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Sleep deprivation affects the sensitivity of proactive and reactive action monitoring: A behavioural and ERP analysis

Céline Ramdani[a,b,*], Laurence Carbonnell[a,c], Arnaud Rabat[a], Cédric Meckler[a], Boris Burle[b], Thierry Hasbroucq[b], Franck Vidal[b,a]

[a] Institut de Recherche Biomédicale des Armées, Toulon, France
[b] Laboratoire de Neurobiologie de la Cognition, Aix-Marseille Univ/CNRS, Marseille, France
[c] Laboratoire de Psychologie Cogntive, Aix-Marseille Univ/CNRS, Marseille, France

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ABSTRACT

We studied the impact of sleep deprivation on action monitoring. Each participant performed a Simon task after a normal night of sleep and after 26 h of awakening. Reaction time (RT) distributions were analyzed and the sensitivity of the error negativity (Ne/Ne like) to response correctness was examined.

Results showed that (1) the Simon effect persisted for the longest RTs only after sleep deprivation and (2) the sensitivity of the Ne/Ne like to correctness decreased after sleep deprivation, especially on incongruent trials. This suggests that after sleep deprivation (1) the ability to inhibit prepotent response tendencies is impaired and (2) the sensitivity of a response monitoring system as revealed by the error negativity is less sensitive to performance.

In conclusion, action monitoring was affected by sleep deprivation as revealed by distributional analyses and the sensitivity of the Ne/Ne like to performance, which may be attributed to the fragility of prefrontal structures to sleep deprivation.

1. Introduction

Failing to respond correctly in a timely fashion to a stimulus, is a hallmark of sleep deprived state (Doran et al., 2001; Lim et al., 2008). Response accuracy under speed-stress may rely, at least in part, on "action monitoring" that is on the ability (i) to overcome prepotent response tendencies and (ii) to detect and correct errors. The "error negativity" (Ne) (Falkenstein et al., 1991) or "Error-Related Negativity" (ERN) (Gehring et al., 1993), an event-related potential starting just after electromyographic (EMG) onset and peaking just after mechanical error commission in reaction time (RT) tasks, is widely considered as an index of action monitoring (Holroyd et al., 2002). Different research groups (Hsieh et al., 2007; Murphy et al., 2006; Tsai et al., 2005) investigated the effect of sleep deprivation (SD) on the Ne. While Murphy et al. (2006) reported no effect of SD on Ne amplitude, both Tsai et al. (2005) and Hsieh et al. (2010) found that SD was associated with smaller Ne. The latter results suggest that SD impairs action monitoring. Therefore, both performance and EEG data converge in suggesting that SD impairs action monitoring.

Reaction Time Distribution and EMG patterns analyses under speed-stress allow to dissociate two complementary modes of action monitoring that contribute to response accuracy that will be respectively termed "reactive control" and "proactive control" in what follows.

1.1. Reactive control: detection and correction of errors

On numerous trials, the overt correct response is preceded by a covert incorrect response which can be evidenced by the presence of a subthreshold EMG burst associated with the incorrect response (Smid et al., 1990). These so-called "partial errors" are successfully suppressed, preventing a full performance error (Burle et al., 2002).

Thus, with respect to action monitoring, three categories of trials should be distinguished: full performance errors, partial errors, and "pure" correct trials. Partial error trials reflect the implementation of a reactive control that remedies errors before they turn into full performance errors.

Analysing the Laplacian transforms of EEG activity for all responses sorted as a function of the EMG pattern leading to correct, partial error and error responses showed that the Ne amplitude (Vidal et al., 2000, 2003) depends on the category of trial. The amplitude of the Ne is maximal for errors, minimal for correct responses and intermediate for partial errors. Such a gradation suggests that the negativities observed for the three types of responses reflect

* Corresponding author at: Institut de Recherche Biomédicale des Armées, Antenne Toulon, Boulevard Sainte Anne, BP 20548, 83041 Toulon Cedex 09, France. Tel.: +33 4 83 16 28 51; fax: +33 4 83 16 28 59.
E-mail address: celineramdani@hotmail.fr (C. Ramdani).

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reactive control (Vidal et al., 2000, 2003). This notion is further supported by the fact that on one hand, the Ne-like is also sensitive to participant's performance (Allain et al., 2004) and that on the other hand, source analyses indicate that the Ne and Ne/like are generated by similar structures (Roger et al., 2010; Hoffmann and Falkenstein, 2010). A meta-analysis (Ridderinkhof et al., 2004a) indicates that the generator of the Ne is located within the Rostral Cingulate Zone which is known to be largely involved in cognitive control (Desmet et al., 2011). As the Ne starts just after EMG onset, it is likely that it reflects a reaction to correctness of the action monitoring system. As such, it can also be considered as an index of reactive control.

1.2. Proactive control: overcoming prepotent responses

Distribution analyses reveal that a proactive (i.e. preventive) control is implemented for correct trials in a behavioural paradigm known as the "Simon task".

This task, which is known to be particularly error- and partial error-prone (Craft and Simon, 1970; Hasbroucq et al., 1999, 2009; Burle et al., 2002), provides experimental contexts and theoretical models for analyzing proactive action monitoring when irrelevant external stimulus information elicits response impulses that interfere with goal-directed actions (Kornblum et al., 1990; Ridderinkhof, 2002; Van den Wildenberg et al., 2010). In the most common version of the Simon task (Hommel, 2011), the participants have to choose between a left- and a right-hand key press according to the colour of a visual stimulus presented a few degrees either to the left or the right of a fixation point. The performance expressed both in terms of error rate and RT is better when the required response corresponds spatially to the irrelevant stimulus location (congruent association) than when it does not correspond (incongruent association). This effect is termed the "Simon effect" (Hedge et al., 1975; Simon, 1990; Hommel, 2011).

A widely accepted interpretation of the Simon effect is that the irrelevant stimulus location automatically engages a response impulse in the spatially corresponding hand via a fast route while the relevant stimulus colour must be translated into the required response according to the task instructions via a slower controlled route (De Jong et al., 1994; Kornblum, 1994; Proctor et al., 1995).

When the stimulus-response association is congruent, the impulse triggered by the irrelevant stimulus location activates the required response, which facilitates response processing. In contrast, when the stimulus-response association is incongruent, the impulse triggered by the irrelevant location activates the non-required response which competes with the required one. This competition occurs at a cost and the performance is degraded.

RT distribution analyses reveal that the size of the Simon effect diminishes as RT increases (De Jong et al., 1994). In order to account for this effect, Ridderinkhof (2002) proposed the activation-suppression hypothesis according to which an active suppression mechanism of the fast route builds up over time. As this inhibitory mechanism is assumed to take time to develop, a reduction of the Simon effect appears with increasing RT. According to this view, faster responses should more likely be impulsive actions that are captured by the irrelevant stimulus dimension (Van den Wildenberg et al., 2010), via the fast route. Conversely, slower responses are less vulnerable to impulsive actions that are captured by the irrelevant stimulus dimension because selective suppression has time to build up and counteracts automatic impulses (Van den Wildenberg et al., 2010). Therefore, the active suppression of the fast automatic route, as revealed by the reduction of the Simon effect with increasing RTs, is assumed to represent a mechanism aimed at preventing the emission of incorrect activations when the required responses competes with the (non-required) automatically activated one (Ridderinkhof, 2002; Van den Wildenberg et al., 2010; Wylie et al., 2009). As this mechanism is aimed at avoiding the emission of prepotent incorrect activations, it can be considered as an action monitoring process. According to the activation-suppression hypothesis, the reduction of the Simon effect as RT increases manifests the implementation of an action monitoring process aimed at preventing the expression of EMG outputs associated to erroneous responses (partial errors and full performance errors, for a review see Van den Wildenberg et al., 2010). Since this process acts *before* the emission of any incorrect EMG activation, it can be considered as proactive (as opposed to the elicitation of the Ne, which occurs *after* incorrect EMG onset). To sum up, RT distribution analyses, because of their sensitivity to the fast route suppression, are efficient tools to explore proactive action monitoring and the impact of SD on these processes.

The first aim of the present study was to examine the effect of a 26 h SD on reactive and proactive control. In the event that this control is impaired, SD was expected to reduce the difference in Ne between the three response types: errors, partial errors and pure correct responses. In the event that proactive control is impaired, SD was expected to counteract the decrease of the Simon effect for long RTs.

2. Material and method

2.1. Participants

Six men and six women (mean age: 26; range: 21–37) volunteered in this experiment. They were all right-handed, no smokers and had a normal or a corrected to normal vision. Participants were nurses students at the Ecole du Personnel Paramédical des Armées de Toulon. They were paid 200 Euros for their participation. Informed written consent was obtained according to the declaration of Helsinki and the local ethics committee approved the experiment.

2.2. Task

The stimuli were digits 1, 2, 3, 4, 5, and 6 (2 cm height, 0.63° vertically) presented either to the right or the left of a central fixation point (a central cross 0.4 cm height and 0.4 cm width); the distance between the fixation point and each digit subtended 1.24° of visual angle. Half of the participants responded with the right thumb for even digits on the right button of a response pad (Neuroscan®) and with the left thumb on the left button for odd digits; the other half performed the reverse mapping. They were asked to respond as soon and as accurately as possible after the apparition of the stimulus. When the stimulus was presented on the same side as the correct response, the stimulus-response association was congruent. When the stimulus was presented on the side opposite to the correct response the stimulus-response association was incongruent. A block contained 50% of congruent trials and 50% of incongruent ones. A trial began with the presentation of a stimulus. Participants' responses turned off the stimulus and 500 ms later the next stimulus was presented. If participants had not responded 800 ms after stimulus onset, the stimulus was turned off and the next stimulus was displayed 500 ms later.

2.3. Design and procedure

Participants were comfortably seated in an armchair in a sound attenuated and air-conditioned Faraday cage. They faced a faradised screen on which the stimuli were presented. Participants were asked to maintain their gaze on the fixation point.

At the beginning of an experimental session, participants practiced one training block of 145 trials to reach a stable level of RT performance. Then, they were required to complete 16 blocks

of 145 trials each. A block lasted about 2 min 30 s. There was 1-min break between two blocks and 5 min break every four blocks. Between each block, if participants committed more than 10% of errors, they were asked to commit fewer errors in the following block. The training block was discarded from statistical analyses.

The experiment comprised two experimental sessions differing by the level of vigilance: (1) the "control condition": the participant performed the task after a normal night of sleep and (2) the "SD condition": the participant performed the task after 26 h of awakening (the day and the night before the test). Each participant performed both sessions. The order of conditions was counterbalanced between participants. Participants were asked not to take stimulating substances or alcohol the day and the night before the SD session and the day and the night before the control condition. For the night of SD, participants arrived at the laboratory at approximately 9:30 p.m. and were then kept awake throughout the night, watched by a member of the laboratory. Recreational and non-tiring activities were proposed (DVD, games, books, etc.). Participants were filmed on the whole night. The night before the control condition they slept at home. We gave them a sleep diary to evaluate the quality and the quantity of their sleep. The task started at 9:00 a.m. for both conditions. The two vigilance conditions were separated from 2 to 4 weeks and each participant performed these two conditions the same day of the week. Monday was eliminated to avoid the week-end effect.

2.4. Electrophysiological recordings

Electroencephalogram (EEG), electromyogram (EMG), and electro-oculogram (EOG) were recorded continuously from preamplified Ag/AgCl electrodes (BIOSEMI Active-Two electrodes, Amsterdam). The signal was filtered and digitized on-line (bandwidth: 0–268 Hz, 3 db/octave, sampling rate: 1024 Hz).

For EEG, 64 recording electrodes were disposed according to the 10/20 system with CMS-DRL as reference and ground. A 65th electrode on the left mastoid was used to reference the signal offline.

Electrodes for vertical and horizontal EOG were respectively at Fp1 and below the left eye, and at the outer canthus of the left and right eyes.

For EMG, two electrodes were pasted on the skin of the thenar eminence of each thumb over the *flexor pollicis brevis* about two centimetres apart.

2.5. Data processing

2.5.1. Chronometric data processing

Trials with RT shorter than 100 ms were rejected. The overall RT was fractioned into premotor time (PMT): from stimulus onset to EMG onset, and motor time (MT): from EMG onset to the mechanical response (Botwinick et al., 1966). PMT can be considered as an index of central processing whereas MT reflects the implementation of peripheral motor processes.

We have distinguished correct trials containing a partial error, from correct trials without partial error and these trials were called correct responses.

Error rate was calculated by dividing the number of errors (an error was defined as a mechanical response, i.e. a button press on the non-required side) by the number of effective trials (correct responses, partial errors and errors). In addition to usual analyses of the mean values of chronometric data, we performed distribution analyses. To this aim, we used the "Vincent averaging" or "Vincentization" technique (Ratcliff, 1979; Vincent, 1912). To make these analyses, we binned the RT distribution of correct response in ten classes of equal size (same number of trials) and we computed the mean of each class. From the individual vincentized distributions, "delta plots" were estimated (Ridderinkhof et al., 2004b). Delta plots were obtained by plotting for each bin, the size of the congruity effect (incongruent minus congruent) as a function of the RT.

RT were analyzed by means of ANOVAs. The ANOVAs involved three within-participants factors: State of vigilance (control, SD), Congruity (congruent, incongruent) and Bin.

Delta plots were submitted to trend analyses to study the shape of the curves.

According to the activation-suppression hypothesis, the tendency to react impulsively is revealed by the relationship between errors and RT. The smaller the RT, the higher the likelihood the response has been activated via the fast route. On congruent associations, the fast route leads to the correct response whereas on incongruent associations, it leads to the incorrect one. Plotting accuracy rates as a function of RT (conditional accuracy function or CAF) thus permits to study the strength of the fast route. Now, some correct trials contain partial errors. In CAFs, these peculiar trials are classified as correct responses although the first EMG activation is incorrect. This is why we chose to study here incorrect activation rates (error rates + partial error rates) as a function of RT (conditional incorrect activation function or CIAF), instead of accuracy rates.

To determine the incorrect activation rate for each condition, PMTs of errors and correct responses and latencies of partial errors distributions were binned in five bins of equal size (same number of trials). Incorrect activation rates were calculated for each bin. To this aim, we plotted the incorrect activation rate as a function of all PMT distribution (PMT of correct responses, PMT of errors and latency of partial errors, this is the time between the stimulus appearance and the onset of the incorrect EMG activation in correct responses containing a partial error).

CIAFs were realized for each condition: control–congruent, control–incongruent, SD–congruent and SD–incongruent.

Percentage data cannot be normally tested by ANOVA as their means and variances are closely related. However, the arcsine transform is efficient to stabilizing the variances of these data (Winer, 1970). All percentages were therefore transformed accordingly before being submitted to ANOVAs with state of vigilance, congruity and bin as within-participants' factors.

2.5.2. Electrophysiological data

After the experimental session, electrophysiological data were numerically filtered (EEG: high-pass = 0.02 Hz). Response-related activities were averaged time-locked to EMG onset. Ocular artefacts were subtracted (Gratton et al., 1983). Then, trials in which the subtraction was deemed unsatisfactory, or containing other artefacts, were rejected on the basis of trial-by-trial visual inspection of the monopolar recordings: 9.6% of the trials were rejected in the control condition and 10.2% in SD one.

EEG was averaged time-locked to EMG onset, detected by visual inspection of each trial (Hasbroucq et al., 1999).

The Laplacian transformation was applied on the monopolar averages, after spherical spline interpolation, with 3 as the degree of spline and a maximum of 15° for the Legendre polynomial (Perrin et al., 1987), approximation parameter Lambda: 1.0e−005.

Based on the Laplacian transformed grand averages obtained at Cz, we determined the latency of the negative peak of the Ne and that of the positive peak preceding it, which was taken as representing the onset of the Ne. We then defined two time windows of 40 ms, each centred on the mean latency of each peak (the positive and the negative ones). For each participant in each condition, we measured the peak-to-peak amplitude (which is thus baseline-free) defined as the difference between the surfaces under the curve for each window.

These values were then submitted to repeated-measures canonical analyses of variance (ANOVA). The ANOVA involved two

Table 1
Results of mixed-effect ANOVAs with state of vigilance (S) and congruity effect (C) as factors.

	Control condition		Sleep deprivation condition		Significant effect
	Congruent	Incongruent	Congruent	Incongruent	
Correct RT (ms)	421	429	446	458	S*, C**, S × C*
Correct PMT (ms)	337	344	348	360	C**, S × C*
Correct MT (ms)	84	85	98	98	S**
Error rate	7.44%	7.6%	7.00%	9.67%	S × C**
Incorrect activation rate	16.44%	17.75%	18.82%	23.31%	S*, S × C**

* $P < 0.05$.
** $p < 0.01$

within-participants factors: state of vigilance (control, SD) and congruity (congruent, incongruent) for mean results.

3. Result

3.1. Behavioural data

3.1.1. Pure correct trials: RT (Table 1)

RT was longer in SD condition (452 ms) than in control condition (425 ms) ($F(1, 11) = 5.96$, $p < .05$). RT was also longer on incongruent associations (443 ms) than on congruent associations (433 ms) ($F(1, 11) = 17.38$, $p < .01$). These two main effects were superseded by a two-way interaction between state of vigilance and congruity ($F(1, 11) = 5.89$, $p < .05$). The effect of congruity was significant in control condition (incongruent minus congruent = 8 ms; $F(1, 11) = 11.40$, $p < .01$) and in SD condition (incongruent minus congruent = 12 ms; $F(1, 11) = 19.23$, $p < .01$) but it was more important after SD than after a normal night of sleep.

3.1.2. Vincentizations and delta plots (Fig. 1)

An important feature of RT distribution in the Simon task is the fact that the Simon effect (incongruent minus congruent) decreases and can even disappear as RTs become longer (De Jong et al., 1994; Ridderinkhof, 2002). The statistical analysis highlighted a nearly significant second-order interaction between state of vigilance, congruity and bin ($F(9, 99) = 1.93$, $p = .056$). In control condition, we found an interaction between bin and congruity ($F(9, 99) = 9.92$, $p < .001$). The Simon effect decrease and even disappear as RTs increased (Table 2) On the other hand, after SD, there was no interaction between bin and congruity ($F(9, 99) = 1.72$, $p = .09$).

Concerning the analysis of delta plots in SD condition and in control condition, polynomial contrast analyses showed a linear–linear interaction ($F(1, 11) = 4.62$, $p < .05$). This interaction revealed that the shape of the curve was different depending on the state of vigilance. More specially, the shape of the end of the curve was different: linear trend was significant on control condition ($F(1, 11) = 5.16$, $p < .05$) but not on SD condition ($F(1, 11) < 1$). To sum up on control condition congruity effect decreased and disappeared when RTs became longer. On SD condition, the congruity effect decreased less and did not disappear. The increase of Simon effect after SD, observed on mean RT was therefore not global all over the distribution but can be attributed to trials with longest RT in which the congruity effect did not disappear.

Table 2
Statistical effect of congruity in each bin on control condition.

	Size of Simon effect: incongruent RT minus congruent RT (ms)	
Bin 1	13	$F(1, 11) = 62.95$, $p < 0.001$
Bin 2	14	$F(1, 11) = 57.25$, $p < 0.001$
Bin 3	14	$F(1, 11) = 53.55$, $p < 0.001$
Bin 4	13	$F(1, 11) = 32.89$, $p < 0.001$
Bin 5	11	$F(1, 11) = 21.97$, $p < 0.001$
Bin 6	9	$F(1, 11) = 10.22$, $p < 0.01$
Bin 7	5	$F(1, 11) = 2.07$, $p = 0.18$
Bin 8	3	$F(1, 11) < 1$
Bin 9	1	$F(1, 11) < 1$
Bin 10	−2	$F(1, 11) < 1$

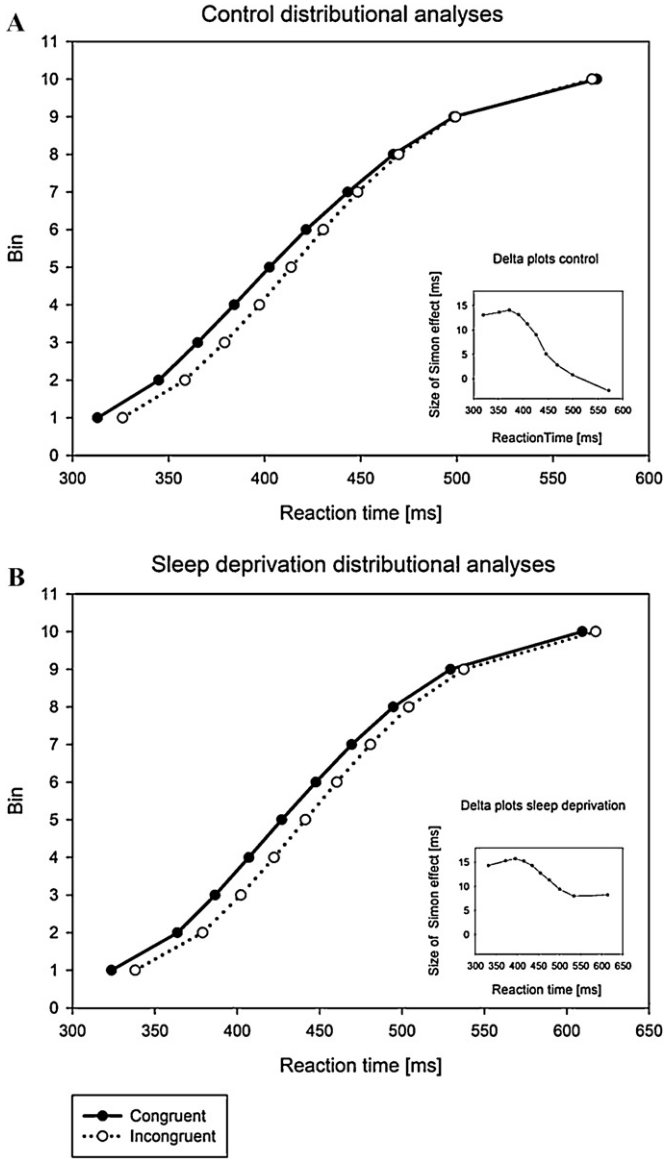

Fig. 1. Distributional analysis for congruent and incongruent associations. (A) Control condition. (B) Sleep deprivation condition. *Insets:* delta plots for each condition (control and sleep deprivation). After sleep deprivation the shape of the end of the curve differs in the 2 conditions, revealing an impairment of the inhibitory mechanism of the fast/automatic route.

3.1.3. Correct response: premotor time (PMT) (Table 1)

As expected, we found a main effect of congruity on PMT which essentially reflected the duration of central motor processes (342 ms on congruent condition and 354 ms on incongruent condition ($F(1, 11) = 14.67$, $p < .01$). Whereas there was no main effect of state of vigilance ($F(1, 11) = 2.06$, $p = .18$), we observed an interaction between congruity and state of vigilance ($F(1, 11) = 9.28$, $p < .02$). This interaction revealed that the congruity effect was more important after SD (incongruent minus congruent = 12 ms; $F(1, 11) = 17.25$, $p < .01$) than in control condition (incongruent minus congruent = 8 ms; $F(1, 11) = 9.18$, $p < .05$).

3.1.4. Correct response: motor time (MT) (Table 1)

In accordance with previous studies, ANOVA revealed no effect of congruity (91 ms on congruent condition and 91.5 ms on incongruent condition ($F(1, 11) = 2.21$, $p = .16$)). There was a main effect of state of vigilance on MT: MT increased of 13.5 ms after SD (98 ms) compared to control condition (84.5 ms) ($F(1, 11) = 12.69$, $p < .01$) which corresponds to an 16% increase of MT.

Most of the SD effect on overall RT was attributable to an increase of MT which reflects the duration of peripherical motor execution processes.

3.1.5. Error rates (Table 1)

There was no main effect of congruity on error rate (congruent: 7.28%, incongruent: 8.63%, $F(1, 11) = 2.93$, $p = .11$) and no main effect of state of vigilance (SD: 8.34%, control: 7.58%, $F(1, 11) = 1.05$, $p = .32$).

There was, however, an interaction between these two factors (state of vigilance and congruity effect ($F(1, 11) = 23.66$, $p < .001$). Contrast analyses showed that there was an effect of stage of vigilance only on incongruent associations so only when the stimulus-response association was more controlled (SD: 9.67%, control: 7.60%; $F(1, 11) = 8.88$, $p < .02$). On congruent associations, when the stimulus-response association was more automatic, there was no effect of state of vigilance ($F < 1$).

In short, the error rate increased after a night of SD only in incongruent stimulus-response associations, when the automatic route had to be inhibited.

3.1.6. Incorrect activation rates (Table 1)

The incorrect activation rate is the sum of errors and partial errors.

Mean incorrect activation rate increased from 17.15% on control condition to 20.77% on SD condition ($F(1, 11) = 6.42$, $p < .05$). The increase of incorrect activation rate on incongruent associations (20.53%) compared to congruent associations (17.38%) was not statistically significant ($F(1, 11) = 4.31$, $p = .06$). ANOVA revealed an interaction between state of vigilance and congruity ($F(1, 11) = 20.25$, $p < .001$). Contrasts analyses showed that incorrect activation rate increased after SD only when the stimulus-response association was incongruent (increase of 5.57% from control condition to SD condition; $F(1, 11) = 11.80$, $p < .01$). On congruent stimulus-response associations, this increase, after a SD, was not significant (increase of 1.67% from control condition to SD condition; $F(1, 11) = 1.78$, $p = .21$).

3.1.7. Conditional incorrect activation functions (Fig. 2)

There was an interaction between congruity and bin ($F(1, 11) = 17.08$, $p < 001$): The congruity effect present for the first bin decreased and even disappeared as RT increased (Incongruent minus congruent: bin 1: 20.41% ($F(1, 11) = 26.24$, $p < 001$; bin 2: 3.46%, $F(1, 11) = 3.06$, $p = .11$; bin 3: −3.63%, $F(1, 11) = 2.20$, $p = .16$; bin 4: −4.47%, $F(1, 11) = 11.03$, $p < .01$; bin 5: −1.76%, $F(1, 11) < 1$).

As for mean incorrect activation rate, ANOVA revealed an interaction between congruity effect and state of vigilance ($F(1, 11) = 21.93$, $p < .001$). This interaction showed that there was an effect of state of vigilance only on incongruent condition ($F(1, 11) = 12.69$, $p < .01$) and not on congruent condition ($F(1, 11) < 1$).

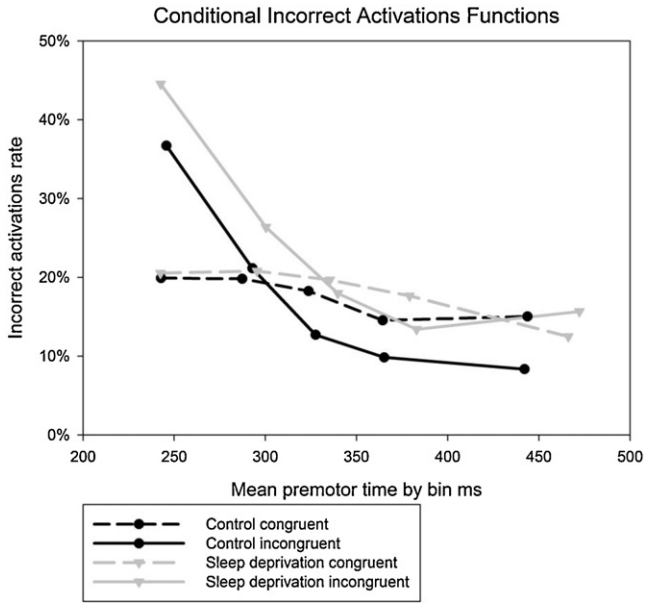

Fig. 2. Conditional incorrect activations functions for congruent and incongruent associations in control condition and sleep deprivation condition. For both conditions, incorrect activations are associated with fastest reaction times on incongruent associations. The effect of sleep deprivation on incorrect activation rate on incongruent associations appears all over the distribution, not only for a subcategory of trials, showing a global effect of sleep deprivation on incorrect activation rate for incongruent stimulus-response associations.

The effect of state of vigilance on incorrect activation rate on incongruent condition appeared all over the distribution, not only for a subcategory of trials. It was a global effect of SD on incorrect activation rate when the stimulus-response association was incongruent.

3.2. Electroencephalographical data

Errors elicited a large negative wave, maximal at central electrode Cz, peaking around 140 ms after the EMG onset. The same negative wave was obtained for partial errors, peaking around 100 ms after EMG onset of partial errors. A smaller wave peaking at the same topographical site was also obtained for correct responses, peaking around 150 ms after EMG onset of these responses.

3.2.1. Sensitivity of the Ne/Ne-like (Figs. 3 and 4)

On Laplacian data, in ANOVA, in addition to state of vigilance and congruity, we introduced a third within-participants factor: the correctness of the trial (correct responses, partial errors and errors).

The statistical analysis highlighted a significant second-order interaction between these three factors ($F(2, 22) = 4.72$, $p < .02$).

Amplitude of Ne on errors (Fig. 3) was smaller in SD condition than in control condition ($F(1, 11) = 15.56$, $p < .01$). ANOVA revealed a trend ($F(1, 11) = 4.73$, $p = .052$) to congruity effect. Statistical analyze did not showed any interaction between congruity and state of vigilance ($F(1, 11) = 2.53$, $p = 0.14$).

Amplitude of Ne-like on partial errors (Fig. 3) decreased on SD condition compared to control condition ($F(1, 11) = 5.62$, $p < .05$). There was no main effect of congruity ($F < 1$) and no interaction between these two factors was showed ($F(1, 11) = 2.41$, $p = .15$).

Amplitude of Ne-like on correct responses (Fig. 3) was not statistically different before and after SD ($F(1, 11) = 1.41$, $p = .26$). There

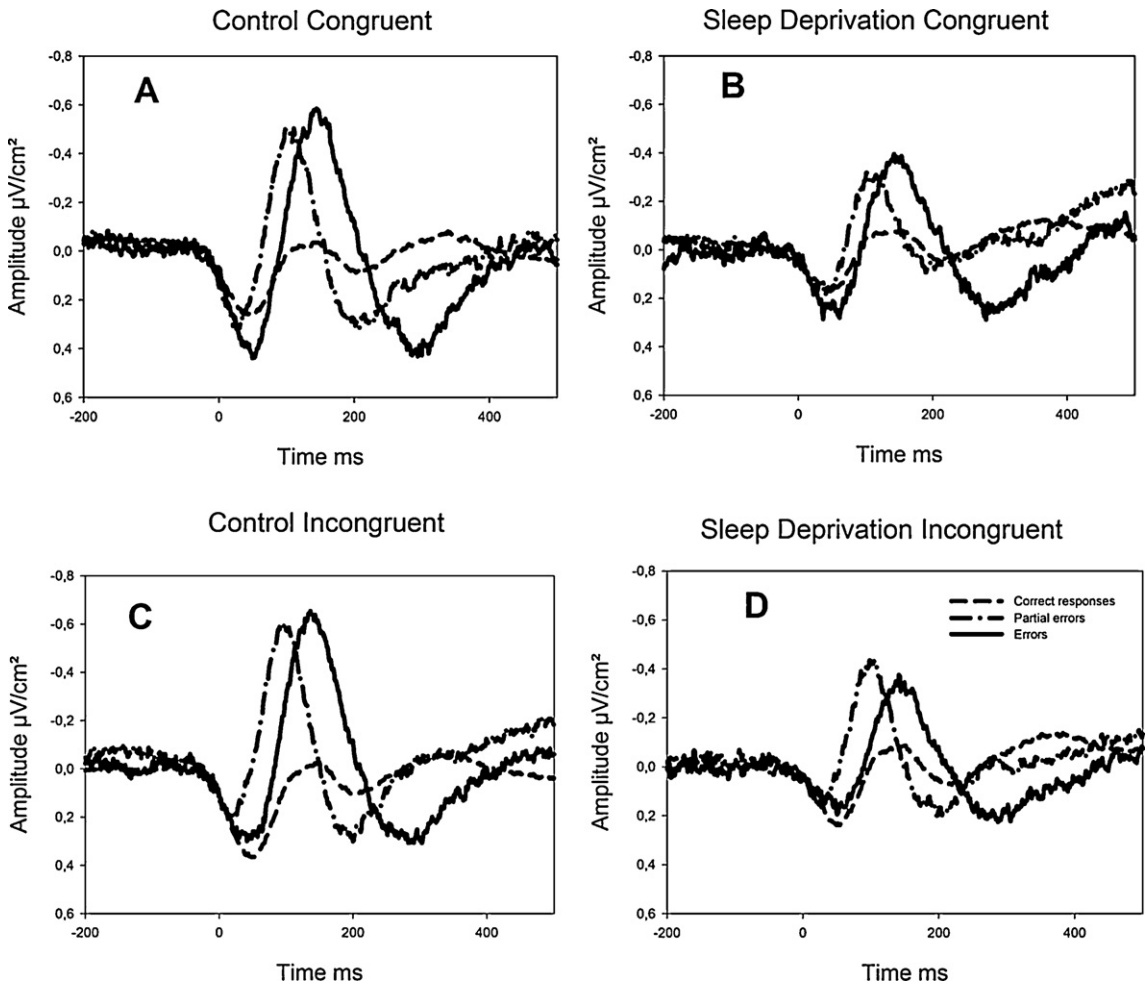

Fig. 3. Grand averages (Cz) of the three categories of trials (in dash: correct responses, in dash-dot: partial errors and in solid: errors) for the Laplacian transformed data. The zero of time indicates the relevant EMG onset: for correct responses, it corresponds to the EMG burst that triggered the correct mechanical response. For errors, it corresponds to the EMG burst that triggered the error. For partial errors, the relevant EMG is the small EMG burst occurring on the incorrect hand before the EMG burst triggering the correct response. (A) Amplitude of the Ne on congruent associations, on control condition. (B) Amplitude of the Ne on congruent associations, on sleep deprivation condition. (C) Amplitude of the Ne on incongruent associations, on control condition. (D) Amplitude of the Ne on incongruent associations, on sleep deprivation condition.

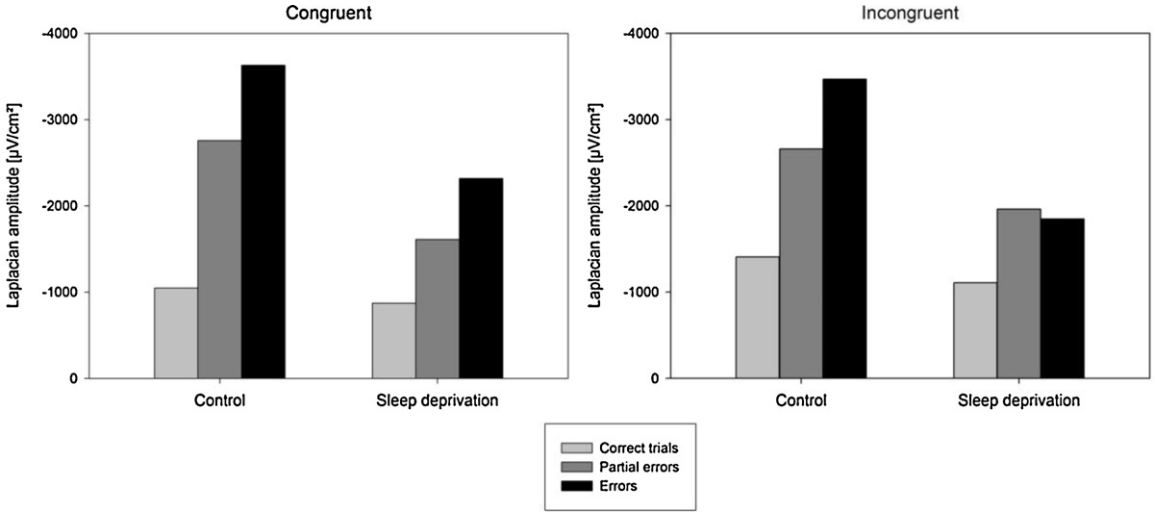

Fig. 4. Amplitude of the Ne/Ne like after Laplacian transform as a function of the correctness and synchronized on the EMG onset; In grey, it is the amplitude of Ne on correct responses, in dark grey the amplitude of Ne on partial errors and in black, the amplitude of Ne on errors. On the left side, on congruent associations, the sensitivity of the Ne/Ne-like amplitude decrease after sleep deprivation and modulation between different trials persists. On the right side, on incongruent associations, the sensitivity of the Ne/Ne-like amplitude decreases after sleep deprivation and the modulation between partial errors and errors is not found.

was a main effect of congruity ($F(1, 11) = 14.03$, $p < .01$), amplitude being larger on incongruent than on congruent stimulus-response associations. No interaction between these two factors was showed ($F(1, 11) = 2.56$, $p = .13$).

In control condition, there was a simple effect of the correctness of trial ($F(2, 22) = 17.71$, $p < .001$): The amplitude of Ne-like on partial error was smaller than the amplitude of Ne on error ($F(1, 11) = 6.09$, $p < .02$) and the amplitude of Ne-like in correct trial was smaller than the amplitude of Ne-like in partial error ($F(1, 11) = 11.08$, $p < .01$). There was no effect of congruity ($F < 1$) and no interaction between congruity and correctness of the trial ($F(2, 22) = 2.02$, $p = .15$).

In SD condition, there was also a simple effect of the correctness of the trial ($F(2, 22) = 5.84$, $p < .01$) and no effect of congruity ($F < 1$). But, there was an interaction between these two factors ($F(2, 22) = 8.48$, $p < .01$). This interaction revealed that the effect of the correctness of the trial on Ne persisted on congruent trials ($F(2, 22) = 9.28$, $p < .01$) and only a trend subsisted on incongruent trials ($F(2, 22) = 3.32$, $p = .055$). On congruent trials, the amplitude of Ne-like in partial error was smaller than the amplitude of Ne in error ($F(1, 11) = 5.12$, $p < .05$) and the amplitude of Ne-like in correct trial was smaller than the amplitude of Ne-like in partial error ($F(1, 11) = 6.14$, $p < .05$). On incongruent trials, if the amplitude of Ne-like in correct trial was still smaller than the amplitude of Ne-like in partial error ($F(1, 11) = 5.80$, $p < .05$), the amplitude of Ne-like in partial error was not different from the amplitude of the Ne in errors ($F < 1$).

In other words, the sensitivity of Ne and Ne-like to the correctness of the trial decreased after a night of SD in particular on incongruent trials.

4. Discussion

The goal of the present study was to evaluate the impact of SD on proactive and reactive action monitoring.

4.1. Proactive action monitoring

Proactive action monitoring was investigated by examining the time course of the Simon effect (Simon, 1990) through RT distribution analyses.

Consistent with previous studies (Murphy et al., 2006; Tsai et al., 2005; Hsieh et al., 2010; Scheffers et al., 1999), SD impaired participants' performance: mean RT increased and accuracy decreased. On congruent associations, RT increased while error rate did not change and on incongruent associations, both RT and error rate increased. Moreover, after a night of SD the overall size of the Simon effect increased both on RTs and error rates (Table 1). Distribution analysis suggests that this overall increase was caused by an impairment of the action monitoring process that prevents the expression of EMG outputs associated to erroneous response (partial errors and full performance errors) (see Van den Wildenberg et al., 2010).

Consistent with previous studies (Ridderinkhof et al., 2004b; Burle et al., 2002, 2005), in the control condition, distribution analyses revealed that the Simon effect decreased and disappeared when RT increased. In contrast, after SD, the Simon effect decreased less and did not disappear for long RTs. According to the suppression activation hypothesis of Ridderinkhof et al., 2004b; Ridderinkhof, 2002), two explanations could account for the reduction of the Simon effect for long RTs: (1) SD impaired the ability to inhibit the response activated by the fast route and/or (2) SD increased the strength of the fast route relative to that of the slow route.

Conditional incorrect activation function (CIAF) (Fig. 2) allowed us to study the strength of the fast route on incongruent trials. The stronger the fast route, the higher the probability that an incorrect activation occurs before the suppression mechanism takes place (fastest RTs). Hence, an increase of the strength of the fast route between two conditions is expressed by a different pattern of incorrect activation rate for shorter RTs. As expected, on control condition, CIAF showed an increase in the proportion of fast incorrect activations on incongruent trials as compared to congruent trials. A strengthening of the fast route should result in a selective increase of fast incorrect activations on incongruent trials only. On the contrary, after SD, CIAF revealed a non-selective increase of incorrect activation rate whatever the RT, that is to say that the increase of mean incorrect activation rate observed on incongruent trials could not be explained by a selective increase in the proportion of fast incorrect activations. Then, this increase of incorrect activation rate cannot be attributed to a strengthening of the fast route. The increase of the Simon effect after a SD is thus due to an impairment of the suppression mechanism; the present results suggest that a night of SD compromises the ability to inhibit response activation via the fast route. In line with Wylie et al. (2009), we consider that this decrease in fast route inhibition reveals an impairment of cognitive control processes. Now, since fast route inhibition takes place preventively, that is before EMG onset, it is concluded that proactive action monitoring is impaired after SD. Such an explanation allows accounting for the increase of the Simon effect both on RTs and on incorrect activations rates after a SD.

4.2. Beyond SD effects on proactive action monitoring

While the impairment of proactive control could account for the increase of the Simon effect and the increase of incorrect activation rate on incongruent associations after SD, it cannot by itself account for all the increase of RT on both congruent and incongruent associations.

EMG recordings were used to fraction the overall RT (Botwinick et al., 1966) into PMT and MT. MT reflects the implementation of peripheral motor processes whereas PMT can be considered as an index of more central processing. SD specifically increased the congruity effect on PMTs. In contrast, after SD, MT increased equally for incongruent and congruent associations. In fact, the increase of RT after SD was mainly explained by an increase of MT. This motor effect is in line with the results of De Gennaro et al. (2007) who showed, with Transcranial Magnetic Stimulation, that the excitability of the cortico spinal pathway was decreased after SD. Moreover, the coregistration of EEG with TMS allowed the authors to consider that this effect "... should be mainly explained by the contribution of peripheral factors" (p. 1284). More specifically, one can easily imagine that sleepiness decreases muscle tone. Indeed, the firing rate of orexinergic neurons (which stimulate both cortical activation and muscle tone) largely decreases from active to quiet waking (Lee et al., 2005). Given, that muscle tension before the response signal is known to decrease MT (Possamaï et al., 2002; Sanders, 1980) it is therefore likely that the increase of MT observed here is a by-product of a decrease in muscle tone induced by SD. It is noteworthy and somewhat counter intuitive that peripheral (motor) processes are less robust to SD than are more central ones.

4.3. Reactive action monitoring

The Ne is a widely acknowledged central index of action monitoring (Simons, 2010). As the Ne develops after EMG onset and is sensitive to performance (small on correct responses, larger on partial errors and even larger on full performance errors) it should be considered as revealing reactive action monitoring. Some studies have reported a reduction of Ne amplitude after a 24 h SD (Tsai et al., 2005; Hsieh et al., 2007, 2010; Scheffers et al., 1999). Murphy et al. (2006) however showed no significant difference in Ne amplitude

after wakefulness of 20 h. As noted by Hsieh et al. (2010) there was no conclusive evidence supporting the idea that SD impairs action monitoring, as revealed by the Ne (on errors). The interpretation of the effects of SD on reactive control as revealed by Ne modulations (when present) on errors may present difficulties: it is established that when participants trade accuracy for speed, the Ne is decreased on errors while the error rate increases (Falkenstein et al., 1991; Gehring et al., 1993). Previous studies (Murphy et al., 2006; Tsai et al., 2005; Hsieh et al., 2010; Scheffers et al., 1999) found that SD increase the error rate. Therefore, variation in the decrease of the Ne on errors might just be a by-product of error rate increase and not an effect of SD per se. This question has been addressed by Tsai et al. (2005) and the authors have concluded that SD *directly* affects the sensitivity of the system generating the Ne. Our results are consistent with the conclusion of these authors as the decrease of the Ne on errors after SD is manifest in congruent condition, where SD does not affect the error rate. This indicates that SD has a direct effect on reactive control. More specifically, by examining the modulation of amplitude of the Ne/Ne-like as a function of performance, we will argue in the following, that SD decreases the sensitivity of reactive control to response correctness (Fig. 4).

In line with previous results (Vidal et al., 2000), in the control condition (Fig. 3A and C) of the present study, the amplitude of the Ne/Ne-like was sensitive to correctness: small on correct responses, large on errors and intermediate on partial errors. After SD (Fig. 3B and D), the amplitude of the Ne on errors was smaller than in control condition. The same effect held on partial errors. On the contrary, the amplitude of the Ne-like on correct responses did not statistically decrease after SD.[1] Moreover, differences in Ne/Ne-like amplitude between errors and partial errors on the one hand and between partial errors and correct responses on the other hand were also reduced after SD (Figs. 3 and 4). These differences were even more reduced on incongruent associations than on congruent associations to such an extent that there was no more difference between errors and partial errors after SD. Therefore, the modulation of amplitude due to correctness, from correct responses to partial errors, and from partial errors to errors is decreased after SD, which reveals an impairment of the sensitivity to correctness of the reactive control system. To sum up, the sensitivity of the reactive control system was directly impaired by SD and all the more so that the required response involved more controlled processes.

SD is not the only variable altering the sensitivity of the Ne/Ne-like. In fact, reduced modulations of the Ne/Ne-like have been reported in prefrontal damaged patients (Gehring et al., 2000) and in schizophrenic patients (Ford, 1999; Kim et al., 2006; Mathalon et al., 2002; Bates et al., 2002), two pathologies often attributed to prefrontal impairment (Sullivan et al., 1998). In this context, we suggest that the decrease of the sensitivity of Ne/Ne-like is somehow linked to a prefrontal impairment caused by SD. This study offers new empirical insights into how SD could affect prefrontal cortex in agreement with the frontal lobe hypothesis of SD (Harrison et al., 2000). According to this hypothesis, "the prefrontal cortex may well be among the first brain regions to suffer as a consequence of SD" (p. 246).

To summarize: (1) proactive control (ability to overcome prepotent response tendencies) was affected by a SD and (2) reactive control (ability to detect and correct errors) had a weaker sensitivity to performance after a SD.

[1] This is not due to a lack of statistical power be because the number of correct responses was largely higher than the number of partial errors and errors. Therefore, a global decrease of brain activity could not account for the decrease of amplitude of Ne on errors and partial errors because this decrease was not found for correct trials.

Conflicts of interest

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

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