High-Frequency Heart Rate Variability Reactivity and Trait Worry Interact to Predict the Development of Sleep Disturbances in Response to a Naturalistic Stressor

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Abstract

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Sasha MacNeil

High-frequency heart rate variability (HF-HRV) reactivity was proposed as a vulnerability factor for stress-induced sleep disturbances. Its effect may be amplified among individuals with high trait worry or sleep reactivity. This study evaluated whether HF-HRV reactivity to a worry induction, sleep reactivity, and trait worry predict increases in sleep disturbances in response to academic stress, a naturalistic stressor. A longitudinal study following 102 undergraduate students during an academic semester with well-defined periods of lower and higher academic stress was conducted. HF-HRV reactivity to a worry induction, trait worry using the Penn State Worry Questionnaire, and sleep reactivity using the Ford Insomnia Stress Reactivity Test were measured during the low stress period. Sleep disturbances using the Pittsburgh Sleep Quality Index were assessed twice during the lower stress period and three times during the higher stress period. Greater reductions in HF-HRV in response to the worry induction predicted increases in sleep disturbances from the lower to the higher academic stress period. Trait worry moderated this association: individuals with both higher trait worry and greater HF-HRV reactivity to worry had larger increases in stress-related sleep disturbances over time, compared to participants with lower trait worry and HF-HRV reactivity. A similar, but marginally significant effect was found for sleep reactivity. This study supports the role of HF-HRV reactivity as a vulnerability factor for stress-induced sleep disturbances. The combination of high trait worry and high HF-HRV reactivity to worry might identify a subgroup of individuals most vulnerable to stress-related sleep disturbances.

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Contribution of Authors

Sasha MacNeil: Ms. MacNeil contributed to this thesis by participating in study design, data collection and cleaning, and writing of the abstract, introduction, method, and discussion.

Sonya Deschênes: Dr. Deschênes contributed to this thesis by running all statistical analyses and writing the results section.

Warren Caldwell: Mr. Caldwell contributed to this thesis by participating in study design, data collection and cleaning, and providing input and recommendations for points of clarification and wording in the manuscript.

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Thienh-Thanh Dang-Vu: Dr. Dang-Vu contributed to this thesis by providing input and recommendations for points of clarification and wording in the manuscript.

Jean-Philippe Gouin : This study is part of a larger project supervised by Dr. Gouin. Dr. Gouin designed this project, supervised the data collection and cleaning phases of this project, and supervised writing of the manuscript, providing direction and feedback for writing.

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General Introduction

There has been growing interest in the role of heart rate variability (HRV) as a potential biomarker for vulnerability to psychopathology (Beauchaine, 2015; Thayer & Lane, 2009). The Neurovisceral Integration Model (Thayer & Lane, 2000) posits that this physiological measure may index a neurophysiological system involved in physiological, emotional, cognitive, and behavioural regulation (Thayer & Lane, 2000; Thayer & Lane, 2009). HRV is a measure of the fluctuations in the temporal distance between consecutive heart beats that is determined by the interplay between sympathetic and parasympathetic influences upon the heart (Thayer & Lane, 2000). This model proposes that HRV indexes the efficiency of a neural circuit, the central autonomic network, promoting flexible responsiveness to environmental changes (Thayer & Brosschot, 2005; Thayer & Lane, 2000). The central autonomic network, comprised of cortical and subcortical structures, provides an integrated regulation system through which the brain sends efferent signals to control visceromotor, neuroendocrine and behavioural responses that are essential for adaptability and goal-directed behaviour (Thayer & Brosschot, 2005). The principal output of the central autonomic network is communicated to the heart with efferent preganglionic sympathetic neurons via the stellate ganglia and parasympathetic neurons via the vagus nerve. In addition, approximately 90% of fibers within the vagus nerve are afferent fibers transmitting inputs from visceral, humoral, and environmental sources back to the central autonomic network (Benarroch, 1993). As such, HRV indexes a system integrating central and peripheral information to coordinate appropriate autonomic and behavioural responses to environmental challenges (Thayer & Lane, 2000).

The heart is dually innervated by the excitatory sympathetic and inhibitory parasympathetic influences from the central autonomic network. According to the Neurovisceral Integration model, at rest, the prefrontal cortex maintains an inhibitory influence upon the amygdala, which further inhibits sympatho-excitatory neurons involved in energy mobilization and maintains the inhibitory activity of parasympathetic neurons upon the sinoatrial node of the heart via the vagus nerve, allowing a state of energy conservation (Thayer & Brosschot, 2005; Thayer & Lane, 2000; Thayer & Lane, 2009). The vagus nerve acts as a brake upon the heart, maintaining heart rate at a slower pace by providing constant but changing levels of inhibition on the sinoatrial node (Thayer & Lane, 2000). The interplay of the faster-acting parasympathetic (in milliseconds) and slower-acting sympathetic (in seconds) outputs of the central autonomic

network at the sinoatrial node of the heart produces beat-to-beat variability marking an adaptive organism, measured as HRV (Thayer & Brosschot, 2005). However, the high frequency component of HRV (HF-HRV) is a measure of vagally-mediated parasympathetic influence on cardiac activity (Bernston et al., 1997). During the respiration cycle, vagal parasympathetic neurons are inhibited during the inspiration phase, leading to decreases in HF-HRV, and are again activated during expiration, leading to increases in HF-HRV. HF-HRV arises from these fast-acting millisecond fluctuations of vagally-mediated parasympathetic control of the heart (Bernston, Cacioppo, & Quigley, 1993). Resting levels of HF-HRV thus index the inhibitory strength of the central autonomic network and of the parasympathetic nervous system upon the sinoatrial node of the heart. Greater resting HF-HRV levels are thought to indicate increased flexibility in adjusting the autonomic modulation of cardiac activity to the environment, indexing better adaptation capacities (Thayer & Lane, 2000; Thayer & Lane, 2009).

In response to stress, the prefrontal cortex becomes hypoactive, leading to withdrawal of the inhibitory effects of parasympathetic activity and disinhibition of sympathetic excitatory autonomic processes upon the heart in order to adaptively respond to the threat (Arnsten & Goldman-Rakic, 1998; Bernston & Cacioppo, 2004). Based on the Neurovisceral Integration Model, this decrease in parasympathetic influence on the sinoatrial node of the heart leads to reductions in the fluctuations of temporal distance between consecutive heart beats, decreasing HF-HRV (Thayer & Sternberg, 2006). Given the integration of afferent and efferent inputs from cerebral, visceral, humoral, and environmental sources via the vagus nerve, such decreases in HF-HRV to stress associated with a reduction in parasympathetic influence are considered part of the adaptive general response to perceived threats (Berntson & Cacioppo, 2004). Therefore, change in HF-HRV to stress, or HF-HRV reactivity, is another measure of vagally-medical parasympathetic regulation.

Although decreases in HF-HRV in response to physical stressors is considered an adaptive part of the normal response to threat (Bernston & Cacioppo, 2004), it has been proposed that excessive HF-HRV to emotional stressors is less adaptive (Beauchaine, 2015). In particular, HF-HRV reactivity to emotional stressors, such as worry, may not be adaptive given that situational demands requiring energy mobilization are not present (Gouin, Deschenes, & Dugas, 2014). Thus, in the absence of objective threat, greater HF-HRV reactivity may be less adaptive. Empirical evidence has supported this view, such that individuals with excessive HF-HRV

reactivity to emotional stressors were at increased risk for the development of psychopathology (Beauchaine et al., 2001; Beauchaine, Hong, & Marsh, 2008; Boyce et al., 2001; Calkins et al., 2007; Crowell et al., 2005; Fortunato, Gatzke-Kopp, & Ram, 2013; Gouin et al., 2014; Gouin et al., 2015; Hughes & Stoney, 2000; Monk et al., 2001).

Introduction

According to contemporary models of insomnia, stress is an important precipitating factor for the onset of sleep disturbances (Morin, 1993; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Spielman, 1986). Cross-sectional studies indicate that a range of psychosocial stressors, including work and financial stressors (Akerstedt, 2006; Huang et al., 2014; Kalimo, Tenkanen, Harma, Poppius, & Heinsalmi, 2000; Knudsen, Ducharme, & Roman, 2007), academic stress (Lund, Reider, Whiting, & Prichard, 2009), interpersonal conflicts (Fortunato & Harsh, 2006), and negative life events (Taylor et al., 2016) are associated with increased subjective and objective sleep disturbances. Experimental studies show that acute laboratory stressors before sleep, such as giving a speech task (Hall et al., 2004), watching aversive films (Baekeland, Koulack, & Lasky, 1968), or the anticipation of a stressful speech task after sleep (Gross & Borkovec, 1982) lead to delayed sleep onset latency, lower sleep efficiency, more frequent nocturnal awakenings, and earlier morning awakenings. Daily diary studies indicate that individuals who report more stress than usual on a given day tend to experience poorer sleep the following night (Akerstedt et al., 2012; Morin, Rodrigue, & Ivers, 2003; Winzeler et al., 2014). Further, in longitudinal studies, higher work stress (Linton, 2004; Ota et al., 2009), academic stress (Galambos, Howard, & Maggs, 2011; Galambos, Vargas Lascano, Howard, & Maggs, 2013), family life stress (Bernert, Merrill, Braithwaite, Van Orden, & Joiner, 2007), and the occurrence of major life events (Drake, Pillai, & Roth, 2014) predict greater risk for insomnia symptoms and syndrome over time.

Despite evidence supporting stress as a precipitating factor for sleep disturbances, laboratory and epidemiological studies indicate that there are marked individual differences in the susceptibility to and extent of sleep disruptions following stress exposure (Bonnet & Arand, 2003; Drake, Pillai, & Roth, 2014; Drake, Richardson, Roehrs, Scofield, & Roth, 2004). Individual differences in self-reported sleep reactivity, defined as the tendency to experience sleep disruptions following sleep challenges such as stress (Drake, Friedman, Wright, & Roth, 2011), have been associated with greater sleep disturbances in response to caffeine intake close

to bedtime (Drake, Richardson, Roehrs, Scofield, & Roth, 2004), following rotating shift work schedules (Kalmbach, Pillai, Cheng, Arnedt, & Drake, 2015), and during periods of increased work stress (Petersen, Kecklund, D'Onofrio, Nilsson, & Akerstedt, 2013). In longitudinal studies, greater sleep reactivity was associated with increased risk for the onset and persistence of insomnia disorders over time (Kalmbach, Pillai, Arnedt, & Drake, 2016; Pillai, Steenburg, Ciesla, Roth, & Drake, 2014).

We recently proposed that high-frequency heart rate variability (HF-HRV) may represent a vulnerability factor for stress-induced sleep disturbances (Gouin et al., 2015). HRV represents the fluctuations in the time intervals between consecutive heart beats (Berntson et al., 1997). During waking restfulness, the high-frequency (HF) component of HRV is an indicator of vagal-dependent parasympathetic influences to the sinoatrial node of the heart. Given that the brain stem nuclei regulating HF-HRV integrate ascending projections from the viscera through the vagal nerve and efferent projections from the prefrontal cortex to the amygdala and central autonomic network (Porges, 2007; Thayer & Lane, 2009), low HF-HRV has been conceptualized as a peripheral marker of poor physiological and emotional regulation (Beauchaine, 2015; Proges, 2007; Thayer & Lane, 2000). Consistent with this perspective, low HF-HRV has been associated with greater heart rate, blood pressure, cortisol and negative emotional responses to stress (Gouin, Deschênes, & Dugas, 2014; Hansen, Johnsen, & Thayer, 2009; Melzig, Weike, Hamm, & Thayer, 2009; Papousek, Schulter, & Premsberger, 2002; Shinba et al., 2008; Souza et al., 2007; Weber et al., 2010).

Waking HF-HRV is also associated with sleep quality. Among community participants and individuals with depression and panic disorder, reduced HF-HRV during resting wakefulness was associated with greater self-reported sleep disturbances, including worse subjective sleep quality, longer sleep onset latency, and more nocturnal awakenings (Brosschot, Van Dijk, & Thayer, 2007; Hovland et al., 2013; Werner, Ford, Mauss, Schabus, & Blechert, 2015). Among individuals with an insomnia disorder, lower HF-HRV was associated with greater severity of insomnia symptoms and increased daytime sleepiness (Farina et al., 2013; Yang et al., 2011). In large community samples, short sleep duration and poor sleep efficiency were associated with reduced HF-HRV (Castro-Diehl et al., 2016; Jackowska, Dockray, Endrighi, Hendrickx, & Steptoe, 2012). Lower HF-HRV at rest is thus associated with poorer sleep quality.

HF-HRV reactivity, defined as the HF-HRV response to challenges, has also been related to sleep quality. Whereas some authors argue that greater reactivity to challenges represents an adaptive physiological stress response (Porges, 2007), others have noted that excessive reactivity is associated with poor emotion regulation and greater risk for psychopathology, especially when HF-HRV reactivity is elicited using emotional tasks (Beauchaine, 2015). Only a handful of studies have examined the association between HF-HRV reactivity and sleep quality. Lower HF-HRV during a stress task was associated with poorer sleep among children, depressed patients, and women with breast cancer (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014; El-Sheikh, Erath, & Bagley, 2013; Palesh et al., 2008). Greater HF-HRV decreases to an emotional task increased the longitudinal association between a psychosocial stressor and poor sleep in children (Keller, Kouros, Erath, Dahl, & El-Sheikh, 2014), and HF-HRV reactivity to a cognitive task had a protective effect on stress-related sleep disturbances (El-Sheikh, Hinnant, & Erath, 2015). Although there are some inconsistencies likely related to different methodological approaches, the extant literature suggests that HF-HRV reactivity to emotional tasks is related to sleep quality. However, the directionality of the association between sleep and HF-HRV reactivity is unclear; that is, these studies do not clearly tease apart whether poor sleep alters HF-HRV reactivity, whether high HF-HRV reactivity increases risk for poor sleep, or if a third variable may be affecting this association.

Laboratory and longitudinal studies suggest that HF-HRV might be a marker of individual differences in sleep reactivity to stress. In an experimental study, individuals with reduced waking HF-HRV experienced more situational insomnia in response to different sleep challenges, compared to those who did not exhibit such sleep reactivity (Bonnett & Arand, 2003). In a small longitudinal study of individuals selected for high and low self-reported sleep reactivity, lower HF-HRV during a worry induction predicted longitudinal increases in sleep disturbances from well-defined periods of lower and higher academic stress (Gouin et al., 2015). This study provided initial evidence that HF-HRV reactivity to a worry induction might predict vulnerability to stress-related sleep disturbances. However, this study was limited by a small sample selected for extreme scores on sleep reactivity. Replication of these results in a broader, unselected sample is thus warranted.

Emerging research suggests that individuals who are more vulnerable to stress-induced insomnia experience greater worry in response to stress (Drake, Pillai, & Roth, 2014). Worry is a

chain of repetitive, negative affect-laden and uncontrollable thoughts (Borkovec & Ray, 1998). Individuals with insomnia often report trouble initiating or maintaining sleep because of uncontrollable and excessive worry (Armstrong & Dregan, 2014; Urponen, Vuori, Hasan, & Partinen, 1988). In cross-sectional studies, individuals reporting greater work- and health-related worries or higher repetitive negative thinking were more likely to report delayed sleep onset latency and decreased sleep efficiency (Akerstedt et al., 2002; Akerstedt, Kecklund, & Axelsson, 2002;, Jean-Louis et al., 2009; Pereira, Meier, & Elfering, 2013). In daily diary studies, days with greater daily and pre-sleep worry were associated with longer sleep onset latency, worse sleep efficiency, and decreased total sleep time, especially among high trait worriers (McGowan, Behar, & Luhmann, 2016; Pillai, Steenburg, Ciesla, Roth, & Drake, 2014; Syrek, Weigelt, Peifer, & Antoni, 2016; Weise, Ong, Tesler, Kim, & Roth; 2013). Although it has been suggested that increased worry may be an epiphenomenon, a consequence of periods of increased wakefulness in bed (Omvik, Pallesen, Bjorvatn, Thayer, & Nordhus, 2007), experimental studies showed that worry induction before sleep was associated with longer subsequent objective and subjective sleep onset latency and poorer sleep quality (Brosschot, 2010; Zoccola, Dickerson, & Lam, 2009), suggesting a causal relationship between worry and greater sleep disturbances.

According to the Perseverative Cognition Hypothesis, worry and rumination are forms of repetitive negative thought maintaining the mental representations of anticipated future or past stressful events, thereby prolonging stress-related cognitive and physiological arousal (Brosscot, 2007; Brosschot, Pieper, & Thayer, 2005; Brosschot, van Dijk, & Thayer, 2002; Brosschot, Verkuil, & Thayer, 2010; Pieper & Brosschot, 2005). In line with this theory, individual differences in worry intensity moderated cardiovascular reactivity, such that greater stress-related worrying led to prolonged cardiac reactivity to stress (Verkuil, Brosschot, de Beurs, & Thayer, 2009; Verkuil, Brosschot, Meerman, Esther, & Thayer, 2012). These cardiovascular effects of stress-related worry have been found to extend up to two hours beyond the worry episode (Pieper, Brosschot, van der Leeden, & Thayer, 2010), and into the night (Brosschot, 2010; Brosschot, van Dijk, & Thayer, 2002; Brosschot, van Dijk, & Thayer, 2007). Furthermore, daily diary and longitudinal studies indicate that increased worry and rumination mediate the association between stress exposure and delayed sleep onset latency (Radstaak, Geurts, Beckers, Brosschot, & Kompier, 2014), poor sleep quality (Van Laethem et al., 2015), and risk of

insomnia syndrome (Drake, Pillai, & Roth, 2014). These data thus suggest that high trait worry might represent a risk factor for vulnerability to stress-induced sleep disturbances.

High HF-HRV reactivity may interact with psychological vulnerabilities to stress, as indexed by sleep reactivity and trait worry, to increase vulnerability to sleep disturbances. Individuals with high trait worry experience more perseverative cognition in response to stress (Verkuil, Brosschot, Gebhardt, & Thayer, 2010; Verkuil, Brosschot, & Thayer, 2007). Similarly, individuals with high sleep reactivity report greater perceived stress and emotional and cognitive hyperarousal (Fernandez-Mendoza et al., 2010). Further, there is emerging evidence indicating that individuals with low resting HF-HRV and high HF-HRV reactivity have greater difficulty disengaging from negative repetitive thoughts (Gillie, Vasey, & Thayer, 2015) and regulating their emotions more broadly (Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013; Yaroslavsky et al., 2016). This suggests that individuals with a double risk profile of high HF-HRV reactivity and high trait worry or high sleep reactivity might be particularly susceptible to stress-induced sleep disturbances.

The goals of the present study were to replicate and extend prior findings by testing whether individual differences in HF-HRV reactivity to an emotional task would predict the development or exacerbation of self-reported sleep disturbances in response to academic stress (Gouin et al., 2015), and whether trait worry and sleep reactivity would interact with HF-HRV to prospectively predict changes in sleep disturbances under stress. To examine this question, sleep was assessed across an academic semester, an ecologically valid and relatively standardized period of increasing stress (Bolger & Eckenrode, 1991; Jernelov et al., 2009; Malarkey, Pearl, Demers, Kiecolt-Glaser, & Glaser, 1995). Indeed, sleep disturbances due to academic stress have been reported to increase throughout the academic semester as students are approaching final examinations (Feld & Shusterman, 2015; Lund, Reider, Whiting, & Prichard, 2010), providing a unique opportunity to examine individual differences in stress-related sleep disturbances. It was hypothesized that HF-HRV reactivity during worry would prospectively predict increases in self-reported sleep disturbances in response to academic stress, and that individuals with high trait worry or high sleep reactivity and greater HF-HRV reactivity would be at greatest risk for sleep disturbances.

Method

Participants

One hundred and twenty full-time healthy undergraduate students participated in the study in exchange for course credit. Exclusion criteria for this study were being over thirty years of age, smoking more than 3 cigarettes per week, and having a chronic medical condition. Eighteen participants were excluded from the present analyses due to missing HF-HRV, sleep reactivity, or trait worry data. The excluded participants tended to be older (M_{age} = 22.28, SD_{age} = 2.22) than included participants (M_{age} = 21.00, SD_{age} = 2.20), p = .03, but did not differ on sex, trait worry, sleep reactivity, or baseline sleep disturbances.

Included participants were recruited from psychology (48.0%) and exercise science (52.0%) undergraduate programs. A majority of participants were in their first year of university (54.9%), 14.7% were in their second year, and 22.6% were in their third year. The sample was predominantly female (77.5%) and Caucasian (59.8%). Thirteen of the 102 included participants were taking oral contraceptives, and sixteen had used other over-the-counter or prescribed medication in the past week, including the use of pain relievers and anti-inflammatories (69%), allergy medication (13%), muscle relaxants (6%), antidepressants (6%), acne medication (6%), and medication for acid deficiency (6%). No participants reported taking any sleep medication. This study was approved by Concordia University's Institutional Review Board. Informed consent was obtained from all participants.

Measures

Sleep disturbances. The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a self-report questionnaire measuring sleep quality and disturbances. The instructions were altered so that participants responded to the questions based on their sleep quality in the last week. This scale contains 19 items composing seven subscales: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. A global score ranging from 0 to 21 is computed from the seven subscales, with higher scores indicating greater sleep disturbances. In this sample, the PSQI demonstrated adequate internal consistency, with Cronbach's alphas of the seven component scores ranging from .60 to .72 across the five measured time points. A cut-off score of 5 has been demonstrated to correctly identify 88.5% of good and poor sleepers, with a sensitivity of 89.6% and specificity of 86.5% (Buysse et al., 1989). At Time 1, 28.3 % of participants had a PSQI score above 5.

Sleep reactivity. The Ford Insomnia Response to Stress Test (FIRST) (Drake, Richardson, Roehrs, Scofield, & Roth, 2004) is a 9-item questionnaire measuring sleep disturbances in response to common stressors. Items are scored on a 4-point Likert scale ranging from 1 (*Not likely*) to 4 (*Very likely*). Higher scores represent greater vulnerability to stress-related sleep disturbances. The FIRST demonstrated high internal consistency in this sample (Cronbach's alpha = .83), and excellent 2-week test-retest reliability (Drake, Richardson, Roehrs, Scofield, & Roth, 2004) and high temporal stability at 6 and 12 months (Jarrin, Chen, Ivers, Drake, & Morin, 2016) in other studies. A cut-off score of 18 identified individuals at high risk for insomnia, with sensitivity of 62% and specificity of 67% (Kalmbach, Pillai, Arnedt, & Drake, 2016). In this sample, 43% of participants met this cut-off score.

Trait worry. The Penn State Worry Questionnaire (PSWQ) (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item measure of the general factor of trait worry. Items are rated on a 5-point Likert Scale ranging from 1 (*Not at all typical of me*) to 5 (*Very typical of me*). The PSWQ has demonstrated good psychometric properties, with excellent internal consistency (Cronbach's alpha = .93) in this sample and excellent test-retest reliability (r = .92) after an 8-week interval (Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ has also demonstrated good convergent validity, with positive correlations between PSWQ scores and scores on other measures of emotional disturbances, psychological problems, and maladaptive coping strategies in undergraduate students (Meyer, Miller, Metzger, & Borkovec, 1990). A cut-off score of 57 has been shown to have optimal sensitivity, correctly classifying 94% of patients with generalized anxiety disorder (Fresco, Mennin, Heimberg, & Turk, 2003). In this study, 30.2 % of participants scored above this clinical cut-off.

Psychological distress. To serve as a manipulation check, the short form of the Depression, Anxiety, and Stress Scale (DASS-21) (Lovibond & Lovibond, 1993) was administered at each time point as a measure of subjective distress in the past week. It consists of 21 items forming three subscales: depression (e.g.: I felt that life was meaningless), anxiety (e.g.: I experienced trembling), and stress (e.g.: I found it difficult to relax). Each item is scored on a Likert Scale from 0 (i.e., Never/Did not apply to me at all) to 3 (i.e., Almost Always/Applied to me almost all of the time). Total scores are obtained by summing the items of each subscale, with higher scores indicating more severe symptoms. This scale has excellent internal consistency, with Cronbach's alpha of .90 (Henry & Crawford, 2005). In the present sample, the

internal consistency of the DASS-21 ranged from .91 to .92 in the lower stress period, and from .94 to .95 in the higher stress period.

Heart rate variability. Participants were fitted with a chest belt hardwired with a digital inter-beat interval recorder Polar RS800CX (Polar Electro Oy; Finland: Kempele). The telemetric inter-beat interval recording device recorded the interval between successive R-spikes of consecutive QRS complexes, using a sampling rate of 1000 samples per second. Recording artifacts were identified and corrected using CardioEdit (Brain Body Center, University of Illinois at Chicago, 2007). Artifact correction was performed using integer arithmetic (i.e., dividing or adding intervals between heartbeats to correct missed or spurious R-spike detections). HF-HRV was calculated from edited interbeat intervals using CardioBatch (Brain Body Center, University of Illinois at Chicago, 2007), a software that implements the moving polynomial method proposed by Porges and Bohrer (Porges et al., 1980). The moving polynomial filter was applied to the heart rate time series to remove the influence of aperiodic processes. A bandpassed filter was applied to the detrended residualized heart rate time series to extract the variance associated with fluctuations in interbeat intervals across the respiration cycle (0.12-0.40 Hz). HF-HRV was calculated sequentially over 30-second epochs within each task and then averaged to minimize the impact of violation of the stationarity assumption. Each HF-HRV estimate was transformed using a natural logarithm to normalize its distribution. In a validation study, HF-HRV estimates derived during each 30-s epoch were strongly correlated (r = .88-.95) with the total 5-min HF-HRV averaged estimate (Lewis, Furman, McCool, & Porges, 2012). This method provides an optimal assessment of vagally-mediated HF-HRV. Theoretical and empirical research suggests that resting HF-HRV and HF-HRV reactivity represent different dimensions of autonomic constraint (Bernston, Cacioppo, & Quigley, 1991; Graziano & Derefinko, 2013). Importantly for HF-HRV reactivity, both the magnitude and direction of change in HF-HRV to a task (i.e., vagal withdrawal vs. augmentation) have been associated with different psychological outcomes (Beauchaine, 2015, Keller et al., 2014). As recommended by Bernston and colleagues (1991), HF-HRV reactivity was determined by subtracting the average HF-HRV during the worry period from the average HF-HRV during the resting period. In this context, higher HF-HRV reactivity represented greater vagal withdrawal, that is greater decreases in HF-HRV from the resting period to the worry period (Yaroslavsky et al., 2013; Yaroslavsky et al., 2016).

Procedure

Participants were followed longitudinally over the course of an academic semester. In the first month of the semester (i.e., the lower academic stress period), participants completed the self-reported DASS, PSQI, and FIRST questionnaires online and came to the laboratory to complete heart rate recordings (Time 1). Participants were instructed to refrain from exercising or drinking caffeinated beverages at least 2 hours before the laboratory session. The heart monitor was fitted around the participants' chests at the base of the sternum. Participants underwent a baseline resting heart rate recording for 5 minutes while seated. Following the baseline recording, participants completed a worry induction task. For the worry induction, participants were asked to engage in a 5-minute worry period induced based on Hofmann et al.'s protocol (Hofmann, Schulz, Heering, Muench, & Bufka, 2010). Participants were provided with a definition of worry ("a chain of negative thoughts about something that can have negative consequences for you in the future"), and were asked to worry about a topic they identified worrying about most often and most intensely.

One week following the laboratory session, participants completed the PSWQ and a repeated assessment of the PSQI and DASS online (Time 2). During the second to last week of the semester (Time 3), the last week of the semester (Time 4), and the first week of exams (Time 5), conceptualized in this study as the higher academic stress period, participants completed repeated assessments of the DASS and PSQI (See Figure 1 for a depiction of the assessment schedule).

Statistical Analysis

Multilevel modeling was used to test the interactions between HF-HRV reactivity to a worry-inducing task, trait worry (PSWQ), sleep reactivity (FIRST), and trajectories of change over time in self-reported sleep disturbances during the academic semester (PSQI). The growth model was centered at Time 1. Intra-individual change in self-reported sleep disturbances was modelled as a function of the wave of assessment (Time 1 through Time 5). Separate models were estimated for the three-way interactions between HF-HRV reactivity, time, and PSWQ scores and interactions between HF-HRV reactivity, time, and FIRST scores. A total of five multilevel models were estimated. In Model 1, an unconditional means model was estimated for PSQI scores and the intraclass correlation (ICC) was calculated. ICC reflects the proportion of PSQI score variance that is accounted for by between-person clustering (Peugh, 2010). An

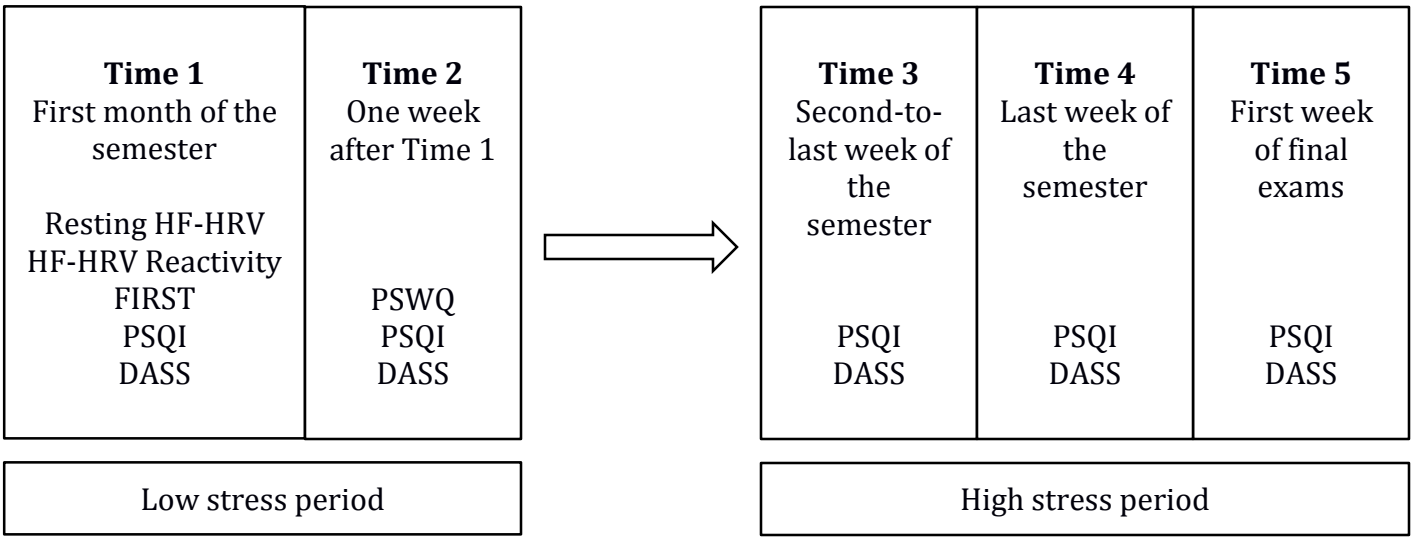

Figure 1. Assessment schedule

unconditional growth model was tested in Model 2. We hypothesized that, on average, PSQI scores would increase during the academic semester. Proportional reduction in variance was calculated by subtracting the residual variance of the more complex models from the residual variance in the unconditional means model and dividing this value by the residual variance in the unconditional means model, as a measure of the magnitude of the effect size (Singer & Willett, 2003).

The next steps tested the associations between level-2 predictors and change over time in PSQI scores. Model 3 tested the time × HF-HRV reactivity interaction, Model 4 tested the time × HF-HRV reactivity × PSWQ scores interaction, and Model 5 tested the time × HF-HRV reactivity × FIRST scores interaction.

Resting HF-HRV was included as a covariate in all models given meta-analytic evidence that covarying resting HF-HRV increases the association between HF-HRV reactivity and adaptive outcomes (Graziano & Derefinko, 2013). All main effects and two-way interactions were included in Models 3, 4 and 5. Covariates in the models included sex, ethnicity, and past week medication use other than oral contraceptives because these have been found to influence HF-HRV (Liao et al., 1995). Program of study was included as a covariate to account for the slightly different exam schedules at the end of the semester. Also, the number of hours of work on a weekly basis was included as a covariate because this may represent a source of additional stress for students. Interactions between time and each covariate were also included in models 3, 4, and 5. Simple slopes analyses were planned following a statistically significant time × HF-HRV reactivity × PSWQ or FIRST scores interaction by constructing slopes of change over time in PSQI scores at two levels of the moderators, HF-HRV reactivity and PSWQ or FIRST scores (1 SD above and below the mean). Bonferroni correction for multiple comparisons was used to minimize inflated type I (family-wise) error. Analyses were conducted using the xtmixed procedure in Stata version 14.1 (StataCorp LP, College Station, TX) with maximum likelihood estimation and using an unstructured covariance matrix.

Results

As a manipulation check, change in psychological distress over the academic semester was examined. In a multilevel model, there was a significant linear increase in psychological distress on the DASS total score over the 5 time points (b = .62, SE = .24, p = .01). Using the stress subscale of the DASS, there was a significant linear increase in perceived stress from the

lower to the higher stress period (b = .21, SE = .07, p = .003), providing support for the academic stress model. In addition, a paired sample t-test indicated that there was a statistically significant mean reduction in HF-HRV from the resting phase (M = 6.73, SD = .12) to the worry induction phase (M = 6.47, SD = .12), $t(101) = 3.85$, $p < .001$. Despite the average decrease in HF-HRV during the task, 34.3% of the sample experienced an increase in HF-HRV in response to the worry induction task, highlighting the individual differences in HF-HRV reactivity to the task. There was also a significant correlation between resting HF-HRV and HF-HRV reactivity (r = .24, $p < .05$), such that individuals with higher resting HF-HRV tended to have greater HF-HRV reactivity. Table 1 presents the means and SDs as well as the bivariate correlations between resting HF-HRV, HF-HRV reactivity, PSWQ and FIRST scores measured at baseline, and PSQI scores measured at each time point.

Table 2 describes the results of the multilevel analyses predicting change over time in PSQI scores for Models 1 to 5. In the unconditional means model (Model 1), the ICC was .51 which indicates substantial between-person variability in PSQI scores. Model 2 demonstrated a significant linear increase over time in PSQI scores during the study period (b = .14, SE = .07 p = .05) indicating that overall, self-reported sleep disturbances tended to increase during the academic semester. Linear time accounted for 13.54% of the variance in PSQI scores.

Model 3 examined whether HF-HRV reactivity accounted for variability in change over time in PSQI scores. There was a statistically significant time × HF-HRV reactivity interaction in predicting PSQI scores (b = .21, SE = .10, p = .046). Model 3 accounted for approximately 23.27% of the random slope variance compared to Model 2. We additionally examined whether there was a significant time × resting HF-HRV × HF-HRV reactivity interaction on PSQI scores, however no statistically significant interaction was found (b = -.02, SE = .09, p = .81).

In Model 4, a significant time × HF-HRV reactivity × PSWQ interaction was found (b = .02, SE = .01, p = .009). A statistically significant time × HF-HRV reactivity × PSWQ interaction (b = .01, SE = .01, p = .02) was also found in a model without covariates. Model 4 accounted for a total of 57.38% of the random slope variance in PSQI scores compared to Model 2. Decomposing the interaction revealed that high HF-HRV reactivity with high PSWQ scores were associated with a statistically significant increase in PSQI scores over time (Figure 2), whereas high HF-HRV reactivity with low PSWQ scores, low HF-HRV reactivity with high PSWQ scores, and low HF-HRV reactivity with low PSWQ scores were not significantly

Table 1.

Means (SD) and Pearson's bivariate correlations

	HF-HRV resting	HF-HRV reactivity	PSWQ	FIRST	PSQI-1	PSQI-2	PSQI-3	PSQI-4	PSQI-5	Mean (SD)	Observed Range
	Baseline Assessment				Low Stress		High Stress				
HF-HRV resting	1.00	.24*	-.11	.02	.01	.11	.03	.05	-.06	6.73 (1.17)	3.46 – 9.40
HF-HRVr		1.00	-.16	.05	.08	.10	.16	.26**	.20 ^{t}	0.26 (0.68)	-1.58 – 2.29
PSWQ			1.00	.52**	.15	.33**	.41**	.31**	.43**	49.70 (15.07)	23 – 80
FIRST				1.00	.31**	.39**	.31**	.39**	.39**	17.03 (7.82)	0 – 36
PSQI-1					1.00	.54**	.37**	.49**	.45**	4.75 (3.04)	0 – 16
PSQI-2						1.00	.60**	.56**	.64**	4.40 (2.36)	0 – 13
PSQI-3							1.00	.65**	.71**	5.17 (2.81)	0 – 13
PSQI-4								1.00	.68**	5.22 (2.76)	0 – 14
PSQI-5									1.00	5.10 (2.85)	0 – 12

Note. HF-HRV resting = resting high frequency heart rate variability measured at baseline; HF-HRVr = high frequency heart rate variability reactivity to worry induction at baseline ; PSWQ = Penn State Worry Questionnaire measured at baseline; FIRST = Ford Insomnia Response to Stress Test; PSQI-1 = Pittsburgh Sleep Quality Index, time 1; PSQI-2 = Pittsburgh Sleep Quality Index, time 2; PSQI-3 = Pittsburgh Sleep Quality Index, time 3; PSQI-4 = Pittsburgh Sleep Quality Index, time 4; PSQI-5 = Pittsburgh Sleep Quality Index, time 5. *$p < .05$, **$p < .01$, ^{t} < .10.

Table 2.

Multilevel models predicting time-varying PSQI scores by HF-HRV reactivity and trait worry

	Model 1	Model 2	Model 3	Model 4	Model 5
Fixed effects coefficients (SE)					
Intercept	4.61 (.60)**	3.99 (.59)**	2.70 (1.70)	-.64 (2.04)	1.13 (1.76)
Program (Psych)	-.05 (.43)	.04 (.43)	.64 (.56)	.76 (.53)	.37 (.55)
Sex (female)	-.12 (.50)	-.11 (.50)	-.14 (.67)	-.46 (.65)	-.47 (.66)
Medication use (yes)	1.49 (.57)**	1.59 (.56)**	1.98 (.74)**	1.70 (.71)*	1.73 (.75)*
Work hours	.03 (.03)	.04 (.03)	.03 (.03)	.02 (.03)	.02 (.03)
Ethnicity (non-White)	-.06 (.44)	.02 (.44)	.48 (.58)	.67 (.55)	.58 (.56)
Resting HF-HRV			.12 (.25)	.30 (.22)	.19 (.24)
Time		.14 (.07)*	.68 (.43)	.34 (.50)	.66 (.44)
HF-HRVr			-.02 (.42)	3.46 (1.36)*	1.03 (.97)
PSWQ				.05 (.02)*	
Time × HF-HRVr			.21 (.10)*	-.59 (.33)†	-.21 (.24)
Time × PSWQ				.01 (.00)†	
HF-HRVr × PSWQ				-.07 (.03)**	
Time × HF-HRVr × PSWQ				.02 (.01)**	
FIRST					.10 (.04)*
Time × FIRST					.01 (.01)
HF-HRVr× FIRST					-.07 (.05)
Time × HF-HRVr × FIRST					.03 (.01)†
Random effects coefficients (SE)					
Intercept	3.71 (.62)	4.09 (1.09)	3.95 (1.06)	3.25 (.97)	3.47 (.99)
Slope		.17 (.07)	.13 (.07)	.07 (.06)	.11 (.06)
Residual	3.49 (.25)	3.02 (.25)	3.02 (.25)	3.02 (.25)	3.01 (.25)

Note. Coefficients are unstandardized. Two-way interactions between each covariate and time are also included in the models (data not shown). PSQI = Pittsburgh Sleep Quality Index; HF-HRVr = high frequency heart rate variability reactivity; PSWQ = Penn State Worry Questionnaire; FIRST = Ford Insomnia Response to Stress Test; Model 1 = unconditional means

model; Model 2 = unconditional growth model; Model 3 = HF-HRV worry reactivity × time model; Model 4 = HF-HRV worry reactivity × PSWQ × time model. *$p < .05$, **$p < .01$, $^{t} < .10$.

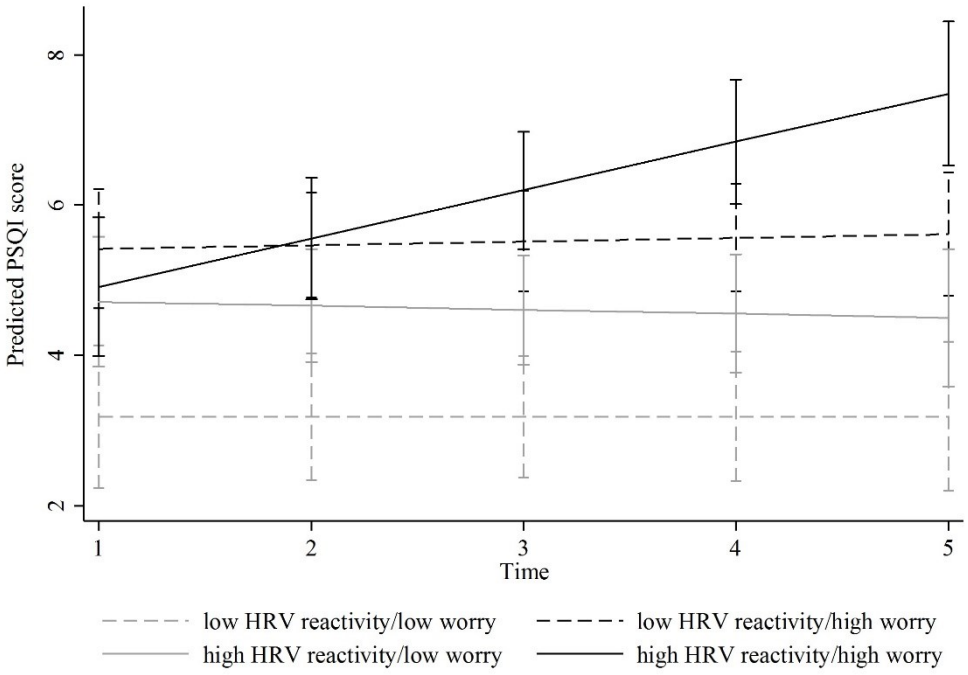

Figure 2. Interaction between HF-HRV reactivity and trait worry predicting sleep disturbances from a lower to higher academic stress period.

associated with change over time in PSQI scores. After Bonferroni correction, only the slope of change over time in PSQI scores for high HF-HRV reactivity with high PSWQ scores was found to be significantly different from zero (Table 3).

In Model 5, though there was a main effect of FIRST scores in predicting PSQI scores (b = .10, SE = .04, p = .01), the time × FIRST interaction was not statistically significant (p = .48; see Table 3). In Model 5, the time × HF-HRV reactivity × FIRST interaction was marginally significant, b = .03, SE = .01, p = .059. A pattern of results similar to the trait worry and HF-HRV reactivity interaction emerged with the combination of high sleep reactivity and high HF-HRV reactivity leading to the largest increases in sleep disturbances over time. Model 5 accounted for a total of 34.25% of the random slope variance in PSQI scores compared to Model 2. However, the time × HF-HRV reactivity × FIRST interaction was not statistically significant (b = .02, SE = .01, p = .18) when covariates were excluded from Model 5. Taken together, these results suggest that those with high HF-HRV reactivity to a worry-inducing task and high trait worry experienced greater increases in sleep disturbances during a period of higher academic stress.

Exploratory Analyses

An exploratory analysis was conducted to examine whether the time × HF-HRV reactivity × PSWQ and the time × HF-HRV reactivity × FIRST interactions were driven by some specific subscales of the PSQI. Models 4 and 5 were therefore conducted with each of the seven PSQI subscales as the outcome. For the time × HF-HRV reactivity × PSWQ interaction, we found that no single subscale was driving the effect found with the overall PSQI scores given that no model was statistically significant when conducted separately. For the time × HF-HRV reactivity × FIRST interaction, there was a statistically significant 3-way interaction with the sleep quality subscale (b = .01, SE = .001, p = .02) and the daytime dysfunction subscale (b = .01, SE = .004, p = .03). However, these exploratory analyses should be interpreted with caution given the number of statistical tests performed.

Discussion

The goal of this study was to examine whether HF-HRV reactivity, self-reported sleep reactivity, and trait worry predicted changes in sleep disturbances in response to increasing academic stress. As expected, there was an overall increase in stress from the lower to the higher academic stress period. There was an overall increase in self-reported sleep disturbances

Table 3.

Simple slopes analysis of the HF-HRV reactivity × PSWQ × Time interaction

	b	*se*	*p*
Low HF-HRV reactivity & low PSWQ	.00	.14	.99
High HF-HRV reactivity & low PSWQ	-.05	.13	.67
Low HF-HRV reactivity & high PSWQ	.05	.11	.67
High HF-HRV reactivity & high PSWQ	.64	.13	< .001

Note. Low and high HF-HRV reactivity correspond to 1 *SD* below (-.42) and above the mean (.94), respectively; low and high PSWQ scores correspond to 1 *SD* below (34.69) and above the mean (64.71), respectively.

associated with increasing academic stress, although individual differences in stress-related sleep disturbances were notable. HF-HRV reactivity to a worry induction during the lower-stress period predicted the development or exacerbation of sleep disturbances from periods of lower to higher academic stress. In addition, HF-HRV reactivity interacted with trait worry to predict individual differences in sleep disturbances. During the lower academic stress period, individuals with higher trait worry and higher HF-HRV reactivity evidenced the most sleep disturbances, compared to their counterparts with lower HF-HRV reactivity and/or lower trait worry. Longitudinally, simple slopes analysis revealed that only the individuals with higher trait worry paired with greater HF-HRV reactivity experienced increases in self-reported sleep disturbances during the higher academic stress period. A similar pattern of results was found for the interaction between sleep reactivity and HF-HRV reactivity, but the interaction was only marginally significant. Thus, the combination of higher trait worry and greater HF-HRV reactivity to worry might represent a profile of heightened vulnerability to the effects of stress on sleep disturbances.

In a previous, smaller study of individuals selected based on their high or low levels of sleep reactivity, HF-HRV during a worry induction predicted longitudinal increases in self-reported sleep disturbances in response to increased academic stress (Gouin et al., 2015). The present study replicates these results in a larger sample of participants that were not selected for sleep reactivity. During the lower stress period, the mean PSQI score was 4.58 (SD = 2.7), a score below the clinical cut-off for poor sleep. During the higher stress period, the mean PSQI score increased to 5.16 (SD = 2.81), a value above the clinical cut-off for poor sleep. Importantly, HF-HRV reactivity to the worry induction assessed during the lower stress period predicted longitudinal changes in sleep quality. That is, the magnitude of the association between HF-HRV reactivity and sleep quality increased from the lower stress to the higher stress periods, suggesting that HF-HRV reactivity forecasted the development or exacerbation of sleep disturbances during a naturalistic stressor. Current theoretical models have conceptualized HF-HRV as a marker of physiological, cognitive, and emotion regulation as well as a transdiagnostic risk factor for psychopathology (Beauchaine & Thayer, 2015; Porges, 2007; Thayer & Lane, 2009). These results provide further evidence that HF-HRV reactivity might also be a vulnerability factor for stress-induced sleep disturbances.

The combination of high HF-HRV reactivity to worry and high trait worry led to the

largest increases in stress-related sleep disturbances. Although this study was not designed to evaluate putative mechanisms of the relationship between HF-HRV and stress-related sleep disturbances, past literature suggests potential processes underlying this association. Bonnet & Arand (1997) argue that individuals with higher baseline physiological arousal have more difficulties sleeping, and that further increases in arousal may exacerbate sleep disturbances. Lower HF-HRV has been associated with increased physiological arousal in response to stress, including larger and longer-lasting elevations in heart rate, diastolic blood pressure, cortisol, and tumor necrosis factor-α (Souza et al., 2007; Weber et al., 2010), and greater startle response to unpleasant stimuli (Melzig, Wieke, Hamm, & Thayer, 2009). Further, lower HF-HRV and higher HF-HRV reactivity are related to greater anxiety and depression in response to stress in cross-sectional (Fortunato & Harsh, 2006; Friedman, 2007; Jonsson, 2007; Kemp et al., 2010), experimental (Ingjaldsson, Laberg, & Thayer, 2003; Kogan, Allen, & Weihs, 2012; Rottenberg, Salomon, Gross, & Gotlib, 2005) and prospective (Gentzler, Santucci, Kovacs, & Fox, 2009; Gouin et al., 2015; Gouin, Deschenes, & Dugas, 2014) studies. Furthermore, individuals with higher trait worry are known to develop greater physiological and cognitive arousal in response to stress (Verkuil, Brosschot, de Beurs, & Thayer, 2009). Together, individuals with both higher trait worry and higher HF-HRV reactivity to worry seemingly exhibit a particular risk profile wherein they experience the largest increases in physiological, emotional, and cognitive arousal. This state of hyperarousal might then interfere with sleep onset and maintenance (Riemann et al., 2010).

Individuals with high HF-HRV reactivity and high trait worry may not only experience more cognitive and emotional arousal, but may have greater difficulty regulating their emotions and disengaging from repetitive negative thoughts. Cross-sectional studies have demonstrated that individuals with more self-reported emotion regulation difficulties tend to have lower resting HF-HRV (Williams et al., 2015) and slower HF-HRV recovery following negative emotions (Berna & Nandrino, 2014). HF-HRV also seems to modulate the effectiveness of emotion regulation strategies. Individuals who exhibited low resting HF-HRV with large HF-HRV reactivity to a sad film clip experienced more symptoms of depression in response to maladaptive emotion regulatory strategies compared to those with higher resting HF-HRV and HF-HRV reactivity (Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013; Yaroslavsky et al., 2016). Similarly, among individuals with low resting HF-HRV, thought suppression strategies

were associated with smaller declines in negative intrusions compared to individuals with high HF-HRV (Gillie, Vasey, & Thayer, 2015). These studies suggest that HF-HRV reactivity may moderate the efficacy of emotion regulation strategies. Importantly, a breadth of research shows reciprocal effects between emotion regulation and sleep, such that daytime emotion regulation difficulties can disrupt sleep, which can further impair emotion regulatory processes, especially in the context of elevated stress (Gruber & Cassoff, 2014). Further experimental studies are needed to test these potential mechanisms.

Individuals with greater sleep reactivity exhibited greater sleep disturbances during the lower stress period. Furthermore, sleep reactivity interacted with HF-HRV reactivity to predict changes in sleep disturbances over time. Individuals with both high sleep reactivity and high HF-HRV reactivity showed the largest increases in PSQI scores during the higher academic stress period. Research has shown that individuals reporting greater sleep reactivity experience greater physiological arousal (Drake, Richardson, Roehrs, Scofield, & Roth, 2004), are more vulnerable to stress-related cognitive intrusions (Drake, Pillai, & Roth, 2014), and are at greater risk for developing insomnia in response to major life events (Drake, Richardson, Roehrs, Scofield, & Roth, 2004; Jarrin, Chen, Ivers, & Morin, 2014; Peterson, Kecklund, D'Onofrio, Nilsson, & Akerstedt, 2013). Although only marginally significant, these results suggest that HF-HRV reactivity to a worry induction magnifies the impact of self-reported sleep reactivity on vulnerability to stress-induced sleep disturbances.

Past research has observed a significant association between resting HRV and sleep quality (Castro-Diehl et al., 2016; Jackowska, Dockray, Endrighi, Hendrickx, & Steptoe, 2012). In contrast, there was no significant association between resting HF-HRV and PSQI scores at Time 1 ($r = .01$, $p = .99$) in the present study. This is surprising given that in a smaller sample drawn from the same population we found a much stronger and negative association between resting HRV and PSQI (Gouin et al., 2015). However, in this last study, participants were selected for high and low sleep reactivity. Meta-analyses have highlighted that the association between resting HF-HRV and adaptive functioning was higher in clinical or at risk samples, compared to community samples (Graziano & Derefinko, 2013; Sharhrestani, Stewart, Quintana, Hickie, & Guastella, 2015). Healthy undergraduate students may represent a group with a restricted range of sleep quality and HF-HRV, diminishing the likelihood of observing the hypothesized associations between the two constructs. This highlights the importance of

replicating these findings in a clinical population of individuals with insomnia.

Strengths, Limitations, and Future Directions

The longitudinal design of this study whereby HF-HRV reactivity assessed at baseline was used to predict prospective changes in sleep disturbances provides confidence in the directionality of the relationship between HF-HRV and self-reported sleep disturbances. This is important given that sleep deprivation has also been shown to lead to decreases in HF-HRV (Dettoni et al., 2012; Glos, Fietze, Blau, Baumann, & Penzel, 2014; Zhong et al., 2005). Academic stress is an ecologically-valid paradigm allowing the examination of individual differences in stress reactivity to a relatively standardized naturalistic stressor. However, academic stress is a relatively controllable and predictable stressor that is time-limited. Future research should examine whether HF-HRV reactivity and trait worry predict changes in sleep disturbances in response to chronic or uncontrollable stressors and challenges. Indeed, in a laboratory study, HF-HRV was found to predict greater responses to unpredictable rather than predictable stressors (Gorka et al., 2013). Further, HF-HRV naturally fluctuates during sleep, with increases in HF-HRV observed during deep sleep and decreases in HF-HRV observed during REM sleep (Busek, Vankova, Opavsky, Salinger, & Nevsimalova, 2005). Considerable evidence has found stress to be associated with altered physiological arousal not only during the day, but also during sleep (Brosschot, van Dijk, & Thayer, 2007; Riemann et al., 2010; Sanford, Suchecki, & Meerlo, 2015). Future studies should use actigraphy or polysomnography to obtain objective measurements of sleep disturbances and to assess changes in night-time arousal. Although individuals self-reporting chronic medical conditions were excluded from the study, a number of participants were using over-the-counter medications. In this study, over-the-counter medication use was associated with poorer sleep quality at baseline. It is possible that some of these medications may indicate the presence of an underlying acute illness or may have been used for their hypnotic properties. Future studies should better assess the potential confounding effect of over-the-counter medication use on stress-related sleep disturbances. Furthermore, given the a priori hypotheses regarding vagal-dependent parasympathetic activity, the present study did not include measures of sympathetic activity. Future studies should assess sympathetic activation to examine whether the effect observed in the present study is specific to parasympathetic activity, or whether they interact (Erath & El-Sheikh, 2015). Additionally, the predominantly female sample and the limited statistical power to test three-way interactions

should be noted as limitations to this study. Finally, this study was conducted with undergraduate sample who were not seeking treatment for, or diagnosed as having a sleep disorder. Future studies should assess whether these results would replicate in a population of individuals with chronic insomnia.

In summary, this study suggests that high trait worry and high HF-HRV reactivity are markers of vulnerability to stress-induced sleep disturbances. The combination of high trait worry and high HF-HRV reactivity to worry might help identify a subgroup of individuals more vulnerable to stress that may benefit from preventive stress management, worry, and/or sleep interventions.

General Discussion

In this study, individuals with both high trait worry and greater HF-HRV reactivity to a worry induction were at increased risk for the development of sleep disturbances during a period of higher academic stress. According to the Neurovisceral Integration Model (Thayer & Lane, 2000), resting HF-HRV indexes the top-down inhibitory influence of cortical and subcortical structures on cardiac activity, promoting energy conservation and adaptability. In turn, HF-HRV reactivity represents the inhibition of the prefrontal cortex to stress, leading to top-down activation of sympatho-excitatory influences and withdrawal of parasympathetic influences upon the heart for context-appropriate resource mobilization (Thayer & Lane, 2000; Thayer & Lane, 2009). Although some have demonstrated that decreases in HF-HRV under physical stress is an adaptive process associated with the normal physiological response to threat (Bernston & Cacioppo, 2004), others have shown that HF-HRV decreases in response to emotional stressors in particular are related to maladaptive outcomes (Beauchaine, 2015). This study provides further support that excessive HF-HRV reactivity to a worry induction constitutes a risk factor for the development of stress-induced sleep disturbances.

Activation of the stress response to emotional stressors is thought to be ineffective in the absence of situational demands requiring energy mobilization (Gouin et al., 2014). Specifically, excessive HF-HRV reactivity to personally-relevant worry may indicate the deployment of physiological, behavioural, and affective resources which are incongruent with the external context. For individuals with high trait worry and greater HF-HRV reactivity to a worry induction, exposure to repeated worry states activates the stress response. . It is known that chronic activation of the stress response due to emotional stress leads to pathogenic outcomes

(Sapolsky, 2014). As such, HF-HRV reactivity to worry may indicate vulnerability to psychopathology via a pathway of context-inappropriate sensitivity of the stress response to worry.

The Hyperarousal Model of Insomnia assumes an interplay between excessive affective, cognitive, and physiological arousal in the onset and maintenance of sleep disturbances (Riemann et al., 2010). In addition, this model states the importance of inhibiting processes associated with wakefulness for sleep onset and maintenance (Riemann et al., 2010). The Neurovisceral Integration Model (Thayer & Lane, 2000) also highlights the important role of inhibitory processes for adaptive and context-appropriate responding. Excessive HF-HRV reactivity to worry may represent deficits in one's physiological capacity to inhibit stress responses in the absence of objective threat. Similarly, high trait worry has been associated with poor attentional control and difficulty inhibiting cognitive intrusions or disengaging from negative automatic thoughts (Friedman & Miyake, 2004; Gillie, Vasey, & Thayer, 2015). As such, the combination of both high trait worry and greater HF-HRV reactivity to worry may index deficits in physiological and cognitive-emotional inhibitory feedback loops under stress, leading to a state of hyperarousal that promotes sleep disturbances by impairing inhibition of wakefulness at bedtime. When inhibitory feedback loops are compromised, this may lead to the development of positive feedforward loops which promote perseveration and continued activation of systems (Thayer & Lane, 2000). Thus, individuals with high trait worry and greater HF-HRV reactivity to their worry episodes may enter a cyclical feedforward pattern whereby worry events and over-activation of the stress response interfere with inhibitory processes required for sleep by perpetuating physiological and emotional arousal.

Seeing as a large proportion of the population presents with clinical (approximately 10%) and sub-clinical (approximately 40%) symptoms of insomnia (Morin et al., 2011), it is important to further determine the clinical utility of HF-HRV as a biomarker for vulnerability to stress-induced sleep disturbances. Further research should consider the best practices for and standardization of the measurement of HF-HRV with the goal of developing a valid and reliable clinical index of typical and atypical resting HF-HRV and HF-HRV reactivity. Additionally, whereas the current study provides evidence for the role of HF-HRV reactivity to personally-relevant worry as a risk factor for the development of stress-induced sleep disturbances, research has found differential associations between psychopathology and HF-HRV reactivity as a

function of the type of stimuli used to assess HF-HRV reactivity. For instance, HF-HRV reactivity to sadness and fear inductions have been related to internalizing symptomatology (Boyce et al., 2001; Cho, Philbrook, Davis, & Buss, 2017; Fortunato et al., 2013; Gouin et al., 2014; Hinnant & El-Sheikh, 2009; Kovacs et al., 2016;Rottenberg et al.,2007), but not HF-HRV reactivity to happiness or anger inductions (Fortunato et al., 2013). In contrast, blunted HF-HRV reactivity to happiness inductions have been associated with externalizing symptoms (Hinnant & El-Sheikh, 2009), as well as greater HF-HRV reactivity to cognitive and social stressors (Beauchaine et al., 2001). HF-HRV reactivity to fear and sadness inductions were not associated with externalizing symptoms independently (Cho et al., 2017; Fortunato et al., 2013), but have been associated with comorbid internalizing and externalizing symptoms (Pang & Beauchaine, 2013). Given these symptom-specific associations, future research is needed to better understand the clinical relevance of the type of stimuli used to elicit HF-HRV reactivity for specific psychological disorders. This line of research can further lead to the development of interventions targeted towards enhancing emotional regulation and reducing context-inappropriate stress response.

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