



Full-length Article

Repeating patterns of sleep restriction and recovery: Do we get used to it?



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ABSTRACT

Despite its prevalence in modern society, little is known about the long-term impact of restricting sleep during the week and 'catching up' on weekends. This common sleep pattern was experimentally modeled with three weeks of 5 nights of sleep restricted to 4 h followed by two nights of 8-h recovery sleep. In an intra-individual design, 14 healthy adults completed both the sleep restriction and an 8-h control condition, and the subjective impact and the effects on physiological markers of stress (cortisol, the inflammatory marker IL-6, glucocorticoid receptor sensitivity) were assessed. Sleep restriction was not perceived to be subjectively stressful and some degree of resilience or resistance to the effects of sleep restriction was observed in subjective domains. In contrast, physiological stress response systems remain activated with repeated exposures to sleep restriction and limited recovery opportunity. Morning IL-6 expression in monocytes was significantly increased during week 2 and 3 of sleep restriction, and remained increased after recovery sleep in week 2 ($p < 0.05$) and week 3 ($p < 0.09$). Serum cortisol showed a significantly dysregulated 24 h-rhythm during weeks 1, 2, and 3 of sleep restriction, with elevated morning cortisol, and decreased cortisol in the second half of the night. Glucocorticoid sensitivity of monocytes was increased, rather than decreased, during the sleep restriction and sleep recovery portion of each week. These results suggest a disrupted interplay between the hypothalamic-pituitary-adrenal and inflammatory systems in the context of repeated exposure to sleep restriction and recovery. The observed dissociation between subjective and physiological responses may help explain why many individuals continue with the behavior pattern of restricting and recovering sleep over long time periods, despite a cumulative deleterious physiological effect.

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1. Introduction

Patterns of restricting sleep during the week and 'catching up' over the weekend are prevalent in modern society (Hansen et al., 2005; Monk et al., 2000; National Sleep Foundation, 2010; Tsui and Wing, 2009; Wing et al., 2009). These sleep patterns are not commonly thought of as deleterious; however, there is limited empirical evidence to support this belief. Given the wealth of accumulated evidence that insufficient sleep is associated with elevated health risks (e.g., cardiovascular disorders (Grandner et al., 2013), metabolic disorders (Knutson et al., 2007), and chronic pain conditions (Finan et al., 2013)), gaining a better understanding of the impact of these common sleep patterns is essential.

Sleep loss can be conceptualized a physiological stressor, with both subjective (psychological) and physiological effects (described further below). The multiple systems involved in the physiologic stress response are homeostatic and tightly inter-related (Almawi et al., 1996; de Kloet, 2000) and include the sympatho-adrenal, the hypothalamic-pituitary-adrenal (HPA), as well as the inflammatory system. Inflammatory cytokines serve as chemical messengers and are negatively controlled by cortisol, a glucocorticoid (GC) that is the main output hormone of the HPA axis (reviewed in Chrousos, 2009). Impaired GC sensitivity has been reported in response to various acute and chronic stressors (Herman et al., 1995; Miller et al., 2002; Stark et al., 2001), and GC sensitivity is one possible mechanism by which observed increases in inflammatory markers can be explained.

The HPA system is perhaps the most studied stress response system, and is known to typically habituate when faced with repeated or ongoing stressors (Grissom and Bhatnagar, 2009).

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However, sleep loss is a unique stressor because it is a biological resource necessary for regulation of multiple physiological systems, including the stress response system (Hamilton et al., 2007; McEwen, 2006). Further, in extreme cases, sleep is necessary for survival itself (Everson et al., 1989; Montagna et al., 1995). No prior research has examined whether humans can adapt to chronic patterns of insufficient sleep and limited recovery, or studied the impact of this common real-world pattern on stress-response systems. As described below, the impact of single episodes of sleep loss and (to a lesser extent) recovery sleep has been tested, however it remains unknown whether these results remain true when patterns of restricted sleep and recovery become chronic.

Within single episodes of experimental sleep loss, subjective ratings of sleepiness, positive mood, and self-reported physical functioning appear to show response stabilization, or acclimation. For example, subjective experiences of pain (Haack and Mullington, 2005) and sleepiness (Van Dongen et al., 2003) stabilize after a few days of sleep restriction or sleep deprivation (or deteriorate more slowly), despite ongoing sleep loss. On a physiological level, multiple markers of the stress system have been found to increase following a single episode of sleep loss, including cortisol (Balbo et al., 2009; Guyon et al., 2014) and the inflammatory marker interleukin [IL]-6 (Haack et al., 2007; Irwin et al., 2006; Pejovic et al., 2013; van Leeuwen et al., 2009; Vgontzas et al., 2004). Although habituation to acute stressors is a key feature of the HPA system (Grissom and Bhatnagar, 2009), it is unknown whether this classic pattern of habituation can be applied to the physiological stress of repeated exposures to sleep loss with limited recovery sleep, given that sleep loss is a unique physiological stressor.

Little is known about the impact of repeated episodes of sleep loss or the role of recovery sleep. To our knowledge, the current study protocol tests the longest model of chronic sleep restriction to date. Everson and colleagues have conducted studies of repeated exposure to sleep loss and recovery in an animal model, and have documented changes in metabolic indices (weight, food intake), and pathological organ and bone changes (Everson and Szabo, 2009, 2011). Recovery from sleep loss has been rarely studied, but using a five night sleep restriction/two night recovery protocol, van Leeuwen and colleagues showed that IL-6 mRNA levels remained elevated after two nights of recovery sleep (van Leeuwen et al., 2009). These data provide preliminary support that 'catching-up' on sleep over the weekend might be insufficient to restore stress-response systems, and contribute to ongoing responses to repeated exposure to sleep loss over time. These limited data highlight a critical gap in our understanding of consequences of insufficient sleep, as it is the real-world experiences of repeated episodes of sleep loss and limited recovery sleep that are most likely to have a long term impact on health.

This study modeled real-world sleep-wake patterns of sleep restriction and recovery in the laboratory environment to investigate effects on multiple stress system markers, using an intensified model of sleep restricted to four hours of sleep on weekdays and extended to eight hours on weekends. This amplification of the magnitude of difference between weekdays/weekends was chosen in part due to the aim of assessing the impact of these patterns under highly controlled experimental conditions that can be maintained for a period of weeks, rather than the months or years that adults often will maintain these milder patterns of sleep restriction and recovery in the real world.

Based on previous research, we hypothesized that there would be a response stabilization or habituation across repeated episodes of sleep loss in subjective domains, but poor habituation and an incomplete recovery in physiological domains. If true, these findings could help explain why patterns of inadequate sleep persist, namely, because there would be no perceived negative impact of

these behavior patterns. Additionally, this study was specifically designed to extend previous research demonstrating that sleep loss results in increases in serum or plasma IL-6 (Haack et al., 2007; Irwin et al., 2006; Pejovic et al., 2013; van Leeuwen et al., 2009; Vgontzas et al., 2004) by focusing on monocytes, and whether the expected increased expression of inflammatory mediators can be explained by changes in the sensitivity of monocytes to cortisol.

2. Methods

2.1. Experimental model

The hypothesis was tested using a sleep restriction condition consisting of three weeks of a repeating pattern of five nights of sleep restricted to 4 h/night (0300–0700 h) followed by two nights of recovery sleep with 8 h/night (2300–0700 h). This model was designed to mirror commonly observed patterns of moderately restricting on weeknights and recovering sleep on weekend nights that often occur in the general population for periods of months or years (National Sleep Foundation, 2010), albeit with an amplified sleep restriction pattern on weeknights (see Fig. 1). This amplified sleep restriction period was designed to maximize the potential that the effects of what are often much longer periods of milder sleep restriction and recovery that occur in the real world could be captured in a relatively short three-week in-laboratory experimental protocol. The sleep control condition consisted of three weeks with a nightly sleep opportunity of 8 h. In an intra-individual randomized balanced design, participants underwent two 25-day in-hospital stays (restricted sleep condition and sleep control condition) separated by more than two months. Each 25-day stay started with an adaptation and a baseline day, followed by three weeks of either the repeated exposure to sleep restriction/recovery or control sleep, and ended with an additional night of full sleep (totaling 25 days).

2.2. Participants

This study was approved by the Institutional Review Board for the Protection of Human Subjects at the Beth Israel Deaconess Medical Center (BIDMC). Participants were recruited via community advertisements. Seventeen healthy young women and men were studied. Fourteen participants completed both 25-day-in-hospital conditions; three participants could only complete one of the two 25-day-in-hospital conditions due to change in work/family-related requirements (see Fig. 2).

Participants were between the ages of 18–35 years, had a body mass index (BMI) between 18.5 and 30 kg/m², a daily sleep duration between 7 and 9 h (verified by sleep diary data over a 2-week period), began their habitual sleep period within one hour of 11 pm (to ensure normal entrainment) and had blood chemistry levels within the normal range. Female participants were eligible if they had regular menstrual cycles and no significant discomfort during pre-menses/menses. Exclusion criteria included presence or past history of major medical problems, psychiatric disorders or sleep disorders. Additional exclusion criterion included pregnant/nursing status, regular medication use other than oral contraceptives, and donation of blood or platelets three month prior to or in-between study stays.

2.3. Study protocol

2.3.1. Screening & randomization

Participants were initially screened over two visits to the hospital and were evaluated by a study physician for the exclusion

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