Research Article

Inhibition of Return, but Not Facilitation, Disappears Under Vigilance Decrease Due to Sleep Deprivation

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Abstract. In this study, we assessed whether unspecific attention processes signaled by general reaction times (RTs), as well as specific facilitatory (validity or facilitation effect) and inhibitory (inhibition of return, IOR) effects involved in the attentional orienting network, are affected by low vigilance due to both circadian factors and sleep deprivation (SD). Eighteen male participants performed a cuing task in which peripheral cues were nonpredictive about the target location and the cue-target interval varied at three levels: 200 ms, 800 ms, and 1,100 ms. Facilitation with the shortest and IOR with the longest cue-target intervals were observed in the baseline session, thus replicating previous related studies. Under SD condition, RTs were generally slower, indicating a reduction in the participants' arousal level. The inclusion of a phasic alerting tone in several trials partially compensated for the reduction in tonic alertness, but not with the longest cue-target interval. With regard to orienting, whereas the facilitation effect due to reflexive shifts of attention was preserved with sleep loss, the IOR was not observed. These results suggest that the decrease of vigilance produced by SD affects both the compensatory effects of phasic alerting and the endogenous component involved in disengaging attention from the cued location, a requisite for the IOR effect being observed.

Keywords: sleep deprivation, tonic alertness, phasic alertness, inhibition of return, facilitation

Behavioral and cognitive effects of sleep deprivation (SD) are well known. Sleep loss increases sleepiness (Carskadon & Dement, 1979; Gillberg, Kecklund, & Akerstedt, 1994) and impairs cognitive performance (Casagrande, Violani, Curcio, & Bertini, 1997; Heuer, Kohlisch, & Klein, 2005; Martella, Plaza, Estevez, Castillo, & Fuentes, 2012; Sagaspe et al., 2006). Several approaches are used for manipulating SD. In the present study, we are interested in using an approach that represents a realistic match to what occurs in real life, when people are requested to sustain wakefulness for 24 hr (e.g., a physician in an intensive care unit, a fireman in a disaster evacuation, or a pilot on long-haul flights; Barger et al., 2006; Tsai, Young, Hsieh, & Lee, 2005). When SD is manipulated in this manner, two potential sources of influence may affect the performance: sleep loss per se and the circadian variation (Dijk & Czeisler, 1995; Lavie, 2001). This type of SD, namely, extended wakefulness beyond the usual evening time sleep onset, is particularly useful because it reproduces the normal partial sleep deprivation experienced by night workers.

These aspects are important to understand the consequences of sleep loss, given that many people are required to work under sleep loss conditions. In fact, sleepiness due to reduced sleep has been associated with occupational and transportation accidents, industrial catastrophes, and medical errors (Barger et al., 2006; Connor, Whitlock, Norton, & Jackson, 2001; Philip & Åkertstedt, 2006).

Many studies have suggested that performance decrements after sleep loss are primarily due to attentional alterations (Dinges, 1992; Kjellberg, 1977), although attention in those studies is primarily defined as a unitary entity. From a cognitive neuroscience approach (Posner & Fan, 2008), however, attention is thought to be a set of independent neural networks that perform specific computations related to selecting relevant objects and locations (the orienting network), solving cognitive conflict by selecting the target between competing distractors (the executive network), or achieving and maintaining a general activation state of the cognitive system to maximize task performance (the alerting network). Regarding this latter network, two types of alertness have been described (Raz & Buhle, 2006). Tonic alertness or vigilance refers to sustained activation over a period of time, whereas phasic alertness refers to nonspecific activation experienced when, for instance, a warning signal is presented before the target presentation.

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Regarding the orienting network, attention shifts might occur endogenously or exogenously. Attention can be endogenously (voluntarily) oriented toward the most probable location of information that is relevant to current goals, also involving the executive network. Additionally, attention can be exogenously (automatically) captured by salient stimuli, although the individual does not have the intention of orienting his attention to that object or location. Our primary objectives in this study were to assess the effect of reduced vigilance due to a period of moderate sleep loss on several components of the orienting and alerting networks and to evaluate their interaction.

To disentangle the different operations involved in shifting attention, researchers have frequently used Posner's cuing task (Posner & Cohen, 1984). To assess exogenous orienting of attention, a spatial cue is presented either to the left or right side of fixation, followed by a target either at the same location as the cue (cued location-valid trial) or on the opposite side (uncued location-invalid trial). With informative peripheral cues, that is, when the cue signals the most likely location of the forthcoming target, facilitatory effects are usually observed across several cue-target stimulus onset asynchrony (SOA). In other words, valid trials produce faster reaction times (RTs) than invalid trials, which is an effect known as the validity effect. With uninformative peripheral cues, that is, when the cue does not provide any information about the location of the forthcoming target (50% of valid trials), the effects on target responses strongly depend on the cue-target SOA value. When the cue-target SOA is brief (e.g., less than 300 ms), a validity effect is observed as with informative peripheral cues. Nevertheless, when the cue-target SOA is longer (i.e., more than 300 ms), this facilitatory effect is usually reversed (i.e., RTs are slower for valid than for invalid trials), which is an effect traditionally known as inhibition of return (IOR) (Posner, Rafal, Choate, & Vaughan, 1985). To assess the endogenous orienting of attention, central instead of peripheral cues are used. A central cue might consist of an arrow pointing to either the left or right location; as with peripheral cues, the target can appear at the cued location, that is, the location to which the arrow points (valid trial), or the opposite uncued location (invalid trial). Attention shifts are now under the conscious control of the participant, involving the executive network, and only validity (facilitatory) effects are usually observed, irrespective of whether the central cue is informative with respect to the target location. The cuing task, therefore, seems to be a suitable procedure to assess the effects of decreasing vigilance due to periods of moderate sleep deprivation, or extended wakefulness, on the diverse mechanisms involved when people perform orienting tasks.

Although the cuing task allows measuring the efficiency of both facilitatory and inhibitory mechanisms involved in the orienting network, most studies have focused on the effects of sleep deprivation on the facilitatory mechanisms, with contrasting results. For instance, several studies have reported a general slowing in RTs with sleep deprivation, or a general slowing with central compared with spatial cues (Martella, Casagrande, & Lupiáñez, 2011), but there were no effects on specific orienting mechanisms (Casagrande, Martella, DiPace, Pirri, & Guadalupi, 2006). However, other studies have reported specific effects of sleep loss primarily on endogenous attentional shifts (Trujillo, Kornguth, & Schnyer, 2009), the reorienting mechanism that operates in invalid trials when peripheral predictive cues were used (Bocca & Denise, 2006; Fimm, Willmes, & Spijkers, 2006; Versace, Cavallero, De Min Tona, Mozzato, & Stegagno, 2006), and the reorienting costs when peripheral unpredictive cues were employed (Roca et al., 2012). These results have suggested that sleep deprivation might affect not only task performance by slowing general responding but also diverse components of the orienting network primarily involving exogenous shifts of attention with predictive cues, and components involved in endogenous attention shifts.

However, predictive cues, although peripherally presented, involve the executive network, because attention must be held on the cue location once the exogenous cue attracts attention automatically to that location before the target is presented. Therefore, to assess the effects of sleep deprivation on reflexive mechanisms of the orienting network with minimal involvement of executive attention, the use of uninformative exogenous cues is preferable. Additionally, by manipulating the cue-target SOA, we might be able to assess the effect of sleep deprivation not only on the facilitatory but also the inhibitory component of the orienting network, that is, IOR. Several authors have argued that the mechanisms involved in IOR are crucial for an organism to survive (Fuentes, 2004; Fuentes, Vivas, Langley, Chen, & González-Salinas, 2012; Klein, 1988, 2005). IOR plays the role of avoiding reiterative explorations of already attended locations and objects by favoring a bias of the organisms for novelty (Chen, Fuentes, & Zhou, 2010; Fuentes, 2004). Therefore, any effect of SD on the inhibitory mechanisms involved in orienting attention is of crucial relevance to assess the deleterious effects of sleep loss on attention-dependent activities.

Regarding the alerting network, although SD is generally accepted as a powerful means to reduce tonic alertness or vigilance (Killgore, 2010; Lim & Dinges, 2008), the effects of sleep loss on phasic alertness have been less frequently studied, although recent studies did not observe any effects on alertness (e.g., Martella et al., 2011; Trujillo et al., 2009). However, increasing phasic alertness might counteract the deleterious effects of low arousal due to sleep loss. Posner (1978) has demonstrated that auditory stimuli acting as warning signals reduce RTs to visual targets more than visual stimuli do to auditory targets. This result suggests that auditory stimuli are better able to automatically activate the alerting mechanism than visual stimuli. Posner (2008) argued that a larger alerting effect is generally observed in people who have difficulty maintaining alertness. Thus, it might be anticipated that under reduced vigilance, the inclusion of a warning tone might help to increase the alertness of the participants, thus resulting in better performance. Finally, in line with previous findings, we expect the increase of phasic alerting to interact with the orienting network by improving the effect of the exogenous visual cue on target responses (Callejas, Lupiáñez, Funes, & Tudela, 2005; Callejas, Lupiáñez, &

Tudela, 2004; Fuentes & Campoy, 2008). Regulation of the functioning of orienting and conflict networks by a warning signal has been already observed in patients diagnosed with mild cognitive impairment (Fernández et al., 2011), or with Lewy bodies dementia (Fuentes et al., 2010). Accordingly, larger facilitation and IOR effects are expected when a warning tone acting as an alerting stimulus is presented.

In the present study, the participants were required to perform the exogenous cuing task at two critical moments. At 6:00 pm, it was assumed that participants exhibited a high level of arousal coinciding with the "forbidden zone for sleep" described by Lavie (2001). At 5:00 am, it was assumed that participants exhibited the lowest level of arousal corresponding to the primary "sleep gate." Two potential factors may affect the decrease of vigilance at that time: sleep loss per se and the circadian variation (Dijk & Czeisler, 1995; Lavie, 2001). Because we are looking for a possible interaction between vigilance and spatial orienting, it possible to anticipate that such an interaction, if found, could be due to a decrease of vigilance produced by each of the two above-mentioned sources. In our study, we were only interested in manipulating vigilance in general, whatever the component primarily involved in this variation. Therefore, the aim of our study was to evaluate the effects of 24 hr of prolonged wakefulness on visuospatial attention.

According to previous studies (Casagrande et al., 2006; Martella et al., 2011), prolonged wakefulness (circadian factors and sleep loss) should cause decreased vigilance, that is, a general slowing in RTs (Dinges, 1992), an increase in subjective sleepiness, and a decrease in body temperature.

Moreover, we propose the following three hypotheses about the effect of decreased vigilance on visuospatial orienting: (1) If decreased vigilance does not affect orienting (Fan, Raz, & Posner, 2003; Fernandez-Duque & Posner, 1997), prolonged wakefulness should produce an increase in reaction times, but it should not affect the orienting mechanisms of attention (i.e., the benefits in valid trials and costs in invalid trials). (2) If decreased vigilance affects the orienting mechanisms, in addition to the slowing of RTs, we should find a diminished efficacy in orienting attention mechanisms (engagement, shift, and disengagement). (3) If an increase of phasic alertness counteracts the deleterious effects of low arousal due to sleep loss, we should find no effect on orienting under warning condition.

Method

Participants

Eighteen males (mean age, 23 ± 2.6 years) signed an informed consent before participating as volunteers in the study. The participants were all right handed according to a Hand Preference Index $\geq$ .85, as assessed by the Lateral Preference Questionnaire (Salmaso & Longoni, 1985). They were all naïve to the purpose of the experiment, and all of the participants reported normal or corrected-to-normal vision. We selected the participants after a structured interview. This interview allowed us to exclude people who had extreme behavior (with regard to coffee, it was considered to be acceptable to consume up to four cups of Italian coffee, equivalent to a maximum 240 mg of caffeine per day). To be eligible to participate in the experiment, the participants had to be nonsmokers and drug-free and report both normal sleep duration (7.5–8.5 hr per day) and schedule (going to sleep at 11:30 pm ± 60 min and waking up at 7:30 am ± 60 min). Those who had reported sleep, medical, or psychiatric disorders were not included in the study. The local ethical committee approved the study.

Apparatus and Stimuli

Stimuli were presented on a 21-inch color VGA monitor (HP Hewlett Packard 71). An IBM compatible Asus PC running E-Prime software controlled the presentation of stimuli, timing operations, and data collection. Responses were collected using the computer keyboard. The display consisted of two white boxes (3 degrees of the visual angle), one to the left and the other to the right of the fixation point; the center of each box subtended 6 degrees of the visual angle. The fixation point consisted of a white plus sign (0.8 degrees of the visual angle). The visual cue consisted of the brightening of one of the two boxes. For the auditory warning, a 98-dB and 2,000-Hz sound, lasting 50 ms, was used (Callejas et al., 2005). A headphone set was employed to deliver the alerting signal. The target stimulus was a white asterisk (1 degree of the visual angle), which was randomly presented in the center of one of the two boxes. All of the visual stimuli were presented on a black background.

Procedure

Participants were seated 57 cm directly in front of a computer monitor, and their heads were held steady with a chin/head rest. The sequence of events for each trial is shown in Figure 1. The fixation point was displayed for a variable time (range, 200–1,000 ms), followed by a cue stimulus that appeared to the left or to the right of the fixation cross. The cue was presented for 100 ms and then removed. The cue never predicted the position of the target. In 25% of the trials, before the presentation of the cue, a 50-ms auditory warning was presented. After the offset of the cue, a delay period of either 100 ms, 700 ms, or 1,000 ms occurred, depending on the SOA condition (200 ms, 800 ms, or 1,100 ms). Subsequently, the target was displayed in one of the two boxes for 1,500 ms or until the participant responded. If no response was given within 1,500 ms after the target onset, the next trial began. Additionally, 40 catch trials, in which no target stimulus was presented after the cue, were run to minimize the frequency

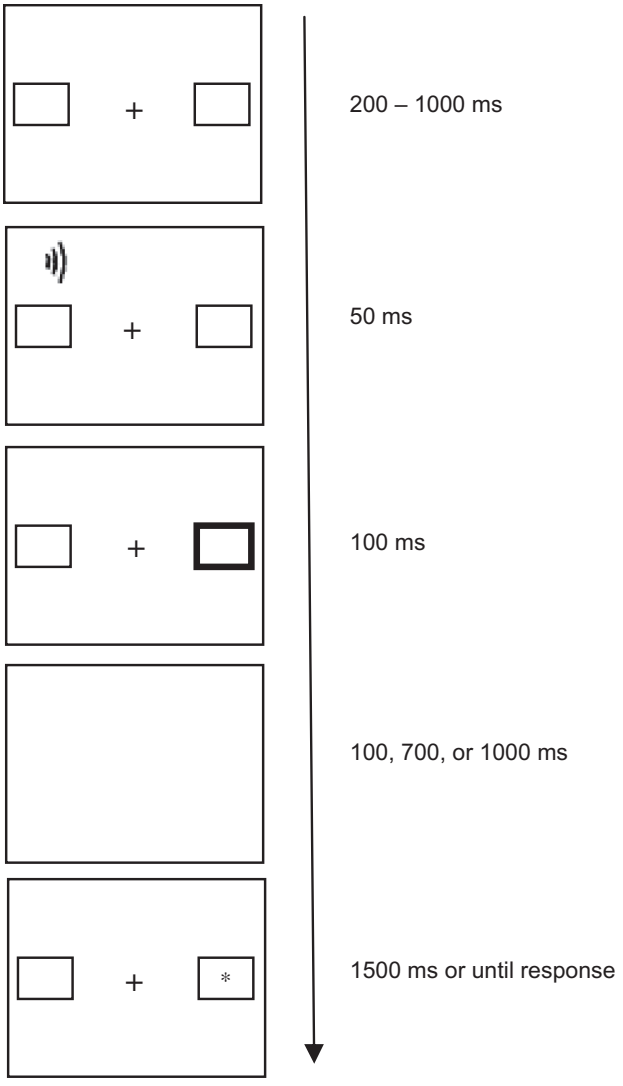

Figure 1. An experimental trial sequence (cued trial), from top to bottom.

of the anticipatory responses. For all of the trials, the presentation of both cues and targets was equally likely at both left and right locations. The participants completed one 24-trial practice block and three experimental blocks. Each practice block consisted of 210 trials that lasted approximately 15 min. A short break was provided at the end of each practice block. The trials were presented randomly throughout the experiment.

Task

The participants were told to look at the fixation point throughout the experiment. As soon as the target stimulus was presented, the participants had to perform a simple detection task by pressing the space bar on the computer keyboard.

Complementary Measures

We used a one-dimensional Visual Analog Scale (VAS; Curcio, Casagrande, & Bertini, 2001), which required each participant to respond to the question: "How do you feel right now with respect to the adjective sleepy?" The participants responded by making a stroke with a pen on a 100-mm long line. The stroke corresponded to the point that indicated the intensity of the self-evaluation. The VAS was anchored at one end with "not at all" and at the other end with "very much." The distance of the mark from the left end of the line was considered to be the dependent variable. Additionally, the peripheral body temperature was measured using an oral thermometer to evaluate the circadian rhythmicity of the participants.

General Procedure

The experiments were run on two nonconsecutive days, separated from one another by approximately 7 days. On the first day, the participants came individually to the laboratory and were subjected to a brief interview aimed at obtaining information on sleep duration and schedule, the presence of any disease, and the use of drugs. The participants completed the Lateral Preference Questionnaire. If the participant was considered to be suitable to participate in the research, he was given detailed information about the procedure. The participants signed the informed consent to participate in the research, and they performed a brief training task. At the end of this session, the participants were reminded to maintain regular sleeping hours throughout the study duration and were given a sleep log in which they were instructed to record immediately after awakening (no more than 20 min) for a week. Approximately 1 week later, the participants returned to the laboratory for the experimental session, which consisted of 24 hr of continuous wakefulness.

During the 24-hr period of sleep deprivation, the participants did not drink or eat anything containing caffeine (e.g., coffee, tea, chocolate), and they were continuously monitored by trained research assistants. The participants had two breaks: one for lunch (approximately 2:00 pm) and one for dinner (approximately 8:00 pm). On the day of the experiment, after the participants had slept their usual 8 hr, they arrived at the laboratory at 9:00 am and were kept awake for 24 hr. They performed the task at 6:00 pm (baseline – BSL) and at 5:00 am (sleep deprivation – SD). The task was performed individually in a sound attenuated air-conditioned room. The environmental temperature was set to 25 °C. The laboratory was constantly illuminated with artificial light of 500 lux, and the cabin in which the experiment was conducted was illuminated with approximately 20 lux.

Before and after the covert attention task, the participants performed other cognitive tasks (e.g., attentional and memory tasks). The VAS scores and body temperature were recorded hourly.

Table 1. Mean correct reaction times and *SD*s (between parentheses) for two experimental sessions: Baseline (BSL) and Sleep Deprivation (SD). Warning signal (present/absent), Visual cue (cued/uncued), SOA (200 ms, 800 ms, 1,100 ms) conditions have been differentiated

	BSL		SD	
SOA and warning	Cued	Uncued	Cued	Uncued
200 ms				
Present warning	369.45 (50.54)	377.31 (62.40)	447.21 (63.71)	460.38 (67.67)
Absent warning	387.83 (60.93)	401.83 (60.24)	469.65 (64.75)	488.17 (69.04)
800 ms				
Present warning	353.92 (59.77)	348.88 (60.68)	418.69 (66.68)	420.01 (71.85)
Absent warning	366.84 (56.90)	351.77 (63.70)	436.67 (62.03)	432.06 (72.41)
1,100 ms				
Present warning	354.00 (49.76)	341.40 (57.32)	429.79 (77.11)	431.26 (79.43)
Absent warning	363.71 (54.16)	354.78 (68.13)	431.07 (73.45)	422.69 (75.88)

Design

All of the data are presented as the mean ± *SD*. The experiment consisted of a 2 (Session) × 2 (Warning Tone) × 3 (SOA) × 2 (Cuing) repeated-measures factorial design. There were two sessions (BSL and SLD), two warning conditions (absence, presence of the tone), three SOA values (200 ms, 800 ms, and 1,100 ms), and two cuing conditions (cued, uncued). The subjective measures of sleepiness and body temperature were separately analyzed using one-way ANOVAs considering Session (1–24 hr) as the within-subjects factor.

Results

RTs faster than 100 ms (0.13% of the trials) or slower than 800 ms (0.05% of the trials) were removed from the data analyses. Given the small percentage of errors in the target detection task, accuracy data were not reported. Table 1 shows the mean (± *SD*) RTs for each experimental condition for sessions BSL and SLD.

The RT analysis showed significant effects of Session, $F(1, 17) = 61.64$; $p < .000001$; partial $\eta^2 = 0.98$, with longer RTs in the SLD compared to the BSL session (440.64 ms vs. 364.31 ms, respectively); Warning, $F(1, 17) = 18.87$; $p < .001$; partial $\eta^2 = 0.52$, with RTs on the present-warning condition (396.02 ms) being faster than RTs on absent-warning condition (408.92 ms); and SOA, $F(2, 34) = 46.68$; $p < .000001$; partial $\eta^2 = 0.73$, with RTs decreasing with longer SOAs. The primary effect of cuing was not significant ($F < 1$). The following interactions were statistically significant. The Cuing × SOA interaction, $F(2, 34) = 13.11$; $p < .001$; partial $\eta^2 = 0.41$ revealed a facilitation effect for the 200 ms SOA, $F(1, 17) = 8.15$; $p = .001$; partial $\eta^2 = 0.32$, whereas no differences were found between the cued and the uncued conditions for the longer 800 ms and 1,100 ms SOAs (*p*s > .05). The Session × Cuing interaction was also significant, $F(1, 17) = 6.46$; $p = .02$; partial $\eta^2 = 0.27$, showing a larger effect of sleep deprivation for uncued trials, although the effect was significant for both cued and uncued trials, $F(1, 17) = 51.52$; $p = .000002$; partial $\eta^2 = 0.70$ and $F(1, 17) = 70.16$; $p < .000001$; partial $\eta^2 = 0.79$, respectively. The Warning × SOA interaction was significant, $F(2, 34) = 6.29$; $p = .005$; partial $\eta^2 = 0.27$, although this interaction was qualified by the significant three ways Session × Warning × SOA interaction, $F(2, 34) = 3.63$; $p = .037$; partial $\eta^2 = 0.18$. RTs were faster for the present-warning than for the absent-warning condition in all SOA values for the baseline session, $F(1, 17) = 37.80$, $p < .0001$; partial $\eta^2 = 0.69$ for the 200 ms SOA; $F(1, 17) = 5.70$, $p = .002$; partial; $\eta^2 = 0.25$ for the 800 ms SOA, and $F(1, 17) = 5.14$, $p = .036$; partial $\eta^2 = 0.23$ for the 1,100-ms SOA, but only for the 200-ms and 800-ms SOAs, for the sleep deprivation condition, $F(1, 17) = 29.83$, $p < .0001$; partial $\eta^2 = 0.62$ and $F(1, 17) = 6.09$, $p = .02$; partial $\eta^2 = 0.26$, respectively. No other interactions were significant.

To better characterize the effects of sleep deprivation on orienting and alerting effects as a function of SOA, we analyzed separately the RT data for sessions BSL and SLD.

Data analyses from the BSL session showed significant primary effects of Warning, $F(1, 17) = 19.80$; $p < .001$; partial $\eta^2 = 0.53$, and SOA, $F(2, 34) = 22.49$; $p < .00001$; partial $\eta^2 = 0.57$. As expected, the alerting tone produced a faster response than when the tone was not presented, and RTs were faster with longer SOAs. The Cuing × SOA interaction was significant, $F(2, 34) = 12.68$; $p < .001$; partial $\eta^2 = 0.39$. The facilitation effect was observed just for the brief SOA, $F(1, 17) = 4.32$; $p < .05$; partial $\eta^2 = 0.20$; the IOR effect was marginally significant at the long 800-ms SOA, $F(1, 17) = 2.67$; $p = .12$; partial $\eta^2 = 0.13$ and significant at the longest 1,100-ms SOA, $F(1, 17) = 4.73$; $p < .05$; partial $\eta^2 = 0.21$. We observed the expected bi-phasic pattern of facilitation with short cue-target intervals and IOR with long cue-target intervals at BSL. No other effects or interactions reached statistical significance.

Data analyses from the sleep deprivation session showed, as in the baseline session, significant effects of Warning, $F(1, 17) = 10.31$; $p = .005$; partial $\eta^2 = 0.37$ and SOA, $F(2, 34) = 39.82$; $p < .001$; partial $\eta^2 = 0.70$,

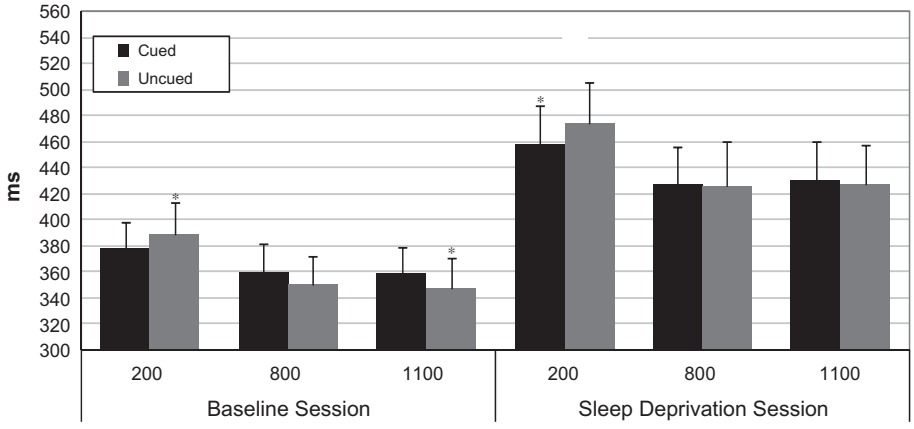

Figure 2. Mean RT and standard error in BSL and SLD sessions as a function of the SOA (200 ms, 800 ms, 1,100 ms) and the cue type (cued and uncued). An asterisk indicates a significant difference between cued and uncued condition.

although these two effects were qualified by the significant Warning × SOA interaction, $F(2, 34) = 5.58$; $p < .001$; partial $\eta^2 = 0.25$. That is, the effects of the alerting tone were observed just for the 200-ms and 800-ms SOAs, $F(1, 17) = 29.83$; $p < .0001$; partial $\eta^2 = 0.62$ and $F(1, 17) = 6.09$; $p = .02$; partial $\eta^2 = 0.26$, respectively, but not for the longest 1,100-ms SOA ($F < 1$). The Cuing × SOA interaction was also significant, $F(2, 34) = 4.47$; $p = .018$; partial $\eta^2 = 0{,}21$ (Figure 2). A similar effect of facilitation was observed, as in the baseline session, for the short SOA, $F(1, 17) = 10.41$; $p < .01$; partial $\eta^2 = 0.38$. The Warning × SOA × Cuing was not significant, $F(1, 17) = 1.01$; $p = .374$. However, contrary to the baseline session, no IOR was observed with the longer SOAs (Fs < 1).

The results of subjective sleepiness (VAS) and body temperature measures are shown in Figure 3. The ANOVA performed on sleepiness scores showed a significant primary effect of Session, $F(6.8, 116.4) = 17.73$; $p < .00001$; partial $\eta^2 = 0.51$; with Greenhouse-Geisser correction; $\varepsilon = 0.2977$). The trend analyses revealed just a significant linear component, $F(1, 17) = 85.64$; $p < .0000001$; partial $\eta^2 = 0.83$, indicating that subjective sleepiness increased with time. Regarding body temperature, an expected effect of circadian rhythmicity was found. The repeated-measures ANOVA revealed significant differences in the hourly measures, $F(23, 391) = 3.26$; $p < .000001$; partial $\eta^2 = 0.32$, and planned comparisons revealed a significant reduction of temperature mainly in the early morning (5:00–7:00 am; $p < .001$).

Discussion

Recently, there has been renewed interest in the effects of extended wakefulness on human performance. This interest stems from prolonged operations required in industrial and military settings, as well as in disaster evacuations, emergency medical operations, long-haul flights, and forest fire eradication. The requirement of continuous performance for extended time periods has driven laboratory and field studies on the effects of extended wakefulness on human performance (Caldwell, Caldwell, Brown, & Smith, 2004; Casagrande et al., 2006; Fallet, Maruff, Collie, Darby, & McStephen, 2003; Maccari, Martella, Marotta, Banjai, et al., 2012; Maccari, Martella, Marotta, Sebastaiani, et al., 2012; Marotta et al., 2012; Martella, Marotta, et al., 2012; Martella et al., 2011; Petrilli, Jay, Dawson, & Lamond, 2005; Sebastiani et al., 2012).

It is usually recognized that performance decrements after sleep loss are primarily due to attentional deficits (Dinges, 1992; Kjellberg, 1977), which indicates that attention is a unitary construct. Cognitive researchers agree that attention is a multidimensional ability (Fan et al., 2003; Posner & Petersen, 1990) and that it can be considered concretely as an organic system (Posner & Fan, 2008). This system involves almost three specialized neural networks and neuromodulators subserving different attentional functions: (1) alerting; (2) orienting; and (3) executive control. The alerting system is associated with the frontal and parietal areas of the right hemisphere, which is modulated by noradrenaline (Sturm & Willmes, 2001; Witte & Marrocco, 1997). Neural correlates of alerting effects are associated with extrastriate regions, which suggest that increased phasic alertness results in a top-down modulation of neural activity in the visual processing areas (Thiel, Zilles, & Fink, 2004). The executive system involves the anterior cingulated and the lateral prefrontal cortex, which are modulated by dopamine (Bush, Luu, & Posner, 2000; MacDonald, Cohen, Stenger, & Carter, 2000). The orienting system is mediated by a network lateralized to the right superior parietal cortex and the right inferior frontal gyrus (Corbetta & Shulman, 2002), which is modulated by the cholinergic system (Thiel, Zilles, & Fink, 2005). Specifically, the neural correlates of the orienting system are observed in the anterior cingulate cortex, which is more active during valid compared to neutral cue trials. The neural correlates of reorienting of attention, that is, brain activity to invalid as compared to valid cued trials, are evident in several brain

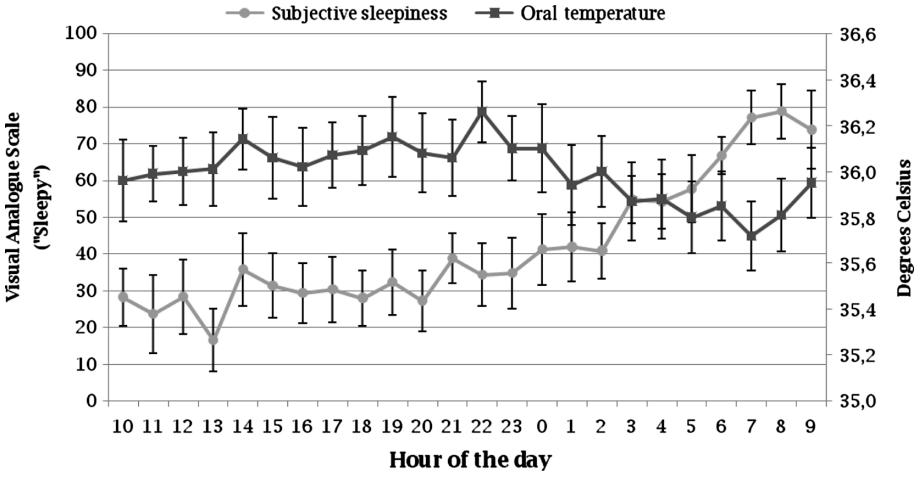

Figure 3. Mean and standard error measures of subjective sleepiness (in mm) and oral temperature through the 24 hr of sleep deprivation.

regions, including the left and right intraparietal sulcus, the right temporo-parietal junction, and the middle frontal gyrus bilaterally. These data suggest that the frontal and parietal regions are specifically involved in reorienting rather than orienting attention to a spatial position (Thiel et al., 2004).

It is well known that SD affects the alerting system, as reflected by reaction time tasks (Urrila, Stenuit, Huhdankoski, Kerkhofs, & Poskka-Heiskanen, 2007; Van den Berg & Neely, 2006). Changes in the cerebral blood flow may account for the cognitive deficits observed during SD. Neuroimaging studies have shown that changes in cerebral activation occur as a function of SD and that these changes are correlated with variations in cognitive performance (Drummond, Gillin, & Brown, 2001; Drummond et al., 2000; Thomas et al., 2000). Specifically, a significant decrease in overall brain activation has been recorded after SD (Thomas et al., 2000); this impairment has shown to correlate with declines in performance. The neural activity decreases have been observed primarily in the thalamus (Thomas et al., 2000), temporal (Wu et al., 1991), and prefrontal and parietal cortices (Thomas et al., 2000). Several neural regions are associated with the spatial orienting of attention. However, the decrease of neural activity observed under SD conditions depends on numerous factors (e.g., the duration of SD, the circadian time in which the neuroscan is made, the type of activity performed during the SD period, and the eventual task performed during the scanning of neural activity). No study has measured the neural activity of individuals who were executing an orienting task.

The aim of the present study was to evaluate the functioning of both facilitatory and inhibitory effects involved in the attentional orienting network under conditions of extended wakefulness. The results confirmed the effectiveness of the present experimental manipulations to reduce the vigilance level. In fact, a general slowing in RTs was observed in the nocturnal session, in line with previous studies (Casagrande et al., 2006; Martella et al., 2011). In addition, at 5:00 am, self-rating scores indicated high sleepiness and low-level body temperature, confirming the reduced arousal state at that time. Regarding phasic alerting, latencies in responding were effectively reduced by the warning signal, although the effect varied for BSL and SLD sessions as a function of the cue-target SOA. The warning tone improved responding times in all of the experimental conditions during the baseline session; it was useful to partially compensate for the deleterious effect of SD on target responses. However, it was true only for 200-ms and 800-ms SOA values. The phasic alerting provided by the warning tone was inefficient to compensate for the slowing in responses produced by sleep loss when the participants had to wait a long time for the target to be presented. To the best of our knowledge, this study is the first to report a deficit in the efficiency of phasic alerting under SD conditions. Although the warning signal effect was significant, it did not interact with the cuing effect as described in previous studies (Callejas et al., 2004, 2005; Fernández et al., 2011; Fuentes & Campoy, 2008; Fuentes et al., 2010). A possible explanation for the failure of the warning tone to improve orienting is that the percentage of trials with the warning tone was lower than in earlier studies, which might have been sufficient for a general effect of alertness to be observed but insufficient for improving more specific processes related to shifting attention to the cued location. Further research in which the percentage of trials including the warning signal is varied might better elucidate the effects of phasic alerting on orienting attention under sleep deprivation conditions.

Regarding attentional orienting, we replicated the standard bi-phasic pattern (facilitation with the short SOA and IOR with the longer SOAs) of previous studies that used the Posner's cuing task. However, that was true only for the baseline session. In the nocturnal session, only the facilitation effect resisted SD, and the inhibitory effect signaled by the IOR effect vanished. The manner in which the IOR procedure was implemented in the present study might clarify the nature of the deleterious effects of sleep deprivation on the inhibitory component of the orienting network.

A critical feature of the cuing task, which seems to be a requisite for observing IOR, is that participants are able to disengage their attention from the cued location and move it back to fixation before the target is presented. In the cue-back procedure, a central cue is presented at fixation once the peripheral cue is off and before the target is presented. That central cue serves to disengage and move attention from the cued location in a reflexive way, which encourages inhibition rather than facilitation of attention at the peripheral cue location. The rather scarce attentional control setting imposed by the simple detection task, together with the increased attentional engagement by the central cue, might foster the early appearance and long-lasting characteristic of IOR that are usually observed with target detection tasks (Klein, 2000; Langley, Fuentes, Vivas, & Saville, 2007). In the present study, however, we did not use the cue-back procedure. Instead, the operations necessary to move attention back to fixation were expected to occur as part of the participants' strategy to maximize the target detection. Although IOR has been generally observed with and without the cue-back procedure, several authors have proposed that in the absence of an exogenous fixation cue, IOR depends, at least in part, on the endogenous disengagement of attention from the peripherally cued location (Klein, 2005; Klein, Castel, & Pratt, 2006). Given that we did not use the cue-back procedure, it is possible that in our study, it was the more endogenous component of IOR to be affected by lack of sleep. In other words, the deprivation-reduced vigilance conditions would affect those endogenous processes that lead to IOR rather than to IOR per se. This concept is further supported by IOR studies that used participants who show poor attentional control, such as patients diagnosed with schizophrenia (Fuentes, 2001; Mushquash, Fawcett, & Klein, 2012), the elderly (Castel, Chasteen, Scialfa, & Pratt, 2003, Langley et al., 2007), patients with Alzheimer's disease (Faust & Balota, 1997), and patients with Parkinson's disease (Poliakoff et al., 2003).

Whereas reflexive orienting mechanisms seem to be preserved under sleep deprivation conditions, the endogenous components involved in orienting attention, which it is assumed to be subserved by the executive network of the attention system, are seriously affected. We propose that using similar procedures to those used in this study, future research should be conducted to determine the conditions and extension under which sleep loss affects specific components of the attention system. For instance, by increasing the percentage of warning signal trials, phasic alerting might compensate longer for the low arousal levels imposed by the sleep loss. The processes that lead to IOR might likely require more time to develop, instead of being fully deteriorated by sleep deprivation conditions. The inclusion of longer cue-target intervals than those used in the present study might reveal the time course of the inhibitory components under normal and sleep deprivation conditions.

However, other explanations can be advanced. The high intersubject variability of the effects of SD (Banks & Dinges 2007) may, in part, account for many experimental results of SD effects on performance. In fact, the neurobehavioral deficits from SD vary significantly among individuals and are stable within individuals (Van Dongen, Baynard, Maislin, & Dinges, 2004). Based on a vast review of SD research, Banks and Dinges (2007) suggested a trait-like differential vulnerability to SD. In line with this hypothesis, it was found that after 48 hr of SD, the deactivation of a neural network, including posterior cerebellum, right fusiform gyrus, precuneus, left lingual and inferior temporal gyri, was effective only in the participants who showed an impairment in memory performance but not in those who were able to maintain higher performance (Bell-Mcginty et al., 2004). This variability in neural and behavioral responses to SD, confirmed also by other studies (Chee et al., 2006, 2008; Chuah & Chee, 2008; Vandewalle et al., 2009), in conjunction with the intensity of vigilance decrease produced by SD, may partially account for the results of this study. To assess this hypothesis, we should increase the number of participants so that we can evaluate whether the absence of the IOR is present only in people with a high vulnerability to SD, that is, in those who have a very strong decrease in arousal, as indicated by a sharp slow-down in RTs.

Conclusions

This study is the first to report a deficit in the efficiency of IOR under reduced vigilance conditions. This effect seems to be consistent with the deactivation of certain brain areas observed under SD conditions. Our results were obtained using an approach that represents a good match to what occurs in real life, when people are requested to sustain wakefulness for 24 hr (e.g., a physician in an intensive care unit, a fireman in disaster evacuation, or a pilot on long-haul flights). Whereas this experimental paradigm represents a point of strength in this study, it is also the main cause of the study's weakness, because it allows us to not balance the baseline and SD conditions. This balance would have returned an effect of SD condition stronger than that observed (in fact, one cannot exclude a practice effect); however, the intention to reproduce a realistic condition has led us to not balance the two experimental conditions. Another limitation of the study is the only male population, an option which limits the generalizability of the observed results. Finally, a higher number of participants would have allowed us to split the sample on the basis of vulnerability to SD and to evaluate how the IOR and the effectiveness of the warning signal depend on the general level of arousal. Future studies will address these limitations.

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