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Impaired inhibition after total sleep deprivation using an antisaccade task when controlling for circadian modulation of performance ☆

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HIGHLIGHTS

- Inhibition after TSD when taking into account circadian modulation of performance.
- Inhibition assessed with antisaccade, go no-go and incompatibility tasks.
- Impaired antisaccade performance but no changes for the two neuropsychological tasks.
- Circadian modulation of performance may reveal or mask cognitive impairments.
- Task demands and resultant recruitment of cortical regions may explain the results.

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ABSTRACT

Objective: Sleep deprivation affects several cognitive functions subserved by the prefrontal cortex. Conflicting results have, nonetheless, been reported for inhibitory function, which could be explained by methodological bias. The present study aimed to assess the effects of sleep deprivation on response inhibition using a particularly suitable inhibition test, the antisaccade, while controlling for circadian influences on performance. For this purpose, testing was conducted at: (1) the same time of day in both the control and sleep deprivation conditions; and (2) at a time of day when inhibitory performance has been found not to be at its lowest level. Two other neuropsychological tasks (go no-go and incompatibility) were used for comparison.
Methods: Twelve healthy young participants performed the three tasks in the early afternoon after a normal night and after a total sleep deprivation (TSD) night in a study with a balanced, crossover design.
Results: TSD significantly impaired the error rate, the latency, and the intra-individual coefficient of variation of latency in the antisaccade task. None of these parameters were affected in the two neuropsychological tasks.
Conclusions: When circadian modulation of performance is controlled, TSD impairs inhibition assessed by an antisaccade test. This result emphasizes that it is crucial to control for circadian effects when assessing cognitive performance in TSD studies since the time of testing may reveal or mask cognitive and behavioral impairments. The discrepant findings obtained with the go no-go and incompatibility tests are probably explained by the specific task demands and differences in recruitment of prefrontal regions.

1. Introduction

Sleep debt is becoming increasingly common, especially in the most developed countries, and insufficient sleep is known to lead to serious adverse consequences in daily life such as motor vehicle accidents. In addition to the increased occurrence of "microsleep" episodes, impaired motor and cognitive performance has been implicated in these deleterious effects of sleep deprivation (see [1] for review). For several decades, numerous authors have aimed at understanding the mechanisms of effects of sleep deprivation on cognitive functions. According to Wilkinson [2], sleep deprivation alters performance exclusively via a reduction in "the nonspecific arousal level of the body", thus only when cognitive tasks are long and monotonous. This view has been challenged on the basis that some short-lasting and stimulating tests are also altered after sleep deprivation [3]. More specifically, Harrison [3] formulated a prefrontal cortex (PFC) vulnerability hypothesis because the most impaired tests are those known to involve the frontal cortex, such as working memory tasks or executive functions (see [4] and [1] for reviews).

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Inhibition is an important executive function, and its dysfunction is one of the most frequent consequences of PFC brain damage [5,6]. The inhibition process is a mechanism that actively suppresses reaction to distracting information that is in direct competition with the information relevant to the subject's goals [7]. Various tasks have been used to evaluate sleep deprivation effects on the inhibition process, but their results are conflicting (see [8] for a review). For example, deleterious effects of sleep deprivation were reported with the go no-go [9], Hayling [3,10], and "finding embedded figures" [11] tests, but not with the Stroop task [12–14] nor with an incompatibility [15] or a random letter generation [16] task, nor with antisaccade tasks [17–19]. However, it is difficult to compare the results, as study designs differ widely from each other. Among these discrepant designs is the time of the day at which tests were performed in each condition. Indeed, in some studies, performance in the sleep deprivation and in the control conditions were not tested at the same time of the day although circadian variations in performance have been reported for many tasks, including cognitive tasks (see [20] and [21,22] for reviews). These conflicting results could also be due to the fact that most of these neuropsychological tests involve both executive and non-executive cognitive functions [23], which may have influenced the resulting neural correlates of successful response inhibition.

Eye movements are particularly relevant in assessing mechanisms of attention and inhibitory control impairments, as the cortical and sub-cortical structures involved in eye movements partly overlap with the structures implicated in these cognitive processes (i.e., mainly frontoparietal and temporal brain regions for the visuospatial attentional oculomotor tasks [24–26] and dorso-lateral prefrontal cortex for the inhibitory oculomotor task [27–29]). Oculomotor tasks have previously shown their sensitivity in revealing cognitive impairment after sleep deprivation [30]. An interesting oculomotor task used to study inhibition is the antisaccade task [31]. In this task, the participants are asked to fix their gaze on a central stimulus until a peripheral visual stimulus is presented, and then to look to its mirror position in the opposite visual field. This task requires two main processes: (1) suppression (or inhibition) of a reflexive saccade towards the peripheral stimulus (prosaccade); and; (2) generation of a volitional saccade to the mirror position (antisaccade) [32–34]. A conflict occurs between the prepotent response (i.e., the reflexive saccade), which must be inhibited, and a volitional response (i.e., an antisaccade), which must be generated [34,35]. This saccadic paradigm is essentially a motor task and, unlike neuropsychological tasks, it engages a limited range of cognitive processes [24–26]. Increased error rate (i.e., production of prosaccades instead of the requested antisaccades), usually accounted for by a deficit in inhibition, has been found in numerous neurological and psychiatric disorders affecting the frontal cortex [32,36,37]. The antisaccade latency is another useful parameter since it provides information about the efficiency of the voluntary generation process. Consequently, the different parameters of eye movements can improve understanding of sleep deprivation effects. The three previous studies that used antisaccade tasks to evaluate cognitive inhibition after total sleep deprivation (TSD) did not reveal any impairment [17–19]. It is, however, possible that impaired performance could not be evidenced because the influence of circadian rhythms on inhibitory performance was not taken into account (see [21,22] for reviews). Indeed, Crevits' [17] and Gais' [19] studies compared, in the same subjects, the performance in the morning after TSD to those in the evening in the control condition. Circadian variation in performance could have hindered a putative sleep deprivation effect on the antisaccade results. Moreover, in the three previous studies, antisaccade performance in the control condition was assessed in the early morning, a time of day when inhibitory performance is at its lowest [38]. It is thus likely that degradation in performance after TSD was more difficult to detect than if testing in control conditions had been done at a time of day when performance of inhibitory tasks is better [38]. The primary objective of the present study was thus to investigate the effect of TSD on the inhibition process using an antisaccade task while controlling for circadian influences on performance. This was done: (1) by testing all participants at the same time of day, and in both the control and sleep deprivation conditions; and (2) by evaluating the performance at a time of day when inhibitory performance has been found not to be at its lowest level [38] in order to detect a decrease in performance in the sleep deprivation condition. We hypothesized that this experimental paradigm would provide evidence of impaired antisaccade performance after TSD.

A secondary objective of this study was to assess and compare, in the same experimental study, the inhibition of a prepotent response with saccadic eye movements and classic neuropsychological tests. For this purpose, we used the go no-go and incompatibility tasks, which share with the antisaccade task a common need to execute volitional action and inhibit a prepotent motor response. The versions of the neuropsychological tasks that we chose have been shown to be impaired in various pathologies, including patients with frontal dysfunctions [39–42].

2. Materials and methods

2.1. Participants

Twelve healthy undergraduate students (4 women and 8 men; age: 21.5 ± 1.3 years) were selected for this study. Screening excluded participants having sleep difficulties or regular naps during the day, and those who took medication that affected sleep or sleepiness. Participants suffering from neurological, psychiatric, cardiovascular, respiratory, hepatic, renal, or metabolic pathologies were also excluded. All participants reported habitually sleeping 7 to 9 h, were non-smokers, and had no history of alcohol abuse. All had normal or corrected-normal vision. The participants signed an informed consent form before the experiment and received financial compensation. The Caen Northwest III ethics committee approved this experiment (No. CCPPRB 2005-18).

2.2. Design

The study was conducted using a balanced, crossover design. The 12 participants underwent tests in both experimental conditions (i.e., normal night (NN) and TSD night) separated by a 2-week interval to avoid test–retest effects. Six participants started by the NN condition and 6 started by the TSD condition. Three days before each experimentation night, actigraphs were worn by the participants to verify that they respected a normal sleep-wake schedule with: (1) 7–9 h of sleep per night; (2) awakening between 7 and 8 AM, and; (3) abstinence from sports or naps 24 h prior to testing. Participants continued to wear the actigraphs during the nights (sleep deprivation and normal) and throughout the day in which they performed the inhibition tests. Sleep diaries were also completed in which participants recorded all their activities and hours of sleep. Before the sleep deprivation or normal night, participants arrived in the laboratory between 6 and 7 PM, completed a short practice session of an antisaccade paradigm for 10 min, and had a standardized dinner. The 2 sleep-deprived participants stayed in the laboratory all night and were supervised by experimenters. The 2 nonsleep-deprived participants were escorted home at 10 PM and retired at their usual bedtime (between 11 and 12 PM). They awoke between 7 and 7:30 AM and were brought to the laboratory by an experimenter. We chose to have participants slept at home as sleep in the laboratory is often disrupted and the quality of sleep is usually better at home than in a laboratory [43]. The 4 participants took a standardized breakfast together in the laboratory at 8:30 AM and were not allowed to drink caffeinated beverages. The antisaccade and neuropsychological tests were all performed in the early afternoon from 2 to 3 PM, after a standardized lunch. During both the night and the morning that preceded the tests, the participants were allowed to carry out quiet activities (e.g., reading, writing, conversing), while vigorous activities (e.g., exercise) or stimulating activities (e.g., electronic games) were prohibited. No food or beverages other than water were