

Respiratory Sinus Arrhythmia Moderates the Association Between Stress and Insomnia

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ABSTRACT

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The Spielman 3P model of insomnia proposes that precipitating factors, such as stress, may contribute to the development of insomnia in good sleepers. Previous research has found that respiratory sinus arrhythmia (RSA) may also interact with stress to exacerbate sleep disturbances, resulting in lower subjective sleep quality in good sleepers. The goal of this study is to examine the association between stress, RSA, and sleep quality among individuals with an insomnia disorder. It is hypothesized that RSA will moderate the association between stress exposure and subjective and objective measures of sleep quality among individuals with primary insomnia. Sixty-two individuals with chronic insomnia participated in this cross-sectional study. They completed the Insomnia Severity Index (ISI) and the Pittsburg Sleep Quality Index (PSQI) to assess subjective sleep quality, as well as a daily stress diary for fourteen consecutive days. Objective sleep efficiency was calculated from polysomnography recording during an overnight study at the laboratory. RSA was collected during a five-minute resting baseline task. Results indicated that RSA moderated the association between daily stress and PSQI and ISI scores, with individuals with lower RSA exhibiting a stronger association between daily stress and worst subjective sleep, compared to those with high RSA. RSA did not moderate the association between average daily stress and sleep efficiency in the present sample. These results suggest that among the subset of patients with low RSA, stress exposure may perpetuate subjective sleep complaints.

Keywords: insomnia, stress, sleep quality, insomnia severity, sleep efficiency, respiratory sinus arrhythmia, moderation, cross-sectional

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Insomnia is characterized by the inability to fall asleep and/or maintain sleep more than three times per week for at least one month (American Psychiatric Association, 2013). These symptoms are associated with daytime impairments, which include loss of productivity, impaired cognitive function, and low mood (American Psychiatric Association, 2013). Insomnia is one of the most prevalent sleep disorders, affecting approximately 10% of the population worldwide and typically manifesting more often in women than men (Chaput et al., 2018).

Spielman, Caruso, and Glovinsky (1987) proposed the 3P model of insomnia to identify different factors involved in the development and maintenance of an insomnia disorder. First, individuals who are predisposed to the disorder may present with specific biological and psychological characteristics, such as having a family history of insomnia, that increase the likelihood of sleep disruptions. Next, precipitating factors are acute life events or changes in a daily schedule that trigger the onset of sleep disturbances (e.g., a chaotic divorce or serious argument with a friend). These events are perceived by the individual as stressful and may contribute to difficulty initiating or maintaining sleep. Finally, perpetuating factors, such as maladaptive sleep habits, (e.g., staying in bed for too long) may prolong sleep disturbances after the stressor has ended.

Spielman and colleagues (1987) proposed that stressful life events can play a key role in the onset of an insomnia episode. According to Lazarus and Folkman (1984), stress is defined as an imbalance between the perceived demands of environmental stimuli and the emotional and cognitive resources available to confront these demands (Lazarus & Folkman, 1984). The association between stress and insomnia has been observed in several studies. In a sample of healthy adults, Fortunato and Harsh (2006) found that those who experienced more interpersonal conflict, work demands, and job ambiguity were more likely to report poorer sleep quality.

Similarly, Deguchi and colleagues (2017) found that occupational stressors, such as role conflict and having an anxious temperament, increased the risk of good sleepers developing symptoms of insomnia in adults without a sleep disorder.

In another study using good sleepers, Åkerstedt, Kecklund, and Axelsson (2007) examined the effect of varying levels of stress on sleep. The participants reported on their stress every second hour during the day and filled out a diary about the level of stress experienced at bedtime for six weeks. Low stress was defined as experiencing little or no stress at all, whereas high stress was defined as experiencing some to very much stress. The researchers found that reporting high stress or worry during the day was related to lower sleep efficiency (i.e., proportion of time in bed sleeping), high wake after sleep onset, and longer time spent in slow wave sleep. These results suggest that significant stress during the day or over time may decrease time spent asleep, potentially leading to subsequent sleep disturbances.

Åkerstedt et al. (2012) found similar results. Participants were asked to keep a sleep/wake diary for forty-two consecutive days. Before bed, they were asked to report any situations during the day that might impact their sleep and reported the quality of their sleep upon awakening. The researchers found that experiencing stress during the day negatively influenced daily sleep quality day-to-day, indicating that increased stress or worrying before bed were indicative of poor subjective sleep in good sleepers. Another longitudinal study by Gosling and colleagues (2014) found that occupational stress, such as lack of job marketability, increased the risk of experiencing sleep disturbances in good sleeping adults. Further, Pillai and colleagues (2014) found that exposure to numerous stressors within the past year was a vulnerability factor for developing an insomnia disorder.

Despite evidence that an abundance of stressors may increase an individual's risk for developing insomnia, not everyone who is exposed to stress develops insomnia. Previous research has found that the risk for developing insomnia is greater for those who exhibit higher levels of sleep reactivity. Sleep reactivity refers to the tendency to experience sleep disruption in response to stress. In a longitudinal study, Drake, Pillai, and Roth (2014) asked participants to report their level of sleep reactivity, the number of stressful life events they had experienced in the past year, and insomnia symptoms. The results indicated that more stressful life events were associated with an increased risk for insomnia over time, and that participants with high sleep reactivity at baseline may be at an increased risk of developing insomnia (Drake, Pillai, & Roth, 2014; Pillai et al., 2014).

Recent conceptual models suggest that high-frequency heart rate variability, also referred to as respiratory sinus arrhythmia (RSA), is a biomarker of sleep reactivity. Heart rate variability (HRV) is defined as the variability in the interbeat intervals of the heart (i.e., the length of time between two successive R peaks; Shaffer & Ginsberg, 2017). The variability in time intervals between heartbeats is due to the antagonistic innervations of the SA node by the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS), two subsystems within the autonomic nervous system (ANS), which slow and quicken the heartbeat, respectively (Appelhans & Luecken, 2006; Berntson et al., 1997). A network of cortical and subcortical structures are involved in the regulation of HRV. Projections from the prefrontal cortex are sent to the amygdala, and then to the medulla, which controls autonomic function that regulates breathing and heart rate. The medulla sends parasympathetic output via the vagus nerve to the sinoatrial (SA) node of the heart, the heart pacemaker. The tonic inhibitory parasympathetic output to the SA node is gated by respiration. Wherein expiration slows heart rate via the tonic

PNS activation and inspiration quickens heart rate via inhibition of parasympathetic output to the SA node (Berntson, Cacioppo, & Quigley, 1993). The magnitude of change in the heart rate associated with the respiratory cycle reflects the magnitude of vagal inhibition and the related level of parasympathetic activity. This phenomenon of cardiac vagal control is known as RSA, a biomarker of vagally-mediated parasympathetic activity.

Current literature has suggested that low RSA is observed more frequently in those with insomnia (Dodds, Miller, Kyle, Marshall, & Gordon, 2017; Shaffer & Ginsberg, 2017). Previous studies have found that while stress is a precipitating factor of the development and maintenance of insomnia, individual differences in RSA may moderate this relationship. In an experimental study of good sleepers, Bonnet and Arand (2003) examined the relationship between situational insomnia, which refers to experiencing poorer sleep due to potentially stressful circumstances as well as less sleep efficiency and lower heart rate variability. Participants spent five to seven consecutive polysomnography days and nights at the lab and were subjected to psychomotor performance testing. The researchers found that those with less RSA had decreased sleep efficiency when the participants were exposed to stressful circumstances in the laboratory before bed. Thus, HRV may be a biomarker of sleep reactivity.

In a study with good sleeping students, Gouin et al. (2015) examined whether RSA moderated the association between stress and insomnia. They hypothesized that experiencing a naturally occurring stressor during periods of lower and higher stress would lead to subsequent sleep disturbances for participants who exhibited low RSA. RSA was assessed during a period of resting wakefulness, where the participants were asked to close their eyes and relax for five minutes, as well as during a worry induction task, where the participant was requested to think about a very stressful situation for five minutes. The researchers found that those with low RSA

during worry predicted frequent sleep disturbances during subsequent stressful periods. These results support the idea that increased stress negatively affects the sleep of individuals with low RSA more compared to those with high RSA.

In an extension and replication of the previous study by Gouin et al. (2015), MacNeil et al. (2017) also wanted to determine if participants with low RSA had an increased risk for sleep disturbances during periods of high stress. Using a longitudinal design to depict low and high stress periods, the researchers posited that participants who exhibited high RSA reactivity (i.e., phasic changes in HRV due to being presented with challenges) during a worry task would have the greatest risk of experiencing poor sleep during high academic stress (Berntson et al., 1997). During the low stress academic period (i.e., the first month of school), participants' resting HRV, measured when seated, and RSA reactivity, measured during a worry task, were collected (MacNeil et al., 2017). Participants with high RSA reactivity during worry were more likely to report experiencing sleep disturbances during periods of high stress. Collectively, these results suggest that RSA may serve as a biomarker of sleep reactivity that moderates the strength of the relationship between stress and insomnia.

As Spielman, Caruso, and Glovinsky's (1987) model of insomnia has postulated, stress appears to influence the onset and course of insomnia. Stressors have been found to negatively affect sleep (Drake, Pillai, & Roth, 2014; Pillai, Roth, Mullins, & Drake, 2014). Prior studies suggest that RSA may act as a moderating variable between stress and insomnia. Though previous research has found that low RSA at rest and during worry induction may influence the relationship between stress and insomnia in participants without a sleep disorder, no studies have analyzed this relationship in the context of participants with a chronic insomnia disorder (Gouin

et al., 2015; MacNeil et al., 2017). Furthermore, previous research has focused on how HRV may moderate the impact of stress on subjective but not objective sleep measures.

The aim of the present study is to determine whether HRV moderates the relationship between stress and chronic insomnia using both objective and subjective sleep quality measures among individuals with an insomnia disorder. First, it is hypothesized that low RSA at baseline will moderate the association between high average daily stress and increased severity of insomnia among individuals with an insomnia disorder. Furthermore, it is anticipated that low RSA will interact with high stress to decrease subjective sleep quality. Lastly, low RSA is expected to moderate the relationship between high stress and low objectively-measured sleep efficiency.

Method

Participants

Sixty-two individuals with an insomnia disorder, as assessed using the Structured Clinical Interview for DSM-5, participated in the study. Exclusion criteria for this study included a chronic, unstable medical condition (e.g., diabetes, hypertension) or a severe psychiatric disorder (e.g., schizophrenia, bipolar disorder), the presence of another sleep disorder (e.g., sleep apnea, restless leg syndrome), drinking excessive amounts of alcohol during an average week, and using psychotropic medication or sleep medication more than once per week. Participants were also excluded if they worked night shifts within the past year or travelled through at least one time zone. All included participants were over 18 years old and received a score of above twenty-six in the MoCA cognitive screening test (Nasreddine et al., 2005). Moreover, all participants were

excluded if they had an apnea-hypopnea index of more than five. Participants were recruited through advertisements (i.e., posters, website) and clinical referrals.

During the recruitment process, thirteen participants were excluded at intake, whereas twelve participants were excluded after their first PSG night due to evidence of another sleep disorder. One participant was excluded from the statistical analyses due to unreadable electrocardiogram (ECG) data. Additionally, one participant was excluded due to an incomplete stress diary, for a final sample of sixty-two. Participants ranged from 21 to 82 years old, with an average body mass index (BMI) of 24.47 ($SD = 5.14$). Most of the participants were female (77.4%), middle-aged (51.28 ± 15.83), and Caucasian (73.8%).

Procedure

Participants were asked to spend two polysomnography (PSG) nights at the sleep lab. However, only data from their second PSG night was considered in this present study to account for the first night effect, which refers to the concern that participants may experience poorer sleep than usual during their first night at the lab due to an unfamiliar sleep environment (Hirscher et al., 2015). All participants gave their informed consent to participate in this study and this study was approved by Concordia University's Institutional Review Board. They received a compensated amount of \$50 CAD per every PSG night they attended.

Participants were invited to the sleep lab for an initial screening night after being administered the Structured Clinical Interview for DSM-IV (SCID) for eligibility. They were invited back for an additional pre-treatment PSG night, provided they did not have any sleep disorders that were concurrent with their insomnia disorder.

Most participants arrived for the evening between six-thirty and eight o'clock, changed into their night clothes, and sat still as the researchers applied head, chin, and eye electrodes using the Ten-Twenty System. ECG electrodes were placed last and in Lead II position. Electrode application was completed in one to two hours, and the participants retired to their bedrooms after all electroencephalogram (EEG) signals were verified. Lab volunteers monitored the signals during the night to ensure that they were stable. Additionally, participants reported their sleep quality and the severity of their insomnia symptoms either during the initial or second PSG night at the lab via the Insomnia Severity Index (ISI; (Morin et al., 2011) and Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

Morning tasks were conducted when the participants awakened after their second PSG. The participants were woken up no later than 8 AM the following morning to account for circadian patterns. They were offered standard breakfast and non-caffeinated tea, and they were asked to refrain from engaging in any excessive exercise. First, participants were instructed to sit upright and breathe as normally as possible with their eyes open for five consecutive minutes. This period was used as their resting baseline. Next, participants engaged in other tasks unrelated to this study. After completing the morning tasks, the researchers removed and cleaned all electrodes. Before the participants left the lab, they were given stress diaries to complete for 14 consecutive days following their lab visit.

Measures

Sleep Disturbances

The ISI (Morin et al., 2011) is a subjective measure used to assess the severity of insomnia and its associated daytime impairments for the past two weeks (e.g., “how

satisfied/dissatisfied are you with your current sleep pattern?”). This measure contains seven items which are rated on a Likert scale ranging from zero to four. A score between 15 and 28 is indicative of moderate to severe insomnia, whereas a score between zero and fourteen suggests sub-threshold insomnia or no clinically significant insomnia. The ISI exhibited good internal consistency for this sample, with a Cronbach’s alpha of 0.73.

The PSQI (Buysse et al., 1989) assesses subjectively reported sleep quality and sleep patterns in the past month (e.g., “during the past month, how would you rate your sleep quality overall?”). It is a measure with nineteen items which compose seven components, including: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction over the last month. On a global scale ranging from 0 to 21, scores of seven or over suggest the presence of sleep disturbances. Higher scores are indicative of worse sleep quality. In this sample, the Cronbach’s alpha was 0.72, demonstrating good internal consistency.

Daily Stress Diary

A stress diary was administered for fourteen consecutive days, which included items assessing having to work hard and fast during the day, experiencing long, difficult work days, and interpersonal conflict. Reliability estimates for the first day of the diary indicated good internal consistency (Cronbach’s alpha = 0.74).

Polysomnography

The PSG night required setup for the recording of EEG, electromyography (EMG), electrooculogram (EOG), and ECG data. These data were collected using Domino version 2.8.0 from SOMNOmedics. Furthermore, the data were recorded at a sampling rate of 512 Hz for

forty-two participants and a sampling rate of 256 Hz for the remaining twenty-one participants (EEG, EOG bandpass filter 0.3 to 35 Hz; EMG bandpass filter 10 to 100 Hz) due to a technical error during data collection. Sleep from the pre-treatment PSG was scored according to the American Academy of Sleep Medicine (AASM) scoring manual (Berry et al., 2015). Wonambi was used to score sleep data, using the mastoids as reference points and the middle of the forehead as ground (O'Byrne et al., 2018). Sleep efficiency, which is calculated by dividing the total sleep time (TST) by the total time spent in bed during the night, was analyzed from the second PSG night.

Heart Rate Variability

During the PSG night, pre-gelled ECG electrodes were applied to the chest and rib areas in a Lead II position. The data were recorded at a sampling rate of 512 Hz. All ECG data was recorded using Domino 2.8.0. Artefact detection and correction was conducted on Mindware Heart Rate Variability (HRV) Analysis Software. Though the automatic R-peak detection feature was used, the data were manually checked for errors and discrepancies. The data were cleaned in 30-second epochs to minimize the possibility of error. The ECG data of one participant could not be accurately cleaned due to increased presence of artefacts during the resting baseline. The first five minutes before sleep was cleaned for this participant, as a proxy for their resting RSA.

RSA was derived from the ECG data collected at the resting task. Participants remained seated in an upright position during measurement to meet stationary requirements for HRV (Berntson et al., 1997). RSA (bandpass: 0.12 – 0.42) was calculated across 60-second epochs. An average RSA value was calculated per participant. There was average inter-rater agreement regarding how the data were cleaned ($ICC = 0.79$).

Statistical analysis

Pearson correlation coefficients were generated to examine associations amongst the variables. Three separate moderation models were generated to determine the interaction effect of resting RSA with daily stress on subjective sleep quality (i.e., ISI, PSQI) and objective sleep quality (i.e., sleep efficiency). Hierarchical multiple regression was used to perform the moderation analyses. Age and sex were included as covariates. All analyses and descriptive statistics were calculated using IBM SPSS Statistics Version 25.0. The moderation analyses were conducted using the Process macro v3.4 (Hayes, 2013).

Results

Descriptive Statistics

Demographic information is described in Table 1. Missing values were excluded from analyses using pairwise deletion for sleep efficiency ($n = 1$). These values were found to be missing completely at random ($\chi^2(10, 64) = 12.82, p = .234$).

Table 1

Demographic information for scale variables

Variable	Mean	Standard Dev.	Kurtosis	Skewness
Age	50.38	16.09	-0.96	-0.13
Stress	1.81	0.54	-0.29	0.70
Objective SE	77.9	11.32	0.06	-0.81
ISI	17.28	3.94	-0.25	0.41
PSQI	10.16	2.87	-0.53	-0.06
RSA	5.45	1.21	0.51	-0.10

Bivariate Correlations

Bivariate Pearson's correlations between all variables were generated (see Table 2). Baseline RSA was found to be positively related to stress ($r = .28, p = .05$) and negatively related

to age ($r = -.46, p = .001$). Moreover, a negative relationship was found between sleep efficiency and age ($r = -.44, p = .001$), suggesting that sleep efficiency decreases with age. Lastly, the ISI and PSQI were found to be positively related ($r = .54, p = .001$), indicating that the two measures are interrelated in the present sample.

Table 2

Bivariate correlation matrix for all variables

Variables	1	2	3	4	5	6
1. Age	1.00					
2. Stress	-.18	1.00				
3. Obj SE	-.44**	.06	1.00			
4. ISI	.09	.12	-.16	1.00		
5. PSQI	.09	.13	-.09	.54**	1.00	
6. RSA	-.46**	.28*	.03	.07	.18	1.00

Note. * $p < .05$, ** $p < .001$

Hierarchical Multiple Regression Analyses

Three separate regression-based moderation models were generated using the Process version 3.4 macro for SPSS (Hayes, 2018). When ISI was entered as the outcome variable, the model was found to be marginally statistically significant ($F(5, 56) = 2.06, p = .08, R^2 = .16$). An interaction effect between stress and baseline RSA on the ISI was significant ($F(1, 56) = 7.65, p = .01, b = -2.00, p = .01, 95\% \text{ CI } [-3.45, -0.55]$). The interaction term explained 12% of the variance in insomnia severity ($R^2_{\text{chg}} = .12$). This finding suggests that baseline RSA moderates the relationship between stress and insomnia severity for those with primary insomnia. Simple slope analysis indicates that at low RSA at baseline and increased daily stress predicted greater insomnia severity (see figure 1; $b = 4.09, p = .01, 95\% \text{ CI } [1.06, 7.12]$). The simple slope values for moderate RSA and high RSA at baseline were not statistically significant (see table 3).

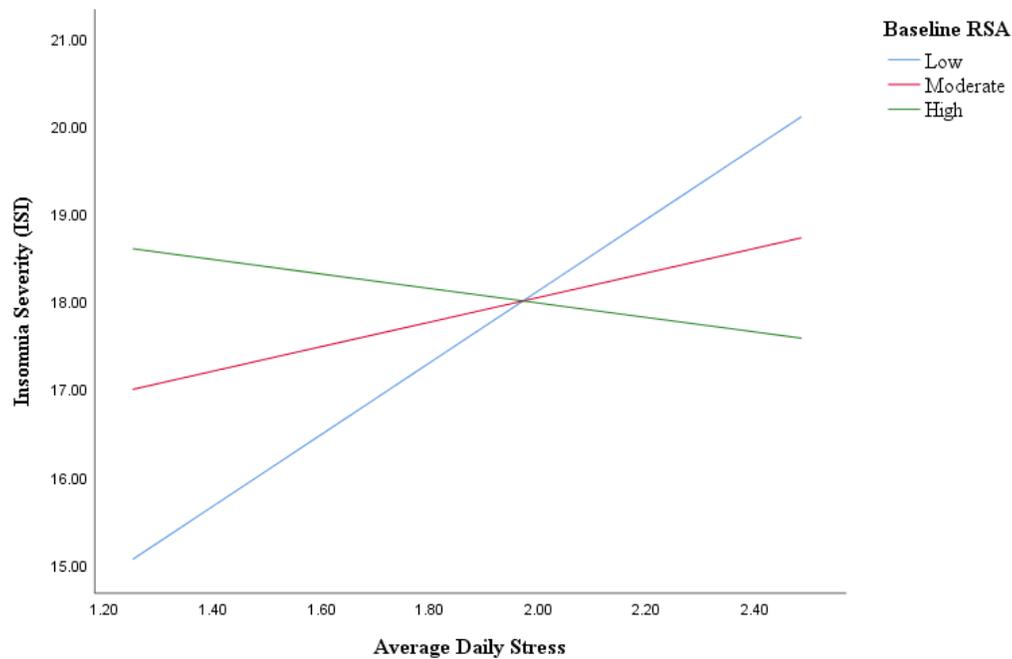
Table 3*Moderation results for the three outcome variables*

Outcome	Effect	<i>b</i>	SE	<i>t</i>	<i>p</i>	LLCI	ULCI
ISI	Stress	12.48	4.32	-1.11	.01	3.83	21.14
	RSA	3.95	1.36	2.89	.01	1.23	6.67
	Interaction	-2.00	.72	2.90	.01	-3.45	-.55
PSQI	Stress	8.60	2.99	2.88	.01	2.61	14.59
	RSA	2.95	.94	3.14	.002	1.06	4.83
	Interaction	-1.38	.50	-2.76	.01	-2.38	-.38
SE	Stress	-6.49	11.92	-.55	.58	-30.40	17.41
	RSA	-4.28	3.82	-1.12	.27	-11.94	3.38
	Interaction	1.27	2.01	.63	.53	-2.77	5.31

Note. SE = sleep efficiency as measured by polysomnography.

Figure 1

Baseline RSA x Average Daily Stress on Insomnia Severity (ISI)



Note. Line graph depicting conditional effect of low RSA moderating the relationship between stress and insomnia severity. Groupings for low, moderate, and high RSA generated based on 16th, 50th, and 84th percentiles, respectively

The PSQI was entered as the next outcome variable in the moderation analysis. The overall model was found to be significant ($F(5, 56) = 3.15, p = .01, R^2 = .22$). There was an

interaction effect between average daily stress and baseline RSA on the PSQI, ($F(1, 56) = 7.59, p = .01, b = -1.38, p = .01, 95\% \text{ CI } [-2.38, -.38]$), indicating that RSA moderates the relationship between stress and sleep quality. In this model, the interaction term explained 11% of the variance in the PSQI ($R^2_{\text{chg}} = .11$). A conditional effect of stress predicting sleep quality at differing levels of baseline RSA was found for low RSA, suggesting that low RSA at baseline with increased daily stress predicted poor sleep quality in those with primary insomnia (see figure 2; $b = 2.82, p = .01, 95\% \text{ CI } [.72, 4.91]$). The simple slope values for moderate and high RSA at baseline were not statistically significant (see table 4).

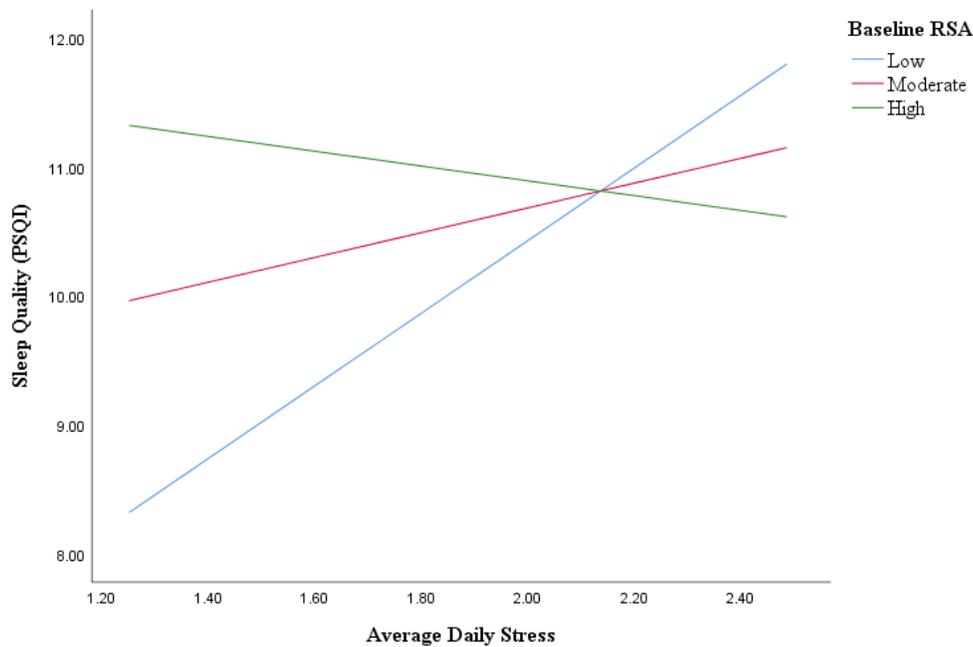
Table 4

Simple slope analysis for RSAxStress on ISI

Moderator	<i>b</i>	SE	<i>p</i>	LLCI	ULCI
Low	4.09	1.51	.01	1.06	7.12
Moderate	1.40	.97	.1	-.54	3.34
High	-.83	1.12	.1	-3.06	1.41

Figure 2

Baseline RSA x Average Daily Stress on Sleep Quality (PSQI)



Note. Line graph depicting conditional effect of low RSA moderating the relationship between stress and sleep quality. Groupings for low, moderate, and high RSA generated based on 16th, 50th, and 84th percentiles, respectively

Table 5

Simple slope analysis for RSAxStress on PSQI

Moderator	<i>b</i>	SE	<i>p</i>	LLCI	ULCI
Low	2.82	1.05	.01	.72	4.91
Moderate	.96	.67	.1	-.38	2.30
High	-.57	.77	.1	-2.12	.97

Polysomnography-derived sleep efficiency was entered in the moderation model as the final outcome variable ($F(5, 56) = 3.06, p = .02, R^2 = .22$). The interaction effect for RSA and stress on sleep efficiency was found to be statistically non-significant ($F(1, 53) = .40, p = .02, b = 1.27, p = .53, 95\% \text{ CI} [-2.77, 5.31]$), which suggests that RSA does not moderate the relationship between average daily stress and sleep efficiency in the present sample.

Discussion

The goal of this study was to test whether RSA would moderate the relationship between stress and insomnia severity among individuals with primary insomnia. Specifically, it was hypothesized that low RSA would moderate the relationship between high average daily stress and increased subjective insomnia severity, decreased subjective sleep quality, and decreased objective SE. An interaction between high stress and low RSA on subjectively reported insomnia severity was found. Likewise, RSA moderated the relationship between high stress and poor subjective sleep quality. Thus, low RSA and high daily stress were associated with poorer sleep quality and increased severe insomnia severity. However, a statistically significant interaction between average daily stress and low RSA on objective SE was not found, indicating a discrepancy in subjective and objective sleep parameters. These results indicate that low RSA may be a risk factor for stress-related subjective sleep disturbances among those with an insomnia disorder. These findings are consistent with previous research on good sleepers, who may also be more prone to sleep disturbances due to the combined effect of increased stress and low RSA (Gouin et al., 2015; MacNeil et al., 2017).

The Spielman 3P model explains how sleep disturbances may begin as acute, non-clinical, transient symptoms that develop into an insomnia disorder depending on presence of predisposing, precipitating, and perpetuating factors (Spielman, Caruso, & Glovinsky, 1987). Stress may serve as a precipitating factor for the onset of insomnia symptoms, such that experiencing high stress during the day (i.e., increased demands at work) may lead to transient sleep disturbances at night that become more permanent over time, particularly if stress and perpetuating factors, such as poor sleep hygiene practices, are maintained. Various studies have found a link between stress and symptoms of insomnia, suggesting that higher levels of stress

may increase subjectively reported sleep disturbances in healthy good sleepers (Friedman et al., 1995; Drake et al., 2014). One study determined that poor sleepers experienced more stressful life events during the year that their insomnia developed compared with good sleepers (Healey et al., 1981). Another study found that the chronicity of stress may moderate the relationship between stress and the onset of insomnia in good sleepers (Pillai et al., 2014). These previous results support the notion of stress as a precipitating factor to insomnia in healthy participants.

The present study revealed that stress may also be a maintenance factor in a subset of individuals diagnosed with an insomnia disorder. Past studies have yielded that increased stress is related to sleep disturbances (Vgontzas et al., 1998). Morin et al. (2003) found that while participants with insomnia and good sleepers reported the same number of minor stressful events on average, those with insomnia rated the events as more impactful. Those with insomnia also rated more major negative life events as intense than good sleepers, suggesting that those with an insomnia disorder show a stronger association between stress and sleep than good sleepers. Likewise, Hall and colleagues (2007) found that increased perceived stress was associated with low RSA and decreased delta power (i.e., slow wave sleep) during NREM sleep in a cross-sectional study of individuals with primary insomnia. These results imply that experiencing more life stress is related to decreased parasympathetic output during sleep in those with insomnia. Therefore, while the Spielman 3P model has posited that stress assists as a risk factor for the development of insomnia in good sleepers, the current study suggests that stress may also serve to maintain or bolster insomnia in a subset of individuals with diagnosed chronic insomnia.

Similar results were found in a study by Bonnet and Arand (2003). They recruited healthy good sleepers to determine the presence of situational insomnia due to laboratory-administered stressors, which included two nights of advanced bedtime. The participants

experienced one night of baseline rest before one night of phase-advance sleep by 3 hours prior to their usual bedtime. Then, they were invited back to the lab for an additional baseline night, followed by phase-advance sleep of 6 hours prior. The researchers found that among good sleeping participants without a history of poor sleep, those who had lower RSA had reduced sleep efficiency in response to different sleep challenges. These results suggest that sleep reactivity may serve as a precipitating factor for the development of insomnia in good sleepers. Moreover, low RSA may be a predisposition for insomnia, particularly when presented with increased stress.

Hall and colleagues (2004) randomized healthy participants into either a stress group, wherein they told that they would be delivering an oral speech upon awakening, or a control group and measured both subjective and objective sleep quality. Whereas the control group would be asked to read a popular magazine for six minutes in the morning, the stress group were informed that they would be delivering an oral speech upon awakening. The researchers found that participants in the stress condition reported subjectively worse sleep than those in the control group. Moreover, the stress group experienced blunted parasympathetic modulation throughout the course of the night, whereas the control group exhibited a steady increase in parasympathetic modulation during the evening. Consistent with the findings of the present study, these results imply that being exposed to a stressor before bed may lead to poor subsequent sleep, which is reflected in both decreased parasympathetic output and subjective sleep.

In another study with good sleeping university students, Gouin and colleagues (2015) found that lower RSA during a worry induction task predicted decreased sleep quality, but not increased insomnia severity, during both low and high stress academic periods. A replication of this study found similar results, wherein low RSA reactivity during a high stress academic period

predicted increased sleep disturbances (MacNeil et al., 2017). These findings propose that among healthy individuals without an insomnia disorder, those with low RSA reactivity may be at a higher risk for experiencing sleep disturbances in response to stress compared to those with high RSA reactivity. This could suggest that low RSA reactivity is a physiological vulnerability factor for stress-related insomnia symptoms in good sleepers.

The present study extended Gouin and colleagues' (2015) findings with individuals diagnosed with an insomnia disorder, further suggesting that the interaction between high stress and low RSA may contribute to decreased sleep quality in both healthy good sleepers and those with chronic insomnia. Though Gouin et al. (2015) and MacNeil et al. (2017) found that low RSA moderated the relationship between high stress and decreased sleep quality, they did not find significant results related to insomnia severity. This deviation between these previous studies and the results of the present study may have been due to the populations sampled. The current study gathered a sample of individuals diagnosed with an insomnia disorder. Furthermore, these researchers examined RSA during a worry induction task (Gouin et al., 2015; MacNeil et al., 2017), whereas the current data were collected at rest for a measure of basal cardiovascular activity. RSA may vary across resting and stress tasks, such that basal RSA activity may moderate the relationship between stress and insomnia severity and RSA reactivity may not. Therefore, this study may have found that low RSA moderated the relationship between high stress and increased insomnia severity due to having a pool of participants with chronic insomnia, as well as the measurement of RSA solely at rest.

While the hypothesis that RSA would moderate the relationship between stress and objective sleep efficiency was not supported in the present study, research has yielded mixed findings regarding the occurrence of objective poor sleep in those with insomnia (Feige et al.,

2013). Some studies have found that individuals with insomnia experience objectively worse sleep than good sleepers, whereas other researchers have posited that there is little difference between the objective sleep of those with insomnia and good sleepers (Linden et al., 2016; Fernandez-Mendoza et al., 2010). One reason for this divergence in results between good sleeping participants and those with insomnia may be due to sleep state misperception, or mistakenly perceiving sleep as wakefulness (International Classification of Sleep Disorders, 2014; Bianchi et al., 2013). This issue can be detected by examining subjective sleep reports (e.g., ISI, PSQI) and comparing them with objective sleep reports (e.g., PSG, Actiwatch; Tang & Harvey, 2004). In the present study, participants reported moderate insomnia severity via the ISI and poor sleep quality via the PSQI on average, though objective SE from the PSG suggested that most participants had experienced moderate to good sleep during the evening. These findings may indicate a discrepancy between subjective and objective sleep measures.

A potential explanation for this inconsistency between measures may be due to the high number of female participants in the current study. Females have been found to experience higher sleep state misperception than males and are likewise more likely to experience insomnia in their lifetime (American Psychological Association, 2013). Given that many studies display this high female to male ratio in recruitment, sleep state misperception may be present as a confounding variable often in insomnia research (Feige et al., 2013; Tang & Harvey, 2004; Bianchi et al., 2013). Consequently, sleep state misperception may have influenced the subjective SE results within the present sample, suggesting that participants may have mistakenly over-reported their poor sleep quality and increased insomnia severity. This would yield conflicting results between the subjective measures, the ISI and PSQI, and objective SE. However, the interaction between stress and HRV predicting subjective sleep is also found in

good sleepers (MacNeil et al., 2017; Gouin et al., 2015). Therefore, sleep-state misperception cannot fully explain this phenomenon. Instead, the initial influence of low RSA may contribute to the phenomenon of sleep-state misperception in participants with an insomnia disorder. Hyperarousal, or an increased physiological and psychological responsiveness to stimuli, has been found to decrease RSA and subjective sleep quality (Jerath, Beveridge, & Barnes, 2018). This could suggest that the presence of low vagally-mediated parasympathetic activation in either good sleeping participants or those with insomnia negatively affects perceived quality of sleep.

Though novel strengths of this study include the recruitment of participants with an insomnia disorder and the analysis of both subjective and objective measures of sleep quality, there are some limitations to be addressed. First, average daily stress was assessed using a three-question measure administered over fourteen consecutive days after the second PSG visit. The daily stress diaries allowed for the consistent assessment of stress over time and beyond the laboratory. Nevertheless, three questions may not have been sufficient to encapsulate the varying types of stress encountered during an average day (e.g., financial strain, medical issues). Furthermore, while the diaries were also a measure of sleep reactivity, the Ford Insomnia Response to Stress Test (FIRST; Jarrin et al., 2016) could have provided additional information on trait vulnerability to sleep reactivity. Other studies have used the FIRST to measure sleep reactivity and vulnerability to situational insomnia. Drake and colleagues (2004) found that good sleepers who scored higher on the FIRST had lower objective SE and increased stage one sleep latency. Furthermore, they found that sleep reactivity may be a risk factor for the development of an insomnia disorder. In the current study, participants with greater stress reported lower subjective sleep quality and increased insomnia symptoms, but not lower objective SE. However,

as this study is cross-sectional, the results could not be used to interpret the direction of the relationships found in the data.

A second limitation pertains to the measurement of RSA. As mentioned, RSA was only assessed at rest and using different sampling rates. Previous research has measured RSA reactivity during a worry task and at a stationary sampling rate (Gouin et al., 2015; MacNeil et al., 2017). Porges (2007) has suggested that vagal influence on the heart decreases when a stressor emerges. Thus, measuring RSA at baseline and during a stress-induction task with a consistent sampling rate may have provided an opportunity to further explore how parasympathetic function interacts with stress to develop and maintain insomnia symptoms. Additionally, RSA was only measured during the morning. Past studies examining nocturnal RSA have found that parasympathetic activity may be blunted during the evening (Jarrin et al., 2018) compared to good sleepers (Spiegelhalder et al., 2011). These findings suggest that good sleeping participants with low baseline and nocturnal RSA may be at risk for developing an insomnia disorder.

Another limitation was the sole measurement of parasympathetic activity. Though RSA is widely accepted as a biomarker of vagally-mediated parasympathetic activity and is used extensively in stress research (Berntson, Cacioppo, & Quigley, 1993), extracting a measure of sympathetic activity (i.e., PEP, R-R) would have allowed for the assessment of both divisions of the ANS. Investigation of sympathetic function with parasympathetic activity in those with insomnia is important, as the two systems share a semi-symbiotic relationship. Moreover, some researchers have found increased sympathetic reactivity in participants with chronic insomnia (Carter et al., 2018), suggesting that individuals with an insomnia disorder may demonstrate both parasympathetic and sympathetic dysfunction.

A final limitation pertains to the large age range of the participants included in the study. Insomnia is most likely to affect middle-age and older individuals, with complaints regarding the maintenance rather than the initiation of sleep emerging more in these populations (American Psychological Association, 2013). In contrast, younger individuals tend to report more difficulty initiating sleep than maintenance, suggesting that insomnia may develop differently with age. Moreover, one study has suggested that different factors may influence sleep disturbances in older individuals with insomnia, such as reduced physical health (Morgan, 2000). This variation may have been reflected in the subjective measures (e.g., PSQI) used in the present sample, which included participants aged 21 to 82 years old. Moreover, HRV has also been found to decrease with age due to lower vagal cardiovascular activity (de Meersman, 1993); this could have limited the current results further. Therefore, as insomnia may be heterogeneous in accordance with age and older individuals are more likely to have low RSA, older participants may have reported poorer sleep quality and more insomnia symptoms than younger participants.

There are numerous future directions that should be explored. First, conducting a longitudinal study of good sleepers and participants with an insomnia disorder would allow for the contrast of differences in RSA, sleep quality, and average daily stress levels between these two groups. While utilizing a cross-sectional design has significant benefits, such as being low cost to perform and providing a way to exhaustively analyze a specific point in time, there are some disadvantages. Primarily, it does not specifically describe cause and effect relationships. Thus, associations within the data may be difficult to interpret because the design does not expose directionality between variables. Moreover, a cross-sectional design does not allow for the analysis of multiple timepoints. A longitudinal design would help to determine the specific directionality of relationships between variables, as well as allow for the analysis of both groups

of participants at multiple timepoints. Maes et al. (2014) found that those with insomnia displayed low RSA during slow wave sleep compared to healthy good sleepers, suggesting that participants with insomnia may differ in vagal parasympathetic function across sleep stages. Past studies have also found that those with objectively short sleep (i.e., < 6 hours) experienced less parasympathetic activation than the healthy control group (Jarrin et al., 2018; Spiegelhalder et al., 2011). It is important to determine how environmental stress may interact with physiological mechanisms in those who are and are not vulnerable to experiencing sleep disturbances.

A second direction which future research may wish to focus is how decreased parasympathetic activation may relate to the emotion regulation of individuals with insomnia. It has been proposed that vagally-mediated parasympathetic activation may be associated with emotion regulation, or the ability to respond to environmental changes with a range of controlled emotions and flexibility (Beauchaine, 2015; Williams et al., 2015). Those with insomnia and low RSA may exhibit emotion dysregulation, though the directionality of this relationship could not be determined with the present study design. Future studies may wish to explore the presence of low RSA and insomnia severity during an emotion regulation task to determine the specific faucets of emotion dysregulation that may be present during an insomnia disorder.

A final key future direction would be to compare the sleep, RSA, and stress levels of individuals with chronic insomnia before and after treatment for their insomnia. This comparison would be useful to determine changes in vagal-cardiac output, sleep quality, presence of stressors, and insomnia severity across more than one timepoint. A difference of better sleep, lower reported stress, and increased RSA at post-treatment may demonstrate the effectiveness of cognitive-behaviour therapy for insomnia (CBTi), which teaches patients about sleep hygiene and offers long-term solutions to coping with insomnia and decreasing its symptoms (Jarrin et

al., 2016). Jarrin et al. (2016) found that those with insomnia who attended CBTi-based therapy sessions for as short as six weeks experienced more fulfilling sleep. The participants also experienced decreased parasympathetic activation during the night, suggesting that the treatment may have been effective in altering physiological activation in those with insomnia.

Consistent with earlier research, the current study determined that low RSA moderates the relationship between average daily stress levels and subjective sleep quality and insomnia severity. The Spielman 3P model of insomnia identifies stress as a precipitating factor for the development of insomnia in good sleepers (Spielman, Caruso, & Glovinsky, 1987), though the present study suggests that stress may also serve as a maintenance factor for insomnia for a subset of individuals diagnosed with an insomnia disorder. Treatments for insomnia must incorporate techniques that assist in the lowering of stress and the enhancement of diurnal and nocturnal RSA. By identifying the combined effect of increased stress and low RSA as a risk factor implicated in the development of an insomnia disorder, therapeutic interventions can be adapted to include methods effective in reducing stress and increasing RSA before their sleep disturbances become chronic over time. Furthermore, the identification of stress and RSA as maintenance factors for insomnia may assist in the recovery of those who already have an insomnia disorder.

Previous research has found that insomnia has chronicity rates ranging from 45% to 75% at follow up periods spanning one to seven years (American Psychological Association, 2013), making it imperative to treat persistent sleep disturbances to improve quality of life. In order to encapsulate and analyze potential components of insomnia disorder, such as RSA, future studies should further explore ANS activity at rest and during stress longitudinally. Additionally, the inclusion of a healthy control group would extend the present literature on the development of

sleep disorders and explore the potential factors involved in the maintenance of sleep disturbances in those with a diagnosed insomnia disorder.

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