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Poor sleep quality and exaggerated salivary cortisol reactivity to the cold pressor task predict greater acute pain severity in a non-clinical sample

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ABSTRACT

Poor sleep is often independently associated with greater pain sensitivity and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis (e.g., greater basal cortisol and exaggerated stress-induced cortisol reactivity). However, the interactions among sleep, pain, and the HPA axis have not been adequately evaluated. In this study, 40 healthy adults provided self-report regarding perceived sleep quality over the past month prior to completion of an acute noxious physical stressor (i.e., cold pressor task; CPT). Following the CPT, they reported on the severity of pain experienced. Salivary cortisol was sampled before, immediately following, and during recovery from CPT. Using bootstrapped confidence intervals with a bias correction, results showed that poor sleep quality was significantly associated with greater reports of CPT-induced pain severity and greater cortisol reactivity (i.e., increase from baseline). Furthermore, greater cortisol reactivity to the CPT was found to significantly mediate the relationship between poor sleep and pain severity.

1. Introduction

Over recent years it has become increasingly evident that sleep quality is highly predictive of pain experiences as demonstrated in laboratory and clinical settings (Smith and Haythornthwaite, 2004). Specifically, research examining clinical pain reports and the responses of individuals exposed to controlled laboratory stimuli has documented reliable relations between poor sleep quality and increased pain severity (Raymond et al., 2001; Mystakidou et al., 2009; Edwards et al., 2009). At present, the mechanisms by which poor sleep quality exerts its nocent effects on the experience of pain have not been fully characterized, although the hypothalamic-pituitary-adrenal (HPA) axis has been proposed as a potential mediator of this relationship (Canivet et al., 2008). The HPA axis and its constituent neurohormones, particularly cortisol, are commonly examined in studies as an index of neuroendocrine stress reactivity. Previous research testing whether sleep quality predicts cortisol responses to stress has predominantly involved psychosocial stressors such as public speaking or mental stress test (Wright et al., 2007; Raikkonen et al., 2010). Acute pain represents a noxious physical stressor that also has been shown to elicit significant cortisol responses (Goodin et al., 2012), yet no studies to date have addressed whether sleep quality predicts cortisol reactivity to a noxious stressor and the resultant pain response. A direct examination of whether cortisol reactivity to a noxious stressor mediates the relationship between sleep quality and reports of pain severity may help to elucidate the physiological mechanisms linking poor sleep with pain sensitivity and is warranted at this time.

It has been revealed that poor sleep is directly associated with increased basal activity of the HPA axis, and it has further been suggested that poor sleep may potentiate the reactivity of this system to threat and challenge (Vgontzas and Chrousos, 2002; Buckley and Schatzberg, 2005; Meerlo et al., 2008). Support for this suggestion was provided in a recent review that reported robust relationships between poor sleep quality and subsequent dysregulation of the cortisol response to various stressors (Balbo et al., 2010). In particular, poor sleep quality has been shown to predict exaggerated cortisol responses to psychological stressors (Raikkonen et al., 2010) and physiological stressors (Hori et al., 2011); however, it remains to be determined whether poor sleep also predicts cortisol response to a noxious physical stressor.

That poor sleep seems to promote exaggerated cortisol responses to stress is particularly relevant here because the HPA axis and cortisol have previously been found to be implicated in pain perception. In laboratory-based studies of healthy adults, it has been demonstrated that exposure to a cold pressor task (CPT) resulted in a significant increase in salivary cortisol from baseline,

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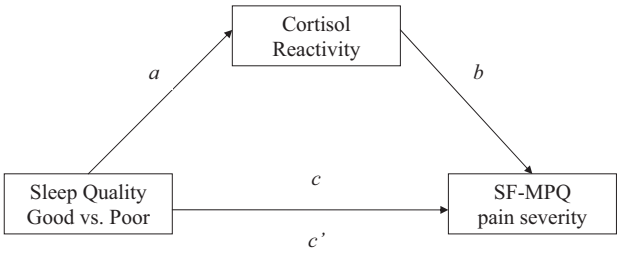

Fig. 1. Putative study model.

and this increase was significantly related with greater reports of pain intensity and pain unpleasantness (Zimmer et al., 2003; Goodin et al., 2012). Further, HPA axis activation (e.g., increased cortisol) has been associated with elevations in patient-related pain severity in samples with chronic widespread pain (Neeck and Riedel, 1999; Neeck, 2000). However, it is noteworthy that some previous experimental and clinical studies found inverse relationships between cortisol and pain, such that greater basal cortisol was associated with less severe pain (al'Absi et al., 2002) and diminished cortisol reactivity was associated with greater pain perception (Geiss et al., 1997). Thus, it appears that additional research is needed to further elucidate that nature of the relationship between cortisol and pain.

On balance, there is preliminary and indirect support for the view that poor quality sleep, by acting on stress systems like the HPA axis, may sensitize individuals to the experience of pain. However, it appears that no previous studies have evaluated whether sleep quality predicts aberrant HPA axis-related responses (i.e., cortisol) to a noxious physical stressor, and, in turn, whether cortisol response is related to reports of pain. Using a cold pressor task (CPT) and questionnaires, we tested three hypotheses. First, poor sleep quality will be significantly related with greater reported pain following the CPT. Second, poor sleep quality will also be significantly related with an increased cortisol response to the CPT. Third, the relationship between poor sleep quality and greater reported pain will be significantly mediated by the increase of cortisol in response to the CPT. Fig. 1 displays our putative study model.

2. Methods

2.1. Participants

Participants were 40 healthy adults, recruited from a college campus using posted advertisements, and individuals of both sexes were eligible for study enrollment. The sample was predominantly young adults (mean age = 20.2 ± 2.8 years old; range 18–24), with an equal number of men and women (50% women). Mean body mass index was 22.93 ± 3.28, which falls within the "ideal weight" range as determined by the National Institutes of Health (NIH, 1998). The majority indicated their race as either non-Hispanic white (35%) or Asian, Pacific Islander (35%), with the remainder being African American (25%) or of Hispanic decent (5%). Individuals were unable to participate if they met any of the following criteria: (a) age less than 18 or over 45 years; (b) ongoing chronic pain problems; (c) diagnosed sleep disorder or taking medication for sleep; (d) circulatory disorders; (e) history of cardiac events; (f) history of metabolic disease or neuropathy; (g) pregnant; (h) currently using prescription analgesics, tranquilizers, antidepressants, or other centrally acting agents; (i) use of nicotine, (j) use of prescription medication (e.g., corticosteriods, oral contraceptives), (k) psychiatric disorders (e.g., depression), or (l) chronic or acute health problems that affect the neuroendocrine or immune system. This study was carried out in accordance with the University's appropriate guidelines for ethical conduct of research. Informed consent was obtained in accordance with approved protocol guidelines of an Institutional Review Board. All participants were compensated for their participation.

2.2. Procedures

Prior to the laboratory session, participants were asked to not use nonprescription medications or alcohol within 24 h of their appointment. Participants were asked to refrain from exercise and consumption of caffeine for at least 2 h prior to the testing session. To minimize potential error associated with the collection of oral fluid samples, participants were asked to not eat foods that may cause bleeding of the gums (e.g., potato chips) or brush their teeth for at least 2 h prior to the testing session. This is because blood leakage from microinjuries of the oral mucosa may confound the measurement of salivary cortisol (Kivlighan et al., 2004). All study procedures were carried out between the hours of 4 P.M. and 7 P.M. to control for diurnal variations in neuroendocrine parameters and because afternoon sessions have been associated with greater cortisol responses (Dickerson and Kemeny, 2004).

Upon arrival to the study site, participants rested comfortably in a chair for 15 min to adapt to the experimental setting. During this time participants completed behavioral and psychological questionnaires that assessed perceived sleep quality and negative affect. Participants then provided a saliva sample for cortisol assessment (initial sample). The initial sample was intended to familiarize participants with the saliva collection procedures and is not included in data analysis. Participants then rested an additional 15 min and subsequently provided a second saliva sample (baseline sample) that was collected prior to the initiation of the CPT. Additional salivary cortisol samples were collected immediately following termination of the CPT and at various intervals during recovery (15, 20, 25, 30 and 40 min following initiation of the CPT). These sampling time-points were chosen based on a meta-analysis of prior research showing that peak changes in cortisol occur at approximately 30 min following stressor onset (Dickerson and Kemeny, 2004). Also following completion of the CPT and cortisol sampling, participants completed a short questionnaire describing their pain experiences.

2.3. Acute pain stressor

2.3.1. Cold pressor task

The CPT procedure is a psychophysiological pain test that involved a NESLAB RTE-10 liter water bath (Thermo Electron Corporation, Portsmouth, New Hampshire) filled with circulating cold water maintained at approximately 4 °C (±0.2 °C). Participants were instructed to place their dominant hand into the cold water up to their wrist. In an effort to maximize participants' exposure to the CPT and promote a corresponding cortisol response, standardized instructions asked participants "please try to keep your hand immersed in the water for at least 2 min or we may not be able to use your data". However, participants were then immediately informed that they could remove their hand from the water at any time should it become intolerable. Unbeknownst to participants, the maximum allowable duration of the CPT was 300 s. While prior research has used different cutoff times, our 300 s cutoff is consistent with many previous studies (Walsh et al., 1989). Whether participants completed the entire CPT, or terminated the task prior to the allotted maximum time of exposure, the duration of exposure was recorded and classified as cold pressor pain tolerance (CPTo).

2.4. Questionnaires

2.4.1. Pittsburgh Sleep Quality Index (PSQI)

Sleep quality was measured before completion of the CPT using the PSQI. The PSQI is a self-rated questionnaire that retrospectively assesses sleep quality and disturbances over a one month time interval (Buysse et al., 1989). Nineteen individual items generate seven "component" scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. Each of the seven component scores is weighted equally on a scale from 0 to 3, 0 indicating no difficulty and 3 indicating severe difficulty. The sum of scores for these seven component scores yields one global score, ranging from 0 to 21. Higher scores indicate worse sleep quality, and a global PSQI score > 5 is consistent with poor sleep quality. The seven component scores of the PSQI have previously been shown to possess good internal consistency (α = .83), and the overall global score has demonstrated good test-retest reliability (r = .87) (Buysse et al., 1989.) In the current study, internal consistency for the PSQI components was acceptable (α = .75)

2.4.2. Positive and Negative Affect Schedule (PANAS)

Negative affect was also measured prior to the CPT using the negative affect subscale of the Positive and Negative Affect Schedule (Watson et al., 1988). Given the positive relationship between negative affect and pain reports (Staud et al., 2006), this subscale was included to examine the influence of general negative affect on key study variables and determine the need for statistical control. The negative affect subscale (PANAS-neg) includes 10 negative affects (e.g., distressed, upset), and participants were asked to indicate on a five-point Likert scale (1 = not at all, 5 = very much so) the strength of the emotion for them. The total negative affect score for each participant was the sum of the 10 items, with a possible range of 10–50. The PANAS has good psychometric properties (Watson et al., 1988) and the internal consistency of the scale was adequate in the current study (α = 0.77).

2.4.3. Short Form-McGill Pain Questionnaire (SF-MPQ)

The Short-Form McGill Pain Questionnaire (SF-MPQ) allows quantitative, multidimensional pain ratings to be obtained in a brief period of time (Melzack, 1987). In the current study, respondents rated 15 pain descriptors on a scale from 0 (none) to 3 (severe) following the CPT and cortisol sampling, and a sum of all rankings was used to compute a total pain rating score. The SF-MPQ is a reliable and valid instrument commonly used in clinical and research applications (Melzack, 1987). The instructions used in the current study asked participants about "the painful procedure you