SLEEP REACTIVITY AND PARASYMPATHETIC CONTROL WHEN RETURNING TO SLEEP

By

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Abstract

Insomnia is among the most common health problems and is associated with lower parasympathetic control. Sleep reactivity is associated with development of chronic insomnia. The purpose of this study was to examine whether sleep reactivity is associated with parasympathetic control in response to a sleep-relevant stressor. Parasympathetic control was operationalized using respiratory sinus arrhythmia (RSA). Sleep reactivity was operationalized as a score of 14 or higher on the Ford Insomnia Response to Stress Test (FIRST). Participants were 33 healthy young adults required to adhere to a fixed eight-hour sleep schedule for three nights before an in-laboratory sleep study. Physiological signals were recorded for two five-minute baseline periods of resting wakefulness prior to lights out. Participants were woken after the first five minutes of contiguous N2 sleep in the third NREM period and kept awake for 15 minutes, then allowed to return to sleep. In a multiple linear regression, the interaction between baseline RSA and sleep reactivity predicted RSA when returning to sleep. Individuals with high sleep reactivity had relatively low parasympathetic control when returning to sleep. People with high sleep reactivity may benefit from interventions to increase parasympathetic control during awakenings from sleep.
Background and information

Insomnia

Insomnia is among the most common health problems today and has serious consequences (Harvey, 2001). It can be a situational, recurrent, or persistent problem. Insomnia can also follow an intermittent pattern, presenting sleep difficulties recurrently with stressful events. Acute insomnia is often associated with life events or changes in sleep schedule, but typically subsides after the event subsides, but for some, the sleep disturbance persists after the initial cause (Morin & Benca, 2012). Chronic insomnia affects approximately 30% of the general population (Roth, 2007) and has a high rate of comorbidity with medical and psychiatric disorders (Morin & Benca, 2012; Ohayon & Reynolds III, 2009; Roth et al., 2006; Simon & VonKorff, 1997). Insomnia remains an important public issue because of its negative impact on society in terms of physical and social performance, work performance, and quality of life.

Definition and criteria

The predominant complaint in insomnia remains the same across diagnostic criteria. Diagnostic criteria include: a long-standing difficulty initiating or maintaining sleep, including waking up too early, or non-restorative sleep or poor quality sleep, for at least three months, in addition to daytime distress or impairment (Harvey, 2001; Roth, 2007; Roth et al., 2011; American Psychiatric Association, 2013). These daytime consequences include fatigue, reduced concentration and memory, irritability, and low mood and anxiety (Billiard & Bentley, 2004).

Epidemiology

Prevalence

Epidemiological studies consistently find that roughly one-third of the United States adult population report sleep problems (Ancoli-Israel & Roth, 1999; Grandner & Kripke, 2004;
National Sleep Foundation 2005), specifically with about one-third of adults reporting insomnia symptoms (Ohayon, 2002). As it stands, 10%-15% of the population meets the criteria for insomnia (Breslau et al., 1996; Cirignotta et al., 1975.; Ohayon, 1996, 1997, 2002; Roth et al., 2006) with 10%-15% experiencing associated daytime impairments (Morin et al. 2006). In primary care settings, approximately 10%-20% of individuals complain of significant insomnia symptoms, with overall prevalence estimates of broadly defined insomnia at approximately 23.6% in an epidemiological study (Ohayon, 2002; Roth, 2007; Roth et al., 2006, 2011).

Insomnia is a more prevalent complaint among women than among men, with a gender ratio of about 1.41:1 (Zhang & Wing, 2006) and also has a higher prevalence among older adults (Roth, 2007). Not only are women more likely than men to have insomnia, but those that do have higher levels of comorbid depression, but not comorbid anxiety (Taylor et al., 2005).

**Comorbidities**

Although insomnia can be an independent disorder, it is most frequently observed as a comorbid condition with another medical condition or mental disorder (Roth et al., 2006; Taylor et al., 2007). Upon analyses of available literature, people with insomnia have a higher prevalence of comorbid medical problems than those without insomnia and people with chronic medical problems have higher prevalence of insomnia than people without those same disorders (Kessler et al., 2012; Taylor et al., 2007). However, symptoms of anxiety and depression that do not meet criteria for a specific mental disorder may also be present (American Psychiatric Association, 2013).

Among these comorbid conditions, depression and anxiety are the most common psychiatric disorders (Harvey, 2001; Roth, 2007). A study found that 20% of people with insomnia report clinically significant depression, making people with insomnia almost 10 times
more likely to have depression than people without insomnia. That same study found 19.3% of people with insomnia report clinically significant anxiety, which is 17 times more likely than people without (Taylor et al., 2007; Taylor et al., 2005). In a study of 772 people, 9.6% showed clinically significant levels of depression, but among people in the sample with insomnia, 20% had depression (Taylor et al., 2005). Overall, 40%-50% of individuals with insomnia present with a comorbid mental disorder (Harvey, 2001).

**Consequences**

Chronic insomnia makes other disorders more difficult to treat. Chronic insomnia is often associated with a wide range of adverse effects, including mood disturbances, difficulties with concentration, and memory (Harvey, 2001). While treatment of the comorbid disorder may resolve symptoms of the comorbid disorder, it does not often improve or resolve the insomnia (Roth, 2009). In fact, insomnia complicates the primary disorder treatment with insomnia being one of the most common residual symptoms (Benca, 1996).

People with insomnia also have a higher incidence of cardiovascular and neurologic disease, high blood pressure, and pulmonary, urinary, and gastrointestinal problems, and chronic pain, after controlling for depression and anxiety levels (Kessler et al., 2012; Taylor et al., 2007). Chronic insomnia is a clinically significant risk factor associated with development of Type 2 diabetes, independent of other comorbid conditions frequently associated with insomnia and diabetes (e.g. age, race, obesity, smoking, alcohol consumption, periodic limb movements, depression) (Vgontzas et al., 2009). Specifically, chronic insomnia is associated with a 1.84 times higher risk of diabetes (Vgontzas et al., 2009). In another smaller study, people with chronic insomnia and short sleep duration (< 5 hours per night) as measured by polysomnography
had a risk of hypertension at 3.5 to 5 times higher than subjects who slept greater than 6 hours and had no sleep complaint (Vgontzas et al., 2009).

**Insomnia and treatment of Major Depressive Disorder**

Insomnia is part of the diagnostic criteria for diagnosis of Major Depressive Disorder (MDD), but insomnia might persist and is a commonly reported residual symptom even after effective treatment and remission of depressive symptoms (Carney et al., 2007; Nierenberg et al., 1999). Many patients with MDD who experience full symptomatic remission after antidepressant treatment, for example Cognitive Behavioral Therapy (CBT) or pharmacotherapies still have residual depressive symptoms (Carney et al., 2007; Nierenberg et al., 2010). These residual insomnia symptoms that occur with a MDD episode may cause such patients significant distress and further complicate recovery. Insomnia symptoms have also been found to increase risk for subsequent MDD (Karp et al., 2004; Perlis et al., 1997). Variability in residual symptoms confers greater risk for relapse and subsequent episodes, which insomnia is among the most variable after MDD treatment (Karp et al., 2004; Paykel, 1994). Residual insomnia rates following remission suggest that between 30 and 55% of those who recover from depression have residual insomnia (Carney et al., 2011; Carney et al., 2007; Nierenberg et al., 2010). Preliminary evidence shows that adjuvant treatment of insomnia can cause a greater beneficial effect on insomnia and depression and lead to a better outcome than just treating depression alone (Fava et al., 2006; Manber et al., 2008). Whether insomnia is co-occurring or independent of associated problems is not always easily determined, but is critical to treatment strategies for individual patients (NIH 2005). It is also possible that insomnia serves to worsen and exacerbate symptoms for people with chronic illnesses (Broström et al., 2004; Taylor et al., 2005).
Development of Insomnia

Age and gender are the most clearly and well-defined risk factors for insomnia as reported by the State-of-the-Science Conference held by the National Institutes of Health in June 2005 (Roth, 2007). Individuals with insomnia are also more likely to have a family history of the disorder (Beaulieu-Bonneau et al., 2007), which suggests a genetic component, a common environmental factor, or a learned part. However, sleep-wake regulatory gene abnormalities have not yet been identified in insomnia (Morin & Benca, 2012). The complaint of poor sleep and the presence of mental health problems are strong predictors of chronic insomnia compared to physical health problems. Individuals with a history of subjective poor sleep were at significantly higher risk of developing chronic insomnia (Singareddy et al., 2012).

Sleep reactivity

Sleep reactivity is a significant risk factor for insomnia, in part by exacerbating the effects of stress-induced cognitive intrusions. It is also a precipitant of depression, as mediated by insomnia (Drake et al., 2014) and is associated with polysomnographic changes in response to stressors and subsequent development of insomnia. In 2004, Drake et al. demonstrated vulnerability to sleep disturbance can be reliably assessed and is associated with physiological hyperarousal as measured by heart rate. While chronic insomnia is associated with lower parasympathetic control (Bonnet & Arand, 1998), the relationship between sleep reactivity and parasympathetic control has not been reported. One measure of sleep reactivity is the Ford Insomnia Response to Stress Test (FIRST; Drake et al., 2004). The FIRST has been validated by identifying individuals at risk for disturbed sleep using polysomnography in response to caffeine (Sheehan et al., 1998). Previous longitudinal studies have shown higher FIRST scores were associated with increased risk of developing insomnia. This highlights the importance of
investigating the relationship between changes in stress and sleep-reactivity at the level of the individual.

The National Sleep Foundation 2015 poll found greater stress was associated with less sleep and worse sleep quality (National Sleep Foundation 2015). Approximately 1 in 10 people (12%) reported severe or very severe stress in the previous 7 days and another 31% reported moderate levels of stress. People with severe or very severe stress were getting less sleep than they felt they need with 49 minutes less sleep per night on average. In contrast those with no or only mild stress were only two minutes shy of their desired sleep need per night on average. Those with severe or very severe stress were more than twice as likely to report poorer sleep quality (very poor, poor or fair) compared with those with mild or no stress (83% v. 35%). Also, 67% of those with severe or very severe stress reported difficulty sleeping in the past 7 days compared with only 25% of those with no or mild stress.

Currently, there is very little data directly linking vulnerability to acute sleep disturbance to the subsequent development of chronic insomnia. Although there is a subset of individuals susceptible to acute sleep disturbance, this characteristic may be completely coincidental and reflect individuals with high-exposure to sleep-disruptive stressors (Drake et al., 2008). The existing data suggest a trait of sleep reactivity to stress may exist and this predisposition is hypothesized as genetic (Drake et al., 2008). A twin study found significant genetic influence on three factors: insomnia, daytime sleepiness, and obesity. It showed that more than 50% of the variance in the risk for insomnia can be attributed to genetic factors (Watson et al., 2006). This is consistent with previous twin studies similarly demonstrating genetic factors strongly influence insomnia. Based on this, if sleep reactivity is a significant risk factor for insomnia, there should be significant familial aggregation of sleep reactivity (Drake et al., 2008).
Stress reactive insomnia

Whether accompanying another disorder or only occasional, insomnia can be influenced by other factors like stress. In general, how individuals view stressful life events has been shown as major factors increasing risk for insomnia (Healey et al., 1981). An individual with stress reactive insomnia will typically have normal sleep, but can have that sleep easily disturbed in response to stressors. Stress reactive insomnia differs from chronic insomnia in that it may last only a single night, whereas chronic will persist for months or years. However, stress reactive insomnia can develop into chronic insomnia (Drake et al., 2014).

Autonomic Arousal

People with insomnia show increased activation of the autonomic nervous system, which is evidenced by physiological arousal measures including heart rate variability (HRV), metabolic rate, body temperature, hypothalamic-pituitary-adrenal (HPA) axis activity, norepinephrine secretion, and neuroendocrine measures (Bonnet & Arand, 1998; Bonnet & Arand, 2010; Riemann et al., 2010). HRV provides a measure of arousal in that it is regulated by both sympathetic and parasympathetic nervous system activities (Roth, 2007).

Parasympathetic control

Average heart rate is not a reliable measure of autonomic arousal (Bonnet & Arand, 1998; Drake et al., 2004). A more selective measure to provide this information noninvasively is heart rate variability (HRV) which refers to the beat-to-beat changes in heart rate over time (Kawachi, 1997; Stein & Pu, 2012). HRV results from a dynamic relationship between sympathetic and parasympathetic nervous system influences which make up the autonomic nervous system (ANS). This relationship can be the combined activation, combined inhibition, or single activation/inhibition of the sympathetic and parasympathetic nervous system (Allen et
al., 2007). However, studies suggest the vagus nerve is implicated in HRV within the respiratory frequency band (Allen et al., 2007; Berntson et al., 1991). High-frequency heart rate variability (0.12-0.40 Hz), which is often referred to as respiratory sinus arrhythmia (RSA) is reflective of overall parasympathetic control (Porges, 1995).

Parasympathetic control has not been well studied in relation to sleep reactivity. The purpose of this study was to examine whether sleep reactivity is associated with parasympathetic control while resting and in response to a sleep-relevant stressor (mid-night awakening).

Based on all previously discussed, hypotheses were as follows:

**Study Hypotheses**

Hypothesis 1: Stress-reactive insomnia will be negatively associated RSA at rest.

Hypothesis 2: Stress-reactive insomnia will be negatively associated with RSA during an awakening from sleep.

Hypothesis 3: Stress-reactive insomnia will moderate the relationship between RSA at rest and during an awakening from sleep, such that individuals with greater stress-reactive insomnia will show decreases in RSA of greater magnitude than individuals with less stress-reactive insomnia.

**Materials and methods**

**Participants and Demographics**

The sample size was thirty three healthy young adults (63.6% female, mean age=20.06 ± years, SD=2.26) meeting the below criteria.

**Recruitment and Inclusion/Exclusion Criteria**

Research participants were recruited through the use of advertisements and fliers posted around the University of Arizona campus. Inclusion criteria included English as a first language, average habitual sleep onset latency less than 20 minutes, average habitual total sleep time
between 5 to 9 hours, and a weeknight bedtime between 9pm and 1am. Exclusion criteria included significant health problems, history of a head injury (i.e., loss of consciousness greater than five minutes), sleep disorders (e.g., obstructive sleep apnea, narcolepsy), use of medication with effects on sleep or memory (e.g., diphenhydramine), daily use of nicotine or illegal drugs (e.g. marijuana, cocaine), history of psychiatric disorders (e.g. bipolar disorder, psychotic disorders) and current DSM-IV Axis I disorders. Participants were asked to abstain from naps, alcohol use, and restrict habitual caffeine use to less than two cups or glasses per day.

**Measures**

**Stress reactive insomnia**

Participants answered the Ford Insomnia Response to Stress Test (FIRST) during the baseline and screening appointment. The FIRST is a self-assessment of vulnerability to sleep reactivity to stress with good internal consistency. It was essential to monitoring stress-proneness in certain situations affecting sleep; specifically, the self-report measures vulnerability to stress-induced insomnia (Drake et al., 2004; Gouin et al., 2015). It is a 9-tem self-report to measure traits of sleep reactivity in nine different situations, asking how likely a participant is to have difficulty getting a good night’s sleep before those events. For example, one question asks about sleep before an important meeting the next day, and another asks after watching a frightening movie or TV show. This is to gauge likelihood of sleep disturbance even if the individual has not experienced any situation of that sort recently (Drake et al. 2014). Sleep reactivity was operationalized using the FIRST with a cutoff of 14 or higher identifying high sleep reactivity.

**Parasympathetic control**
EKG methodology was used to provide information on autonomic functioning through respiratory sinus arrhythmia (RSA) to analyze changes in heart rate before sleep at rest, at and during awakening, while staying awake, and at return to sleep to observe how this induced-stress reflects on parasympathetic measures. RSA is the log of the variance in the interbeat interval (IBI) series in the frequency of respiration (0.12 – 0.40 Hz). EKG data was collected using a modified Lead II placement with 0.3 Hz highpass and 70 Hz lowpass filters, then sampled at 400 Hz. EKG data was extracted and converted to interbeat interval. A time series at 10 Hz was then fit to the interbeat interval series, and filtered in the respiratory frequency to pass 0.12 to 0.40 Hz. The natural log of the variance of this filtered time series was thus taken as the index of RSA (cf. Allen et al., 2007).

Procedure

Screening and baseline

Participants were screened over the phone to determine preliminary eligibility based on the above inclusion and exclusion criteria. During this first visit, participants provided informed consent and began initial testing to determine further eligibility. They were screened for presence of DSM-IV disorders using the MINI (Mini International Neuropsychiatric Interview) (Sheehan et al., 1998). Exclusion criteria included DSM-IV Axis I classified disorders as assessed with the MINI. They were also screened for current or past insomnia disorder as assessed with the Duke Structured Sleep Interview and for risk of Obstructive Sleep Apnea through the STOP-BANG and completed other measures including the FIRST. If participants were verified to meet inclusion and exclusion criteria, they were scheduled for an overnight in-laboratory sleep study. The time scheduled was based on habitual sleep schedule and participant
preference with total sleeping time of eight hours and a bedtime as early as 9pm to as late as 1am.

**In-laboratory sleep study**

Subjects were asked to adhere to a fixed eight-hour sleep schedule at home for three days before in-laboratory nocturnal sleep study. In order to control for extraneous influence on sleep, participants were asked to maintain a regular bedtime between 9pm-1am and an awakening and rise time between 5am-9am. They were also asked to abstain from napping and restrict caffeine use to 2 cups or glasses before 12pm. To monitor compliance, participants were asked to complete consensus sleep diaries every morning upon awakening for the 3 to 5 days prior to the overnight sleep study. The sleep diaries include information on actual bed time, rise time, and subjective experience of sleep (Carney et al., 2012).

Participants arrived at the laboratory 2 hours before bed-time. Sensors were affixed. Electrodes were placed on the participant’s head according to the 10-20 electrode system at the sites FP1, FP2, F3, F4, FZ, C3, C4, CZ, PZ, O1, and O2. Electrodes were referenced to contralateral mastoid during data collection. Bilateral electroocculogram, submental electromyogram, anterior tibialis electromyogram, respiratory effort (respiratory inductance plethysmography), respiratory flow (nasal pressure transducer and thermister), hand electromyogram (EMG), and blood oxygen were all recorded. EKG was recorded using a modified lead II placement.

Prior to lights-out, physiological signals were recorded for two baseline periods each five minutes long first with eyes open and then eyes closed. After baseline recordings, participants were allowed to sleep. They were woken after five minutes of stage N2 sleep in the second NREM period by the experimenters calling their name over the intercom.
RSA was derived from the electrocardiogram (EKG) and was recorded and analyzed at multiple time points – at rest while sitting quietly before lights out for the evening, compared to RSA at this point of sudden awakening, over the duration of the awakening, and during the return to sleep. Participants were asked to remain awake for fifteen minutes while listening to auditory stimuli as part of a memory test that will be reported elsewhere. If participants began to fall asleep during this period, they were asked to remain awake. After this fifteen minute period, participants were allowed to resume sleeping. Auditory stimuli were stopped when participants resumed N2 sleep as evidenced by presence of sleep spindles or K complexes. Participants were then allowed to sleep until the end of the eight hour schedule at which they were awoken and sensors were removed. Participants were then debriefed and provided with compensation.

**Results**

The mean FIRST score was 14.60, SD(3.68) with a minimum reported score of 9 and maximum reported score of 22. Of the total participants, 48.48% were above the FIRST cut-off of 14 for high sleep-reactivity. Other descriptive statistics, including demographics, questionnaires, and RSA variables are reported in Table 1.

Analyses will be presented organized by each hypothesis.

*Hypothesis 1: Stress-reactive insomnia will be negatively associated with parasympathetic control at rest.*

There was no difference in RSA during baseline eyes open rest between those who scored below 14 on the FIRST (M=7.05, SD=1.05) and those who scored 14 or higher on the FIRST (M=7.02, SD=1.02), t(31)=0.09, p=0.93.

*Hypothesis 2: Stress-reactive insomnia will be negatively associated with parasympathetic control during an awakening from sleep.*
T-tests showed there was no significant difference in overall RSA during the awakening period between those who scored below 14 on the FIRST (M=66.45, SD=10.17) and those who scored 14 or above on the FIRST (M=69.66, SD=9.22), t(31)=-0.95, p=0.35. Through the 15 minute stay awake period, there was no difference in RSA between those who scored below 14 on the FIRST (M=7.09, SD=1.36) and those who scored above 14 (M=6.88, SD=0.96), t(31)=0.52, p=0.61. Likewise, there was no difference in RSA during the return to sleep period between participants who scored below 14 (M=7.42, SD=1.45) and those participants who scored above 14 (M=7.19, SD=0.91), t(31)=0.55, p=0.59. For the entire awakening period including the stay awake time and return to sleep time, there was no difference in those who scored below 14 on the FIRST (m=7.20, SD=1.37) and those who scored above 14 (M=7.04, SD=0.94), t(31)=0.37, p=0.71.

**Hypothesis 3: Stress-reactive insomnia will moderate the relationship between parasympathetic control at rest and during an awakening from sleep, such that individuals with greater stress-reactive insomnia will show decreases in parasympathetic control of greater magnitude than individuals with less stress-reactive insomnia**

In the multiple linear regression model, the relationship between baseline RSA and RSA during return to sleep moderated by FIRST score was statistically significant, F(3, 29)=29.49, p<.0001, R²=.75 (see Figure 1.). Higher baseline RSA predicted significantly higher RSA during return to sleep, F(1, 29)=83.33, p<.0001. There was no significant main effect of FIRST score on RSA during return to sleep, F(1, 29)=0.90, p=.35. The interaction between baseline RSA and FIRST score was significant, F(1, 29)=4.25, p<.05. When returning to sleep, participants with non sleep reactivity (FIRST scores <14) had higher RSA with values 124% of
their baseline (b=1.2372, SE=.15, t= 8.08, p<.0001), while participants with sleep reactivity (FIRST scores >14) had RSA values 77% of their baseline (b=0.77, SE=.16, t= 4.74, p<.0001).

Discussion

The goal of this study was to examine the relationship between parasympathetic control and sleep reactivity. This included measures of parasympathetic control during resting wakefulness and when returning to sleep after being awoken. There were no significant influences of sleep reactivity on parasympathetic control at rest. Similarly, there were also no significant differences between sleep reactive and non-sleep reactive groups in their absolute level of parasympathetic control when returning to sleep after being awoken. However, sleep reactivity moderated the relationship between parasympathetic control at rest and when returning to sleep. While non-sleep-reactive participants had higher parasympathetic control when returning to sleep relative to their baseline, sleep-reactive participants had relatively low parasympathetic control.

Sleep reactive individuals appear to decrease, rather than increase their parasympathetic control relative to baseline when resuming sleep. These results suggest that parasympathetic control may in part explain how sleep reactivity disrupts sleep. It extends findings about autonomic arousal in chronic insomnia to people at risk for developing insomnia, finding that arousal, at least in terms of parasympathetic control, may only be altered in certain situations. Chronic insomnia and sleep reactivity are believed to show 24-hour arousal (Bonnet & Arand, 2010; Drake et al., 2006); however, the present study’s results do not show this. Rather, finding ways of increasing parasympathetic control when returning to sleep may help prevent those with high sleep reactivity from developing chronic insomnia. Much of the current evidence in the role of parasympathetic control is correlative; further studies can elucidate whether arousal is causal,
coincidental or a perpetuating or predisposing factor for primary insomnia (Billiard & Bentley, 2004) because this suggests it is not a 24-hour arousal.

**Implications and future directions**

Parasympathetic control during the period of sleep onset and throughout the night may prove to be an important link between stress and sleep (Drake et al., 2004). Further research aimed at nonpharmacological and pharmacological interventions to increase parasympathetic control can work to decrease likelihood of developing a chronic insomnia disorder and can aim to target autonomic activity with multiple measures, rather than just RSA, and include measures of sympathetic nervous system activity. Studies that induce experimental manipulations of parasympathetic control would give clarity to the mechanisms underlying sleep reactivity and the role of parasympathetic control on sleep reactivity. Participants in the current study were primarily female. Increasing male participation, in addition to a wider age range, would provide a more representative sample overall. In the future, rather than excluding comorbidities, specific ones can be an examined variable. Studying the relationship between sleep reactivity and parasympathetic tone in individuals with depression, for example, would allow for testing if the relationship found in this study remains consistent across comorbidities. It could be interesting to test whether sleep reactivity is associated with lower parasympathetic control while returning to sleep among people with comorbid psychiatric disorders. Lastly, additional time points such as a follow-up sleep study would provide information on the time-frame of sleep reactivity. Subsequent participant follow-up would illuminate if the relationship seen between sleep reactivity and parasympathetic tone remains constant over time, specifically if changes in RSA precede changes in sleep or sleep reactivity, or the opposite relationship.

**Limitations**
This study had only the single measure of autonomic activity with no other measures of physiological arousal assessed, during a single overnight in-laboratory sleep study. The population studied was relatively homogenous with an age range from 18 to 26 and significantly more female than male participants. Participants were also ineligible if they had existing comorbidities. Additionally, other studies use higher cut offs to define sleep reactivity. Although lower cut offs for sleep reactivity are used in this study, abnormalities in parasympathetic control are seen. If there is more of a sample with higher sleep reactivity, larger differences may be elucidated.

Conclusion

In summary, this is the first study to examine the relationship between sleep reactivity and parasympathetic control as measured by the FIRST and RSA in response to a sleep-related stressor. Individuals with high sleep reactivity were shown to have lower parasympathetic control when returning to sleep compared to individuals with low sleep reactivity. Future studies should continue to explore this relationship and look into interventions related to and that target parasympathetic control which perhaps may benefit those susceptible to stress-related sleep disturbance.
References


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http://doi.org/10.4088/JCP.v60n0403


http://doi.org/10.1016/j.sleep.2009.07.008


and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10.


http://doi.org/10.1016/j.sleep.2011.10.033


Appendix A

**Table 1 – Ford Insomnia Response to Stress Test**

When you experience the following situations, how likely is it for you to have difficulty sleeping? Circle an answer even if you have not experience these situations recently.

<table>
<thead>
<tr>
<th>Situation</th>
<th>Not likely</th>
<th>Somewhat likely</th>
<th>Moderately likely</th>
<th>Very likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before an important meeting the next day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After a stressful experience during the day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After a stressful experience in the evening</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After getting bad news during the day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After watching a frightening movie of TV show</td>
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<td></td>
</tr>
<tr>
<td>After having a bad day at work</td>
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<tr>
<td>After an argument</td>
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<tr>
<td>Before having to speak in public</td>
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<tr>
<td>Before going on vacation the next day</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Drake et al., 2004
Figure 1.

Table 1. Demographic and subjective sleep characteristics of study participants

<table>
<thead>
<tr>
<th></th>
<th>Low FIRST</th>
<th>High FIRST</th>
<th>t value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>20.24 (2.54)</td>
<td>19.88 (2.00)</td>
<td>0.45</td>
<td>0.6549</td>
</tr>
<tr>
<td>FIRST</td>
<td>11.59 (1.42)</td>
<td>17.19 (3.08)</td>
<td>6.77</td>
<td>&lt;.0001 *</td>
</tr>
<tr>
<td>RSA baseline</td>
<td>7.04 (1.03)</td>
<td>7.01 (1.00)</td>
<td>0.07</td>
<td>0.9429</td>
</tr>
<tr>
<td>RSA RTS</td>
<td>7.42 (1.45)</td>
<td>7.19 (0.91)</td>
<td>0.55</td>
<td>0.5861</td>
</tr>
</tbody>
</table>

*p<.0001