hr per night. About one third had sleep that was less than 85% efficient. Participants reported an average of 3.5 stressors per day, with the most severe stressor reported as *somewhat* serious.

We tested whether protocol compliance was related to daily stress data or biological markers. The number of completed diary entries were not significantly related to the number of reported stressors ($\beta = -0.18$, $SE = 0.28$, $p = .53$), severity of stressors ($\beta = 0.13$, $SE = 0.12$, $p = .30$), or reports of daily health symptoms ($\beta = 0.37$, $SE = 0.23$, $p = .11$). Number of completed days of actigraphy was not significantly related to minutes of sleep per night ($\beta = -15.26$, $SE = 10.33$, $p = .14$), or nightly sleep efficiency ($\beta = 0.28$, $SE = 0.78$, $p = .72$). Finally, salivary cortisol sampling compliance was not significantly related to daily cortisol output ($\beta = 0.03$, $SE = 0.42$, $p = .94$).

We then tested whether demographic information (age, gender, and ethnicity) predicted childhood family environment variables. None of the demographic variables predicted childhood family risk ($ps > .05$) or parental warmth ($ps > .05$). We also tested whether demographic information predicted daily cortisol output or sleep variables. None of the demographic variables predicted daily cortisol output ($ps > .05$). As well, neither age nor gender predicted stress or sleep; however, ethnicity significantly predicted participants' reports of stress severity ($\beta = 0.38$, $SE = 0.14$, $p = .009$),

Table 1
Descriptive Statistics of Study Variables

Variable	M (SD)	Range	Pearson's r Warmth	Risk
Daily process measures				
No. of stressors	3.55 (1.91)	0.57–8.43	.02	.01
Severity of stressor	3.17 (0.81)	1.80–4.71	−.08	.04
Sleep minutes	419.69 (61.18)	259.50–548.42	−.13	.05
Sleep efficiency	83.68 (6.03)	62.81–93.38	−.12	−.01
Total AUC	9.07 (4.29)	1.31–36.13	−.03	−.11
Background measures				
Risky families	1.98 (0.50)	1.08–3.23	−.67*	—
Parental warmth	34.23 (7.53)	11.00–48.00	—	−.67*

Note. Values of daily process variables were aggregated across days. Possible ranges for number of stressors: 0–16; stress severity: 1 to 5; risky families: 1 to 5; parental warmth: 0 to 48. AUC = area under the curve.
* $p < .001$.

indicating that minority participants rated daily stressors as more severe than White participants. Ethnicity was included as a covariate in subsequent analyses.

We performed Pearson's correlations to assess whether characteristics of the childhood psychosocial environment were related to reports of the number or severity of daily stressors, nightly sleep parameters, or cortisol output. All results were nonsignificant (see Table 1).

Main Effects on Biological Markers

Daily variables. We first ran a series of Level 1 unconditional models to determine whether there was sufficient variance in each of the daily variables to justify examining the relationships between daily variables. Overall, findings show that there was significant variability in sleep minutes (variance = 2,264.18, $p <$ .001) and sleep efficiency (variance = 22.60, $p <$ .001) across the monitoring period. There was also significant variability in the number (variance = 3.05, $p < .001$) and severity (variance = 0.46, $p < .001$) of stressors across the week. Cortisol output also varied significantly (variance = 1.99, $p < .001$).

Sleep. We then generated a series of Level 1 models to assess main effects of whether stress during the day impacted sleep that night. Neither the number of daily stressors ($\beta = -1.94$, $SE = 2.82$, $p = .49$), nor the severity of the worst stressor ($\beta = -4.56$, $SE = 4.97$, $p = .36$) was related to the minutes of sleep per night. As well, neither the number of daily stressors ($\beta = 0.25$, $SE = 0.24$, $p = .31$), nor the severity of the worst stressor ($\beta = -0.39$, $SE = 0.41$, $p = .34$) was related to nightly sleep efficiency.

We next generated a series of Level 2 models to assess the main effects of whether childhood family environment predicted nightly sleep and found that neither family risk (sleep minutes: $\beta = 8.01$, $SE = 14.70$, $p = .59$, sleep efficiency: $\beta = 0.02$, $SE = 1.46$, $p = .99$) nor parental warmth (sleep minutes: $\beta = -1.26$, $SE = 0.89$, $p = .16$, sleep efficiency: $\beta = -0.11$, $SE = 0.09$, $p = .24$) significantly predicted nightly sleep.

Cortisol. We next conducted a similar set of analyses of the main effects between stress and cortisol. Results showed that neither the daily number of stressors ($\beta = 0.16$, $SE = 0.19$, $p = .40$), nor the severity of the worst stressor ($\beta = 0.23$, $SE = 0.40$, $p = .57$) significantly predicted total cortisol secretion. In addition, neither childhood family risk ($\beta = -0.42$, $SE = 0.67$, $p = .53$) nor parental warmth ($\beta = -0.03$, $SE = 0.05$, $p = .60$) significantly predicted cortisol output. Hence there were no main effects of either stress or family environment on sleep or cortisol.

Do Childhood Psychosocial Factors Moderate the Daily Stress-Biology Relation?

We next tested whether the Level 2 between-person childhood psychosocial factors moderated the relationship between daily stress and cortisol/sleep. In other words, we aimed to determine whether the relationships from daily stress to biological markers were different for individuals from different childhood family environment backgrounds. To do this, we tested whether childhood family environment variables explained variance in the slopes from the Level 1 models reported above.

Risky Families.

Sleep. We first tested whether childhood family conflict, as measured by the Risky Families questionnaire (Taylor et al.,

2006), interacted with stress during the day to predict sleep at night. Results showed that family environment significantly moderated the relation between number of stressors during the day and minutes asleep that night ($\beta = -10.66$, $SE = 4.60$, $p = .02$). Although the interaction is one of continuous variables, for illustrative purposes in Figure 1, we graphed at two arbitrary points at the 25th and 75th percentile along the risky family childhood environment distribution to help readers better visualize the nature of the interaction effect. Results indicate that the more "risky" one's childhood environment, the fewer minutes of sleep individuals got on days in which they experienced a greater number of stressors. We calculated the percentage variance accounted for by the risky childhood environment by comparing differences in variance components in HLM models before and after including environment as a moderator. We found that family risk accounted for 20.3% of the variance in the relation between daily number of stressors and nightly minutes of sleep. Family environment did not moderate the relations between stress severity and sleep quantity or efficiency ($\beta = -13.50$, $SE = 10.53$, $p = .20$, and $\beta = .48$, $SE = .71$, $p = .50$, respectively).

Cortisol. We then tested whether childhood family risk interacted with daily stressors to predict daily cortisol output. Results showed that family risk did not significantly moderate the relation between the daily number of stressors and cortisol output ($\beta = 0.51$, $SE = 0.56$, $p = .37$) nor did it moderate the relation between stress severity and cortisol output ($\beta = 1.68$, $SE = 1.04$, $p = .11$).

Parental warmth.

Sleep. Next we tested whether childhood parental warmth moderated the relation between daily stress and nightly sleep. Parental warmth did not significantly moderate the relation between daily number of stressors and sleep minutes ($\beta = 0.21$, $SE = 0.29$, $p = .48$) or sleep efficiency ($\beta = -0.01$, $SE = 0.04$, $p = .86$). As well, parental warmth did not significantly moderate the association between daily stress severity and sleep minutes ($\beta = -0.06$, $SE = 0.69$, $p = .93$) or sleep efficiency ($\beta = -0.02$, $SE = 0.04$, $p = .60$).

Cortisol. Childhood parental warmth significantly moderated the relation between the severity of stress experienced during the day and daily cortisol output ($\beta = -0.19$, $SE = 0.09$, $p = .04$), such that the less warm one's childhood environment, the more cortisol individuals secreted on days in which they also experienced more severe stress. The significant interaction was graphed
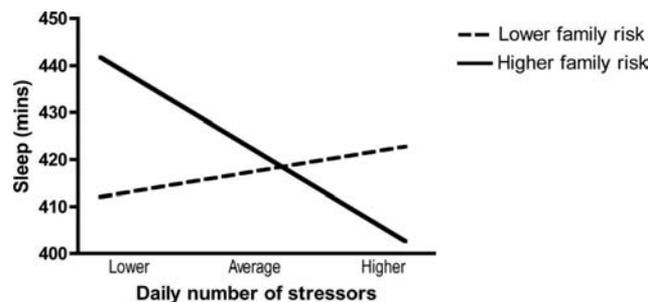

Figure 1. Childhood family risk moderates the relation between daytime stress and nighttime sleep. As childhood family risk increased, on days when young adults experienced a greater number of stressors (relative to their own average), they slept for fewer minutes.

in Figure 2 at two arbitrary points at the 25th and 75th percentile along the continuous childhood parental warmth distribution to illustrate the nature of the interaction effect. We calculated the percentage variance accounted for by childhood parental warmth by comparing differences in variance components in HLM models before and after warmth was included as a moderator. We found that parental warmth accounted for 20.9% of the variance in the relation between daily severity of stressor and cortisol output.

Discussion

The findings from this study provide some evidence that childhood psychosocial environments serve to moderate the relation between stress and biological outcomes. The nature of this interaction was such that among individuals from more difficult childhood environments, days on which individuals experienced a greater number of, or more severe stressors (relative to their average), were associated with less sleep and greater cortisol secretion. Or conversely, the more positive the childhood environment, the more positive the association between daily stress and sleep and the more negative the association between daily stress and cortisol.

The fact that there were no main effects of daily stress on daily cortisol or sleep suggests that, particularly within samples of healthy young adults, approaches that focus on moderating psychosocial influences may be useful in understanding the effects of day-to-day stress on daily biological processes. Given that the majority of previous research on childhood environments and response to stress has been laboratory based or cross-sectional (van Eck et al., 1996), our study extends this research by providing insight into how childhood environments may affect the relationship between naturally occurring stress and daily biological rhythms. These results are consistent with previous studies that have reported that difficult childhood environments predict increased cortisol secretion to stress (Gorman et al., 2002; Hennessy, 1997; Luecken, 1998; Repetti et al., 2002; Rosenblum et al., 2001) and less sleep (Gregory et al., 2006). As well, results are consistent with findings in the literature that parental warmth buffers the effects of stress on cortisol secretion (Caldji et al., 2000; Evans et al., 2007) and is associated with longer sleep (Adam et al., 2007). However, our study builds on previous research by demonstrating
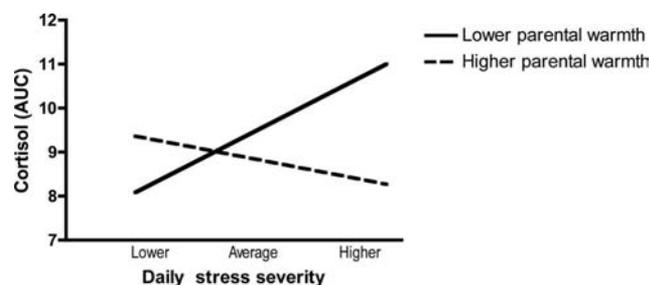

Figure 2. Childhood parental warmth moderates the relation between daily stress and cortisol output. As childhood parental warmth decreased, on days when young adults experienced more severe stressors (relative to their own average), they had higher cortisol output. AUC = area under the curve.

that childhood environmental characteristics moderate the relation between daily, naturally occurring stress and biological outcomes.

In this study we found that the more negative an individual's childhood environment the less they slept on days when they experienced a greater number of stressors (relative to their average). This suggests that these individuals may have developed a heightened sensitivity to potential threats during their childhood to prepare themselves to manage or avoid stressful events (Selye, 1955; Thompson & Calkins, 1996). However, this increased alertness or vigilance for upcoming stress may then have made it more difficult for them to sleep at night (Sadeh et al., 2004).

We also found that, the less positive an individual's childhood environment, the more they secreted cortisol on days when they experienced more severe stress (relative to their average). This is consistent with previous research that has shown that high parental warmth buffers youth from the negative effects of stress on cortisol (Evans et al., 2007). In the present study, a lack of nurturing behavior experienced in childhood may impair children's abilities to regulate their biological responses to stress. As a result, as adults, these individuals may show greater cortisol responses to stressful life situations. In addition, parents who displayed low warmth may have modeled maladaptive emotion regulation skills to their children, which could lead to heightened cortisol secretion in response to stress in adulthood (Repetti et al., 2002). The converse relationship of higher parental warmth being associated with less cortisol secretion on days of more severe stress may possibly be a stress inoculation effect—the idea that some exposures to more mild stress early in life may serve to blunt physiological responses to later life stress (Boyce & Ellis, 2005; Parker, Buckmaster, Sundlass, Schatzberg, & Lyons, 2006).

It is somewhat surprising that, contrary to previous studies (Cartwright & Wood, 1991; Peeters et al., 2003; Schlotz et al., 2006; Smyth et al., 1998; van Eck et al., 1996), we did not find a main effect of stress on biological patterns. This may be due to the fact that, contrary to other studies (Cartwright & Wood, 1991; Peeters et al., 2003), participants in our study were young and free of medical illness. As well, it is possible that the daily stressors that participants reported in this study, such as academic deadlines and financial insecurity, were too mild to elicit a significant change in biological processes across the entire sample; rather, results suggest that these relatively mild stressors are more likely to result in biological changes within individuals from difficult childhood environments.

We note that low parental warmth did not moderate the relationship between stress and sleep, and that risky childhood environments did not moderate the relation between daily stress and daily cortisol secretion. It is unclear why characteristics of the childhood environment would significantly moderate the association between daily stress and some daily biological patterns but not others. Future research is needed to determine the specific pathways through which early life experiences effect adult biological processes.

At a broader level, there are several explanations for why childhood family environments would moderate the stress-cortisol/ sleep relationships in adulthood. First, biological responses to stress may be programmed early in life (Boyce & Ellis, 2005; Meaney, 2001), and persist into adulthood. Second, individuals from difficult childhood environments may develop poor emotion regulation or coping strategies (Cummings, Zahn-Waxler, &

Radke-Yarrow, 1981; Davies & Cummings, 1994; O'Brien, Margolin, John, & Krueger, 1991), which may then lead to heightened biological stress responses in adulthood. Third, there may be genetic factors shared between parents and children that both predispose parents to behaving in ways that are consistent with risky family environments and that make children more reactive to stress. Another possibility is that the effects of difficult childhood environments are actually a proxy for current interpersonal difficulties in adulthood. Recent research, however, has shown that the childhood environment predicts biological profiles in adulthood over and above the current psychosocial environment (Gregory et al., 2006; Miller & Chen, 2007; Rutter & O'Connor, 2004), and hence that this explanation may not be that likely.

Results from this study contribute to the literature by demonstrating that, to understand the relation between daily stress and biological processes in young adults, it is necessary to consider the moderating influence of the childhood environment. More specifically, we demonstrated that difficult childhood environments may make individuals more susceptible to the detrimental effects of daily stress in adulthood. For individuals from more difficult childhood backgrounds, the repeated experiences of daily stress may evoke biological changes that, over time, could lead to wear and tear on the body and, eventually, the onset of disease. For example, previous research has demonstrated that, in a community-based prospective study, adults who reported sleeping less than 7 hr per night on most nights were at increased risk for hypertension (Golieb et al., 2006). As well, evidence suggests that partial sleep deprivation may, over time, result in impaired glucose tolerance and insulin sensitivity, and thus be a risk factor for the development of Type 2 diabetes (Spiegel, Knutson, Leproult, Tasali & Van Cauter, 2005). Thus we suggest that the cumulative effects of these biological changes can have clinical significance over time. As well, our findings provide further insight about daily life experiences with stress and biological responses, and represent a more ecologically valid approach to measuring stress. However, the research was not without limitations.

First, childhood environmental factors were measured retrospectively, and individuals may not have been able to accurately recall family environments years later. Second, because we only measured childhood environmental characteristics, we cannot rule out the influence of unmeasured aspects of the current psychosocial environment on the relation between daily stress and biological outcomes. Third, our measure of daily stress may not have captured certain aspects of the stress process, including whether the stressor was socially relevant (Dickerson & Kemeny, 2004), or whether the stressor occurred within the context of chronic stress (Marin, Martin, Blackwell, Stetler & Miller, 2007; Miller et al., 2007). As well, the generalizability of these study findings may be limited, given that the sample was composed of young and healthy undergraduate students, and was largely women and Asian.

Although this research provides an important step forward in understanding potential pathways through which childhood psychosocial factors create vulnerability for disease later in life, future studies are needed that assess these types of patterns across the life span. Studies of this kind would provide a better understanding of whether there are critical periods that exist during which the childhood psychosocial environment may be particularly protective or potent, as well as for how long childhood environments continue to affect daily stress and biology relationships. Other research suggests that critical periods may occur during the first 2 years of life (Cohen, Doyle, Turner, Alper, & Skoner, 2004; Miller & Chen, 2007), or during puberty (Stroud, Papandonatos, Williamson, & Dahl, 2004; Walker, Sabuwalla, & Huot, 2004). If such critical periods are identified, interventions could be designed to minimize negative and maximize positive environmental characteristics, or to teach coping responses to stress during those periods, that would protect individuals from the harmful effects of stress on health.

The present study provides a step toward this goal by documenting that more difficult childhood environments may have long lasting detrimental effects on how biological processes respond to daily stress in young adulthood. Understanding such moderating influences is important for identifying individual differences in biological responses to daily stress and for targeting interventions that will hopefully improve the health of all individuals throughout the life span.

References

Adam, E. K., Snell, E. K., & Pendry, P. (2007). Sleep timing and quantity in ecological and family context. *Journal of Family Psychology, 21,* 4–19.

Åkerstedt, T. (2007). Altered sleep/wake patterns and mental performance. *Physiology and Behavior, 90,* 209–218.

Ancoli-Israel, S., & Roth, T. (1999). Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation survey. *Sleep, 22,* 347–353.

Boyce, T., & Ellis, B. (2005). Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17,* 271–301.

Caldji, C., Diorio, J., & Meaney, M. J. (2000). Variations in maternal care in infancy regulate the development of stress reactivity. *Biological Psychiatry, 48,* 1164–1174.

Canon, W. B. (1932). *The wisdom of the body.* New York, NY: Norton.

Carpenter, L., Carvalhoa, J., Tyrka, A., Wiera, L., Melloa, A., Melloa, M., Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62,* 1080–1087.

Cartwright, R., & Wood, E. (1991). Adjustment disorders of sleep: The sleep effects of a major stressful event and its resolution. *Psychiatry Research, 39,* 199–209.

Cheeta, S., Ruigt, G., van Proosdij, J., & Willner, P. (1997). Changes in sleep architecture following chronic mild stress. *Biological Psychiatry, 41,* 419–427.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine, 66,* 553–558.

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Association, 298,* 1685–1687.

Cummings, E. M., Zahn-Waxler, C., & Radke-Yarrow, M. (1981). Young children's responses to expressions of anger and affection by others in the family. *Child Development, 52,* 1274–1282.

Dagan, Y., Zinger, Y., & Lavie, P. (1997). Actigraphic sleep monitoring in posttraumatic stress disorder (PTSD) patients. *Journal of Psychosomatic Research, 42,* 577–581.

Dahl, R., & Lewin, D. (2002). Pathways to adolescent health sleep regulation and behavior. *The Journal of Adolescent Health, 31,* 174–184.

Davies, P. T., & Cummings, E. M. (1994). Marital conflict and child adjustment: An emotional security hypothesis. *Psychological Bulletin, 116,* 387–411.

DeLongis, A., Folkman, S., & Lazarus, R. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. *Journal of Personality and Social Psychology, 54,* 486–495.

Derogatis, L. R., Lipman, R. S., Covi, L., Rickels, K., & Uhlenhuth, E. H. (1970). Dimensions of outpatient neurotic pathology: Comparison of a clinical versus an empirical assessment. *Journal of Consulting and Clinical Psychology, 34,* 164–171.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355–391.

Evans, G., Kim, P., Ting, A., Tesher, H., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology, 43,* 341–351.

Gorman, J. M., Mathew, S., & Coplan, J. (2002). Neurobiology of early life stress: Nonhuman primate models. *Seminars in Clinical Neuropsychiatry, 7,* 96–103.

Gottlieb, D. J., Redline, S., Nieto, J., Baldwin, C. M., Newman, A. B., Resnick, H. E., & Punjabi, N. M. (2006). Associations of usual sleep duration with hypertension: The Sleep Heart Health Study, *Sleep, 29,* 1009–1014.

Graham, J. E., Christian, L. M., & Kiecolt-Glaser, J. K. (2006). Stress, age, and immune function: Toward a lifespan approach. *Journal of Behavioral Medicine, 29,* 389–400.

Gregory, A., Caspi, A., Moffitt, T., & Poulton, R. (2006). Family conflict in childhood: A predictor of later insomnia. *Sleep, 29,* 1063–1067.

Gump, B. B., & Matthews, K. A. (1999). Do background stressors influence reactivity to and recovery from acute stressors? *Journal of Applied Social Psychology, 29,* 469–494.

Hall, M. (1998). Sleep as a mediator of the stress-immune relationship. *Psychosomatic Medicine, 60,* 48–51.

Hall, M., Buysse, D., Dew, M., Prigerson, H., Kupfer, D., & Reynolds, C. (1997). Intrusive thoughts and avoidance behaviors are associated with sleep disturbances in bereavement-related depression. *Depression and Anxiety, 6,* 106–112.

Hall, M., Buysse, D. J., Nowell, P. D., Nofzinger, E. A., Houck, P., & Reynolds, C. F. (2000). Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosomatic Medicine, 62,* 227–230.

Hanson, E. K., Maas, C. J., Meijman, T. F., & Godaert, G. L. (2000). Cortisol secretion throughout the day, perceptions of the work environment, and negative affect. *Annals of Behavioral Medicine, 22,* 316–324.

Heim, C., Mletzko, T., Purselle, D., Musselman, D. L., & Nemeroff, C. B. (2008). The dexamethasone/corticotropin-releasing factor test in men with major depression: Role of childhood trauma. *Biological Psychiatry, 63,* 398–405.

Heim, C., Newport, J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., & Nemeroff, C. B. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284,* 592–597.

Hennessy, M. (1997). Hypothalamic-pituitary-adrenal responses to brief social separation. *Neuroscience and Behavioral Reviews, 21,* 11–29.

Holmes, T., & Rahe, R. (1967). The Social Readjustment Rating Scale. *Journal of Psychosomatic Research, 11,* 213–218.

Javaheri, S., Storfer-Isser, A., Rosen, C.L., & Redline, S. (2008). Sleep quality and elevated blood pressure in adolescents. *Circulation, 118,* 1034–1040.

Lin, N., & Ensel, W. M. (1989). Life stress and health: Stressors and resources. *American Sociological Review, 54,* 382–399.

Luecken, L. (1998). Childhood attachment and loss experiences affect adult cardiovascular and cortisol function. *Psychosomatic Medicine, 60,* 765–772.

Marin, T. J., Martin, T. M., Blackwell, E., Stetler, C., & Miller, G. E. (2007). Differentiating the impact of episodic and chronic stressors on hypothalamic–pituitary–adrenocortical axis regulation in young women. *Health Psychology, 26,* 447–455.

Marmot, M., & Wilkinson, R. G. (1999). *Social determinants of health.* Oxford, England: Oxford University Press.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338,* 171–179.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation. *Physiological Review, 87,* 873–904.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24,* 1161–1192.

Miller, G. E., & Chen, E. (2007). Unfavorable socioeconomic conditions in early life presage expression of pro-inflammatory phenotype in adolescence. *Psychosomatic Medicine, 69,* 402–409.

Miller, G. E., Chen, E., & Cole, S. (2009). Health psychology: Developing biologically plausible models linking the social world to physical health. *Annual Review of Psychology, 60,* 501–524.

Miller, G. E., Chen, E., & Zhou, E. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133,* 25–45.

Nicolson, N. A. (2004). Childhood parental loss and cortisol levels in adult men. *Psychoneuroendocrinology, 29,* 1012–1018.

O'Brien, M., Margolin, G., John, R. S., & Krueger, L. (1991). Mothers' and sons' cognitive and emotional reactions to simulated marital and family conflict. *Journal of Consulting and Clinical Psychology, 59,* 692–703.

Parker, G. (1979). Parental characteristics in relation to depressive disorders. *The British Journal of Psychiatry, 134,* 138–147.

Parker, K. J., Buckmaster, C. L., Sundlass, K., Schatzberg, A. F., & Lyons, D. M. (2006). Maternal mediation, stress inoculation, and the development of neuroendocrine stress resistance in primates. *Proceedings of the National Academy of Sciences, USA, 103,* 3000–3005.

Paulsen, V., & Shaver, J. (1991). Stress, support, psychological states and sleep. *Social Science and Medicine, 32,* 1237–1243.

Peeters, F., Nicholson, N. A., & Berkhof, J. (2003). Cortisol responses to daily events in major depressive disorder. *Psychosomatic Medicine, 65,* 836–841.

Phillips, D. I., & Jones, A. (2006). Fetal programming of autonomic and HPA function: Do people who were small babies have enhanced stress responses? *Journal of Physiology, 572,* 45–50.

Pillar, G., Malhotra, A., & Lavie, P. (2000). Post-traumatic stress disorder and sleep—What a nightmare! *Sleep Medicine Reviews, 4,* 183–200.

Reite, M., & Snyder, D. S. (1982). Physiology of maternal separation in a bonnet macaque infant. *American Journal of Primatology, 2,* 115–120.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128,* 330–366.

Rivkees, S. A. (2003). Developing circadian rhythmicity in infants. *Pediatrics, 112,* 373–381.

Rosenblum, L. A., Forger, C., Noland, S., Trost, R. C., & Coplan, J. D. (2001). Response of adolescent bonnet macaques to an acute fear stimulus as a function of early rearing conditions. *Developmental Psychobiology, 39,* 40–45.

Rutter, M., & O'Connor, T. G. (2004). Are there biological programming effects for psychological development? Findings from a study of Romanian adoptees. *Developmental Psychology, 40,* 81–94.

Sadeh, A., Keinan, G., & Daon, K. (2004). Effects of stress on sleep: The moderating role of coping style. *Health Psychology, 23,* 542–545.

Schlotz, W., Schulz, P., Hellhammer, J., Stone, A. A., & Hellhammer, D. H. (2006). Trait anxiety moderates the impact of performance pressure on salivary cortisol in everyday life. *Psychoneuroendocrinology, 31,* 459–472.

Selye, H. (1955). Stress and disease. *Science, 122,* 625–631.

Sleep and Sleep Disorders (2008). Centers for Disease Control and Prevention. Retrieved June 9, 2010, from http://www.cdc.gov/Features/Sleep/

Smyth, J., Ockenfels, M. C., Porter, L., Kirschbaum, C., Hellhammer, D. H., & Stone, A. A. (1998). Stressors and mood measured on a

momentary basis are associated with salivary cortisol secretion. *Psychoneuroendocrinology, 23,* 353–370.

Spiegel, K., Knutson, K., Leproult, R., Tasali, E., & Van Cauter, E. (2005). Sleep loss: A novel risk factor for insulin resistance and Type 2 diabetes. *Journal of Applied Physiology, 99,* 2008–2019.

Stroud, L. R., Papandonatos, G. D., Williamson, D. E., & Dahl, R. E. (2004). Sex differences in the effects of pubertal development on response to CRH challenge: The Pittsburgh Psychobiologic Studies. *Annals of the New York Academy of Sciences, 1021,* 348–351.

Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality, 72,* 1365–1394.

Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry, 60,* 671–676.

Thompson, R. A., & Calkins, S. D. (1996). The double-edged sword: Emotion regulation in high-risk children. *Development and Psychopathology, 8,* 163–182.

Tworoger, S., Davis, S., Vitiello, M., Lentz, M., & McTiernan, A. (2005). Factors associated with objective (actigraphic) and subjective sleep quality in young adult women. *Journal of Psychosomatic Research, 59,* 11–19.

Urponen, H., Vuori, I., Hasan, J., & Partinen, M. (1988). Self-evaluations of factors promoting and disturbing sleep: An epidemiological survey in Finland. *Social Science and Medicine, 26,* 443–450.

Van Cauter, E., Polonsky, K. S., & Scheen, A. J. (1997). Roles of circadian rhythmicity and sleep in human glucose regulation. *Endocrine Reviews, 18,* 716–739.

van Eck, M. M., Nicolson, N. A., Berkhof, H., & Sulon, J. (1996). Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biological Psychiatry, 43,* 69–84.

Walker, E., Sabuwalla, Z., & Huot, R. (2004). Pubertal neuromaturation, stress sensitivity, and psychopathology. *Development and Psychopathology, 16,* 807–824.

Wilhelm, K., & Parker, G. (1990). Reliability of the parental bonding instrument and intimate bond measure scales. *Australia and New Zealand Journal of Psychiatry, 24,* 199–202.