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Determinants of cortisol awakening responses to naps and nighttime sleep

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ABSTRACT

The cortisol awakening response (CAR) is a phenomenon describing the sharp increase in basal cortisol levels shortly after waking from sleep. While extensively studied, little is known about the role of sleep architecture contributing to CAR. Furthermore, the potential for CAR after a shorter bout of sleep – a nap – has not been directly investigated. The current studies thus aimed at assessed sleep duration, time of day, and sleep architecture as potential determinants of the cortisol awakening response.

Saliva samples were collected during the first hour (0, 30, 45, 60 min) following several EEG-monitored laboratory sleep conditions. Those included afternoon naps wherein 17 participants (4 men; ages 18–26) napped for 50 min and 24 participants (11 men; ages 18–24) napped for 90 min. Furthermore, 20 participants (10 men; ages 18–35) visited the lab twice and in addition to staying overnight, napped 90 min in the morning either under placebo conditions or pharmacologically-manipulated sleep conditions (5 mg Zolpidem).

Cortisol increases were observed in response to each sleep condition except to 50-min afternoon naps. Furthermore, CARs were predicted by Stage 2 sleep when following nighttime sleep ($r = .46$, $p = .04$) and by Stage 1 sleep when following placebo morning naps ($r = .54$, $p = .01$).

The current study established cortisol awakening responses to naps and implicates sleep duration and architecture in the generation of CAR to both napping and nighttime sleep. Assessing CAR in conjunction with the specific type of sleep may thus contribute to our understanding of mechanisms underlying positive and negative health effects of napping.

1. Introduction

Cortisol, the end hormone of the hypothalamus-pituitary-adrenal (HPA) axis, is secreted in a 24-h pulsatile circadian rhythm (Buckley and Schatzberg, 2005; Gronfier and Brandenberger, 1998). This basal cortisol rhythm encompasses the cortisol awakening response (CAR), a sharp increase over the first 30–45 min of awakening from sleep. First described in the early 1990s (e.g., (Pruessner et al., 1997)), the CAR is considered a reliable measure of HPA axis activity (Schmidt-Reinwald et al., 1999). However, the CAR is distinct from the circadian variations in HPA axis activity as it is associated with the process of awakening (Wilhelm et al., 2007). Much research has aimed at determining factors that may explain inter-individual differences in the CAR. For example, gender, as well as psychological factors such as depression, stress, and anxiety are associated with integrated volume of cortisol released between wake and peak levels of the CAR (Chida and Steptoe, 2009), while findings regarding the role of demographic and physical health factors such as age, weight or smoking habits are less consistent (Fries et al., 2009; Pruessner et al., 1997; Wust et al., 2000). One factor of particular interest to the generation of the CAR is sleep.

The transition from sleep to wake is essential for the occurrence of the CAR, but results regarding the effects of sleep-related factors, such at time of awakening and sleep duration, have been inconsistent (Federenko et al., 2004; Wilhelm et al., 2007; Wust et al., 2000). Additionally, while sleep is generally defined as the hours spent in bed at night, shorter bouts of daytime sleep, or naps, are common in individuals of all ages (Dinges, 1992). Though shorter in duration and occurring during the day, naps are a form of sleep, raising the question whether naps are followed by cortisol awakening responses as well. Studies reporting post-nap cortisol data

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are sparse and usually do not focus on defining the relationship between shorter periods of sleep and CARs. For example, Vgontzas et al. (2007) found that after a night of sleep deprivation, a 2-h afternoon nap (1400–1600 h) resulted in a prolonged increase in cortisol levels in the hours post nap (1600–2300 h) that was significantly higher than in sleep-deprived participants who did not nap (Vgontzas et al., 2007). However, in a study by Federenko et al. (2004), students who were asked to take an hour-long nap in the early evening (awakening: 1845–2030 h) did not show a cortisol increase (Federenko et al., 2004). These studies indicate the possibility that the circumstances in which napping occurs may influence the generation of a post-nap cortisol increase.

Several factors potentially explain why some nap conditions, but not others, could be related to increases in cortisol following waking. It may be that nap duration plays a role wherein 60 min (Federenko et al., 2004) might be too short but 90 min (Vgontzas et al., 2007) sufficient to induce a CAR. Another explanation related to both sleep duration as well as sleep pressure may be sleep architecture differences. Interestingly, sleep architecture is an aspect of sleep, which has not been investigated in relation to the generation of the CAR following nighttime sleep, much less following naps. Sleep architecture in naps generally follows the same course as night-time sleep, progressing first from light sleep into deeper sleep (Maron et al., 1964). However, time of day appears to affect the distribution of time spent in specific sleep stages, such that morning naps are predominated by rapid eye movement (REM) sleep, while naps taken later in the day have increasingly greater percentages of slow-wave sleep (SWS) (Karacan et al., 1970). Therefore, differences in time of day may influence cortisol levels after waking from a nap through the composition of sleep stages.

In summary, the phenomenon of a CAR occurring after shorter daytime sleep, or naps, has not been thoroughly investigated. Increasing our understanding of the relationship between nap sleep and post-awakening cortisol increases would provide insights into the physiological mechanisms underlying beneficial (Milner and Cote, 2009; Vgontzas et al., 2007) and detrimental effects (Foley et al., 2007) of napping on mental and physiological health.

The data available to date suggests that CAR to napping may be dependent on nap duration, time of day, and/or a factor related to both, nap sleep architecture. The current study aimed at investigating these factors. More specifically, Aim 1 was to assess the potential for a CAR following naps of different durations (50 min versus 90 min) compared to CAR following longer sleep, i.e., nighttime sleep. Aim 2 was to explore the role of time of day by comparing naps taken in the morning to naps taken in the afternoon. To disentangle sleep architecture effects from time of day effects, a pharmacological manipulation condition was added to mimic afternoon nap sleep architecture in morning naps. Lastly, Aim 3 was to assess sleep architecture effects on CAR across all sleep conditions by monitoring sleep using polysomnography (PSG).

2. Methods

To address the above research aims, data from two studies were assessed. Study 1 investigated afternoon naps of different lengths in a between-subject design, while Study 2 assessed nighttime sleep and two morning nap conditions in a within-subject design.

2.1. Participants

A total of 76 healthy, non-smoking adults without major medical problems were recruited through the University of California, San Diego (UCSD) Laboratory for Sleep and Chronobiology, located at the VA San Diego Healthcare System (VASDHS) in La Jolla, California. All participants were fluent English speakers who habitually slept 7–9 h every night with a habitual bedtime between 2200 h and 2400 h and a habitual wake time between 0600 h and 1000 h. Exclusion criteria were sleep disorders; history of diagnosed significant psychopathology, head injury, or substance dependence; seizures; and current use of psychotropic medications. This research was approved by the local IRB and conducted in accordance with the principles of the Declaration of Helsinki. All procedures were carried out with the adequate understanding and written consent of the participants.

For Study 1, 19 participants were assigned to a 50-min afternoon nap condition, while 28 participants were assigned to a 90-min afternoon nap condition. Within the 50-min nap condition, one participant was excluded for consistently elevated cortisol levels and one for missing cortisol samples. In the 90-minnap condition, three participants were excluded for consistently elevated cortisol samples while a fourth was excluded for missing cortisol samples. Hence, our final sample for analysis consisted of $N = 17$ participants (4 males; ages 18–26) for the 50-minnap condition and $N = 24$ participants (12 males; ages 18–24) for the 90-min nap condition.

Further, in Study 2, a total of 29 healthy adults participated in an overnight sleep condition followed by two morning nap conditions (placebo and Zolpidem). Eight individuals had to be excluded due to missing cortisol or polysomnography (PSG) data and one due to insufficient total nap sleep time (less than 40 min spent asleep). Hence, the final sample for the nighttime sleep and morning nap conditions consisted of $N = 20$ participants (10 males; ages 18–35).

2.2. Procedures

2.2.1. Study 1

Using a between-subjects design, participants reported to the Laboratory for Sleep and Behavioral Neuroscience at the VASDHS at 0900 h. At 1230 h, electrodes were applied for standard polysomnographic recording. Subjects were in bed by 1330 h and were then allowed up to two hours of time in bed or until they achieved either 50 or 90 min of sleep according to on-line polysomnography recording (Mednick et al., 2003). Following each nap, four saliva samples were collected at the same times relative to awakening as post nighttime sleep to assess potential cortisol awakening responses following naps.

2.2.2. Study 2

Participants arrived at the Laboratory for Sleep and Chronobiology two hours prior to bedtime, at approximately 2100 h. After electrodes were applied for standard polysomnographic recording, subjects were in bed by 2300 h and slept overnight with PSG monitoring. Participants were awoken by study staff at 0500 h and four saliva samples were taken 0, 30, 45, and 60 min post-awakening. As such, nighttime sleep duration for this study of 6 h is within the range of average reported sleep duration for young adult/student populations (Orzech et al., 2011; Pilcher and Ott, 1998; Tsai and Li, 2004). After 0600 h, participants were free to read and watch TV until 0830 h, at which time they went back to bed for a nap. Participants were allowed up to two hours of time in bed or until they achieved 90 min of sleep as scored on-line by polysomnography. This time of day was chosen for a nap to capitalize on circadian fluctuations in REM sleep, which is highest in the morning. Directly before lights out, participants received either 5 mg Zolpidem or a placebo (see below for more detail) in a double-blind, within-subjects design. Following each nap, another four saliva samples were collected at the same times relative to awakening as post night-time sleep. After 5–10 days assuring drug wash-out and recovery from any sleep changes related to the nap and/or study drugs, participants returned to the lab and those who received