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Effects of Daily Maladaptive Coping on Nightly Sleep in Mothers

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Abstract

Objective—We examined effects of daily rumination and suppression in response to stressors on objective and subjective sleep among mothers.

Design—Participants were 183 mothers, including chronically stressed mothers of children with an autism spectrum disorder (M-ASD; n = 92) and age-matched mothers of neurotypical children (M-NT; n = 91). In an intensive longitudinal design, participants provided reports of daily rumination and suppression, nightly objective actigraphy-defined sleep, and nightly subjective sleep quality for seven consecutive days at baseline, 9 months, and 18 months.

Main Outcome Measures—Total sleep time, sleep fragmentation, sleep onset latency and subjective sleep quality.

Results—Among M-NT with above average depressive symptoms, higher daily rumination was associated with shorter total sleep time. Rumination was associated with more sleep fragmentation among M-NT at the trend level. Rumination was not associated with sleep onset latency among M-NT, or with any sleep outcomes among M-ASD. Suppression was not associated with any sleep outcomes.

Conclusion—We provide novel evidence of the effect of rumination on objectively measured sleep duration among M-NT. Coping was not related to sleep among M-ASD. Given the prevalence of poor sleep among mothers, future work should examine modifiable factors perpetuating sleep disturbance.

Keywords

mothers; rumination; suppression; total sleep time; sleep fragmentation; sleep quality

Introduction

Sleep is an important health behavior, with implications for both psychological and physical health outcomes. For example, short sleep duration is associated with depression (Zhai, Zhang, & Zhang, 2015), hypertension (Wang et al., 2015), and all-cause mortality (Hublin, Partinen, Koskenvuo, & Kaprio, 2007). Given these wide-ranging consequences, it is important to identify factors associated with poor sleep. Growing evidence suggests that ineffective coping in response to stressful events affects sleep. Our focus in the current paper

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is on the effect of coping in response to stressors on sleep among mothers of children with an autism spectrum disorder and mothers of neurotypical children, among whom poor sleep quality is a common challenge (Lee, 2013; Meltzer, 2008).

Most research examining the impact of ineffective coping on sleep has focused on rumination, defined as a perseverative, past-oriented self-focus on the symptoms, causes, and consequences of distress (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008) or on perceived threats or losses (Trapnell & Campbell, 1999). Rumination has been studied both as a dispositional, stable trait measure, and as a state measure that fluctuates in response to daily events (e.g., Puterman, DeLongis, & Pomaki, 2010). Cognitive theories of insomnia posit that perseverative, negatively-toned cognitive processes such as rumination may initiate and maintain sleep difficulties by increasing distress and autonomic arousal (Harvey, 2002). Consistent with this theory, rumination was associated with subjective poor global sleep quality among college students and two online samples (Berset, Elfering, Luethy, Luethi, & Semmer, 2011; Thomsen, Mehsen, Christensen, & Zachariae, 2003; Zawadzki, 2015). Associations between state rumination and objective measures of sleep may depend on depressive symptoms. For example, state rumination was not associated with actigraphy-defined sleep onset latency in a general sample of students (Zoccola, Dickerson, & Lam, 2009), but was found to be positively associated in a sample of students with moderate depressive symptoms (Pillai, Steenburg, Ciesla, Roth, & Drake, 2014). There is a notable paucity of research examining the relationship between rumination and sleep in samples under chronic stress, who typically have poorer sleep (Meltzer, 2008) and a high prevalence of depressive symptoms (Lee, 2013). Moreover, there have been almost no studies of emotional suppression, another common maladaptive coping strategy.

Suppression is defined as inhibiting a response to an emotion stimulus (Gross, 1998). Although the goal of suppression may be to reduce negative emotions, it is often ineffective at doing so (Gross, 1998). Additionally, suppression is associated with increased sympathetic responses to negative emotions and long-term health consequences (DeSteno, Gross, & Kubzansky, 2013; Gross, 1998). Evidence for the effect of emotion suppression on sleep behavior specifically is more limited and mixed. Suppression was not associated with subjective global sleep quality among undergraduate students (Zawadzki, 2015), but avoidance coping more generally has been linked to higher subjective sleep disturbance symptom severity in a longitudinal study of men with cancer (Hoyt, Thomas, Epstein, & Dirksen, 2009). An important limitation of research on the effects of maladaptive coping on sleep is that it has primarily focused on undergraduate students. There is a critical need to understand the emotional processes that contribute to sleep disturbance among other vulnerable samples. In particular, poor global sleep quality is prevalent both among maternal caregivers of children with developmental disabilities and mothers of neurotypical children (Lee, 2013; Meltzer, 2008). However, research among maternal caregivers has used cross-sectional designs, and most studies have relied on self-report measures of sleep quality and lacked comparison groups. The current study seeks to address these important gaps in the literature. By comparing mothers of children with an autism spectrum disorder to mothers of neurotypical children, we are able to elucidate associations that are evident among mothers generally, versus those that are unique to chronically stressed mothers specifically. We examined the effect of daily coping in response to stressors on objective sleep and subjective

sleep quality among chronically stressed mothers of children with an autism spectrum disorder and mothers of neurotypical children. We utilized an intensive longitudinal design in which participants provided data for seven consecutive days at three timepoints over 18 months separated by 9-month intervals, and focused on two common and often maladaptive coping strategies: rumination and suppression. First, we examined between-group differences in total sleep time, fragmentation, sleep onset latency, and sleep quality. Second, we examined the effect of daily coping in response to stressors on nightly sleep, and the extent to which this differed between caregiver groups. Third, we examined whether effects varied as a function of depressive symptoms based on prior research among students (Pillai et al., 2014; Zoccola et al., 2009).

Method

Participants

Participants were chronically stressed maternal caregivers of children with an autism spectrum disorder (M-ASD, n = 92) and age-matched control maternal caregivers of neurotypical children (M-NT, n = 91) enrolled in a larger prospective study that examined the impact of caregiving stress on cellular aging. Participants were recruited from schools, parenting publications, social media, mailings and ads through child development centers, and direct recruitment at the University of California, San Francisco Autism Clinic.

In order for mothers to participate in this study they had to meet the following inclusion criteria: 1) ages 20–50 years, 2) body mass index < 40, 3) English-speaking, 4) premenopausal, and 5) have at least one child between ages 2–16 years. For M-ASD participants, additional inclusion criteria were: 1) care for a child diagnosed with autism spectrum disorder, and 2) Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983) score ≥ 13. For M-NT participants, additional inclusion criteria were: 1) care for a neurologically typical child, and 2) Perceived Stress Scale score ≤ 19. The selection of PSS cutoffs was determined based on national norms (Cohen et al., 1983): 13.68 (SD = 6.57) for women and 12.94 (SD = 6.02) for the targeted age group (aged 35 to 44 years). Therefore, high stress caregivers (i.e., M-ASD) were defined as reporting average or higher PSS scores based on national norms. Low stress controls (i.e., M-NT) were defined as reporting PSS scores less than one standard deviation above the national age-based mean. Thus, a cutoff of 19 was selected. The overlap in PSS scores facilitates analyses with a continuous stress measure irrespective of group status. All individuals were excluded from participating if they reported having a major chronic illness, such as cardiovascular disease or cancer not in remission, carried a diagnosis of an eating disorder or endocrine disorder, met criteria for current or past substance dependence, bipolar disorder or posttraumatic stress disorder diagnosis in the past ten years, were a regular smoker in the past ten years, or used of medications known to affect the immune system, including steroids. An additional exclusion criterion for M-NT only was a diagnosis of current major depressive disorder; however eligible participants presented with a range of depressive symptoms.

Participants completed study measures at three time points: baseline, 9-month follow-up, and 18-month follow-up. Participants were compensated $75 at each time point for completing study measures. The Institutional Review Board at the University of California,

San Francisco approved the study protocol, and all participants provided written informed consent.

Measures

Perceived stress—The 10-item Perceived Stress Scale (Cohen et al., 1983) was used to measure participant evaluations of stresses experienced over the past month. Total scores range from 0–40. Cronbach's alpha was 0.87 at baseline.

Autism severity—Within the M-ASD group, the Child Autism Rating Scale (CARS) was used to characterize child autism severity at baseline (Schopler, Reichler, DeVellis, & Daly, 1980). The CARS is a 15-item behavior rating scale. Total scores range from 15–60, with scores below 30 considered not autistic. Cronbach's alpha was 0.94 at baseline.

Sleep—Total sleep time, fragmentation, and sleep onset latency were behaviorally assessed using wrist actigraphy for seven consecutive nights at each of the three study time points. Participants wore an Actiwatch-2 (Philips Respironics, Bend, OR) continuously on the non-dominant wrist at each study time point. Data were stored in 15-second epochs and scored using validated Minimitter software algorithms to estimate sleep parameters of interest. Total sleep time is defined as the total number of minutes scored as sleep by the software algorithm in a given defined sleep interval. Estimates of sleep continuity were assessed using the sleep fragmentation index, which is a measure of nocturnal movement calculated as follows: ((number of mobile epochs lasting four epochs + number of immobile epochs < 1 min duration/number of immobile epochs > 1 min duration) × 100). Higher levels of fragmentation indicate worse sleep continuity. Sleep onset latency is defined as the total number of minutes taken to fall asleep based on actigraphy estimation. Subjective sleep quality was assessed for seven consecutive mornings at each of the three study time points. In a morning log, participants responded to the question "How would you rate the quality of your sleep last night?" Response options were "very bad" (1), "fairly bad" (2), "fairly good" (3), and "very good" (4).

Coping in response to stressors—For seven consecutive nights at each time point, participants were asked to describe in detail the most stressful event of their day, and then to indicate how they dealt with the stressful situation. The current manuscript focuses on rumination and suppression. To assess rumination, participants were asked to indicate the extent to which "I am unable to stop thinking about the situation." To assess suppression, participants were asked to indicate the extent to which "I tried to hide my negative feelings about the situation and did not tell anyone how I am feeling." Response options were "not at all" (0), "a little bit" (1), "moderately" (2), "quite a bit" (3), and "a lot" (4).

Depressive symptoms—Depressive symptoms were assessed using the Inventory of Depressive Symptoms (IDS; Rush et al., 1986). The IDS is a 30-item self-report measure; four items assessing sleep were removed for the current analyses. Scores range from 0 – 72, with higher scores indicating more severe depressive symptoms. The IDS was administered once per time point, and Cronbach's alphas were ≥ 0.85 at each time point.

Covariates—The following variables were used as covariates: age (in years); time point (0, 9, 18); body mass index (weight in kilograms/squared height in meters); antidepressant medication use at any time point (yes/no).

Data analysis

Descriptive statistics are reported for participant characteristics and predictor and outcome data. Independent samples t-tests and chi-square tests were used to examine differences by caregiver group.

For sleep outcomes and coping responses we used 21 days of data, measured consecutively for 7 days at a time, across 3 discrete weekly bursts. Our coping response predictor variables (i.e., rumination, suppression) were split into their within-subject state and between-subject trait components. Coping state variables were computed by subtracting participant-specific 21-day coping average from each diary entry. Coping trait variables were computed by subtracting the sample average from each participant-specific 21-day average. Depressive symptoms were reported at each of the three discrete bursts but given the stability across time in our sample (baseline to 9-month $r = .74$, baseline to 18-month $r = .74$, 9-month to 18-month $r = .69$), the resulting models include an average of the three. Additionally, we adjusted for assessment month (0, 9, 18) to account for time point specific variance in our sleep outcomes.

To test our hypotheses, data were analyzed using multilevel models. Separate analyses were tested for each coping variable (i.e., rumination, suppression) and sleep outcome (i.e., total sleep time, fragmentation, sleep onset latency, sleep quality). The first model tested the effect of daily coping on sleep across the full sample, controlling for trait coping and covariates. The second model tested the interaction between daily coping and caregiver group to examine whether the effect of daily coping on sleep varied between caregiver groups. The third model tested the three-way interaction between daily coping, caregiver group, and depressive symptoms (sample mean centered) to examine if the effects of daily coping on sleep varied as a function of depressive symptoms at varying levels of caregiver group. Except where otherwise noted, models included a random intercept and slope, and utilized restricted maximum likelihood estimation and an unstructured covariance matrix. Participants with incomplete data at either a single time point ($n = 20$, 11% of sample) or two time points ($n = 12$, 7% of sample) were included in the models. However, coping response data are missing and thus dropped from analysis when participants reported experiencing no daily stressor ($n = 82$, 2% of days).

Results

Participant characteristics

Table 1 presents participant characteristics stratified by caregiver group. Both M-NT and M-ASD participants were in their early 40s on average, and the majority of both groups was white, college educated, and reported a household income of at least $100,000. There were significant between-group differences in income and antidepressant medication use, with fewer M-ASD participants reporting an income over $100,000 and more M-ASD

participants reporting use of antidepressant medication compared to M-NT. On average, participants had more than one child. The youngest child of M-NT participants was younger, on average, than the youngest child of M-ASD participants. Among M-ASD, children had mild to moderate autism severity on average.

Predictor and outcome descriptives

Table 1 presents descriptive data for study variables. On average, both M-NT and M-ASD participants fell asleep in less than twenty minutes, slept less than seven hours per night across all time points, and reported fairly continuous sleep (average percentage of fragmented sleep was < 11%). On average, participants reported using rumination and suppression to cope with stressors less than "moderately" (i.e., 2), likely due to the fact that participants did not report objectively high severity stressors on average. Across time points, average depressive symptoms ranged from 0–33 for M-NT participants and 0–41 for M-ASD participants.

Multilevel models

Intraclass correlations (ICC) were computed using unconditional means models. The ICCs were 0.24 for total sleep time, 0.21 for fragmentation, 0.07 for sleep onset latency, 0.21 for sleep quality, 0.25 for rumination, and 0.24 for suppression. Thus, the percent total variance due to within-person fluctuations ranged from 75–93% for each of these variables.

Effects of daily rumination on sleep—Multilevel models were employed to test whether daily rumination in response to stress predicted nightly sleep duration (i.e. total sleep time), nightly sleep continuity (i.e., fragmentation), nightly sleep onset latency, and sleep quality in our sample of mothers of children with an autism spectrum disorder and mothers of neurotypical children. First, across the full sample and controlling for the covariates, daily rumination in response to stress was not significantly related to nightly total sleep time (B = −2.20, SE = 1.57, $t(140.94) = -1.40$, $p = .16$). Further, the two-way interaction between daily rumination and caregiver group predicting total sleep time was not significant, (B = −1.41, SE = 3.16, $t(140.80) = -0.45$, $p = .66$). However, we observed a significant three-way interaction between daily rumination, caregiver group, and depressive symptoms, which indicated that the effect of daily rumination on total sleep time varied as a function of depressive symptoms differentially between caregiver groups ($t(155.18) = 2.66$, $p = .009$, 95% CI = 0.37, 2.50); this association was independent of a number of covariates, including age, time, body mass index, antidepressant medication, and trait rumination (Table 2). Regions of significance graphs are presented in Figure 1. Specifically, among M-ASD participants, daily rumination was not significantly related to nightly total sleep time regardless of depressive symptoms. In contrast, among M-NT, the association between daily rumination and nightly total sleep time depended on depressive symptoms, such that the greater daily rumination was associated with shorter nightly total sleep time was significant for M-NT with depressive symptoms at the sample mean or higher. Simple slope analyses illustrating the association between daily rumination and nightly total sleep time at 1 standard deviation above ($p = .006$) and below ($p = .023$) the mean of depressive symptoms are provided as supplemental material (see supplemental Figure 1).

Next, we investigated whether daily rumination in response to stress predicted sleep continuity. There was no clear association between daily rumination and sleep fragmentation in the sample as a whole (B = 0.05, SE = 0.12, $t(101.08) = 0.43$, $p = 0.66$); however, analyses revealed a significant two-way interaction between daily rumination and caregiver group (B = −0.52, SE = 0.24, $t(98.07) = 2.18$, $p = .03$; see Table 3). Simple slopes analyses (see Figure 2) revealed that daily rumination was not associated with nightly sleep fragmentation among M-ASD participants (B = −0.18, SE = 0.16, $p = .26$). In contrast, among M-NT, there was a marginally significant association between daily rumination and nightly sleep fragmentation (B = 0.34, SE = 0.18, $p = .06$). These effects did not vary as a function of depressive symptoms, as indicated by a non-significant three-way interaction between daily rumination, caregiver group, and depressive symptoms (p=.15).

In our investigations of the effect of daily rumination in response to stress on sleep onset latency, models included a random intercept only since the random slope model was unable to converge and provide reliable estimates. Across the full sample, the effect of daily rumination in response to stress on nightly sleep onset latency was not significant (B = −0.02, SE = 0.44, $t(2710.87) = −0.046$, $p = 0.96$). The effect of rumination did not depend on caregiver group (interaction B = −0.22, SE = 0.88, $t(2708.32) = −0.25$, $p = 0.80$). The three-way interaction between daily rumination, caregiver group, and depressive symptoms also was not significant (interaction B = −0.057, SE = 0.15, $t(2684.97) = −0.37$, $p = 0.71$).

In our investigations of the effect of daily rumination in response to stress on sleep quality, models again only included a random intercept since the random slope model was unable to converge. There was no significant association between daily rumination and sleep quality in the sample as a whole (B = −0.002, SE = 0.01, $t(2862.34) = −0.17$, $p = 0.87$); however, analyses revealed a marginally significant two-way interaction between daily rumination and caregiver group (B = 0.05, SE = 0.03, $t(2859.16) = 1.96$, $p = .05$; see Table 3). Simple slopes analyses revealed that daily rumination had a positive, but non-significant, relationship with sleep quality among M-ASD participants (B = 0.02, SE = 0.02, $p = .22$). Among M-NT, there was a negative, but non-significant, relationship between daily rumination and sleep quality (B = −0.03, SE = 0.02, $p = .12$). These effects did not vary as a function of depressive symptoms, as indicated by a non-significant three-way interaction between daily rumination, caregiver group, and depressive symptoms (p=.70).

Effects of daily suppression on sleep—Across the full sample, the effect of daily suppression in response to stress on nightly total sleep time was not significant (B = 1.03, SE = 1.18, $t(118.99) = 0.88$, $p = 0.38$). The effect of suppression did not depend on caregiver group (interaction B = −0.87, SE = 2.38, $t(121.62) = −0.37$, $p = .72$). The three-way interaction between daily suppression, caregiver group, and depressive symptoms also was not significant (interaction B = 0.24, SE = 0.41, $t(121.00) = −0.59$, $p = .56$).

A similar pattern of findings was evident for when investigating sleep fragmentation. Across the full sample, the effect of daily suppression on nightly total sleep time was not significant (B = −0.06, SE = 0.11, $t(120.09) = −0.51$, $p = .61$). The effect of suppression did not depend on caregiver group (interaction B = 0.11, SE = 0.23, $t(120.91) = 0.49$, $p = .62$). The three-

way interaction between daily suppression, caregiver group, and depressive symptoms also was not significant (interaction B = 0.07, SE = 0.04, $t(115.07) = 1.74$, $p = .085$).

Across the full sample, the effect of daily suppression in response to stress on nightly sleep onset latency was not significant (B = −0.50, SE = 0.43, $t(156.74) = -1.16$, $p = 0.25$). The effect of suppression did not depend on caregiver group (interaction B = −0.95, SE = 0.86, $t(160.89) = -1.10$, $p = 0.27$). The three-way interaction between daily suppression, caregiver group, and depressive symptoms also was not significant (interaction B = 0.07, SE = 0.15, $t(148.92) = 0.49$, $p = 0.63$).

Finally, we examined the association between daily suppression and sleep quality. Again, the effect of suppression was not significant in the full sample (B = −0.02, SE = 0.01, $t(128.76) = -1.18$, $p = .24$), nor did it depend on caregiver group (suppression × caregiver group interaction, B = −0.04, SE = 0.03, $t(130.85) = -1.48$, $p = .14$), nor did it vary as a function of depressive symptoms (interaction B = −0.003, SE =0.004, $t(124.26) = -0.58$, $p = .56$).

Discussion

The objective of this study was to examine the effects of daily maladaptive coping in response to stressors on nightly sleep among mothers of neurotypical children and mothers of children with an autism spectrum disorder. The main findings that emerged from this work indicated that the effects of rumination on actigraphy-defined sleep were evident only among mothers of neurotypical children, and the most robust effect was for sleep duration. Higher daily rumination was associated with shorter total sleep time specifically among mothers with above average depressive symptoms; in other words, more depressed mothers were more vulnerable to the effects of rumination on sleep duration. However, the effect of depressive symptoms was not evident for other sleep outcomes. Maternal controls who reported higher stress-related rumination demonstrated a trend for poorer sleep continuity, regardless of depressive symptoms. A similar though non-significant pattern was evident for sleep quality. Null findings for the effect of state rumination on sleep onset latency is consistent with previous work among students generally (Zoccola et al., 2009), and in contrast to work among students with moderate depressive symptoms (Pillai et al., 2014). Our findings among mothers of neurotypical children converge with prior evidence primarily derived from convenience samples (Berset et al., 2011; Pillai et al., 2014; Thomsen et al., 2003; Zawadzki, 2015; Zoccola et al., 2009), but our study is the first to document effects on objectively measured sleep continuity specifically.

Surprisingly, we did not observe an effect of rumination on sleep among mothers of children with an autism spectrum disorder. This is despite the fact that mothers of children with an autism spectrum disorder reported significantly higher rumination in response to stressors than mothers of neurotypical children. There are at least two possible reasons for this. First, it is possible that the sleep of mothers of children with an autism spectrum disorder is more potently influenced by external circumstances, such as the need to attend to caregiving responsibilities during the night (Bourke-Taylor, Pallant, Law, & Howie, 2013), than it is by rumination in response to stressors. Second, it is possible that rumination does affect the sleep of maternal caregivers, but that our one-item measure of rumination in response to

stress was not nuanced enough to capture this maladaptive coping process in this sample of maternal caregivers. Instead, rumination on particular types of stressors, such as those related specifically to caregiving, may have a more powerful impact on sleep among maternal caregivers. Consistent with this, previous work has demonstrated that interpersonal stressors are the most impactful on daily mood and well-being and these effects may extend to sleep (Bolger, DeLongis, Kessler, & Schilling, 1989).

Consistent with prior work among students (Zawadzki, 2015), suppression was not associated with subjective sleep quality among mothers. Avoidance coping more generally, characterized by disengagement, denial, and distraction, has been associated with subjective sleep quality among men with cancer (Hoyt et al., 2009). Other emotion coping processes may be more pertinent for sleep among maternal caregivers. For example, worry is a perseverative cognitive process that is future-oriented, associated with sleep disturbance (for a review, see Pillai & Drake, 2015), and may be particularly relevant for maternal caregivers.

Poor global sleep quality is common among mothers with children with an autism spectrum disorder (Lee, 2013). However, many of these studies lacked a control group, thereby precluding the ability to draw conclusions about whether sleep quality is worse among these mothers than among mothers of neurotypical children. An important exception is a small study that found that parents of children with an autism spectrum disorder had significantly shorter sleep duration than parents of neurotypical children; however, both groups experienced poor global sleep quality and short sleep duration on average (Meltzer, 2008). Of note in the current study, mothers of children with an autism spectrum disorder did not have shorter sleep duration, poorer sleep continuity, or worse subjective sleep quality than mothers of neurotypical children at any time point, with the exception of sleep quality at baseline at sleep onset latency at 18 months. Importantly, participants across the full sample experienced short sleep duration on average (i.e., less than seven hours total sleep time), which is associated with adverse health consequences.

Findings for mothers of neurotypical children may have important implications for interventions to improve sleep. Mindfulness-based interventions, such as mindfulness-based cognitive therapy, have been shown to reduce rumination among individuals with histories of depression (Michalak, Holz, & Teismann, 2011), and growing evidence indicates they may be useful for improving sleep. For example, a three-arm randomized controlled trial found that both mindfulness-based stress reduction and mindfulness-based therapy for insomnia were associated with significantly greater improvements in insomnia symptoms compared to an active control condition among adults with insomnia (Ong et al., 2014). Such interventions may work, in part, by developing skills to change one’s relationship to stressful events (Ong, 2016). That is, mindfulness practice may help individuals observe and allow distressing thoughts and emotions instead of engaging in maladaptive coping strategies, such as rumination and suppression. However, future work is needed to test whether decreased rumination mediates the effect of mindfulness training on sleep, and the degree to which effects extend to individuals without chronic insomnia.

Limitations to the current study include the use of single-item measures to assess rumination and suppression. Additionally, the near floor-effects for sleep fragmentation data may have

limited our ability to detect the effects of daily coping on sleep continuity. The focus on mothers may limit generalizability to fathers, which are an important population for further study given previous research documenting shorter sleep duration among fathers of both neurotypical and autism spectrum children compared to mothers (Meltzer, 2008). The current sample was predominantly of higher socioeconomic status, and thus may have had more coping resources available. Finally, the current study was not powered to test the effects of child age and autism severity, which likely have important implications for maternal sleep (Meltzer, 2008; Mindell, Sadeh, Kwon, & Goh, 2015), and warrant further investigation.

In summary, the current study utilized an intensive longitudinal design to provide novel empirical evidence of the effect of rumination on sleep among mothers of neurotypical children. Given the prevalence of poor sleep among maternal caregivers, it is critical that future work continues to examine the unique factors perpetuating sleep disturbance in this population and that may be modifiable with intervention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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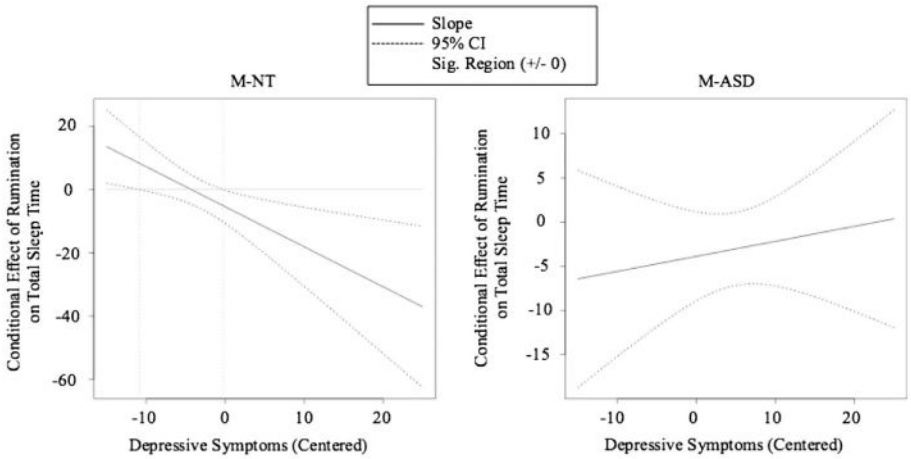

Figure 1.

Regions of significance graphs for the conditional effect of daily rumination on nightly total sleep time. As noted on the x-axis, depressive symptoms were centered such that 0 is the sample average. The solid diagonal line represents the estimated regression coefficient for the effect of daily rumination on total sleep time, at any level of depressive symptoms (i.e., along the x-axis). Among mothers of neurotypical children (left panel), the negative relationship between daily rumination on nightly total sleep time was significant for participants with average or above depressive symptoms. Among mothers of children with an autism spectrum disorder (right panel), the effect of daily rumination on nightly total sleep time was not observed; across the range of depressive symptoms reported, the confidence interval for the regression is shown to include zero (i.e., no effect).

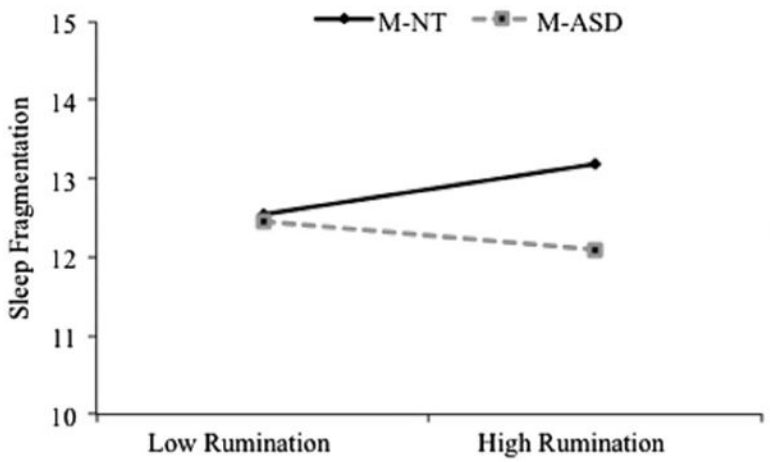

Figure 2.
Predicted sleep fragmentation by caregiver group at 1 SD below and above the mean for rumination.

Table 1

Descriptive Data for Participant Characteristics, Sleep Variables, and Predictors, Reported as M (SD) or % (n)

	Mothers of Neurotypical Children	Mothers of Children with an Autism Spectrum Disorder
Age	41.53 (4.52)	42.27 (5.61)
Caucasian	78.02% (71)	78.02% (71)
College educated	91.01% (81)	82.22% (74)
Income ≥ $100,000 *	84.62% (77)	68.13% (62)
Body mass index	25.09 (4.68)	25.87 (5.66)
Married	84.62% (77)	89.01% (81)
Antidepressant medication use	4.40% (4)	18.48% (17)
Baseline perceived stress **	15.72 (4.37)	21.89 (4.66)
Number of children	1.80 (0.73)	2.02 (0.91)
Age of youngest child *	5.47 (3.24)	7.07 (3.55)
Child autism severity	N/A	33.09 (8.80)
Total sleep time		
Baseline	413.26 (36.09)	403.81 (49.30)
9 months	410.77 (45.58)	400.65 (52.81)
18 months	404.09 (46.66)	396.64 (54.93)
Fragmentation		
Baseline	10.22 (4.05)	10.17 (4.71)
9 months	10.24 (4.28)	10.56 (5.89)
18 months	9.86 (3.68)	9.25 (4.13)
Sleep onset latency		
Baseline	12.24 (9.18)	12.98 (9.15)
9 months	13.52 (11.41)	16.70 (16.17)
18 months *	11.73 (10.59)	16.79 (15.02)
Sleep quality		
Baseline *	2.94 (0.47)	2.75 (0.44)
9 months	2.89 (0.36)	2.84 (0.45)
18 months	2.94 (0.44)	2.83 (0.45)
Weekly Rumination		
Baseline **	0.74 (0.62)	1.21 (0.83)
9 months *	0.78 (0.60)	1.21 (0.92)
18 months *	0.62 (0.58)	0.89 (0.72)
Weekly Suppression		
Baseline *	0.87 (0.70)	1.23 (0.82)
9 months **	0.75 (0.74)	1.31 (0.94)
18 months	0.82 (0.77)	1.04 (0.80)
Depressive symptoms		
Baseline **	8.95 (5.35)	15.62 (7.80)

	Mothers of Neurotypical Children	Mothers of Children with an Autism Spectrum Disorder
9 months**	7.78 (5.68)	15.69 (8.81)
18 months**	7.86 (5.78)	15.73 (8.35)

Note. The nightly sleep and coping strategy variables were averaged to provide timepoint-level estimates.

* p<.05,

** p<.001

Table 2

Multilevel Model Fixed Effects of Nightly Total Sleep Time as a Function of Daily Rumination

	B	SE
Intercept	424.48**	29.80
Time (months)	−0.62*	0.19
Caregiver group	−3.59	7.33
Rumination state	−5.43*	2.66
Rumination trait	−3.10	5.77
Depressive symptoms	−0.24	0.90
Age	0.26	0.62
Body mass index	−0.90	0.62
Antidepressant medication use	14.07	9.76
Caregiver group × Rumination state	1.55	3.68
Rumination state × Depressive symptoms	−1.27*	0.45
Group × Depressive symptoms	−0.57	1.06
Group × Rumination state × Depressive symptoms	1.44*	0.54

Note. Depressive symptoms were sample centered, such that 0 equals the sample average. For caregiver group, 0 = M-NT, 1 = M-ASD.

* $p < .05$,

** $p < .001$.

Table 3

Multilevel Model Fixed Effects of Nightly Fragmentation and Sleep Quality as a Function of Daily Rumination

	Fragmentation		Sleep Quality	
	B	**SE**	**B**	**SE**
Intercept	12.87**	2.49	2.66**	0.27
Time (months)	−0.01	0.02	0.001	0.002
Caregiver group	−0.59	0.56	−0.05	0.06
Rumination state	0.34	0.18	−0.03	0.02
Rumination trait	0.54	0.43	−0.14*	0.05
Age	−0.06	0.05	0.002	0.01
Body mass index	0.00	0.05	0.01	0.01
Antidepressant medication use	1.48	0.82	0.01	0.09
Caregiver group × Rumination state	−0.52*	0.24	0.05	0.03

Note. Depressive symptoms were sample centered, such that 0 equals the sample average. For caregiver group, 0 = M-NT, 1 = M-ASD.

* $p < .05$,

** $p < .001$.