Available online at www.sciencedirect.com
ScienceDirect
Behavior Therapy 50 (2019) 558–570
Behavior Therapy
www.elsevier.com/locate/bt

Effects of Rumination and Worry on Sleep

Olivia H. Tousignant
Nicholas D. Taylor
Michael K. Suvak
Gary D. Fireman
Suffolk University

Recent research suggests that the stress-sleep relationship is mediated by pre-sleep arousal (PSA) and that cognitive arousal has a stronger mediating effect than somatic arousal; however, this has not been directly tested. Using multilevel moderated mediation, we compared the effects of cognitive arousal and somatic arousal within the stress-sleep relationship. We also assessed whether two forms of repetitive negative thought—rumination and worry—are similarly involved in the stress-sleep relationship. Data was collected from 178 participants across the United States via an online platform. Participants completed baseline self-report surveys examining rumination tendencies and worry tendencies. Over the course of 2 weeks, participants completed daily questionnaires assessing daily stress, PSA, and sleep quality. Results indicated that indirect effects from stress to sleep quality via PSA were statistically significant at low and high levels of rumination and worry, and people at high levels of rumination and worry had stronger relationships between stress and PSA. Across all models, cognitive arousal consistently accounted for more of the variance in the stress-sleep relationship as compared to somatic arousal. Implications for the cognitive behavioral treatment of insomnia are discussed.

Nicholas D. Taylor is now at Capital OCD and Anxiety Practice, Austin, Texas.

This research did not receive any grant funding from agencies in the public, commercial, or not-for-profit sectors. This manuscript is based on data also used in the doctoral dissertation of Nicholas D. Taylor.

Address correspondence to Olivia H. Tousignant, Psychology Department, Suffolk University, Boston, MA 02108; e-mail: otousignant-pienkos@su.suffolk.edu

Keywords: sleep; stress; arousal; rumination; worry

THE RECIPROCAL RELATIONSHIP between increased stress and poor sleep is well established as a global risk factor for developing mental and physical illness (American Academy of Sleep Medicine, 2014). Evidence suggests a contributory role of cognition in this reciprocal relationship, such that increased *perceived* stress and poor sleep can increase the perception of poor functioning, which may contribute to the subsequent perception of heightened stress and, thus, poor sleep (Akerstedt et al., 2015; Garde, Albertsen, Persson, Hansen, & Rugulies, 2012; Lin, Liao, Chen, & Fan, 2014; Oh, Im, & Suk, 2016). Within sleep research, subjective sleep *quality* is an especially important outcome of interest due to its role in facilitating a sense of well-being and increased quality of life (Pilcher, Ginter, & Sadowsky, 1997; Weinberg, Noble, & Hammond, 2016). Considering that stress and sleep difficulties are involved in the development of mental and physical illnesses, it is important to continue elucidating mechanisms that perpetuate a maladaptive stress-sleep relationship. Notably, heightened arousal that occurs during the sleep onset period (pre-sleep arousal; PSA) has been established as a strong mediator through which a maladaptive relationship between stress and sleep quality can be perpetuated (Morin, Rodrigue, & Ivers, 2003; Winzeler et al., 2014). Two subcomponents of PSA have been identified: cognitive arousal, defined as intrusive cognitions experienced as uncontrollable, and somatic arousal, likened to physiological arousal (Nicassio, Mendlowitz, Fussell, & Petras, 1985). Currently, it is unclear how these component processes of arousal uniquely contribute to disruptive PSA. For instance, since cognitive arousal is defined broadly as excessive mentation (Nicassio et al., 1985), it does not distinguish between purely maladaptive cognitive activity, such as repetitive negative thought and other types of cognition. Rumination and worry, two forms of repetitive thought, include content with a distinctly negative valence and are oriented toward the past and future, respectively. Aiming to parse out the effects of these maladaptive thought processes on arousal and sleep quality as conceptualized through the empirically established PSA mediation model (Winzeler et al., 2014), we investigate how rumination and worry each influence the mediation of PSA in the relationship between stress and sleep quality. Additionally, no studies have examined whether repetitive negative thought affects specific subcomponents of PSA (cognitive versus somatic arousal) to differentially predict sleep quality. Further exploration of mechanisms that drive PSA may inform cognitive-behavioral interventions for people suffering from poor sleep and high stress.

To date, only two studies (Morin et al., 2003; Winzeler et al., 2014) have compared the strength of the indirect effects of stress on sleep through cognitive versus somatic arousal. Morin and colleagues (2003) found that females with and without insomnia diagnoses experienced more cognitive PSA than somatic PSA. Expanding on Morin et al.'s study by examining multilevel dynamics involved in the PSA mediation, Winzeler et al. (2014) confirmed that total PSA (cognitive plus somatic PSA) mediated the inverse relationship between perceived stress and subjective sleep quality. Moreover, they found that somatic arousal contributed a significant amount of variance to sleep quality on the between-participant level, while cognitive arousal did so on the within-participant level (i.e., relative to their own mean levels, participants who reported decreases in sleep quality also experienced increases in stress and cognitive arousal). This latter finding suggests that individual baseline variables involved in the cognitive processing of stress, such as levels of rumination and worry, may augment the mediation of the relationship between stress and sleep quality by cognitive and somatic arousal. More research is needed to explore these cognitive processes and relationship dynamics (Yeh, Wung, & Lin, 2015). Since Winzeler's findings were based on a young adult female sample, studies using a mixed-gender sample with a wider age range may increase the external validity of the mediation model. Addressing this sample limitation, we examine cognitive arousal and somatic arousal as mediators of the relationship between stress and sleep quality. Further, we compare the moderating effects of rumination versus worry—two types of repetitive negative thought based in the past versus future, respectively—in the mediation of the stress-sleep relationship via cognitive and somatic arousal.

We expect that people who have a baseline tendency to ruminate or worry in response to perceived stressors will exhibit increased PSA as it affects sleep quality. Within sleep literature, Harvey's (2002) Cognitive Model of Insomnia has received strong empirical support for comprehensively delineating the initiation and maintenance of sleep difficulties for people diagnosed with insomnia; however, it is unclear whether this model fits the experiences of healthy sleepers as well. Using a healthy sample, we assess the applicability of Harvey's model to people with subclinical levels of sleep difficulties. Specifically, we aim to explore factors involved in the stress-sleep relationship that may contribute to the development of sleep disorders. Evaluating normative mechanisms involved in Harvey's model that operate in nonclinical samples may elucidate ways that a maladaptive stress-sleep cycle begins to form. This information may help healthcare providers identify people at greatest risk for developing disorders that manifest following high stress and poor sleep quality.

Harvey's theory offers a strong rationale for how we would expect rumination and worry to affect the PSA mediation. The model suggests that cognitive activity that is excessive and negatively valenced initiates and perpetuates a cycle of disturbed sleep and may delay recovery from daytime stress. This delay in recovery can elicit arousal that continues into the pre-sleep period. During the pre-sleep period, increased cognitive activity evokes selective attention toward perceived threat cues, such as internal cognitive and somatic arousal experiences, which can trigger more cognitive arousal and further delay sleep onset. A long delay in sleep onset may result in subsequent perception of poor sleep quality, which can be experienced as a stressor, and thus, affect subsequent sleep experiences. As suggested by the cognitive and somatic components of Harvey's (2002) model, we expect that cognitive arousal and somatic arousal each would account for a significant portion of the variance in the stress-sleep relationship. Further, because Harvey's theory suggests that cognitive arousal is involved in the initiation *and* the perpetuation of a maladaptive stress-sleep relationship, we explore the manner by which cognitive activity, specifically, contributes to poor sleep quality. The question becomes, *To* *what extent does repetitive negative past- versus future-oriented cognitive activity affect sleep quality?* Although rumination, worry, sleep disturbance, and maladaptive stress processing may be transdiagnostic factors common to many disorders (Fairholme et al., 2013; Samtani & Moulds, 2017), no study to date has examined dynamics in their relationships using advanced statistical modeling. Evaluating the degree to which someone's tendency to ruminate versus worry intensifies cognitive and somatic arousal during the pre-sleep period and reduces sleep quality may elucidate intervention priorities for addressing the initial development of sleep onset difficulties. The current study explores the distinct strengthening roles of past-focused rumination and future-focused worry within the stress-sleep relationship as mediated by PSA. Further, the current study contributes to the empirical debate about the functional distinctness and relatedness of rumination and worry, specifically in this study as related to PSA and sleep quality.

Rumination and worry share characteristics of repetitive cognitive mechanisms with negative emotionality and orientation away from the present (Watkins, Moulds, & Mackintosh, 2005), yet evidence suggests that they may have independent effects on sleep processes. For example, an exploratory factor analysis on rumination and worry revealed a three-factor solution including high worry items, high rumination items, and absence of worry items (Carney, Harris, Moss, & Edinger, 2010). Within current research, rumination is described as an experientially avoidant emotion regulation strategy employed in response to perceived discrepancies between actual and preferred outcomes based in the past (Smith & Alloy, 2009). Rumination is generally considered a maladaptive coping strategy that is associated with increased negative affect and reduced inhibition of negative irrelevant information (Donaldson, Lam, Mathews, 2007; Joormann & Gotlib, 2010). It is often employed in response to perceived stressors and may delay recovery from stressful experiences (Cropley, Rydstedt, Devereux, & Middleton, 2015; Ruscio et al., 2015). Additionally, rumination has a significant negative association with subjective sleep quality, and this inverse relationship may be strengthened when rumination is employed during the pre-sleep period following a stress experience (Guastella & Moulds, 2007; Thomsen, Mehlsen, Christensen, & Zachariae, 2003; Zoccola, Dickerson, & Lam, 2009). Therefore, we expect that high rumination tendencies will strengthen the positive association between stress and PSA and the negative association between PSA and sleep quality (Morin et al., 2003).

Worry can be defined as a process of future-oriented perseverative problem-solving on an issue whose outcome is uncertain but could result in a negative outcome (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Common to the human experience, worry-related processes exist in clinical and nonclinical populations (Korte, Allan, & Schmidt, 2016). Although worry-related cognitions can motivate problem-solving and may help with short-term management of distress associated with uncertainty, they can become maladaptive and limiting when used as danger-avoidance mechanism for perceived threats that may not exist (Lee, Orsillo, Roemer, & Allen, 2010; Starcevic & Berle, 2006). For example, high levels of worry may facilitate avoidance through cognitive preoccupation with interpreting internal sensations as indicators of threat (Borkovec, Ray, & Stöber, 1998). As such, an interoceptive avoidance model of worry describes a characteristic aversion to somatic sensations: someone views an initial physiological sensation as threatening, becomes hypervigilant to continued physiological sensations, and initiates excessive anticipatory cognitive processing (i.e., worry) about the meaning of the sensations, thus avoiding the feeling of uncertainty inherent to living (Borkovec, Alcaine, & Behar, 2004). Considering Harvey's model, we expect that both cognitive and somatic arousal processes would contribute to the maintenance of worry-related sleep disturbances. Although the interoceptive model of worry suggests strong somatic involvement in certain disturbances (e.g., Generalized Anxiety Disorder; Borkovec et al., 2004), Harvey's (2002) theory suggests strong involvement of the cognitive component of worry in the initiation and maintenance of sleep disturbances. Additional research suggests that cognitive and somatic arousal components of worry maintain PSA that contributes to the perception of longer sleep-onset latency, decreased sleep quality, and negative consequences of poor sleep (Tang & Harvey, 2004; Wicklow & Espie, 2000). Therefore, we expect that high worry tendencies will strengthen the positive association between stress and PSA and the negative association between PSA and sleep quality.

Despite the common characteristics and moderate correlation between rumination and worry (Segerstrom, Tsao, Alden, & Craske, 2000), evidence suggests that rumination and worry are distinct cognitive processes with differential relationships to sleep (Carney et al., 2010; Yang et al., 2014). To inform intervention efforts, it is important to determine the extent to which rumination and worry contribute common and unique variance to sleep quality outcomes. Thus, we examine rumination and worry as moderators of the PSA mediation model. Considering that repetitive negative thought processes are likely involved in Harvey's (2002) Cognitive Model of Insomnia, we expect that rumination and worry each will strengthen the indirect effect of stress on sleep through PSA, more so through cognitive arousal than through somatic arousal. Although we do not study people diagnosed with insomnia, we investigate those factors that may contribute to the development of insomnia.

In the only study on rumination and worry within the PSA mediation model, a factor analysis showed that two shared components of rumination and worry—(a) dwelling on the negative and (b) repetitive thought—were significantly associated with PSA and sleep quality (Yeh et al., 2015). We expand on the study by Yeh and colleagues (2015) in two ways: we address the distinction of cognitive versus somatic arousal by investigating which form of arousal accounts for more of the variance in the stress-sleep relationship and we analyze relationships between daily measures of stress, PSA, and sleep quality, which is different from the previous study (Yeh et al., 2015) that primarily examined cross-sectional relationships between rumination, worry, PSA, and sleep quality. (In the current study, the term *daily* describes self-report data that is measured each morning in reference to stress, PSA, and sleep quality from the previous day and night.) Hoping to reveal more nuanced information about the effects of rumination and worry on the PSA mediation model, we investigate how relationships between daily measures at the within-participant level are regulated by between-participant variables (levels of rumination and worry). First, we assess whether cognitive or somatic arousal accounts for more variance in the stress-sleep relationship. Second, using multilevel moderated mediation, we compare the strengths of rumination and worry as moderators of the PSA mediation model (i.e., cognitive versus somatic arousal as a mediator of the stress-sleep relationship).

We predict that: (a) cognitive arousal, compared to somatic arousal, will account for more of the variance in the stress-sleep mediated relationship, (b) participants reporting high levels of rumination will experience more cognitive arousal than somatic arousal as it affects sleep quality, and (c) participants reporting high levels of worry will experience more cognitive arousal than somatic arousal as it affects sleep quality.

Method

PARTICIPANTS

The study was approved by a university Institutional Review Board. Data was collected using the Social Sci online platform. An informed consent script was provided for all participants prior to the study, and participants were offered the opportunity to email the investigators to ask questions. Eligibility criteria included that participants were between age 18 and 70 to minimize age-related variability in sleep processes and to maintain measure validity. Exclusionary criteria required that participants were not taking medication(s) that depress or activate the central nervous system, were not addicted to alcohol and/or other drugs, were not taking sleep medication, and were not diagnosed with a sleep disorder, such as sleep apnea.

Among 250 people who initially accessed the study, 6 people (2.4%) were excluded because they participated in the study despite falling outside the specified age range, and 2 participants (0.8%) were excluded from analyses because their reporting patterns seemed spurious (e.g., they reported either maximum or minimum scores across every measure). Two participants met more than one of the exclusionary criteria. The largest source of sample attrition came from participant withdrawal and incomplete data such that 66 participants (26.4%) did not complete a single survey across the 2-week reporting period.

The sample used for analyses consisted of 178 participants (71.2% of the original 250-person sample) for whom there was at least one complete day of data for daily stress, PSA, and sleep quality measures. Among these 178 participants, the average number of days that participants completed the daily diary measures was 9.33 (SD = 5.37). Most participants self-identified as White (n = 132, 74.2%), 23 participants (12.9%) identified as Asian, 12 (6.7%) identified as African American, 6 (3.4%) as Latino/a, and 3 (1.7%) as mixed ethnicity. Two participants (1.1%) did not report their race. This sample included 110 self-identified females (61.8%) and 67 self-identified males (37.6%). One participant (0.6%) did not report gender. On average, participants were 29.95 years old (SD = 10.55, range 18-68) upon entry in the study.

MEASURES

Baseline Self-Report Measures

The Response Styles to Depression Questionnaire, Rumination Subscale (RSDQ; Nolen-Hoeksema, 1991) offered a cross-sectional self-report measure of each participant's baseline tendency to ruminate in response to depressive feelings. The subscale has 22 items and takes approximately four to five minutes to complete. Participants indicate on a 4-point Likert scale the frequency with which they engage in ruminative behaviors in response to feeling stressed, sad, or upset. Sum scores are calculated with higher scores indicating more rumination. The subscale has been shown to have good convergent validity, correlating positively with depression and negatively with positive affect (Knowles, Tai, Christensen, & Bentall, 2005). Within our sample this measure had strong internal reliability (Cronbach's $\alpha = .95$).

The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) provided a cross-sectional self-report measure of each participant's baseline tendency to worry. This scale has 16 items and takes approximately three to four minutes to complete. Participants indicate on a 5-point Likert scale the degree to which worry statements apply to them. The scale contains five reverse-coded items (1, 3, 8, 10, and 11), and sum scores are calculated with higher scores indicating more worry. The measure contains 16 items with strong internal consistency, high test-retest reliability, and predictive validity with worry frequency and worry duration (Verkuil & Brosschot, 2012). Within our sample, this measure had strong internal reliability (Cronbach's $\alpha = .95$).

Daily Self-Report Measures

The Daily Stress Inventory (DSI) provided a daily measure of the impact of a participant's stress experience (Brantley, Waggoner, Jones, & Rappaport, 1987). Reporting on the preceding 24-hour period, participants indicated whether each of 53 events occurred for them (with the option to write in three 'other' events), and they provided an impact rating on a 7-point Likert scale (1 = *occurred but was not stressful* to 7 = *caused me to panic*) for each event that occurred. The measure has strong concurrent validity with measures of stress and sound internal validity ($\alpha = .83$–$.87$; Brantley et al., 1987). We used the impact rating sum as an indicator of perceived daily stress, with higher values indicating higher perceived stress.

The Pre-Sleep Arousal Scale (PSAS) offered a daily measure of cognitive and somatic arousal experienced at bedtime (Nicassio et al., 1985). Participants answered 16 questions total, eight corresponding to cognitive arousal and eight corresponding to somatic arousal. All questions use a 5-point Likert scale (1 = *not at all* to 5 = *extremely)* for rating the intensity of cognitive arousal (e.g., *Thoughts keep running through your head; Being distracted by sounds, noise in the environment; Review or ponder events of the day*) and somatic arousal (e.g., *A jittery, nervous feeling in your body; Shortness of breath or labored breathing; Perspiration in palms of your hands or other parts of your body*). A total score from eight to 40 is calculated for each subscale, and higher scores indicate higher arousal. The subscale scores are added to create a total PSA score. Each subscale demonstrates good test-retest reliability over a three-week interval and shows adequate internal consistency. The PSAS also shows good construct validity and is correlated with measures of sleep disturbance, anxiety, and depression. Prior research also demonstrates that the cognitive subscale and the somatic subscale are correlated with rumination ($r = 0.22$, $p < .001$ and $r = 0.24$, $p < .001$, respectively; Palagini, Moretto, Dell'Osso, & Carney, 2017) and with worry ($r = 0.38$, $p < .001$ and $r = .33$, $p < .001$, respectively; Yeh et al., 2015). The PSAS is a stronger predictor of sleep onset latency than it is of total sleep time or sleep interruptions. Additionally, individuals with insomnia score higher on both subscales than control participants. Within our sample this measure had strong internal reliability (Cronbach's $\alpha = .96$).

The Core Consensus Sleep Diary provided a daily measure of subjective sleep quality. It is a 9-item measure that was developed with the intention of standardizing sleep diary use within research and clinical practice (Carney et al., 2012). To capture self-reported sleep quality, we conducted analyses with participants' answers to the item, *How would you rate the quality of your sleep?* Ratings were captured using a 5-point Likert scale. Higher ratings for this question reflect better subjective sleep quality. This daily measure of sleep quality has been used in prior studies (e.g., Winzeler et al., 2014).

PROCEDURE

Data for the study was collected in two phases using the online survey and recruitment platform, Social Sci. After reviewing inclusionary criteria and the informed consent statement, consenting participants completed a series of self-report, baseline measures including a brief demographic questionnaire, the RSDQ (Nolen-Hoeksema, 1991) to measure baseline rumination, and the PSWQ (Meyer et al., 1990) to measure baseline worry. For up to 14 consecutive days, participants completed the DSI (Brantley et al., 1987), the PSAS (Nicassio et al., 1985), and the Core Consensus Sleep Diary (Carney et al., 2012). To facilitate adherence, each morning participants received email prompts with instruction reminders and a link to the day's surveys. Participants were asked to complete all three daily measures upon waking and to report on the previous day's stress, PSA, and sleep quality. Online survey completion was time-stamped, which allowed us to see the time of survey completion and the duration of time spent responding. All participants included in the data analyses responded within expected limits.

DATA ANALYSES

We used IBM SPSS for data management and the computation of descriptive statistics. To account for change over time and to examine the mediation model purely from a within-subject level, we controlled for each participant's mean level of stress, PSA, and sleep quality. As described by Curran and Bauer (2011), repeated measures designs contain information about both between-person and within-person differences, and sometimes the presence of between-person variance can impact or contaminate the interpretation of within-person processes. Thus, to disaggregate the within-subject processes from the between-subject variance, we followed the procedures recommended by Curran and Bauer (2011). Using Hierarchical Linear Modeling (HLM), we computed a model with participants' mean level of a repeated measure across all assessments as a Level-2 predictor of individual assessments of that measure. To remove the impact of drifts over time in the measure, we included time as a Level-1 predictor. The Level-1 residuals from these models were saved, creating an index of the repeated measures that was disaggregated from between-subject variance and the impact of time. These residual terms were used in the mediation model examining the within-subject associations among stress, PSA, and sleep quality. To account for the skewed distribution of the stress and PSA residuals, we calculated the natural log (ln) and used these variables in our subsequent analyses. Using MPlus software, we conducted a multilevel moderated mediation to examine the moderating effects of rumination and worry on the mediation model comparing the intervening effect of cognitive arousal to that of somatic arousal.

To evaluate our moderated mediation models, we followed guidelines outlined by Muller, Judd, and Yzerbyt (2005). A necessary precondition for testing moderated mediation is that there should be no statistically significant interaction between the moderator and predictor on the outcome when controlling for the mediator. In our study, there were no statistically significant interactions between stress (the predictor) and rumination and worry (the moderators) on sleep quality (the outcome) when controlling for PSA (the mediator). To depict the nature of the moderated-mediations in the current study, we adopted the commonly used practice (Cohen, Cohen, West, & Alken, 2003) of depicting the mediation model at one standard deviation above and below the moderator, including the calculation of percent mediation (which is an indicator of the strength of the *ab-path*). We used the RMediation software package (http://www.amp.gatech.edu/RMediation) to calculate confidence intervals for the indirect effect of stress on sleep quality via cognitive versus somatic PSA. Because multilevel regression does not produce a standardized regression coefficient, we report d (calculated using the formula $d = \frac{2Z}{\sqrt{n}}$) with .20, .50, and .80 as the commonly used cutoffs for small, medium, and large effects, respectively.

Results

PRELIMINARY ANALYSES

Participants' average level of daily sleep quality was 5.17 (SD = 1.53, range = 1–9) and their average level of daily stress impact was 43.00 (SD = 49.57, range = 0–266). Their average level of total PSA was 25.08 (SD = 8.77, range = 16–56), pre-sleep cognitive arousal was 14.06 (SD = 5.65, range = 8–33), and pre-sleep somatic arousal was 11.03 (SD = 3.88, range = 8–28). Participants' mean rumination scores were 30.37 (SD = 13.99, range = 0–63) and mean worry scores were 49.90 (SD = 14.48, range = 16–80).

Multilevel regression indicated a strong association between somatic and cognitive arousal ($r = .70$, $p < .001$); however, this association was not strong enough to suggest that these measures are tapping the same construct. Further, it raises the possibility that cognitive arousal and somatic arousal could account for some unique variance in sleep quality. A statistically significant and moderately strong correlation ($r = .58$, $p < .001$) emerged between rumination and worry, suggesting separate but overlapping constructs. Within the current sample, total PSA, cognitive arousal, and somatic arousal were correlated with rumination ($r = 0.35$, $p < .001$; $r = 0.33$, $p < .001$; and $r = 0.30$, $p < .001$, respectively) and worry ($r = 0.38$, $p < .001$; $r = 0.39$, $p < .001$; and $r = 0.28$, $p < .001$; respectively).

PSA MEDIATION MODEL

Prior to examining somatic and cognitive arousal as potential mediators in the relationship between stress and sleep quality, we first evaluated a model with stress as a Level-1 predictor of sleep quality to estimate *path c*, the relationship between the predictor and outcome when the mediators are not in the model. This indicated a significant relationship between stress and sleep quality (*path c;* B = -.52, cr = -5.63, $p < .001$, d = -.84; cr, critical ratio, is the estimate divided by the standard error of the estimate and is analogous to a z- or t-statistic). We next evaluated the overall PSA variable (combining both somatic and cognitive arousal; Nicassio et al., 1985) as a within-subjects mediator in the relationship between stress and sleep quality by conducting multilevel regressions in Mplus. This approach allowed us to simultaneously compute all paths of mediation (see Figure 1) in one analysis. Results indicated that stress was a statistically significant predictor of PSA (*path a; B* = .12, *cr* = 10.69, *p* < .001, *d* = 1.60) and that PSA was a statistically significant predictor of sleep quality (*path b*; *B* = -3.78, *cr* = -11.44, *p* < .001, *d* = -1.71) when controlling for stress. The indirect effect was also statistically significant (*ab* = -0.45, 95% CI = -0.57 to -0.35). Percent mediation indicated that PSA accounts for about 85.8% of the relationship between stress and sleep quality. Consistent with previous studies (Morin et al., 2003; Winzeler et al., 2014), these results provide support for PSA as a mediator of the relationship between stress and sleep quality. The direct relationship of stress predicting sleep quality, when controlling for PSA, was not statistically significant (*path c'*; *B* = -.07, *cr* = -.88, *p* = .380, *d* = -.13).

Next, we separated PSA into cognitive and somatic components, and included these as two intervening variables in the same multilevel-mediation model. Results indicated that stress predicted cognitive arousal (*path a; B* = 0.14, *cr* = 9.71, *p* < .001, *d* = 1.46) and that cognitive arousal predicted sleep quality (*path b*; *B* = -2.00, *cr* = -7.83, *p* < .001, *d* = -1.17) when controlling for both stress and somatic arousal. The indirect effect was also statistically significant (*ab* = -0.27, 95% CI = -0.37 to -0.19, COV*ab* = 0.00; COV*ab* refers to the covariance of the *ab-path*). Percent mediation indicated that cognitive arousal accounts for about 55.2% of the relationship between stress and sleep quality. Additionally, this model indicated that stress predicted somatic arousal (*path a; B* = .09, *cr* = 7.95, *p* < .001, *d* = 1.19) and that somatic arousal predicted sleep quality (*path b*; *B* = -2.13, *cr* = -5.48, *p* < .001, *d* = -0.82) when controlling for stress and cognitive arousal. The indirect effect was also statistically significant (*ab* = -0.19, 95% CI = -0.28 to -0.12, COV*ab* = 0.06). Percent mediation indicated that somatic arousal accounts for about 26.3% of the relationship between stress and sleep quality. Consistent with our first hypothesis, cognitive arousal accounted for more of the variance in the relationship between stress and sleep quality, compared to the variance accounted for by somatic arousal.

RUMINATION AS MODERATOR OF MEDIATION MODEL

To evaluate our second hypothesis, that participants reporting high levels of rumination will experience more cognitive arousal than somatic arousal as it affects sleep quality, we conducted multilevel regression analyses to examine baseline rumination (a between-subject variable) as a moderator of the relationship between stress, cognitive and somatic arousal, and sleep quality (i.e., *path a, b, c,* and *c'*) in the mediation model (Figure 2). Prior to examining rumination as a moderator of the mediation analyses, we examined whether or not rumination predicted *path c* (the stress-sleep association when the mediators are not in the model); as reported in Table 1, the Rumination × Stress interaction was not statistically significant. We then calculated interaction terms by multiplying the mean centered predictors (i.e., Rumination × Stress and Rumination × PSA), and we included these interaction terms as predictors of the mediator (*path a*) and the outcome (*path b* and

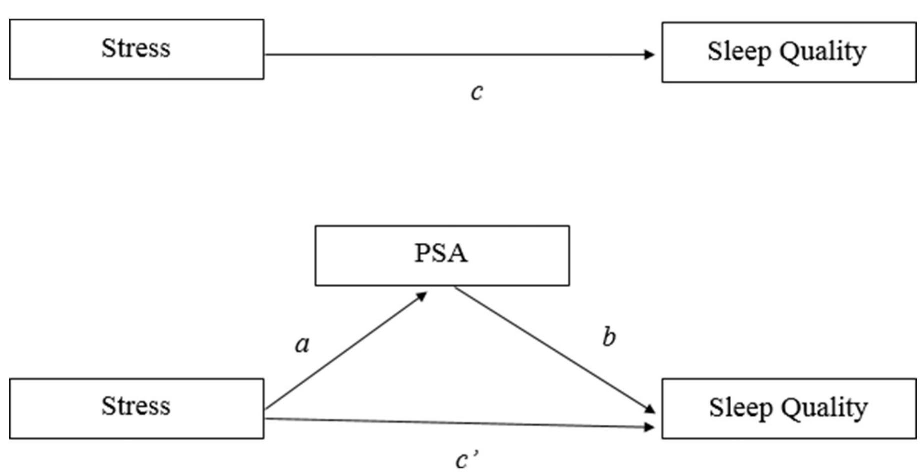

FIGURE 1 Schematic of Within-Subject Mediation Paths. Paths *a* (predictor to mediator), *b* (mediator to outcome when controlling for the predictor), *c* (predictor to outcome), and *c'* (predictor to outcome when controlling for the mediator) represent the relationships involved in assessing mediation.

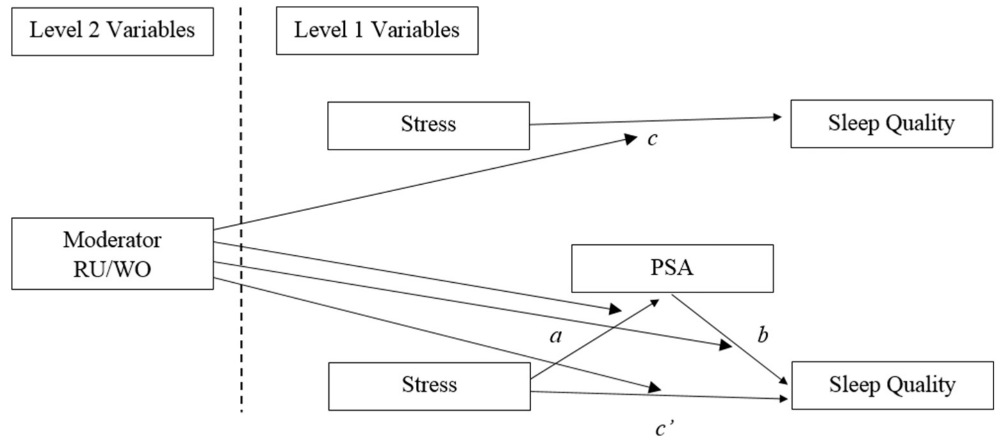

FIGURE 2 Schematic of Moderation and Mediation Model Paths. The multilevel component refers to level-1 daily assessments (within-participant effects) that were nested within individuals (between-participant effects). This analysis is like a standard cross-sectional analysis except that every person has an *a-path* and a *b-path*, thus there can be correlation between *a* and *b*. Paths *a* (predictor to mediator), *b* (mediator to outcome when controlling for the predictor), *c* (predictor to outcome), and *c'* (predictor to outcome when controlling for the mediator) represent the relationships involved in assessing mediation. The box including rumination (RU) and worry (WO) represents the strength of paths *a* and *b* when moderated by baseline rumination and baseline worry (moderated-mediation). The test of mediation is the significance of the joint path (*ab)* from daily stress experience to daily sleep quality through PSA. Random total and random indirect effects are estimated using the method described in Bauer et al. (2006).

path c'). Table 1 reports the coefficients for interaction terms between rumination and each predictor of the mediation model and shows that both *a-paths* (i.e., paths from stress to PSA) significantly varied as a function of rumination whereas the *b-paths* did not. Although we analyzed rumination as a continuous variable, to illustrate the nature of the interactions we examined the mediation model at high levels of rumination and low levels of rumination (i.e., one standard deviation above the mean and one standard deviation below the mean, respectively; M = 31.21, SD = 14.05). At high levels of rumination, stress predicted cognitive arousal (*path a;* B = .19, cr = 8.99, p < .001, d = 1.35) and cognitive arousal predicted sleep quality (*path b*; B = -1.50, cr = -4.63, p < .001, d = -.69). Stress also predicted somatic arousal (*path a;* B = .12, cr = 6.11, p < .001, d = .92) and

Table 1
Coefficients for Interactions Involving the Moderators for the Moderated-Mediation Analyses

Moderator	Path	Predictor	Outcome	*Estimate*	*cr*	*p*	*d*
Rumination							
	c	Stress	Sleep Quality	-0.01	-1.34	.181	-0.20
	a	Stress	PSA_Cog	0.004	3.61	.000	0.54
	a	Stress	PSA_Som	0.002	2.07	.038	0.31
	b	PSA_Cog	Sleep Quality	0.035	1.87	.061	0.28
	b	PSA_Som	Sleep Quality	0.014	0.51	.611	0.08
	c'	Stress	Sleep Quality	-0.005	-0.78	.437	-0.12
Worry							
	c	Stress	Sleep	-0.01	-1.65	.099	-0.25
	a	Stress	PSA_Cog	0.004	3.99	.000	0.60
	a	Stress	PSA_Som	0.002	2.49	.013	0.37
	b	PSA_Cog	Sleep Quality	0.028	1.51	.131	0.23
	b	PSA_Som	Sleep Quality	0.001	0.03	.978	0.00
	c'	Stress	Sleep Quality	-0.004	-0.57	.568	-0.09

Note. PSA_Cog = cognitive pre-sleep arousal, PSA_Som = somatic pre-sleep arousal, *Estimate* = unstandardized regression coefficient, *cr* = critical ratio (estimate/standard error), *p* = p-value, and *d* = effect size estimate.

somatic arousal predicted sleep quality (*path b*; *B* = -1.97, *cr* = -3.99, $p < .001$, *d* = -.60). The indirect effect was statistically significant for cognitive arousal (*ab* = -0.29, 95% CI = -0.43 to -0.16, COV*ab* = -0.08) and somatic arousal (*ab* = -0.23, 95% CI = -0.38 to -0.11, COV*ab* = 0.06). Percent mediation indicated that cognitive arousal accounted for about 52.3% of the relationship between stress and sleep quality and somatic arousal accounted for about 24.5%.

At low levels of rumination, stress predicted cognitive arousal (*path a; B* = .09, *cr* = 5.20, $p < .001$, *d* = .78) and cognitive arousal predicted sleep quality (*path b*; *B* = -2.49, *cr* = -6.12, $p < .001$, *d* = -.92). Stress also predicted somatic arousal (*path a; B* = .07, *cr* = 4.89, $p < .001$, *d* = .73) and somatic arousal predicted sleep quality (*path b*; *B* = -2.36, *cr* = -3.98, $p < .001$, *d* = -.60). The indirect effect was statistically significant for cognitive arousal (*ab* = -0.22, 95% CI = -0.35 to -0.12, COV*ab* = -0.02) and somatic arousal (*ab* = -0.16, 95% CI = -0.27 to -0.07, COV*ab* = 0.06). Percent mediation indicated that cognitive arousal accounted for about 66.5% of the relationship between stress and sleep quality and somatic arousal accounted for about 26.0%. Across rumination levels, the relationship of stress predicting sleep quality when mediators were controlled for was not statistically significant (high rumination, *path c'*; *B* = -.16, *cr* = -1.09, *p* = .275, *d* = -.16, and low rumination, *path c'*; *B* = -.03, *cr* = -.23, *p* = .819 , *d* = -.03).

WORRY AS MODERATOR OF MEDIATION MODEL

We conducted similar analyses to examine the moderating effect of worry on the mediation model (Figure 1). Similarly to rumination, Table 1 indicates that the Worry × Stress interaction was not statistically significant, reflecting that rumination did not predict *path c*. However, in the analyses examining moderated mediation, statistically significant Worry × Stress interactions predicting PSA emerged. Table 1 also depicts the coefficients for the interaction terms between worry and each predictor of the mediation model and shows that again both *a-paths*, but neither *b-path*, significantly vary as a function of the moderator, worry. Although worry was analyzed as a continuous variable, to illustrate the nature of the effects we examined the mediation model at high levels of worry and low levels of worry (i.e., one standard deviation above the mean versus one standard deviation below the mean, respectively; *M* = 50.16, *SD* = 14.78). At high levels of worry, stress predicted cognitive arousal (*path a; B* = .20, *cr* = 9.34, $p < .001$, *d* = 1.40) and cognitive arousal predicted sleep quality (*path b*; *B* = -1.61, *cr* = -5.26, $p < .001$, *d* = -.79). Stress also predicted somatic arousal (*path a; B* = .12, *cr* = 6.98, $p < .001$, *d* = 1.05) and somatic arousal predicted sleep quality (*path b*; *B* = -2.13, *cr* = -4.89, $p < .001$, *d* = -.73). The indirect effect was statistically significant for cognitive arousal (*ab* = -0.31, 95% CI = -0.46 to -0.19, COV*ab* = -0.07) and somatic arousal (*ab* = -0.25, 95% CI = -0.39 to -0.14, COV*ab* = 0.06). Percent mediation indicated that cognitive arousal accounted for about 53.2% of the relationship between stress and sleep quality and somatic arousal accounted for about 26.4%.

At low levels of worry, stress predicted cognitive arousal (*path a; B* = .09, *cr* = 5.72, $p < .001$, *d* = .86) and cognitive arousal predicted sleep quality (*path b*; *B* = -2.42, *cr* = -5.59, $p < .001$, *d* = -.84). Stress also predicted somatic arousal (*path a; B* = .07, *cr* = 5.21, $p < .001$, *d* = .78) and somatic arousal predicted sleep quality (*path b*; *B* = -2.15, *cr* = -3.24, *p* = .001, *d* = -.49). The indirect effect was statistically significant for cognitive arousal (*ab* = -0.22, 95% CI = -0.34 to -0.12, COV*ab* = -0.01) and somatic arousal (*ab* = -0.14, 95% CI = -0.26 to -0.05, COV*ab* = 0.06). Percent mediation indicated that cognitive arousal accounted for about 64.2% of the relationship between stress and sleep quality and somatic arousal accounted for about 23.4%. Across worry levels, the relationship of stress predicting sleep quality when mediators were controlled for was not statistically significant (high worry, *path c'*; *B* = -.15, *cr* = -.94, *p* = .347, *d* = -.14, and low worry, *path c'*; *B* = -.05, *cr* = -.41, *p* = .680, *d* = -.06).

PATTERNS OF FINDINGS

While the *a-path* and the *b-path* were statistically significant across levels of rumination and worry, the *a-path* (stress to PSA) was stronger at high levels of rumination and worry (Table 1). Further, across all three models (i.e., mediation with no moderators, mediation with rumination moderator, and mediation with worry moderator), cognitive arousal consistently accounted for more of the variance in the relationship between stress and sleep quality compared to the amount of variance accounted for by somatic arousal. Although worry has been found to have a strong somatic component (Borkovec et al., 2004) in general, our analyses indicated that, across worry levels, cognitive arousal has a more powerful mediating effect in the relationship between stress and sleep quality. In support of our first, second, and third hypotheses, these patterns of findings highlight the important influence of cognitive arousal on the relationship between stress and sleep for people who ruminate and/or worry.

Discussion

This is the first study to use multilevel statistical modeling to investigate the effects of rumination and worry on the stress-sleep relationship as mediated by cognitive and somatic PSA. Cognitive arousal consistently accounted for more variance than somatic arousal in the multilevel moderated mediation models. Our findings corroborate the results of Winzeler and colleagues (2014), which showed that cognitive arousal mediated the stress-sleep relationship at the within-participant level, with increased cognitive arousal leading to decreased subjective sleep quality. In the current study, the within-participant mediation model revealed significant indirect effects via both cognitive and somatic arousal, with cognitive arousal accounting for more of the variance in the stress-sleep relationship. The within-participant effects remained constant when controlling for between-participant moderating variables. These findings support our first hypothesis and suggest that cognitive arousal reduction may be an important target for reducing PSA to improve the stress-sleep relationship and that initial reduction of cognitive arousal may facilitate sleep quality improvements.

Our pattern of results supports a cognitive primacy effect as indicated by our finding that cognitive arousal accounts for more variance than somatic arousal within the stress-sleep relationship. This pattern is consistent with Harvey's model (2002), a central tenet being that cognitive processes promote an individual's preoccupation with sleep problems. Through repetition of a cycle characterized by narrowing of attention toward sleep-related threat cues (e.g., excessive thoughts) an individual can develop a distorted perception of sleep quality, which may contribute to accumulated stress. Remaining consistent with Harvey's model, we found that somatic arousal also accounts for a substantial portion of the variance in the stress-sleep relationship. This pattern may coincide with the stage of Harvey's model during which internal somatic sensations are perceived as indicators of threat and evoke additional cognitive arousal.

The dual mediating roles of cognitive and somatic arousal may indicate that the utility of Harvey's model for treating people with insomnia generalizes to include healthy people experiencing sleep disruption. It could be that, together, these pieces of Harvey's model (i.e., cognitive and somatic components that form a self-reinforcing cycle that delays sleep onset) signify the etiological root of sleep-related mental disorder. By continuing to explore PSA as a mediator in the relationship between stress and sleep quality, we are working toward understanding important targets of intervention for promoting a healthy stress-sleep relationship.

In partial support of hypotheses two and three, people reporting high levels of rumination and worry had stronger relationships between stress and PSA. These findings suggest that the cognitive-somatic arousal cycle may be especially applicable to people who experience high degrees of repetitive negative thought (as evidenced by significant interactions between rumination and stress as well as worry and stress predicting both cognitive and somatic PSA). The finding that high levels of rumination and worry strengthen relationships between stress and PSA aligns with Garde and colleagues' evidence (2012) supporting a self-reinforcing bidirectional relationship between stress and sleep. It seems that daytime cognitive preoccupation with the previous or upcoming night's sleep quality (a form of stress) may perpetuate disturbed sleep. Regarding clinical intervention implications, people who have disorders characterized by high levels of repetitive negative thought and sleep disturbances may benefit from cognitive arousal reduction most when it occurs following daytime stress experiences. Practicing cognitive arousal reduction strategies throughout the day may improve sleep quality by contributing to decreased PSA.

Across all models, we found that cognitive arousal had a strong mediating effect on the relationship between stress and sleep quality; this finding is consistent with the results from several well-designed sleep studies (e.g., Carney et al., 2010; Garde et al., 2012; Morin et al., 2003; Winzeler et al., 2014). Our results add to the literature by confirming, through multilevel moderated mediation, that cognitive PSA accounts for a substantial portion of the variance within the stress-sleep relationship. It seems that the common elements of rumination and worry (repetitive and negative thoughts), rather than the distinct elements (past versus future focus), may predict poorer sleep quality. These results suggest that interventions aimed at slowing down the perceived pace of thoughts and reducing the negative valence of thought content (e.g., through mindfulness practices) would help people with poor sleep. The current study also suggests substantial benefit to supplementing cognitive treatment with bodily relaxation techniques. To facilitate treatment efficiency and cost-effectiveness, it may be worthwhile to conduct a dismantling design study that compares a cognitive treatment condition to a cognitive plus somatic treatment condition for improving sleep quality. Our study elucidates important avenues through which we can continue learning how to improve the sleep quality of many people.

To facilitate appropriate interpretation of results, it is important to recognize that rumination and worry emerged as significant predictors of the *a-paths* (i.e., stress to PSA), but worry and rumination did not predict the *b-paths* (i.e., PSA to sleep quality). This suggests that the moderating effects of worry and rumination are specific to the effect of stress on both components (cognitive and somatic) of PSA (Table 1).

Our study had three primary limitations. First, measures of stress, PSA, and sleep quality were collected each morning for up to 14 days, and participants reported on experiences that occurred the previous day and night. Although we chose to collect data in this way so that we would not disturb participants' pre-sleep experiences, obtaining all daily measures in the morning prevented us from using more advanced statistical analyses, such as latent growth curve modeling, to examine change over time within the multilevel moderated mediation. Additionally, participants may have been inaccurate reporters about the previous day and night. Finally, the sample was limited in racial and ethnic diversity, which may limit the external validity of our findings. In future sleep research, more effort should be made to oversample from minority populations.

Future research would benefit from including objective measures of stress, PSA, and sleep quality (e.g., through cortisol sampling or actigraphy measures) to supplement self-report measures. This may promote assessment accuracy and compensate for participants' errors in memory when reporting about stress, PSA, and sleep quality. Additionally, to continue investigating the moderating effects of rumination and worry within the mediation model, future research could explore relationships between daily measures of rumination and worry as they relate to within-subject variability of the moderated mediation. This investigation may reveal certain contexts (e.g., when experiencing high daytime rumination and worry levels) in which the relationship between stress and sleep is prone to becoming maladaptive. Additionally, the research literature on mechanisms that contribute to insomnia would benefit from including a measure of repetitive positive thought to assess its relative influence on cognitive and somatic arousal and sleep quality. This avenue of research may help clarify components involved in the etiologies of different psychopathologies associated with sleep problems (e.g., depression compared to bipolar disorder).

Additionally, future research could vary the time of day and evening at which daily measures are collected. For example, on certain days, each participant could complete all three measures in the morning (as done in the current study); on other days, participants could report about stress and PSA during the time that it is occurring. By varying data collection times, we may see whether relationships between within-subject variables shift as a function of data collection time, thus informing sleep research methods.

In sum, the current study demonstrates that cognitive arousal consistently accounts for more of the variance in the mediated stress-sleep relationship as compared to somatic arousal, though cognitive and somatic arousal each have a significant indirect effect in the stress-sleep relationship. These effects endured across all multilevel moderated mediation models. Taken together, these findings suggest that people who have difficulty falling asleep and/or experience poor sleep quality may benefit from cognitive arousal reduction interventions, prior to somatic arousal reduction interventions, to maximize efficiency and cost-effectiveness. The results of the current study are consistent with research demonstrating that PSA is a powerful mediator in the stress-sleep quality relationship and that cognitive arousal is a uniquely powerful mediator at the within-participant level (Morin et al., 2003; Winzeler et al., 2014). Regarding the mediation of the stress-sleep relationship, the present study clarifies that repetitive negative thought processes focused on the past versus future affect PSA similarly, and it provides support for a ubiquitous salutary effect of cognitive arousal reduction in improving sleep quality.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

References

American Academy of Sleep Medicine. (2014). *The International Classification of Sleep Disorders, Third Edition (ICSD-3)*. https://doi.org/10.1378/chest.14-0970

Akerstedt, T., Garefelt, J., Richter, A., Westerlund, H., Hanson, L. L., Sverke, M., & Kecklund, G. (2015). Work and sleep—A prospective study of psychosocial work factors, physical work factors, and work scheduling. *Sleep*, *38*, 1129–1136. https://doi.org/10.5665/sleep.4828

Bauer, D. J., Preacher, K. J., & Gil, K. M. (2006). Conceptualizing and testing random indirect effects and moderated mediation in multilevel models: New procedures and recommendations. *Psychological Methods*, *11*, 142–163. https://doi.org/10.1037/1082 989X.11.2.142

Borkovec, T. D., Alcaine, O. M., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heinberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Borkovec, T. D., Ray, W. J., & Stöber, J. (1998). Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. *Cognitive Therapy and Research*, *22*, 561–576. https://doi.org/10.1023/A:101879000

Borkovec, T. D., Robinson, E., Pruzinsky, T., & DePree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behaviour Research and Therapy*, *21*, 9–16. doi:0005-7967/010009-85303.0/0

Brantley, P. J., Waggoner, C. D., Jones, G. N., & Rappaport, N. B. (1987). A daily stress inventory: Development, reliability, and validity. *Journal of Behavioral Medicine*, *10*, 61–74. https://doi.org/10.1007/BF00845128

Carney, C. E., Harris, A. L., Moss, T. G., & Edinger, J. D. (2010). Distinguishing rumination from worry in clinical insomnia. *Behaviour Research and Therapy*, *48*, 540–546. https://doi.org/10.1016/j.brat.2010.03.004

Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2012). The Consensus Sleep Diary: Standardizing prospective sleep self-monitoring. *Sleep: Journal of Sleep and Sleep Disorders Research*, *35*, 287–302. https://doi.org/10.5665/sleep.1642

Cohen, J., Cohen, P., West, S. G., & Alken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). New York, NY: Routledge.

Cropley, M., Rydstedt, L. W., Devereux, J. J., & Middleton, B. (2015). The relationship between work-related rumination and evening and morning salivary cortisol secretion. *Stress Health*, *31*, 150–157. https://doi.org/10.1002/smi.2538

Curran, P. J., & Bauer, D. J. (2011). The disaggregation of within-person and between-person effects in longitudinal models of change. *Annual Review of Psychology*, *62*, 583–619. https://doi.org/10.1146/annurev.psych.093008.100356

Donaldson, C., Lam, D., & Mathews, A. (2007). Rumination and attention in major depression. *Behaviour Research and Therapy*, *45*, 2664–2678. https://doi.org/10.1016/j.brat.2007.07.002

Fairholme, C. P., Nosen, E. L., Nillni, Y. I., Schumacher, J. A., Tull, M. T., & Coffey, S. F. (2013). Sleep disturbance and emotion dysregulation as transdiagnostic processes in a comorbid sample. *Behaviour Research and Therapy*, *51*, 540–546. https://doi.org/10.1016/j.brat.2013.05.014

Garde, A. H., Albertsen, K., Persson, R., Hansen, A. M., & Rugulies, R. (2012). Bi-directional associations between psychological arousal, cortisol, and sleep. *Behavioral Sleep Medicine*, *10*, 28–40. https://doi.org/10.1080/15402002.2012.636272

Guastella, A. J., & Moulds, M. L. (2007). The impact of rumination on sleep quality following a stressful life event. *Personality and Individual Differences*, *42*, 115–162. https://doi.org/10.1016/j.paid.2006.04.028

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, *40*, 869–893. https://doi.org/10.1016/S0005-7967(01)00061-4

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion*, *24*, 281–298. https://doi.org/10.1080/02699930903407948

Knowles, R., Tai, S., Christensen, I., & Bentall, R. (2005). Coping with depression and vulnerability to mania: A factor analytic study of the Nolen-Hoeksema (1991) Response Styles Questionnaire. *British Journal of Clinical Psychology*, *44*, 99–112. https://doi.org/10.1348/014466504X20062

Korte, K. J., Allan, N. P., & Schmidt, N. B. (2016). Factor mixture modeling of the Penn State Worry Questionnaire: evidence for distinct classes of worry. *Journal of Anxiety Disorders*, *37*, 40–47. https://doi.org/10.1016/j.janxdis.2015.11.001

Lee, J. K., Orsillo, S. M., Roemer, L., & Allen, L. B. (2010). Distress and avoidance in generalized anxiety disorder: Exploring the relationships with intolerance of uncertainty and worry. *Cognitive Behaviour Therapy*, *39*, 126–136. https://doi.org/10.1080/16506070902966918

Lin, S., Liao, W., Chen, M., & Fan, J. (2014). The impact of shift work on nurses' job stress, sleep quality and self-perceived health status. *Journal of Nursing Management*, *22*, 604–612. https://doi.org/10.1111/jonm.12020

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*, 487–495. https://doi.org/10.1016/0005-7967(90)90135-6

Morin, C. M., Rodrigue, S., & Ivers, H. (2003). Role of stress, arousal, and coping skills in primary insomnia. *Psychosomatic Medicine*, *65*, 259–267. https://doi.org/10.1097/01.PSY.0000030391.09558.A3

Muller, D., Judd, C. M., & Yzerbyt, V. Y. (2005). When moderation is mediated and mediation is moderated. *Journal of Personality and Social Psychology*, *89*, 852–863. https://doi.org/10.1037/0022-3514.89.6.852

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: The development of the Pre-Sleep Arousal Scale. *Behaviour Research and Therapy*, *23*, 263–271. https://doi.org/10.1016/0005-7967(85)90004-X

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressed mood. *Journal of Abnormal Psychology*, *100*, 569–582. https://doi.org/10.1037/0021-843X.100.4.569

Oh, W. O., Im, Y., & Suk, M. H. (2016). The mediating effect of sleep satisfaction on therelationship between stress and perceived health of adolescents suffering atopic disease: Secondary analysis of data from the 2013 9th Korea Youth Risk Behavior Web-Based Survey. *International Journal of Nursing Studies*, *63*, 132–138. https://doi.org/10.1016/j.ijnurstu.2016.08.012

Palagini, L., Moretto, U., Dell'Osso, L., & Carney, C. (2017). Sleep-related cognitive processes, arousal, and emotion dysregulation in insomnia disorder: The role of insomnia-specific rumination. *Sleep Medicine*, *30*, 97–104. https://doi.org/10.1016/j.sleep.2016.11.004

Pilcher, J. J., Ginter, D. R., & Sadowsky, B. (1997). Sleep quality versus sleep quantity: Relationships between sleep and measures of health, well-being and sleepiness in college students. *Journal of Psychosomatic Research*, *42*, 583–596. https://doi.org/10.1016/S00223999(97)00004-4

Ruscio, A. M., Gentes, E. L., Jones, J. D., Hallion, L. S., Coleman, E. S., & Swendsen, J. (2015). Rumination predicts heightened responding to stressful life events in major depressive disorder and generalized anxiety disorder. *Journal of Abnormal Psychology*, *124*, 17 26. https://doi.org/10.1037/abn0000025

Samtani, S., & Moulds, M. L. (2017). Assessing maladaptive repetitive thought in clinical disorders: A critical review of existing measures. *Clinical Psychology Review*, *53*, 14–28. https://doi.org/10.1016/j.cpr.2017.01.007

Segerstrom, S. C., Tsao, J. C., Alden, L. E., & Craske, M. G. (2000). Worry and rumination: Repetitive thought as a concomitant and predictor of negative mood. *Cognitive Therapy and Research*, *24*, 671–688. https://doi.org/10.1023/A:1005587311498

Smith, J. M., & Alloy, L. B. (2009). A roadmap to rumination: A review of the definition, assessment, and conceptualization of this multifaceted construct. *Clinical Psychology Review*, *29*, 116–128. https://doi.org/10.1016/j.cpr.2008.10.003

Starcevic, V., & Berle, D. (2006). Cognitive specificity of anxiety disorders: A review of selected key constructs. *Depression and Anxiety*, *23*, 51–61. https://doi.org/10.1002/da.20145

Tang, N. K., & Harvey, A. G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep: Journal of Sleep and Sleep Disorders Research*, *27*, 69–78. https://doi.org/10.1111/j.1365-2869.2004.00424.x

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination relationship with negative mood and sleep quality. *Personality and Individual Differences*, *34*, 1293–1301. https://doi.org/10.1016/S0191-8869(02)001204

Verkuil, B., & Brosschot, J. F. (2012). The online version of the Dutch Penn State Worry Questionnaire: Factor structure, predictive validity, and reliability. *Journal of Anxiety Disorders*, *26*, 844–848. https://doi.org/10.1016/j.janxdis.2012.08.002

Watkins, E., Moulds, M., & Mackintosh, B. (2005). Comparisons between rumination and worry in a non-clinical population. *Behaviour Research and Therapy*, *43*, 1577–1585. https://doi.org/10.1016/j.brat.2004.11.008

Weinberg, M. K., Noble, J. M., & Hammond, T. G. (2016). Sleep well feel well: An investigation into the protective value of sleep quality on subjective well-being. *Australian Journal of Psychology*, *68*, 91–97. https://doi.org/10.1111/ajpy.12098

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphy measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy*, *38*, 679–693. https://doi.org/10.1016/S0005-7967(99)00136-9

Winzeler, K., Voellmin, A., Schafer, V., Meyer, A. H., Cajochen, C., Wilhelm, F. H., & Bader, K. (2014). Daily stress, presleep arousal, and sleep in healthy young women: A daily life computerized sleep diary and actigraphy study. *Sleep Medicine*, *15*, 359–366. https://doi.org/10.1016/j.sleep.2013.09.027

Yang, M., Kim, B., Lee, E., Lee, D., Yu, B., Jeon, H. J., & Kim, J. (2014). Diagnostic utility of worry and rumination: A comparison between generalized anxiety disorder and major depressive disorder. *Psychiatry and Clinical Neurosciences*, *68*, 712–720. https://doi.org/10.1111/pcn.12193

Yeh, Z., Wung, S., & Lin, C. (2015). Pre-sleep arousal as a mediator of relationships among worry, rumination, and sleep quality. *International Cognitive Therapy*, *8*, 21–34. https://doi.org/10.1521/ijct.2015.8.1.21

Zoccola, P. M., Dickerson, S. S., & Lam, S. (2009). Rumination predicts longer sleep onset latency after an acute psychosocial stressor. *Psychosomatic Medicine*, *71*, 771–775. https://doi.org/10.1097/PSY.0b013e3181ae58e8

Received: October 10, 2017
Accepted: September 12, 2018
Available online: 18 September 2018