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Worried sleep: 24-h monitoring in high and low worriers

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ABSTRACT

Background: Commonly used trait measures might not accurately capture the relationship between worry and sleep difficulties in real life.
Methods: In a 24-h ambulatory monitoring study, high and low trait worriers maintained a log of worry and sleep characteristics while actigraphy, heart rates (HR), skin conductance (SC), and ambient temperature were recorded.
Results: Worrying in bed on the night of the recording was associated with longer self-reported and actigraphic nocturnal awakenings, lower actigraphic sleep efficiency, higher HR, lower HR variability, elevated SC level, and more non-specific SC fluctuations compared to not worrying in bed. High trait worriers had higher HR during waking and sleep, and reported shorter total sleep time and poorer sleep quality.
Conclusions: While trait worry is mainly associated with subjective sleep difficulties, worrying in bed impairs sleep according to both subjective and objective sleep parameters, including heightened sympathetic and reduced parasympathetic activation.

1. Introduction

Research relating worrying and insomnia has focused on establishing a link between worry and sleep-onset latency (SOL), finding either a positive association (e.g., Fuller, Waters, Binks, & Anderson, 1997; Harvey, 2000; Nicassio, Mendlowitz, Fussell, & Petras, 1985; Omvik, Pallesen, Bjorvatn, Thayer, & Nordhus, 2007; Wicklow & Espie, 2000) or no association (Carney, Harris, Moss, & Edinger, 2010; Hall, Buysse, Reynolds, Kupfer, & Baum, 1996; Watts, Coyle, & East, 1994). A smaller, less conclusive body of research often showed worry to be positively related to the number of nocturnal awakenings (Fuller et al., 1997; Nicassio et al., 1985), wake time after sleep onset (Åkerstedt, Kecklund, & Axelsson, 2007; Coyle & Watts, 1991; Jansson & Linton, 2006), and daytime dysfunction (Nicassio et al., 1985); and negatively related to sleep efficiency (Åkerstedt et al., 2007; Omvik et al., 2007; Wicklow & Espie, 2000), sleep quality (Smith, Perlis, Smith, Giles, & Carmody, 2000), and total sleep time (Gross & Borkovec, 1982; Jansson & Linton, 2006; Kelly, 2002; Nicassio et al., 1985). Nothing has been reported on early morning awakenings. Inconsistencies in the available research may be caused by whether trait or state estimations of worry were assessed, and whether sleep was measured subjectively and/or objectively.

1.1. Trait vs. state worry

Trait and state measures reflect different aspects of worry. Trait measures, derived from questionnaires and interviews, rely on memory recall, which depends on multiple cognitive processes including retrieving and averaging historical data over time (Piasecki, Hufford, Solhan, & Trull, 2007) and can be biased by beliefs. On the other hand, state worry measures derived from diaries, directly assess experience and behavior in the immediate or near immediate present.

Even if trait measures were faithful representations of average past worry, an average often fails to predict worry at a given future time, for example during monitoring. Trait worry may not account for the larger part of variance in daily worry (Verkuil, Brosschot, & Thayer, 2007) nor trait intrusive thinking explain intrusive thoughts at sleep onset or during nocturnal awakenings (Hall et al., 1996). An experimental design optimal for sleep research should assess state worry, both immediately before sleep and during nocturnal and early morning awakenings.

1.2. Autonomic changes

Worry is reflected to some extent by sympathetic activation, indexed for example by an increase in skin conductance (SC); by

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parasympathetic deactivation, indexed by heart rate variability (HRV) in the respiratory frequency range, also known as respiratory sinus arrhythmia (Berntson et al., 1997); or by both as in heart rate (HR). Higher *state* worry during the day has been consistently associated with higher HR and lower HRV during waking (Brosschot & Thayer, 2003; Brosschot, Van Dijk, & Thayer, 2007; Pieper, Brosschot, Van der Leeden, & Thayer, 2007, 2010), even 2 h after the worry was reported (Pieper et al., 2010), and in the ensuing sleep period (Brosschot et al., 2007; Yoshino & Matsuoka, 2009). This has led some authors to postulate "unconscious stress-related cognition" (Pieper et al., 2010, p. 570) or "unconscious worry" (Brosschot et al., 2007, p. 45). Electrodermal activity has been generally ignored in the context of state worry. *Trait* worry and HR and HRV during sleep were investigated by only one ambulatory study known to the authors which found them not to be associated (Brosschot et al., 2007). Unfortunately, the effects of body movement on cardiac activity were not accounted for. During waking periods inside and outside the laboratory, high trait worriers (HWs) usually have higher HR (Brosschot et al., 2007; Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004; Knepp & Friedman, 2007; Thayer, Friedman, & Borkovec, 1996) and lower HRV (Brosschot et al., 2007; Delgado et al., 2009; Hoehn-Saric et al., 2004; Lyonfields, Borkovec, & Thayer, 1995) than low trait worriers (LWs). State worry may not have occurred in studies that did not find these differences (e.g., Borkovec, Robinson, Pruzinsky, & DePree, 1983; Hoehn-Saric, McLeod, & Zimmerli, 1989; Wilhelm, Trabert, & Roth, 2001). Waking SC levels are usually unaffected by trait worry (Hoehn-Saric et al., 2004; Hoehn-Saric et al., 1989; Wilhelm et al., 2001), although an ambulatory study found significantly reduced SC variance in patients with Generalized Anxiety Disorder compared to non-worried controls (Hoehn-Saric et al., 2004). The relative lack of electrodermal abnormalities in HWs is in stark contrast to the numerous findings of heightened electrodermal arousal in anxiety (see Doberenz, Roth, Wollburg, Breuninger, & Kim, 2010) perhaps because worry is somewhat distinct from other manifestations of anxiety.

An independent line of research implicates abnormal sympathetic and parasympathetic activation in insomnia. Chronic primary insomnia has been labeled a "disorder of 24-h hyperarousal" (Riemann et al., 2010, p. 29); Espie's psychobiological model of insomnia (Espie, 2002) posits a failure to "de-arouse" during the transition from wakefulness to bedtime. HR is higher in a pre-sleep period (Haynes, Adams, & Franzen, 1981; Nelson & Harvey, 2003) and at sleep-onset (Freedman & Sattler, 1982) in people with sleep-onset insomnia. People with chronic insomnia have increased HRs and decreased high-frequency power of HRV during all sleep stages compared to normal sleepers (Bonnet & Arand, 1998). Unlike HR, measures of SC have failed to distinguish people with insomnia from good sleepers (Freedman & Sattler, 1982) although palmar SC is consistently much lower in sleep than in waking (Doberenz, Roth, Wollburg, Maslowski, & Kim, 2011). Vgontzas and colleagues have found evidence for chronic activation of the HPA axis among those with insomnia (Vgontzas et al., 2001; Vgontzas et al., 1998). In addition to these findings, there is evidence that those with insomnia might have a different neurocognitive profile characterized by elevations in high-frequency (i.e., beta/gamma) EEG activity (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997).

1.3. Hypotheses

This study was designed to investigate relations between worry and sleep more comprehensively than previous studies. First, state worry in bed was assessed not only before sleep-onset but also during nocturnal and early morning awakenings. Second, ambulatory measures of sympathetic and parasympathetic activation were combined with actigraphy to assess waking and sleeping levels of activation and rate of de-arousal in the participants' natural environment. Instead of polysomnography, we relied on actigraphy, which gives reasonably valid measures (Lichstein et al., 2006), although it has been found to overestimate sleep in people who sit or lie awake without moving (De Souza et al., 2003).

We hypothesized that only higher *state* worry on the monitoring night would affect objective sleep parameters for that night, delaying physiological de-arousal after sleep onset, and increasing HR and decreasing HRV during sleep. High *trait* worry, on the other hand, would only be associated with *self-reported* prolonged sleep-onset latency (SOL), an increased number and duration of nocturnal awakenings, lower sleep efficiency and sleep quality, and shorter total sleep time in the past month.

2. Methods

2.1. Participants

Thirty-nine high worriers (HWs) and 16 low worriers (LWs) were recruited from the local community with the following advertisement in flyers and on the internet: "How much do you worry? Do you find it easy to dismiss worrisome thoughts? OR Do you worry all the time? Would you like to find a way of dealing with your worries?" A total of 157 people completed the phone screening that included the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and screened for high worriers (PSWQ total score $\geq$ 56, as recommended by Ruscio, 2002) and low worriers (PSWQ total score $\leq$ 42, see Davis, Montgomery, & Wilson, 2002). Excluded was any participant who reported shift work, a history of severe cardiovascular, lung, or neurological disease, uncontrolled thyroid problems, sleep apnea or other sleep disturbances, or who showed signs of cognitive impairment during the phone screening or in direct interactions with the interviewers. Further exclusion criteria were substance abuse or dependence in the past year and a history of, or current, DSM-IV psychotic disorder as diagnosed by the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 2001). Healthy controls were selected not to have a current diagnosis or history of any anxiety or mood disorder. Eligible participants were allowed to continue on stable doses of prescription medicines, but were excluded if they reported taking psychoactive drugs, sleep medications, or drugs with substantial peripheral effects. Seventy-four people were eligible of which 57 decided to participate in the study. Two participants were excluded after the first 24-h monitoring because of reported use of benzodiazepines and sleep medication during the recording period, resulting in a total of 39 high worriers (HW) and 16 low worriers (LW). Participants received monetary compensation for being tested ($50–75 USD).

2.2. Procedure

This investigation was carried out in accordance with the Declaration of Helsinki and was reviewed and approved by the Stanford Institutional Review Board and the VA Palo Alto Veterans Affairs Research Compliance Office. Eligible individuals were invited to an appointment where they gave written informed consent for further assessment. The assessment began with two interviews, the SCID (First et al., 2001) and a sleep interview based upon the research diagnostic criteria for insomnia (Edinger et al., 2004). If no grounds for exclusion were found, participants were asked to wash their hands with soap in preparation for the application of the SC electrodes (Dawson, Schell, & Filion, 1990; Venables & Christie, 1980) and the monitoring devices were connected. Next, participants underwent laboratory testing where they performed posture exercises (lying down, standing, walking) and a worry induction (quiet sitting, relaxing, quiet sitting, worrying, quiet sitting), the results of which will be reported elsewhere.

After the laboratory session, participants were introduced to the electronic diary and reminded not to drink more than one glass of alcohol during the following 24 h. They were instructed to wear the monitoring devices continuously until they returned the next day. Length of recording ranged between 19:04 and 25:03 with a mean of 22:05 (h:min). During monitoring, subjects were prompted by an alarm to fill out a short electronic diary on a handheld device every 2 h during waking. The handheld could also be used to report (a) going to sleep and waking up (including nocturnal awakenings and daytime naps), (b) taking medications, drinking alcohol or caffeinated beverages, or smoking a cigarette, (c) physical exercise, and (d) sensor detachment and replacement. Upon returning to the laboratory the next day, participants filled out questionnaires on a laboratory computer.

2.3. Physiological and actigraphic data

2.3.1. Assessment

Physiological data were recorded with a customized 3-channel ambulatory digital recorder (3991x/3-SIT BioLog, UFI, Moro Bay, CA, USA) worn in a waist pack. Channels were (1) skin conductance (SC) measured by applying 0.5 V DC to