

Sleep Deprivation Triggers Cognitive Control Impairments in Task-Goal Switching

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Abstract

Study Objectives

This study investigates the impact of sleep deprivation (SD) on task-goal switching, a key component of cognitive flexibility.

Methods

Task-goal switching performance was tested after one night of regular sleep ($n = 17$ participants) or of total SD ($n = 18$). To understand the relationships between task-switching performance and other cognitive processes following SD, participants were tested for other key attentional (alertness and vigilance) and executive (inhibition and working memory) functions. Spontaneous eye blink rate (EBR) was also measured as an indirect marker of striatal dopaminergic function.

Results

SD negatively affects task-goal switching as well as attentional and inhibition measures, but not working memory. Changes in task-goal switching performance were not significantly correlated with changes in objective and subjective markers of fatigue and sleepiness, response inhibition, or spontaneous EBR.

Conclusions

Altogether, our results show differentiated effects of SD on key executive functions such as working memory, inhibition, and task-goal switching.

[sleep deprivation](#), [task switching](#), [cognitive flexibility](#), [cognitive control](#), [dopamine](#), [eye blink](#)

Statement of Significance

This study is the first to evidence impact of sleep deprivation on task-goal switching, a key component of cognitive flexibility and cognitive control. Moreover, alterations in task-goal switching were not associated with changes in spontaneous eye blink rate as well as attentional and executive functions. These results are of significance because sleep debt is more and more present in modern society, and cognitive flexibility subtends rapid adaptation to constantly changing situations or environments, a condition usually encountered in everyday life.

Introduction

Cognitive control (also called executive functions or executive control [1]) refers to the mental ability to regulate thoughts and actions in accordance with internally represented goals [1–3]. Cognitive control allows individuals to define a goal, choose a strategy to achieve it, and monitor its execution [1]. Specifically, cognitive control enables an adaptation to novel situations when overlearned routines are insufficient. Cognitive flexibility is a crucial component of cognitive control that subtends rapid adaptation to constantly changing situations or environments, a condition usually encountered in everyday life [4].

Cognitive flexibility is usually investigated in laboratory conditions using task-switching paradigms (for reviews, see Refs. 5 and [6]) in which participants repeatedly switch between two tasks or more. For instance, participants can be presented with colored shapes and instructed to decide between colors (red vs. blue) or shapes (circle vs. square) according to a cue presented previously. Switching between tasks leads to more errors and increased processing time compared with performing the same task [6–8]. The additional time to respond (or higher error rate) in a shifting task condition is called a *switch cost*. It is usually computed by subtracting the mean reaction time (or error rate) for repeated conditions from the reaction time (or error rate) for switch conditions.

Importantly, task switching in itself is not a unitary process. It is rather a complex mechanism that involves several components. Imagine you are photocopying different documents. Some of them are printed double-sided but the others are single-sided and need to be printed onto a single sheet of paper. In addition, consider that the documents are mixed in a fixed order, which you cannot change (i.e., you cannot photocopy the double-sided documents first and then the others, meaning you have to switch between conditions). Succeeding in this situation need at least two distinct sets of operations. First, for each document, you have to keep in mind whether you are photocopying a double-sided document. In the task-switching literature, this stage has been labeled “task-goal activation.” [9, 10] Second, you have to program the settings of the photocopier to a “duplex printing” or a “convert to duplex printing” mode, according to the task goal. This stage has been labeled “task-rule activation” and requires retrieving the rules that allow achieving the goal (i.e., the photocopier instruction manual in this example) in long-term memory [9, 10]. In sum, for any given task, preparation requires setting a goal (“what to do?”; i.e., *task-goal activation*) followed by the activation of the rules (“how to do the task?”; i.e., *task-rule activation*). The task-rule activation stage depends on the complexity of the instructions. Indeed, in the example mentioned above, the task-rule activation depends on the complexity of the photocopier instruction manual. In simple experimental tasks like color and shape discrimination, the task-goal activation is the task to perform (e.g., color discrimination), and the task-rule activation stage is most often achieved by activating the necessary stimulus–response (S–R) mappings (i.e., the S–R instructions) to execute the task [9, 10]. For instance, a production rule for response selection in a color discrimination task might have the following form [10]:

“IF ((GOAL IS TO DO COLOUR-DISCRIMINATION TASK) AND (STIMULUS COLOUR IS RED)) THEN (PRESS RIGHT INDEX-FINGER KEY).”

As can be seen from this instruction set, correct task goal activation is needed to adequately implement the task rule. If you experiment problems or fail in one of these two stages, you will be slower or commit errors, particularly when you have to switch between tasks. However, you could fail because you struggle to retrieve the goal but apply the correct rule, or because you retrieve the correct goal but struggle to apply the rule. This is why the distinction between task-goal and task-rule is essential in the task switching literature. In a switching situation, *task-goal switching* refers to the first component, whereas *task-rule switching* refers to the second one. Rubinstein and colleagues [10] proposed that, during task-goal switching, the current goal is inserted into declarative working memory (WM), and the previous goal is deleted. They also suggested that, during task-rule switching, S–R rules for the current task are loaded into procedural WM. Therefore, a way to investigate the dissociation between task-goal and task-rule switching is to compare tasks involving a strong S–R mapping (memory) load and tasks involving a weak load or an absent one. For instance, Ravizza and colleagues [11, 12] conducted several experiments investigating the impact of S–R rules on task switching. They used odd-man-out test situations (i.e., participants had to find the stimulus that did not match the others). In the weak S–R load condition, responses were spatially congruent to the target stimulus location, that is, participants did not need to learn arbitrary S–R rules. On the contrary, in the strong S–R load condition, participants were required to respond according to an arbitrary rule that had previously been memorized. Because arbitrary S–R mapping involves the learning of a response code (i.e., a rule) that often does not match the target dimension (e.g., red is arbitrarily associated with the left response key, blue with the right response key), switching conditions particularly rely on task-rule switching [5]. In addition, arbitrary S–R mapping gives rise to potential sources of interference as both learning and keeping or retrieving the arbitrary rule in long-term memory are needed to perform the task [13]. By contrast, in nonarbitrary mapping conditions, the response is naturally associated with the target’s features, which decreases the impact of task-rules and

emphasizes task-goal activation.

Besides S–R mappings, the type of cues indicating the task to perform is another element that influences task-rule switching. Depending on its nature, the cue itself can represent an additional rule to learn [5, 6, 14]. A cue can be strong (i.e., transparent, e.g., the word “*color*” used to signal a color judgment task) or weak (i.e., arbitrary, e.g., a triangle indicates the color task and a diamond indicates the shape task). Weak cues increase the involvement of task-rule activation because the information provided by the cue is not sufficient in itself to define the task to perform, and participants must retrieve the meaning of the cue (i.e., the rule) in long-term memory in addition to the main task [5, 6]. Therefore, the use of transparent cues also emphasizes task-goal activation and decreases the impact of task-rules. As expected, arbitrary (weak) cues give rise to higher switch costs than transparent (strong) ones [5, 14, 15].

The functional dissociation between task-goal and task-rule switching has been evidenced using functional neuroimaging and latent factor analyses [16–18]. Task-goal and task-rule switching have been shown to rely on distinct neural processes and substrates. Event-related potentials studies identified an early parietal and frontal positivity associated with task-goal activation, and late parietal positivity and frontal negativity associated with task-rule activation [16]. Functional magnetic resonance imaging studies also found that left anterior regions are differentially involved in task-goal and task-rule representations with the ventrolateral prefrontal cortex (PFC) involved in abstract rules representation, the presupplementary motor area (pre-SMA) involved in rules suppression, and the inferior frontal junction (IFJ) involved in task-goal representation. Posterior regions [i.e., the posterior parietal cortex (PPC) and the intraparietal sulcus (IPS)] are more involved in S–R rules and response-sets representations [16, 17]. In a recent study [18] using latent factor analyses, 20 task pairs were administered to 119 young adults to assess five proposed components of mental set shifting. Task-goal switching was labeled *Judgment shifting* and required participants to switch between varying classification tasks. For example, participants had to determine either the color or the shape of objects presented. Task-rule switching was labeled *mapping shifting*, and participants had to switch between S–R mappings. Three other components, which we will not manipulate in this study, were also assessed, namely, dimension shifting, response set shifting, and stimulus set shifting. Modeling latent factors for each of the components revealed that a model with five separate yet correlated factors fits the data best. Importantly, task-goal switching was more consistently associated with a separate factor than task-rule switching and could not fully be accounted for by a general shifting factor, confirming the importance of dissociating these two stages.

Sleep deprivation (SD) is well known to exert a deleterious impact on various cognitive domains (e.g., Refs. 19–23), but only a few studies investigated its impact on cognitive flexibility [24–27]. These studies found an increased switch cost after a night of total SD. However, several executive functions were mixed in one study (i.e., response inhibition, task switching, and task strategy [27]), which results in difficult comparisons and interpretations. The three other studies used task-switching paradigms that involved arbitrary S–R mapping (e.g., left button for red or circle shape). Therefore, increased SD-related switch costs reported in prior studies [24–26] might be due to (or aggravated by) SD-related impairments in memory load capacities eventually hampering cognitive flexibility. In addition, weak cues were used in all SD studies on task switching also increasing the memory load [24–26]. In sum, these studies arguably evidenced task-rule switching deficits after SD. However, it remains disputable whether task-goal switching in itself is impaired after SD, or if higher switch costs after SD are the consequence of SD-related difficulties to switch the rules during task performance.

To the best of our knowledge, the specific impact of SD on the task-goal component remains unexplored. This information is of importance because task-goal activation and maintenance are key features of most of the theoretical frameworks on cognitive control [3, 28, 29]. During task switching, the goal of the task is generally a more transient representation than the rule, the latter being usually fixed at the beginning of the experiment during instructions. As discussed above, task-goal and task-rule switching processes rely on distinct neural substrates [16, 17]. Likewise, SD does not similarly affect neural activity in all brain regions [30, 31]. It is therefore possible that SD does not affect to the same extent task-goal and task-rule switching processes. In a prior study [32], we measured task-goal switching using a cued match-to-sample task in which the response mapping was congruent with the target location (i.e., nonarbitrary mapping) and the cues were words indicating the task to perform (i.e., transparent cues). Results disclosed improved accuracy switch-cost scores after a short nap, indicating an effect of sleep on task-goal switching. These findings also suggest that SD may have an opposite effect and deteriorate task-goal switching and consecutive accuracy switch-cost scores compared with regular sleep (RS).

Finally, cognitive control abilities are tightly related to central dopaminergic activity. Striatal dopamine is thought to operate as a gating signal that triggers the updating of WM and increases cognitive instability or flexibility [28, 29]. According to the work of Dreisbach et al [33], dopamine plays a central role in the stability–flexibility dilemma. In other words, to follow a goal-directed behavior, a compromise should be reached between maintaining the current goal (i.e., keeping away from distraction) and updating information (i.e., adapting our behavior). Spontaneous eye blink rate (EBR) has been described as an indirect marker of central dopaminergic function [34] linked to cognitive flexibility [33, 35–37]. For instance, healthy people with higher EBR were shown to exhibit increased cognitive flexibility but also reduced cognitive stability [33, 36]. Interestingly, spontaneous EBR increases after SD and positively correlates with sleepiness, which has been interpreted as increased central dopamine activity to counteract the sleep drive [38, 39]. EBR is also a marker of drowsiness or sleepiness [40] and arousal levels [41]. However, recent results [42] have questioned the

plausibility of a dopamine increase after SD. Indeed, SD has been found to downregulate D2 and D3 receptors. Because spontaneous EBR primarily relates to cognitive function via D2-driven modulation [37], one should thus expect an EBR decrease after SD, which is not the case [38, 39]. Furthermore, SD does not affect the impact of methylphenidate, a DA transporter blocker, on D2/D3 receptors [42], indicating that a dopamine increase after SD is also rather improbable. Therefore, if the EBR increase observed after SD is not due to dopamine increase, it should not be associated with a task-goal switching modulation after SD.

In this framework, the present study investigates the impact of one night of total SD on cognitive flexibility using a task-goal switching paradigm with nonarbitrary mapping and strong (transparent) cue conditions that minimize task-rule cognitive load. We predicted that SD would aggravate switch costs, indicating a task-goal switching alteration. We also predicted a spontaneous EBR increase after SD. However, as noted above, this increase might not be associated with the task-goal switching alteration. Importantly, many studies have provided evidence for a deleterious impact of SD on alertness, vigilance, WM, and other mental abilities [19–23]. In this respect, SD-related impairments in cognitive flexibility might be an indirect consequence of the deterioration of these other cognitive functions. Therefore, we tested participants for other key attentional (alertness and vigilance) and executive (inhibition and WM) functions [4, 43].

Material and Methods

Participants

Thirty-eight French-speaking participants gave their written informed consent to participate in this study approved by the Ethics Committee of the Faculty of Psychological Sciences at the Université libre de Bruxelles (ULB). Data for three of them were discarded due to a technical failure during the switching task. The remaining 35 participants (19 females) had a mean age of 21.94 ± 2.52 years old (range = 18–26 years). All participants were right-handed, had no history of medical, neurological, or psychiatric disorders, were free of any medication or drug, and without depression signs (13-item Beck Depression Inventory [44]; score range = 0–5; cutoff score = 8). Participants' chronotype was neutral ($n = 29$), moderate evening ($n = 4$), or moderate morning ($n = 2$) types according to the Morningness–Eveningness Questionnaire [45] (score range = 35–63). Habitual fatigue level was below the cutoff score on the Fatigue Severity Scale [46] (FSS; score range = 1.33–5.11; cutoff score = 5.5). There were four smokers in the SD group and two smokers in the RS group. All smokers smoked less than 10 cigarettes per day.

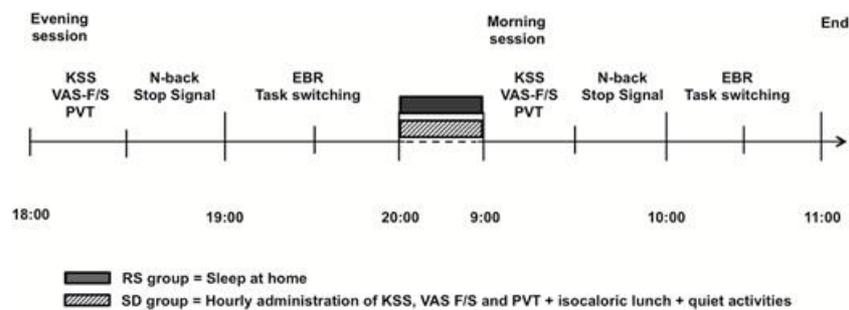
Participants were required to maintain a RS-wake cycle for 3 days prior to the study (i.e., sleep at least 7 hours per night, go to bed before 01:00 am, wake up before 10:30 am, and no naps during the day). Compliance was controlled using actigraphic recordings (ActiGraph wGT3X-BT Monitor, Pensacola, FL). Participants were also asked to complete the St. Mary Hospital questionnaire [47] and a sleep diary after each night of sleep.

Procedure

Participants were assigned to one out of two conditions. They either slept at home between the evening and morning testing sessions (RS; 17 participants) or stayed awake in the laboratory during the entire night (SD; 18 participants). For organizational reasons, participants knew before the testing session in which condition they were included. No difference was found between the two groups for all the above-mentioned inclusion criteria (two-tailed *t*-tests for independent samples; all *p*-values >.26), except for a trend [$t(33) = -2.01$; $p = .052$] on habitual fatigue levels (FSS scores), on average higher in the SD (3.21 ± 1.01) than in the RS (2.56 ± 0.91) group. However, all scores remained below cutoff pathological values.

As illustrated in Figure 1, both SD and RS groups were first tested in the evening starting at 18:00. Participants completed the following tasks, always in the same order: psychomotor vigilance task (PVT), subjective sleepiness (Karolinska Sleepiness Scale, KSS; Visual Analogue Scales for Sleepiness, VAS-S) and fatigue (Visual Analogue Scales for Fatigue, VAS-F) scales, WM (N-back), and response inhibition (Stop-signal) executive tasks (see below for a description of these tasks and scales). At 19:00, spontaneous EBRs were recorded for 3 minutes followed by the task-switching protocol. Afterward, the RS group went home for a regular night of sleep, whereas the SD group stayed in the testing room during the whole night under the constant supervision of two experimenters. During the SD period, participants were asked to remain quiet and seated most of the time. They were allowed to engage in calm activities (e.g., reading, playing society games, and watching movies). Water was available ad libitum, and isocaloric meals were offered hourly. Participants had to refrain from stimulant drinks (e.g., coffee, tea, or energizers) or smoking. The PVT and the KSS were administered hourly to document changes in objective and subjective vigilance levels, respectively, over the night. At 09:00 am, the second testing session started with the exact same protocol administered on the previous evening. Participants assigned to the RS group came to the laboratory in the morning at 09:00 for the second testing session.

Figure 1.



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Schematic representation of the experimental protocol (evening, night, and morning sessions). KSS = Karolinska Sleepiness Scale; VAS-F/S = Visual Analogue Scales for Fatigue/Sleepiness; PVT = Psychomotor Vigilance Task; RS = regular Sleep; SD = sleep deprivation; EBR = spontaneous eye blink rate recording.

Fatigue, Sleepiness, Alertness and Vigilance, and EBR

At the beginning of each session, participants' subjective level of sleepiness and fatigue were assessed using the KSS [48] and VAS-F/S [49].

Objective measures of alertness and vigilance were obtained using the 10-minute version of the PVT [50]. In the PVT, participants were instructed to press a key as fast as possible whenever a millisecond countdown appeared in the middle of a computer screen. Stimuli were randomly presented with an inter-stimuli interval ranging from 2 to 10 seconds. Alertness or PVT speed was estimated using the median reaction times (RTs), less affected by extreme values than the mean [25, 51]. PVT variability as a marker of vigilance [52] was measured using the RTs' coefficient of variation, i.e., standard deviation divided by mean [53].

To record spontaneous EBR, participants were seated in a separate room 1 m away from a wall with a fixation cross at the level of the eyes. They were instructed to relax, sit comfortably, and fixate the cross on the wall. They were allowed to move slightly and to blink but not to turn their head to the right or left. This information helped diverting participants' attention from their blinks. Eye movements and blinks were video-recorded for 3 minutes. Participants were seated in front of the fixation cross during the instructions, and the first 10 seconds of recording were not analyzed to allow for adaptation to the environment. Video-recordings were visually inspected a posteriori to count the number of eye blinks. A blink was defined as a rapid closing and opening of the eyelid: slow eye closures were not counted as blinks. Two independent evaluators counted the EBR, and results were compared. In case of disagreement, the video was recounted for a third time and an agreement decided. Spontaneous EBR was computed as the mean EBR per minute during each recording session and was entered in statistical analyses like in prior studies [54, 55].

WM and Inhibition Tasks

WM N-Back Task

The WM N-back task [56] was adapted from a previous study from our lab [57]. In the 0-back (N0) condition, they had to press the response key only when the digit « 2 » appeared on the screen, i.e., a simple detection task. In the 2-back (N2) condition, they had to press the space bar when the digit currently displayed matched the digit presented two steps earlier in the sequence. Therefore, the task required for successful comparison both the maintenance and the updating of a series of items in WM. Participants performed five blocks of each condition with alternation of 0- and 2-back conditions (see [Supplementary Material](#) for a detailed description of the task procedure). RTs and corrected accuracy scores (hits minus false detections) were averaged over the five blocks per session for the N2 and N0 conditions. WM performance reflecting the updating process (i.e., difference between N2 and N0 conditions) was computed on corrected accuracy scores (WM accuracy = accuracy N0 – accuracy N2) and on mean RTs for correct responses (WM speed = RT N2 – RT N0). Lower scores reflect smaller differences between N2 and N0 conditions, indicating a better performance in the WM updating process.

Inhibition Stop Signal Task

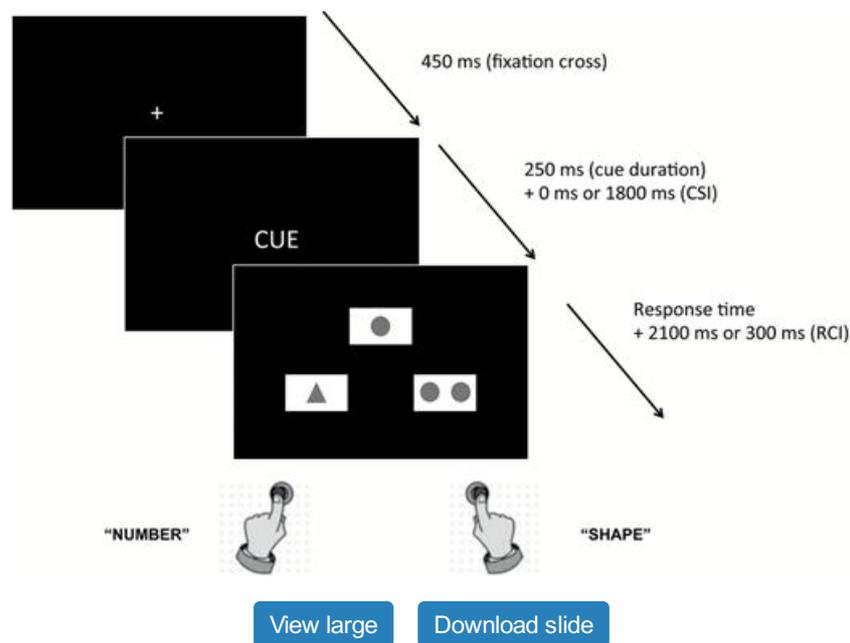
The Stop Signal task evaluating response inhibition abilities was adapted from a previous study from our lab [58] (see [Supplementary Material](#) for a detailed description). Participants were presented with arrows pointing to the left or to the right (go signals) at random intervals. They were instructed to answer as quickly and as accurately as possible to which side the arrow was pointing by pressing the corresponding key. In 20% of the trials, a

stop signal (vertical arrow) was displayed shortly after the horizontal arrow (see [Supplementary Figure S1](#)). In this case, participants were instructed to try not responding, i.e., inhibiting the response initiated after presentation of the go signal. If the participant managed successfully to refrain from responding, then the stop signal delay (SSD) was lengthened in the next stop trial, resulting in a harder response inhibition. If he or she failed (i.e., he or she provided an answer even though the stop signal instructed not to), the SSD on the next stop trial was shortened to facilitate the response inhibition. This SSD adaptive method guarantees a similar task difficulty across participants who work at the edge of their inhibitory capacities. The stop signal reaction time (SSRT) was calculated for both morning and evening sessions. The SSRT is the maximal time delay between the go and the stop signals at which the participant still successfully inhibit the initiated response ($SSRT = \text{mean RT} - \text{SSD}$). SSRT provides an individual index of inhibitory control, with longer SSRT indicating poorer response inhibition [59].

Task-Goal Switching

Task-goal switching was assessed using a cued match-to-sample task from our lab [32]. The task design is illustrated in [Figure 2](#). At the beginning of each trial, an instruction cue was displayed in the center of the screen for 250 milliseconds, followed by a short (0 milliseconds) or a long (1800 milliseconds) cue-stimulus interval (CSI). Participants had to then match the stimulus presented in the upper part of the screen with one of the two stimuli presented in the lower part of the screen according to the instruction cue. Responses were always spatially congruent to the target location, i.e., left key for left answer and right key for right answer.

Figure 2.



Cued match-to-sample switching task. For each trial, a fixation cross is followed by an instruction cue ("shape," "color," "number," or "outline"), after which three figures are presented. Participants are asked to decide which one of the two lower figures matches the top one, depending on the instruction cue, and to press the corresponding key (left key for left figure and right key for right figure).

The task-switching protocol was programmed using Psycscope X software [60, 61] and run on Mac Mini computers. Participants answered on the computer's keyboard using their right and left hand forefingers. RT was recorded for each trial as the time elapsed from target onset to response (in milliseconds). The cue was a written word of the relevant dimension (in French): *COULEUR* (color), *FORME* (shape), *NOMBRE* (number), or *CONTOUR* (outline). For each trial, the three stimuli were presented on a black screen, each within a rectangle [9 cm wide (visual angle = 8.58°) and 5 cm tall (4.77°)] in a triangular disposition, with the target picture on the top ([Figure 2](#)). The geometrical figures were 2.5 cm wide (2.39°) and 1.5 cm tall (1.43°). The distance between the screen and the participants' eyes was approximately 60 cm.

Prior to the task, participants received written instructions that were repeated by the investigator before the session started. They were instructed to press a key ("q" for left and "m" for right on an azerty keyboard) corresponding to the position (left or right, respectively) of the bottom picture that matched the target (top picture) according to the cued dimension. A sample trial figure was included as an example. The participants were instructed to perform as fast as possible while minimizing the number of errors. Each trial began with a fixation cross displayed at the center of the screen during 450 milliseconds, followed by the cue presented for 250 milliseconds. Short and long CSI conditions were presented in a counterbalanced order across participants. Beside warm-up trials, each CSI condition comprised two blocks of 96 trials (with 48 switch and 48 repeat trials in each block). Repeat and switch trials were presented in a pseudo-randomized order controlling for transitions between dimensions and congruency effects. Each

trial involved two dimensions that differ (e.g., shape and color) and two dimensions that were kept constant (e.g., number and outline). Trials were all incongruent ones (i.e., each dimension was associated with a different response) so that it was always possible to know whether the participant responded correctly according to the target dimension. Incongruency was, thus, kept constant during the entire task. The order of the dimensions was pseudo-randomized to control for transitions and to ensure an equal number of presentations for each dimension. In the short CSI (0 milliseconds) condition, the cue was directly followed by the target and the two potential matching figures until a response was provided. In the long CSI (1800 milliseconds) condition, a black screen followed the cue during 500 milliseconds was then replaced by a point in the center of the screen for 250 milliseconds. The black screen–point sequence was repeated twice, followed by another black screen for 300 milliseconds before the presentation of the target and the two potential matching figures. The intertrial interval was set to 300 milliseconds in the long CSI condition and to 2100 milliseconds in the short CSI condition to balance the total time for each trial. Both short and long CSIs were preceded by a training block of 10 trials and administered within a single session that lasted approximately 30 minutes, with a short break between each interval type.

Task-goal switching analyses are based on the percentage of errors (task-switching accuracy) and on mean RTs for trials with correct responses only (task-switching speed). Both measures are complementary and necessary to assess a potential task-switching modulation [62]. The first three trials in each block were considered as warm-up trials and excluded from the analyses. Trials immediately following an error were also discarded from the analyses (9% of trials). RTs' outliers were identified for each participant, each CSI duration (short vs. long), and each task-switching condition (repeat vs. switch trials) using the generalized extreme studentized deviate (GESD) test [62]. This procedure led to discard 3.4% of trials.

Data Analyses

Data are expressed as mean \pm standard deviation, unless mentioned otherwise. Significance level was set at $p < .05$. Partial eta-squared was calculated as a measure for effect size. Actigraphic data were analyzed using ActiLife 6 software (ActiGraph, 2014). Bedtime was visually determined and compared with the information reported by the participants in their sleep diary. Sleep duration was computed using the Cole Kripke algorithm [63]. Separate mixed-design ANOVAs were conducted with between-subject factor Group (RS vs. SD) and within-subject factor Session (evening vs. morning). For task-goal switching accuracy and speed, mixed-design ANOVAs were computed with between-subject factor Group and within-subject factors Session, CSI Duration (short CSI vs. long CSI), and Task Switching (repeat trials vs. switch trials). Interactions with task switching were decomposed using planned comparisons according to our hypotheses (i.e., increased switch costs in the SD group in the morning compared with the evening and the RS group). Interactions were decomposed using Tukey's post hoc comparisons when applicable. The relationships between SD-related changes in task-goal switching performance and changes in other cognitive measures or scales were investigated using Pearson correlations and Bayesian correlations using JASP-software [64]. Changes in fatigue and sleepiness subjective scores (VAS-S, VAS-F, and KSS), task performance (PVT, N-back, and Stop Signal tasks), and spontaneous EBR were estimated by subtracting the results obtained during the evening session from the results obtained during the morning session [Δ Session = morning session – evening session]. p -Values were adjusted for multiple comparisons using Bonferroni correction per variable type compared with the measures of task switching. Correlations were corrected for the three questionnaires scores (VAS-S, VAS-F, and KSS), two PVT measures (PVT speed and variability), and two N-back performances (WM speed and WM accuracy). The difference between two correlation coefficients was computed using the r -to-Fisher- z transformation: $r' = .5 * [\ln(1+r) - \ln(1-r)]$, where r' is the Fisher- z transformed (to a normally distributed variate) Pearson correlation coefficient, r and r' is the standard Pearson correlation coefficient. The significance of the difference between two correlation coefficients is computed as follows: $d = r_1' - r_2'$, where d is the difference between the two Fisher z -transformed correlation coefficients; $sd = \text{Square Root } ((n_1 + n_2 - 6) / ((n_1 - 3) \times (n_2 - 3)))$, where sd is the standard error of the difference between the two normalized (Fisher- z transformed) correlation coefficients, n_1 and n_2 are the two sample sizes (for r_1 and r_2 , respectively). The test statistic d/sd is then evaluated against the t distribution with $df = n_1 + n_2 - 4$ degrees of freedom. The one-sided and two-sided p values are computed as usual, by considering either both sides or only one side of the t -distribution. Here, we chose the two-sided p value.

Results

Sleep Prior to the Experiment

Mean sleep duration during Nights 1 to 3 preceding the experimental night averaged, respectively, 428 ± 59 , 428 ± 57 , and 474 ± 69 minutes in the RS group and 429 ± 74 , 433 ± 71 , and 444 ± 61 minutes in the SD group. A mixed-design ANOVA computed on sleep duration with between-subject factor Group (RS vs. SD) and within-subject factor Night (1 to 3) disclosed a main effect of Night [$F(2,66) = 3.317, p = .042, \eta^2_p = .091$].

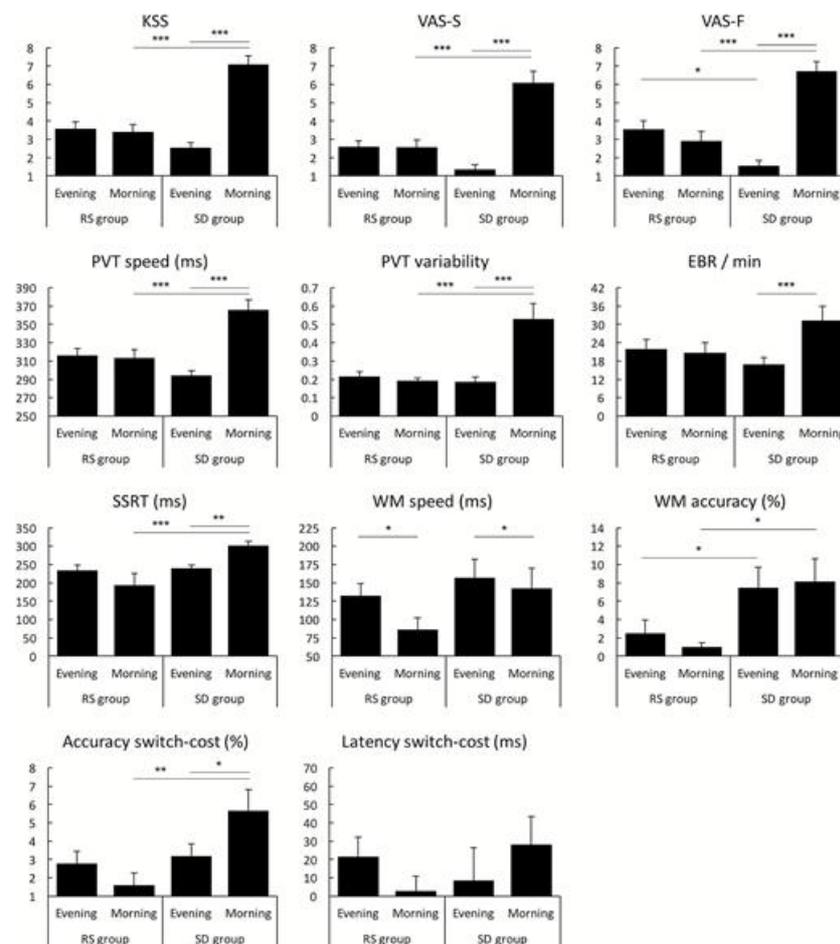
Participants in both groups tended to sleep more during night 3 than during nights 1 ($p = .065$) and 2 ($p = .098$), without any significant difference

between nights 1 and 2 ($p = .982$). The main effect of Group and the Group \times Night interaction failed to reach significance (all p -values $>.357$). Actimetric measures indicate that the RS group slept 397 ± 40 minutes during the experimental night, whereas the SD group did not sleep.

Sleepiness, Fatigue, Alertness, and Vigilance

Figure 3 presents the results and post hoc comparisons for the KSS, VAS-S, VAS-F, and PVT. Means and standard deviations are presented in Supplementary Table S1. Interactions were significant for all variables (all p s $<.001$; see Supplementary Table S2). Post hoc tests showed that scores were similar in RS and SD groups in the evening, but significantly differed in the morning with the SD group being more sleepy, more fatigued, and exhibiting decreased alertness and vigilance.

Figure 3.



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Results for KSS, VAS-S, and VAS-F scores, PVT speed and variability, N-back task, Stop signal task, EBR, and task-goal switching for both groups (RS, SD) and both sessions (evening, morning). RS = regular sleep group; SD = sleep deprivation group; KSS = Karolinska Sleepiness Scale; VAS-F/S = Visual Analogue Scales for Fatigue/Sleepiness; PVT = Psychomotor Vigilance Task; PVT speed = PVT median of RTs; PVT variability = PVT coefficient of variation of RTs; EBR/minute = spontaneous eye blink rate per minute; WM (working memory N-Back) speed = RT N2 – RT N0; WM accuracy = accuracy N0 – accuracy N2; SSRT = Stop Signal Reaction Time; Accuracy and Latency switch costs = switch trials – repeat trials. * $p < .05$; ** $p < .01$; *** $p < .001$.

WM and Inhibition

WM N-Back Task

Figure 3 presents the results and post hoc comparisons for WM speed and accuracy. Means and standard deviations are presented in Supplementary Table S3. The mixed-design ANOVA disclosed a main effect of Group [$F(1,33) = 6.06, p = .019, \eta^2_p = .155$], with RS participants exhibiting a higher accuracy than the SD group. Session and Group \times Session interaction effects failed to reach significance (all p -values $>.258$). The mixed-design

ANOVA computed on WM speed disclosed a main effect of Session [$F(1,33) = 7, p = .012, \eta^2_p = .175$], with participants being faster in WM in the morning than in the evening. Group and Group \times Session interaction effects failed to reach significance (all p -values $>.174$).

Inhibition Stop Signal Task

Figure 3 presents the results and post hoc comparisons for the Stop Signal task. Means and standard deviations are presented in [Supplementary Table S3](#). The mixed-design ANOVA computed on SSRT disclosed a main effect of Group [$F(1,33) = 6.22, p = .018, \eta^2_p = .158$] and a significant Group \times Session interaction [$F(1,33) = 17.23, p < .001, \eta^2_p = .343$]. The Session effect was not significant [$F(1,33) = .69, p = .412, \eta^2_p = .02$]. Post hoc comparisons indicated that, in the SD group, SSRT increased in the morning compared with the evening ($p = .006$). SSRT in the evening and morning sessions did not differ significantly from those in the RS group ($p = .115$). SSRT was significantly higher in the SD group than in the RS group in the morning ($p < .001$) but not in the evening ($p = .996$).

Spontaneous EBR

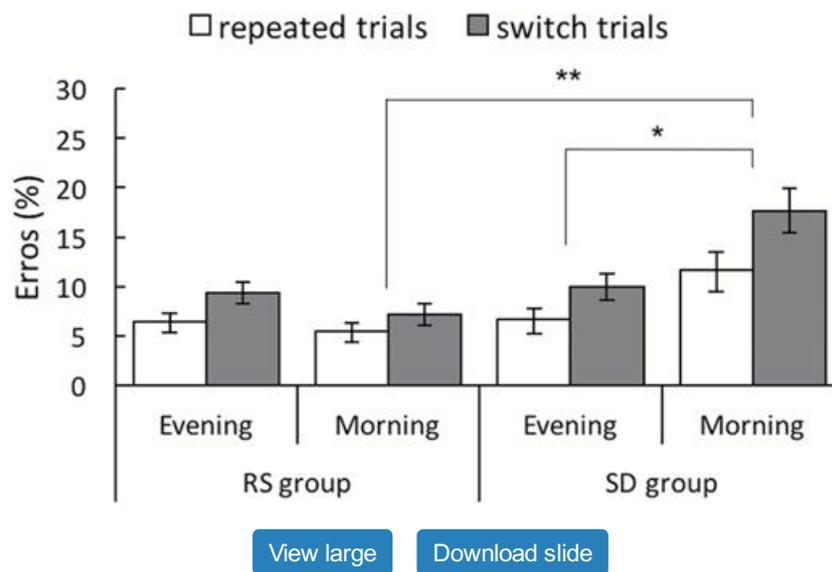
Figure 3 presents the results and post hoc comparisons for spontaneous EBR. Means and standard deviations are presented in [Supplementary Table S3](#). The mixed-design ANOVA computed on mean EBR disclosed a significant main effect of session [$F(1,33) = 7.47, p = .01, \eta^2_p = .185$] and a significant Group \times Session interaction [$F(1,33) = 10.85, p = .002, \eta^2_p = .247$]. The Group effect was not significant [$F(1,33) = .41, p = .525, \eta^2_p = .012$]. Post hoc comparisons indicated that, in the SD group, EBR increased in the morning compared with the evening ($p < .001$) session. In the RS group, EBR did not differ between evening and morning sessions ($p < .979$). No other comparison reached statistical significance (all p -values $>.149$).

The Pearson correlation coefficient between EBR in the evening and morning sessions in the RS group was highly significant ($r = 0.903, p < .001$). This correlation was not significant in the SD group ($r = 0.365, p = .137$). Correlation coefficients were significantly different between the RS and SD groups ($p = .007$).

Task-Goal Switching

For task-goal switching accuracy, the mixed-design ANOVA computed on percentages of errors disclosed main effects of Group [$F(1,33) = 6.97, p = .013, \eta^2_p = .174$], Session [$F(1,33) = 7.49, p = .01, \eta^2_p = .185$], CSI Duration [$F(1,33) = 10.45, p = .003, \eta^2_p = .241$], and Task Switching [$F(1,33) = 51.37, p < .001, \eta^2_p = .609$], as well as Group \times Session [$F(1,33) = 20.61, p < .001, \eta^2_p = .384$], Group \times Task Switching [$F(1,33) = 5.77, p = .022, \eta^2_p = .149$], Group \times Session \times Task Switching [$F(1,33) = 6.55, p = .015, \eta^2_p = .166$], and Group \times Session \times CSI Duration [$F(1,33) = 5.79, p = .022, \eta^2_p = .149$] interaction effects. No other interaction reached significance (all p -values $>.328$). Planned comparisons for the Group \times Session \times Task Switching interaction indicated that accuracy switch-cost scores were significantly higher in the SD group than in the RS group in the morning ($p = .005$) but not in the evening ($p = .689$). Furthermore, accuracy switch-cost scores increased in the SD group in the morning compared with the evening ($p = .041$). Figure 3 presents accuracy switch-cost scores for both groups and both sessions. Figure 4 presents accuracy scores for repeat and switch trials, for both groups, both sessions, and both CSI durations. [Supplementary Table S4](#) presents RTs and switch-cost scores for both groups, both sessions, and both CSI durations.

Figure 4.



Task-goal switching accuracy in the cued match-to-sample task by session (evening, morning) and trials (repeat, switch) for RS and SD groups; RS = regular sleep group; SD = sleep deprivation group. The accuracy switch costs are the difference between switch and repeat trials. ** $p < .01$; * $p < .05$.

Post hoc tests for the Group \times Session \times CSI Duration interaction indicated that participants in the SD group made more errors in the morning than in the evening for both short ($p = .019$) and long ($p < .001$) CSIs. They also made more errors in the morning than participants in the RS group for long CSI ($p = .003$). No other comparison reached statistical significance (all p -values $> .127$).

Concerning task-goal switching speed, the mixed-design ANOVA computed on RTs disclosed a main effect of Task Switching [$F(1,33) = 4.77, p = .036, \eta^2_p = .126$] with participants being faster on repeat trials than on switch trials (878 ± 167 vs. 893 ± 184 milliseconds). The Group \times Session interaction was significant [$F(1,33) = 5.06, p = .031, \eta^2_p = .133$], but post hoc comparisons failed to disclose significant differences (all p -values $> .175$). No other comparison reached statistical significance (all p -values $> .16$). Figure 3 presents accuracy switch-cost scores for both groups and both sessions. Supplementary Table S5 presents RTs and switch-cost scores for both groups, both sessions, and both CSI durations.

Relationships Between Impairments in Task-Goal Switching and Other Variables

In the evening session, we observed a significant negative correlation between EBR and task-goal switching accuracy (across all participants). Participants with lower spontaneous EBR displayed higher accuracy switch costs. This relationship was significant at long CSI ($r = -0.353; p = .037$) but did not reach significance at short CSI ($r = 0.265; p = .124$). However, in the SD group, correlations between Δ Session accuracy switch-cost scores and Δ Session VAS-S, VAS-F, KSS, EBR, PVT speed, PVT variability, Stop signal task SSRT or WM speed, and accuracy did not reach statistical significance (all p -values $> .213$). These results indicate that increases in accuracy switch-cost scores (i.e., worse performance) from morning to evening after SD were not significantly associated with the alterations of other variables after SD (for detailed values, see Supplementary Table S6).

Finally, in the SD group, we assessed the relationships between SD effects on EBR and other cognitive measures or scales (for detailed values, see Supplementary Table S6). No significant correlations were evidenced between Δ Session EBR and Δ Session for the other variables (all p -values $> .167$).

We conducted Bayesian correlational analyses [64] in the SD group to investigate the statistical significance of the null hypothesis (i.e., an absence of association between the variables). Correlations between Δ Session accuracy switch-cost scores and Δ Session VAS-S, VAS-F, KSS, EBR, PVT speed, Stop signal task SSRT, or WM accuracy were considered significantly in favor of the null hypothesis (Bayes Factors < 0.333). Correlations between Δ Session accuracy switch-cost scores and WM speed or PVT variability were considered as inconclusive (Bayes Factors comprised between 0.333 and 3). Finally, correlations between Δ Session EBR and KSS, WM accuracy, or Stop signal task SSRT were also in favor of the null hypothesis while they were considered inconclusive for the other variables.

Discussion

The current study investigated the impact of one night of total SD on task-goal switching in healthy participants. Our results evidence deteriorated task-goal switching performance after SD, characterized by higher accuracy switch costs in the morning compared with the evening in SD participants, and higher accuracy switch costs than in the RS group in the morning but not in the evening. SD also deteriorated objective and subjective markers of fatigue and sleepiness, increased spontaneous EBR, and decreased response inhibition in the Stop Signal task. However, we did not find significant correlations between changes in task-goal switching performance and changes in objective and subjective markers of fatigue and sleepiness, response inhibition, or spontaneous EBR. Finally, SD did not significantly affect WM updating measures in the N-back task.

To the best of our knowledge, the current study is the first to demonstrate that SD negatively affects the task-goal component of task switching. Indeed, prior studies used paradigms investigating task-rule switching [24–26], a related but distinct component of cognitive flexibility [9, 10]. The present findings are also consistent with our previous study that evidenced a beneficial effect of a nap on task-goal switching [32]. In the present study, SD resulted in the reverse effects of napping, supporting the hypothesis of a relationship between sleep-related restorative processes and task-goal switching performance. Task-goal activation and maintenance are key features in most theoretical frameworks of cognitive control [3, 28, 29]. Therefore, task-goal switching alterations observed after SD suggest that failure to adjust or maintain the task goal could be one of the reasons for a deterioration of cognitive control after SD. Our results also extend previous research on task-rule switching [24–26] in showing that both task-goal and task-rule switching can be affected by SD.

The effects of SD on task-goal switching were found on accuracy but not on response latencies. A significant Session \times Group interaction was observed on latency switch costs with a pattern similar to accuracy switch costs (Figure 3), but post hoc tests failed to disclose any significant difference. The latency switch cost usually observed in task switching studies largely relies on the learning and rehearsal of arbitrary associations between stimuli and responses [65, 66], and the preparation mainly benefits the rehearsal or the reconfiguration of S–R mappings (i.e., task-rules). The proposal that retrieving information from long-term memory plays a central role in task switching was developed in the context of the retrieval-demand hypothesis [66]. The authors observed that preparatory effects on switch costs and the retrieval-demands were eliminated when relevant task rules (i.e., the critical S–R associations) were directly provided by the task cues. They also suggested that episodic retrieval might be sufficient to explain the endogenous component (reconfiguration) of task switching. According to them, it would be impossible to represent more than one task rule in WM. Consequently, after a switch, the next task rule has to be retrieved from long-term memory. They also suggested that in some cases the failure to prepare (see also Ref. 67) requires retrieving S–R mappings when the targets are displayed, thus leading to a residual switch cost. This proposal was supported by the fact that participants can only partially prepare, maybe for one S–R association at a time [65]. In line with these elements, we propose that the latency switch cost is mainly affected by task-rule retrieval in episodic memory because these task rules are encoded in long-term memory and their retrieval is predominantly a time consuming process affecting latency switch cost. In contrast, as evidenced by this experiment and previous work from our team [32], the accuracy switch cost appears to be more related to task-goal switching. This is presumably due to the fact that it involves an online maintenance and adaptation of the goal in WM, and less long-term memory retrieval. Failure to maintain or adjust the goal would therefore be characterized by errors and an accuracy switch-cost. The state-instability hypothesis [20] posits that SD induces instability in the capacity to maintain attention and alertness due to the growing influence of sleep initiating mechanisms. A major component of state instability might be task-set instability. The task sets and task-goals can be subject to temporary breakdowns, an effect actually close to the notion of goal neglect [68]. However, the notion of task-set instability is more restricted than the notion of state instability and allows clearer predictions. In particular, it can be predicted that the consequences of SD will be stronger with additional conditions promoting task-set instability, like in a task-goal switching paradigm. Indeed, shifting between tasks by definition involves a temporary task-set instability. Consequently, task-set instability after SD should be stronger in switch than in repeat trials [26], a prediction supported by our data.

It is well known that insufficient sleep leads to a general slowing of response speed and increased variability in performance, particularly for simple measures of alertness and vigilance [22, 69]. Accordingly, we evidenced SD-related changes in subjective and objective measures of sleepiness and fatigue (including alertness and vigilance). RS and SD groups exhibited similar levels in the evening, but differed significantly in the morning, the SD group being sleepier, more fatigued, less alert, and vigilant. In addition, spontaneous EBR were higher after SD. These results are in line with previous findings [38–40, 70]. However, the increase in EBR was not significantly correlated with modulation of subjective and objective markers of sleepiness or vigilance, indicating that SD-related processes causing EBR increase might differ from those affecting alertness and vigilance.

Task switching is one of the cognitive control components that has been most convincingly associated with spontaneous EBR [33, 35, 36, 55]. In this study, we found a negative association between EBR and accuracy switch costs. This association was significant at long CSI, indicating that preparation processes associated with goal adaptation and goal maintenance (proactive control) are associated with EBR and, putatively, with striatal dopamine. Indeed, in previous research, increased EBR was also associated with increased cognitive flexibility and lower switch costs. It was concluded that higher dopamine activity favors an easier disengagement from the previous task [33]. Our results are in line with this proposal. However, the EBR increase observed after SD was not significantly associated with task-goal switching impairment, suggesting that different SD-related processes affect spontaneous EBR and cognitive flexibility. This result is compatible with previous works that have questioned the plausibility

of a dopamine increase after SD [42]. Therefore, if the increase in EBR observed after SD is not due to dopamine increase, it should not be strongly associated with a task-goal switching modulation after SD, which is the case in the present study. Our results, therefore, support the idea that EBR increase after SD is not or only partially related to a dopamine increase or cognitive flexibility impairment.

Previous studies have shown that vigilance is much more affected by SD than executive functions [22, 71], and some studies have failed to disclose a specific effect of SD on executive functions [72]. Executive functions are by essence multidimensional, and operate on and through other cognitive processes. Therefore, any cognitive task recruiting executive functions will recruit other, more basic, processes as well, a phenomenon known as the task impurity problem [73]. It cannot be excluded that low scores previously observed on executive function tasks after SD were due to deficits in nonexecutive components of task performance, like alertness or vigilance, more than deficits in executive functioning per se. Controlling for nonexecutive components in executive tasks can be achieved using control conditions and composite scores such as switch costs, SSRT, or WM updating score. In the present study, WM updating measures were not affected by SD, in agreement with prior studies disclosing null [74, 75] or small effects [76] of SD in a N-back task. On the other hand, SD exerted an impact on response inhibition in the Stop Signal task, which is in agreement with prior findings [77, 78].

The lack of eye tracking or electrooculography (EOG) is a limitation of our study, as this would have allowed the evaluation of pupillary dilation, and trial-to-trial changes in EBR during task, to track potential fluctuations in dopamine related to ongoing task demands [37]. For instance, both EBR during task and pupillary dilation have been associated with cognitive processes such as cognitive inhibition [79] and mental fatigue [80]. Further studies should investigate these aspects. Another limitation of our study is the absence of a direct comparison between task-rule and task-goal switching. Future SD studies should use experimental designs allowing a direct comparison between these two components of task switching.

In conclusion, we showed here that total SD negatively affects both task-goal switching and response inhibition measures, but not WM updating. Furthermore, spontaneous EBR increase after SD was not significantly associated with impaired task-goal switching. Altogether, our results confirm the importance of considering key executive functions such as WM updating, inhibition, and task switching as, at least partially, separated [4, 43] and having a relative sensitivity to the effects of SD.

Supplementary Material

Supplementary material is available at *SLEEP* online.

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References

1. Diamond A. Executive functions. *Annu Rev Psychol*. 2013; 64: 135–168. doi: 10.1146/annurev-psych-113011-143750.

[Google Scholar](#) [Crossref](#) [PubMed](#)

2. Braver TS. The variable nature of cognitive control: a dual mechanisms framework. *Trends Cogn Sci* . 2012; 16(2): 106–113.
[Google Scholar](#) [Crossref](#) [PubMed](#)
3. Miller EK, Cohen JD. An integrative theory of prefrontal cortex function. *Annu Rev Neurosci* . 2001; 24(1): 167–202.
[Google Scholar](#) [Crossref](#) [PubMed](#)
4. Miyake A, Friedman NP. The nature and organization of individual differences in executive functions: four general conclusions. *Curr Dir Psychol Sci* . 2012; 21(1): 8–14.
[Google Scholar](#) [Crossref](#) [PubMed](#)
5. Kiesel A, Steinhauser M, Wendt Met al. Control and interference in task switching—a review. *Psychol Bull* . 2010; 136(5): 849–874.
[Google Scholar](#) [Crossref](#) [PubMed](#)
6. Vandierendonck A, Liefvooghe B, Verbruggen F. Task switching: interplay of reconfiguration and interference control. *Psychol Bull* . 2010; 136(4): 601–626.
[Google Scholar](#) [Crossref](#) [PubMed](#)
7. Jersild AT. Mental set and shift. *Archives of Psychology* . 1927; 14(89): 1–81.
8. Spector A, Biederman I. Mental set and mental shift revisited. *Am J Psychol* . 1976; 89(4): 669–679.
9. De Baene W, Albers AM, Brass M. The what and how components of cognitive control. *Neuroimage* . 2012; 63(1): 203–211.
[Google Scholar](#) [Crossref](#) [PubMed](#)
10. Rubinstein JS, Meyer DE, Evans JE. Executive control of cognitive processes in task switching. *J Exp Psychol Hum Percept Perform* . 2001; 27(4): 763–797.
[Google Scholar](#) [Crossref](#) [PubMed](#)
11. Ravizza SM, Carter CS. Shifting set about task switching: behavioral and neural evidence for distinct forms of cognitive flexibility. *Neuropsychologia* . 2008; 46(12): 2924–2935.
[Google Scholar](#) [Crossref](#) [PubMed](#)
12. Hakun JG, Ravizza SM. Cognitive control: preparation of task switching components. *Brain Res* . 2012; 1451: 53–64.doi: 10.1016/j.brainres.2012.02.046.
[Google Scholar](#) [Crossref](#) [PubMed](#)
13. Dreisbach G, Goschke T, Haider H. The role of task rules and stimulus-response mappings in the task switching paradigm. *Psychol Res* . 2007; 71(4): 383–392.
[Google Scholar](#) [Crossref](#) [PubMed](#)
14. Logan GD, Schneider DW. Interpreting instructional cues in task switching procedures: the role of mediator retrieval. *J Exp Psychol Learn Mem Cogn* . 2006; 32(2): 347–363.
[Google Scholar](#) [Crossref](#) [PubMed](#)
15. Logan GD, Schneider DW. Priming or executive control? Associative priming of cue encoding increases “switch costs” in the explicit task-cuing procedure. *Mem Cognit* . 2006; 34(6): 1250–1259.
[Google Scholar](#) [Crossref](#) [PubMed](#)

16. De Baene W, Brass M. Dissociating strategy-dependent and independent components in task preparation. *Neuropsychologia* . 2014; 62: 331–340. doi: 10.1016/j.neuropsychologia.2014.04.015.
[Google Scholar](#) [Crossref](#) [PubMed](#)
17. Ruge H, Jamadar S, Zimmermann U, Karayanidis F. The many faces of preparatory control in task switching: reviewing a decade of fMRI research. *Hum Brain Mapp* . 2013; 34(1): 12–35.
[Google Scholar](#) [Crossref](#) [PubMed](#)
18. von Bastian CC, Druery MD. Shifting between mental sets: An individual differences approach to commonalities and differences of task switching components. *J Exp Psychol Gen* . 2017; 146(9): 1266–1285.
[Google Scholar](#) [Crossref](#) [PubMed](#)
19. Chee MW, Choo WC. Functional imaging of working memory after 24 hr of total sleep deprivation. *J Neurosci* . 2004; 24(19): 4560–4567.
[Google Scholar](#) [Crossref](#) [PubMed](#)
20. Doran SM, Van Dongen HP, Dinges DF. Sustained attention performance during sleep deprivation: evidence of state instability. *Arch Ital Biol* . 2001; 139(3): 253–267.
[Google Scholar](#) [PubMed](#)
21. Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. *Semin Neurol* . 2005; 25(1): 117–129.
[Google Scholar](#) [Crossref](#) [PubMed](#)
22. Killgore WD. Effects of sleep deprivation on cognition. *Prog Brain Res* . 2010; 185: 105–129. doi: 10.1016/B978-0-444-53702-7.00007-5.
[Google Scholar](#) [Crossref](#) [PubMed](#)
23. Lim J, Dinges DF. Sleep deprivation and vigilant attention. *Ann N Y Acad Sci* . 2008; 1129(1): 305–322.
[Google Scholar](#) [Crossref](#) [PubMed](#)
24. Bratzke D, Rolke B, Steinborn MB, Ulrich R. The effect of 40 h constant wakefulness on task-switching efficiency. *J Sleep Res* . 2009; 18(2): 167–172.
[Google Scholar](#) [Crossref](#) [PubMed](#)
25. Couyoumdjian A, Sdoia S, Tempesta Det al. The effects of sleep and sleep deprivation on task-switching performance. *J Sleep Res* . 2010; 19(1 Pt 1): 64–70.
[Google Scholar](#) [Crossref](#) [PubMed](#)
26. Heuer H, Kleinsorge T, Klein W, Kohlisch O. Total sleep deprivation increases the costs of shifting between simple cognitive tasks. *Acta Psychol (Amst)* . 2004; 117(1): 29–64.
[Google Scholar](#) [Crossref](#) [PubMed](#)
27. Jennings JR, Monk TH, van der Molen MW. Sleep deprivation influences some but not all processes of supervisory attention. *Psychol Sci* . 2003; 14(5): 473–479.
[Google Scholar](#) [Crossref](#) [PubMed](#)
28. O'Reilly RC. Biologically based computational models of high-level cognition. *Science* . 2006; 314(5796): 91–94.
[Google Scholar](#) [Crossref](#) [PubMed](#)
29. O'Reilly RC, Frank MJ. Making working memory work: a computational model of learning in the prefrontal cortex

and basal ganglia. *Neural Comput* . 2006; 18(2): 283–328.

[Google Scholar](#) [Crossref](#) [PubMed](#)

30. Basner M, Rao H, Goel N, Dinges DF. Sleep deprivation and neurobehavioral dynamics. *Curr Opin Neurobiol* . 2013; 23(5): 854–863.
[Google Scholar](#) [Crossref](#) [PubMed](#)
31. Chee MW, Chuah LY. Functional neuroimaging insights into how sleep and sleep deprivation affect memory and cognition. *Curr Opin Neurol* . 2008; 21(4): 417–423.
[Google Scholar](#) [Crossref](#) [PubMed](#)
32. Slama H, Deliens G, Schmitz R, Peigneux P, Leproult R. Afternoon nap and bright light exposure improve cognitive flexibility post lunch. *PLoS One* . 2015; 10(5): e0125359.
[Google Scholar](#) [Crossref](#) [PubMed](#)
33. Dreisbach G, Müller J, Goschke Tet al. Dopamine and cognitive control: the influence of spontaneous eyeblink rate and dopamine gene polymorphisms on perseveration and distractibility. *Behav Neurosci* . 2005; 119(2): 483–490.
[Google Scholar](#) [Crossref](#) [PubMed](#)
34. Slagter HA, Georgopoulou K, Frank MJ. Spontaneous eye blink rate predicts learning from negative, but not positive, outcomes. *Neuropsychologia* . 2015; 71: 126–132. doi: 10.1016/j.neuropsychologia.2015.03.028.
[Google Scholar](#) [Crossref](#) [PubMed](#)
35. Müller J, Dreisbach G, Brocke B, Lesch KP, Strobel A, Goschke T. Dopamine and cognitive control: the influence of spontaneous eyeblink rate, DRD4 exon III polymorphism and gender on flexibility in set-shifting. *Brain Res* . 2007; 1131(1): 155–162.
[Google Scholar](#) [Crossref](#) [PubMed](#)
36. Tharp IJ, Pickering AD. Individual differences in cognitive-flexibility: the influence of spontaneous eyeblink rate, trait psychoticism and working memory on attentional set-shifting. *Brain Cogn* . 2011; 75(2): 119–125.
[Google Scholar](#) [Crossref](#) [PubMed](#)
37. Jongkees BJ, Colzato LS. Spontaneous eye blink rate as predictor of dopamine-related cognitive function-A review. *Neurosci Biobehav Rev* . 2016; 71: 58–82. doi: 10.1016/j.neubiorev.2016.08.020.
[Google Scholar](#) [Crossref](#) [PubMed](#)
38. Barbato G, De Padova V, Paolillo AR, Arpaia L, Russo E, Ficca G. Increased spontaneous eye blink rate following prolonged wakefulness. *Physiol Behav* . 2007; 90(1): 151–154.
[Google Scholar](#) [Crossref](#) [PubMed](#)
39. Barbato G, Ficca G, Beatrice M, Casiello M, Muscettola G, Rinaldi F. Effects of sleep deprivation on spontaneous eye blink rate and alpha EEG power. *Biol Psychiatry* . 1995; 38(5): 340–341.
[Google Scholar](#) [Crossref](#) [PubMed](#)
40. Caffier PP, Erdmann U, Ullsperger P. The spontaneous eye-blink as sleepiness indicator in patients with obstructive sleep apnoea syndrome-a pilot study. *Sleep Med* . 2005; 6(2): 155–162.
[Google Scholar](#) [Crossref](#) [PubMed](#)
41. Bohne A, Savage CR, Deckersbach Tet al. Visuospatial abilities, memory, and executive functioning in trichotillomania and obsessive-compulsive disorder. *J Clin Exp Neuropsychol* . 2005; 27(4): 385–399.

[Google Scholar](#) [Crossref](#) [PubMed](#)

42. Volkow ND, Tomasi D, Wang GJ et al. Evidence that sleep deprivation downregulates dopamine D2R in ventral striatum in the human brain. *J Neurosci* . 2012; 32(19): 6711–6717.
[Google Scholar](#) [Crossref](#) [PubMed](#)
43. Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex “Frontal Lobe” tasks: a latent variable analysis. *Cogn Psychol* . 2000; 41(1): 49–100.
[Google Scholar](#) [Crossref](#) [PubMed](#)
44. Beck AT, Steer RA, Garbin MG. Psychometric properties of Beck Depression Inventory: twenty-five years of evaluation. *Clin Psychol Rev* . 1988; 8(1): 77–100.
[Google Scholar](#) [Crossref](#)
45. Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol* . 1976; 4(2): 97–110.
[Google Scholar](#) [PubMed](#)
46. Krupp LB, LaRocca NG, Muir-Nash J, Steinberg AD. The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. *Arch Neurol* . 1989; 46(10): 1121–1123.
[Google Scholar](#) [Crossref](#) [PubMed](#)
47. Ellis BW, Johns MW, Lancaster R, Raptopoulos P, Angelopoulos N, Priest RG. The St. Mary’s Hospital sleep questionnaire: a study of reliability. *Sleep* . 1981; 4(1): 93–97.
[Google Scholar](#) [Crossref](#) [PubMed](#)
48. Akerstedt T, Gillberg M. Subjective and objective sleepiness in the active individual. *Int J Neurosci* . 1990; 52(1-2): 29–37.
[Google Scholar](#) [Crossref](#) [PubMed](#)
49. Lee KA, Hicks G, Nino-Murcia G. Validity and reliability of a scale to assess fatigue. *Psychiatry Res* . 1991; 36(3): 291–298.
[Google Scholar](#) [Crossref](#) [PubMed](#)
50. Dinges DI, Powell JW. Microcomputer analysis of performance on a portable, simple visual RT task sustained operations. *Behav Res Methods Instrum Comput* . 1985; 17(6): 652–655.
[Google Scholar](#) [Crossref](#)
51. Schmidt C, Collette F, Leclercq Y et al. Homeostatic sleep pressure and responses to sustained attention in the suprachiasmatic area. *Science* . 2009; 324(5926): 516–519.
[Google Scholar](#) [Crossref](#) [PubMed](#)
52. van Zomeren AH, Brouwer WH. *Clinical Neuropsychology of Attention* . Oxford: Oxford University Press, 1994.
53. Fiehmig HC, Steinborn M, Langner R, Scholz A, Westhoff K. Assessing intraindividual variability in sustained attention: reliability, relation to speed and accuracy, and practice effects. *Psychol Sci* . 2007; 49(2): 132–149.
54. Chermahini SA, Hommel B. The (b)link between creativity and dopamine: spontaneous eye blink rates predict and dissociate divergent and convergent thinking. *Cognition* . 2010; 115(3): 458–465.
[Google Scholar](#) [Crossref](#) [PubMed](#)

55. Zhang T, Mou D, Wang C et al. Dopamine and executive function: Increased spontaneous eye blink rates correlate with better set-shifting and inhibition, but poorer updating. *Int J Psychophysiol* . 2015; 96(3): 155–161.
[Google Scholar](#) [Crossref](#) [PubMed](#)
56. Owen AM, McMillan KM, Laird AR, Bullmore E. N-back working memory paradigm: a meta-analysis of normative functional neuroimaging studies. *Hum Brain Mapp* . 2005; 25(1): 46–59.
[Google Scholar](#) [Crossref](#) [PubMed](#)
57. Massat I, Slama H, Kavec M et al. Working memory-related functional brain patterns in never medicated children with ADHD. *PLoS One* . 2012; 7(11): e49392.
[Google Scholar](#) [Crossref](#) [PubMed](#)
58. Massat I, Slama H, Villemonteix T et al. Hyperactivity in motor response inhibition networks in unmedicated children with attention deficit-hyperactivity disorder. *World J Biol Psychiatry* . 2016; 19(2): 101–111.
[Google Scholar](#) [Crossref](#) [PubMed](#)
59. Rubia K, Smith AB, Brammer MJ, Toone B, Taylor E. Abnormal brain activation during inhibition and error detection in medication-naïve adolescents with ADHD. *Am J Psychiatry* . 2005; 162(6): 1067–1075.
[Google Scholar](#) [Crossref](#) [PubMed](#)
60. Cohen JD, MacWhinney B, Flatt M, Provost J. PsyScope: a new graphic interactive environment for designing psychology experiments. *Behav Res Methods Instrum Comput* . 1993; 25(2): 257–271.
[Google Scholar](#) [Crossref](#)
61. International School of Advanced Studies (SISSA/ISAS). *PsyScope X project (version 77 build) [computer software]* .
62. Rosner B. Percentage points for a generalized ESD many-outlier procedure. *Technometrics* . 1983; 25(2): 165–172.
[Google Scholar](#) [Crossref](#)
63. Cole RJ, Kripke DF, Gruen W, Mullaney DJ, Gillin JC. Automatic sleep/wake identification from wrist activity. *Sleep* . 1992; 15(5): 461–469.
[Google Scholar](#) [Crossref](#) [PubMed](#)
64. JASP Team (2018). *JASP (Version 0.8.5) [Computer software]* .
65. Lien MC, Ruthruff E, Remington RW, Johnston JC. On the limits of advance preparation for a task switch: do people prepare all the task some of the time or some of the task all the time? *J Exp Psychol Hum Percept Perform* . 2005; 31(2): 299–315.
[Google Scholar](#) [Crossref](#) [PubMed](#)
66. Mayr U, Kliegl R. Task-set switching and long-term memory retrieval. *J Exp Psychol Learn Mem Cogn* . 2000; 26(5): 1124–1140.
[Google Scholar](#) [Crossref](#) [PubMed](#)
67. De Jong R, Berendsen E, Cools R. Goal neglect and inhibitory limitations: dissociable causes of interference effects in conflict situations. *Acta Psychol (Amst)* . 1999; 101(2-3): 379–394.
[Google Scholar](#) [Crossref](#) [PubMed](#)
68. Duncan J, Emslie H, Williams P, Johnson R, Freer C. Intelligence and the frontal lobe: the organization of goal-

directed behavior. *Cogn Psychol* . 1996; 30(3): 257–303.

[Google Scholar](#) [Crossref](#) [PubMed](#)

69. Killgore WD, McBride SA, Killgore DB, Balkin TJ. The effects of caffeine, dextroamphetamine, and modafinil on humor appreciation during sleep deprivation. *Sleep* . 2006; 29(6): 841–847.

[Google Scholar](#) [Crossref](#) [PubMed](#)

70. McIntire LK, McKinley RA, Goodyear C, McIntire JP. Detection of vigilance performance using eye blinks. *Appl Ergon* . 2014; 45(2): 354–362.

[Google Scholar](#) [Crossref](#) [PubMed](#)

71. Lim J, Dinges DF. A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychol Bull* . 2010; 136(3): 375–389.

[Google Scholar](#) [Crossref](#) [PubMed](#)

72. Tucker AM, Whitney P, Belenky G, Hinson JM, Van Dongen HP. Effects of sleep deprivation on dissociated components of executive functioning. *Sleep* . 2010; 33(1): 47–57.

[Google Scholar](#) [Crossref](#) [PubMed](#)

73. Phillips LH. Do ‘frontal tests’ measure executive functions? Issues of assessment and evidence from fluency tests. In: Rabbitt P, ed. *Methodology of Frontal and Executive Function* . Hove: Psychology Press, 1997:191–213.

74. Miyata S, Noda A, Ozaki Net al. Insufficient sleep impairs driving performance and cognitive function. *Neurosci Lett* . 2010; 469(2): 229–233.

[Google Scholar](#) [Crossref](#) [PubMed](#)

75. Teran-Perez G, Ruiz-Contreras AE, Gonzalez-Robles ROet al. Sleep deprivation affects working memory in low but not in high complexity for the N-back test. *Neurosci Med* 2012; 3(4): 380–386.

[Google Scholar](#) [Crossref](#)

76. Lo JC, Groeger JA, Santhi Net al. Effects of partial and acute total sleep deprivation on performance across cognitive domains, individuals and circadian phase. *PLoS One* . 2012; 7(9): e45987.

[Google Scholar](#) [Crossref](#) [PubMed](#)

77. Acheson A, Richards JB, de Wit H. Effects of sleep deprivation on impulsive behaviors in men and women. *Physiol Behav* . 2007; 91(5): 579–587.

[Google Scholar](#) [Crossref](#) [PubMed](#)

78. Chuah YM, Venkatraman V, Dinges DF, Chee MW. The neural basis of interindividual variability in inhibitory efficiency after sleep deprivation. *J Neurosci* . 2006; 26(27): 7156–7162.

[Google Scholar](#) [Crossref](#) [PubMed](#)

79. Oh J, Han M, Peterson BS, Jeong J. Spontaneous eyeblinks are correlated with responses during the Stroop task. *PLoS One* . 2012; 7(4): e34871.

[Google Scholar](#) [Crossref](#) [PubMed](#)

80. Hopstaken JF, van der Linden D, Bakker AB, Kompier MA. A multifaceted investigation of the link between mental fatigue and task disengagement. *Psychophysiology* . 2015; 52(3): 305–315.

[Google Scholar](#) [Crossref](#) [PubMed](#)

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