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Poor sleep the night before an experimental stress task is associated with reduced cortisol reactivity in healthy women

Caroline E. Wright *, Heiddis B. Valdimarsdottir, Joel Erblich, Dana H. Bovbjerg

Biobehavioral Medicine Program, Department of Oncological Sciences, Mount Sinai School of Medicine, Box 1130, 1425 Madison Avenue, New York, NY 10029, USA

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Abstract

Sleep disruption is a growing problem that may have serious health effects. As stress-induced increases in cortisol are thought to be a key adaptive process it is important to examine how this response is affected by sleep. The current study investigated the association of four sleep parameters (objective/subjectively measured sleep quality and quantity) and subsequent salivary cortisol reactivity (maximal change from baseline) to an experimental stressor in 53 healthy women. Objective actigraphy monitoring and self-report diaries were used to assess sleep. Results revealed that individuals with lower objective sleep quality (wake percentage during sleep) had a blunted response to the experimental stressor. No associations were found between cortisol reactivity and actigraphy-derived sleep quantity, or either of the self-reported sleep variables. Results are discussed with regard to the possible adverse health effects that may result from poor sleep quality and a blunted cortisol response to stress.

Keywords: Actigraphy; Cortisol reactivity; HPA axis; Sleep; Stress; Stroop task; Wake

1. Introduction

Decreased time available for sleep and/or increased sleep disturbance is often associated with a demanding life style and is a growing problem in many Western societies, particularly for women (National Sleep Foundation, 2005; Rajaratman and Arendt, 2001; Soares, 2005). Sleep disturbances resulting from increased workload and other challenges imposed by modern society may present a serious threat to health and well being. There is evidence to suggest that disturbed sleep is associated with increased risk of future disease, such as cardiovascular and coronary heart disease, cancer, and an increased risk of all-cause mortality (Ayas et al., 2003; Hansen, 2001; Heslop et al., 2002; Taskar and Hirchowitz, 2003). Since stress reactivity contributes to health and adaptation to challenging environments, it is important to know how this system is affected by sleep.

The hypothalamic-pituitary-adrenal (HPA) axis plays an important role in a variety of functions that enable an individual to deal with environmental challenges (Van Reeth et al., 2000) as well as the regulation of sleep. Indeed, studies in both humans and animals have demonstrated strong bidirectional relationships between alterations in sleep and regulation of the HPA axis. A large literature has explored the effect of the HPA axis response on subsequent sleep patterns (e.g., Buckley and Schatzberg, 2005; Steiger, 2002), but less work has investigated the association between sleep and subsequent HPA axis activity. The secretion of cortisol by the adrenal is characterized by a spontaneous release over the day (marked by a diurnal pattern with an early morning peak) and by a marked increase in secretion in response to acute stress (stress-induced cortisol reactivity). Some studies have suggested that sleep disturbances are related to an attenuated cortisol awakening response (early morning peak) the following morning, which may in turn have adverse health consequences (e.g., Backhaus et al., 2004; Persson Waye et al., 2003); however, little is known about the association of poor sleep and subsequent cortisol reactivity to acute stress. Studies exploring the effect of sleep disturbance and HPA axis reactivity to acute stressors in rats have found that sleep restriction, as well as total sleep deprivation, prior to novel stressors significantly reduces adrenocorticotropic hormone (ACTH) responses (e.g., Meerlo et al., 2002; Sgoifo

* Corresponding author. Tel.: +1 212 659 5504; fax: +1 212 849 2566.
E-mail address: caroline.wright@mssm.edu (C.E. Wright).

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et al., 2005) although no differences in corticosterone levels were found. A recent experimental study of humans by Capaldi et al. (2005) revealed blunted cortisol reactivity to an experimental stressor among participants with poorer self-reported sleep quality in comparison to those with better sleep quality, although self-reported sleep quantity (hours of sleep) was not related to cortisol reactivity.

Additional research on the relationships between sleep and subsequent HPA axis stress reactivity would be valuable for at least two reasons. First, theorists suggest that the increase in secretion of cortisol under stressful conditions may be initially advantageous for health, providing protection for the individual by increasing energy expenditure and decreasing immune function; however, it is also postulated that a blunted cortisol response to stress may, over time, become detrimental due to excessive activity in bodily defenses (e.g., inflammatory or immune responses) (Fries et al., 2005; Heim et al., 2001). Blunted cortisol responses (possibly acting via inadequate inhibition of inflammatory or immune pathways) have been reported to be predictive of increased sensitivity to pain (Logan et al., 2001), and have been associated with inflammatory diseases including rheumatoid arthritis, multiple sclerosis and osteoporosis (Raison and Miller, 2003). Second, although there has been a wealth of research over the past three decades identifying various factors that can alter human stress responsivity in experimental settings (Dickerson and Kemeny, 2004; Kirschbaum and Hellhammer, 1994; Kudielka et al., 2004a,b; Kudielka and Kirschbaum, 2005), to our knowledge there has only been one previous study (Capaldi et al., 2005) examining the effects of sleep disturbance on subsequent stress-induced cortisol reactivity.

Although the findings of Capaldi et al. (2005) are important, fundamental issues remain to be explored. First, it is not yet known whether the relationship between poor sleep and subsequent cortisol reactivity may be stronger when sleep is assessed the night immediately preceding the acute stressor. Second, objective measurements of sleep, which have been found to be more reliable than self-reported sleep data (Carney et al., 2004) have not yet been examined in relation to cortisol reactivity. Polysomnography is considered the 'gold standard' method for objectively measuring sleep; however, wrist-watch style actigraphy devices, which distinguish sleep from wake based on validated night time movement levels, are considered less burdensome and have been validated as providing objective measurements of sleep quantity and quality that can be collected in the home environment (Ancoli-Israel et al., 2003; Sadeh and Acebo, 2002; Tryon, 1996). Third, sleep quality is reported to be a better predictor of health, quality of life and fatigue in comparison with a basic measure of sleep quantity (e.g., Pilcher et al., 1997). Examination of both sleep quantity and quality in relation to cortisol reactivity is therefore warranted.

The purpose of the present study was to investigate, for the first time, the relationships between objective sleep quantity and quality, measured the night before an experimental stressor, and cortisol responsivity to a standard laboratory challenge the following day. In addition, the study aimed to examine a number of secondary issues highlighted by the previous literature. First, we explored whether the relationships between sleep disturbance and stress-induced cortisol reactivity would be stronger if sleep data collected over the week before (in addition to data from the night before) were included in the analyses. Second, several factors that could account, potentially, for the relationship between sleep and cortisol reactivity (task performance, task appraisal, pre-task fatigue levels, and perceived stress prior to the task) was explored.

In the present study sleep quality and quantity was assessed by actigraphy (objective) and self-report diary (subjective) data collected in the home environment. The primary study hypothesis was that participants with poorer sleep quality, measured the night before an experimental stressor, would have attenuated stress-induced cortisol reactivity, in comparison to participants with higher quality sleep.

2. Methods

The study had two main components. First, participants had a preliminary meeting (Session 1) with study personnel to familiarize themselves with the laboratory setting and with the sleep assessment procedures to be followed for the following 7 days (e.g., daily sleep diaries and continuous actigraphy monitoring). Second, at the end of the 7-day interval there was an experimental component (Session 2) in which participants' psychobiological responses to a laboratory stress task (Stroop) were assessed.

2.1. Participants

Sixty-four healthy women who worked at a major medical center in New York were recruited as part of a larger study investigating psychophysiological responses to stress. Women were excluded if they were not English speaking, had a history of HIV, heart disease, cancer, depression or were taking any prescription medication other than hormonal birth control, were pregnant or had irregular menstrual cycles. Participants were only included if they were free from symptoms of infection (including cold, fever, flu or cold sores) and had successfully completed actigraphy monitoring and daily diaries for all 7 days prior to experimental assessment. Of the 64 women, actigraphy data were not analyzable for six participants due to technical problems with the actigraph device at some time over the week, while a further five women were excluded because they did not provide complete saliva samples sufficient for analyses. Primary analyses were therefore conducted on a final sample of 53 women with a mean age of 37.3 years (±9.9). All participants provided written informed consent. The five excluded women did not differ on any of the demographic measures presented in Table 1 in comparison with those 53 participants who were included in the final analyses.

2.2. Mental stress task

The stress task involved a battery of six Stroop color-word interference trials plus one practice trial (stimulus categories: color, negative, positive, neutral, cancer, heart disease) previously described by Erblich et al. (2003), using a computerized presentation method (Monk et al., 2001). Participants completed a practice trial first, followed by the color Stroop trial. During the standardized color-word Stroop trial, participants were presented with a target word (e.g., green, red, blue, yellow) printed in an incongruent color in the center of a 17 in. color computer screen. Participants were then asked to match the displayed color of the target word with four options at the bottom of the screen using designated computer keystrokes. The remaining trials were presented in a counterbalanced Latin-square randomized order, following an identical procedure. The presentation of stimulus words within trials was programmed to increase in speed when participants performed well in order to maintain the challenging nature of the task constant throughout the testing period. During all trials except the practice, participants heard a recorded voice through headphones stating the names of the four colors in random order, but not