



Impact of menstrual cycle phase on endocrine effects of partial sleep restriction in healthy women

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Summary There is extensive evidence that sleep restriction alters endocrine function in healthy young men, increasing afternoon cortisol levels and modifying levels of other hormones that regulate metabolism. Recent studies have confirmed these effects in young women, but have not investigated whether menstrual cycle phase influences these responses. The effects on cortisol levels of limiting sleep to 3 h for one night were assessed in two groups of women at different points in their menstrual cycles: mid-follicular and mid-luteal. Eighteen healthy, young women, not taking oral contraceptives (age: 21.8 ± 0.53 ; BMI: 22.5 ± 0.58 [mean $\pm$ SEM]), were studied. Baseline sleep durations, eating habits and menstrual cycles were monitored. Salivary samples were collected at six times of day (08:00, 08:30, 11:00, 14:00, 17:00, 20:00) during two consecutive days: first after a 10 h overnight sleep opportunity (Baseline) and then after a night with a 3 h sleep opportunity (Post-sleep restriction). All were awakened at the same time of day. Women in the follicular phase showed a significant decrease ($p=0.004$) in their cortisol awakening responses (CAR) after sleep restriction and a sustained elevation in afternoon/evening cortisol levels ($p=0.008$), as has been reported for men. Women in the luteal phase showed neither a depressed CAR, nor an increase in afternoon/evening cortisol levels. Secondary analyses examined the impact of sleep restriction on self-reported hunger and mood. Menstrual cycle phase dramatically altered the cortisol responses of healthy, young women to a single night of sleep restriction, implicating effects of spontaneous changes in endocrine status on adrenal responses to sleep loss.

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1. Introduction

A number of population-based studies, both cross-sectional and longitudinal, have found that short sleep duration is associated with an increased risk for metabolic disorders such as obesity, the metabolic syndrome and Type 2 diabetes mellitus (Hasler et al., 2004; Choi et al., 2012; Kita et al., 2012). Laboratory-based studies involving either partial or total sleep deprivation for periods ranging from one to several nights have demonstrated acute changes in appetite and endocrine function. These have been interpreted as possible mediators of the impact of chronic short sleep on the increased risk for development of metabolic disorders (Leproult et al., 1997; Spiegel et al., 1999, 2004a; Wu et al., 2008; Markwald et al., 2013). Specifically, sleep loss has been reported to alter the levels of hormones, including cortisol (which affects glucose metabolism), leptin and ghrelin (which contribute to appetite regulation) (Morselli et al., 2012). In addition, sleep restriction has been shown to increase self-reported hunger and preferences for calorie-dense foods, and to disrupt carbohydrate metabolism (Spiegel et al., 1999, 2004a; Buxton et al., 2010). These changes have been reported to occur in the absence of increased tension or anxiety, which were considered to be likely markers of a nonspecific stress response to sleep loss (Spiegel et al., 2004b).

The adrenal hormone cortisol, which regulates many aspects of human physiology, can be measured from most bodily fluids, including saliva, which contains unbound cortisol (Kalman and Grahn, 2004). Under normal sleep/wake conditions, cortisol levels rise from low values during sleep to high values at the time of awakening in the morning, followed by a secondary rise ~30–45 min after awakening, known as the cortisol awakening response (CAR) (Pruessner et al., 1997). Following this morning peak, there is a steady decrease throughout the day to reach daily trough levels by approximately 12 h after awakening (Edwards et al., 2001; Wust et al., 2000; Taheri et al., 2004). Sleep loss alters this daily rhythm by causing a sustained elevation in afternoon cortisol levels, which has been linked to serious metabolic consequences, such as impaired lipid and glucose metabolism and increased risk of insulin resistance and metabolic syndrome (Plat et al., 1999; Whitworth et al., 2005; Anagnostis et al., 2009; Buxton et al., 2010).

Most laboratory studies of the endocrine effects of sleep loss have included only young male participants, probably because of the assumption that menstrual cycles in young women would complicate analyses of results. Studies of menstrual phase effects on spontaneous daily cortisol rhythms have yielded inconsistent findings, depending on their methodology. Results have included delays (Parry et al., 1994) or advances (Parry et al., 2000) of the daily cortisol rhythm during the late luteal phase, relative to the follicular phase, no change in morning cortisol values between the follicular and late luteal phases (Steiner et al., 1999; Kudielka and Kirschbaum, 2003), and no change in the timing and amplitude of cortisol rhythms between the follicular and luteal phases of the cycle (Bloch et al., 1998).

A few studies have examined the impact of sleep loss on cortisol rhythms in women (Patel et al., 2006; Omisade et al., 2010). A post hoc analysis of the self-reported

menstrual cycles of participants in one of these studies suggested that menstrual phase could have modulated the impact of sleep loss. Dividing participants into those studied soon after the end of menstruation (presumed follicular phase) and those studied later (presumed luteal phase) indicated that the latter participants showed less change in cortisol rhythms after sleep loss. Women taking oral contraceptives showed an intermediate response. Because the subgroup sizes were small (3–4) and cycle timing involved only self reports, this post hoc analysis was not included in the original publication (Omisade et al., 2010).

The present study was designed to address this question directly by measuring the Baseline patterns of salivary cortisol levels during waking after a night with a 10 h sleep opportunity in young women during either their mid-follicular or mid-luteal phase, and comparing cortisol patterns to those after a single night with sleep restricted to a 3 h opportunity. Studies reporting that sleep restriction increased hunger ratings involved men exclusively, whereas those that showed no effect have included women (Spiegel et al., 2004a; Schmid et al., 2008; Brondel et al., 2010). Of the studies involving women only, no changes in hunger were reported after 4 nights of progressive sleep loss (Bosy-Westphal et al., 2008) or one night with a 3 h sleep opportunity (Omisade et al., 2010). The possibility that menstrual cycle phase could modulate effects of sleep loss on appetite and on mood changes has not been studied. Therefore, secondary outcome measures in this study included participants' self-reports of mood and hunger ratings.

2. Methods

2.1. Participants

Participants met the following inclusion criteria: self-identified female, 19–25 years of age, BMI of 18–30 (actual range recruited was 18–24.5, except for one in the luteal group with a BMI of 29.1), non-smoker, habitual daytime activity (i.e., no shift work), regular menstrual cycle, no travel over three or more time zones within 6 weeks prior to the study and habitual continuous sleep of 6–9 h nightly without frequent naps. Participants completed a Morningness–Eveningness Questionnaire; anyone with MEQ scores greater than 69 (extreme Morningness) or less than 31 (extreme Eveningness) were excluded (Horne and Ostberg, 1976). Exclusion criteria included a history of eating disorders; affective disorders (including post-traumatic stress disorder); chronic stress, anxiety or depression symptoms in the clinical range; sleep disorders; or ongoing hormone treatments, including hormonal birth control of any kind less than 3 months prior to taking part in the study. Professional or semi-professional athletes were excluded due to the potential for altered menstrual cycles resulting from intense training regimes.

The study was reviewed and approved by the Capital District Health Authority Research Ethics Board (Halifax, Nova Scotia, Canada) in accordance with the Canadian Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans. Potential participants were recruited by word-of-mouth and using notices and online advertising. All participants demonstrated understanding of the study

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