Wandering minds, sleepy brains:
lapses of attention and local sleep in wakefulness.

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Abstract

Attentional lapses are ubiquitous and can negatively impact performance. They correlate with mind wandering, or thoughts that are unrelated to ongoing tasks and environmental demands. In other cases, the stream of consciousness itself comes to a halt and the mind goes blank. What happens in the brain that leads to these mental states? To understand the neural mechanisms underlying attentional lapses, we cross-analyzed the behavior, subjective experience and neural activity of healthy participants performing a task. Random interruptions prompted participants to indicate whether they were task-focused, mind-wandering or mind-blanking. High-density electroencephalography revealed the occurrence of spatially and temporally localized sleep-like patterns of neural activity. This “local sleep” accompanied behavioral markers of lapses and preceded reports of mind wandering and mind blanking. Furthermore, the location of local sleep distinguished sluggish versus impulsive behaviors, mind wandering versus mind blanking. Despite contrasting cognitive profiles, attentional lapses could share a common physiological origin: the appearance of local islets of sleep within the awake brain.
Introduction

The human brain sustains the stream of our conscious experiences. Attention can direct cognitive resources toward the external world and enable the selection and amplification of information relevant to an individual’s current behavioral goals. But attention can also turn inward, as is the case when we focus on internally generated task-unrelated thoughts, a phenomenon usually referred to as mind wandering. Recent investigations have also shown that the stream of thoughts can also come to a pause, as when individuals who are awake are left with the feeling of an empty mind (mind blanking).

Mind blanking and mind wandering can occur spontaneously without our knowledge or will, which raises the question of what are the neuronal mechanisms that control the focus of attention. In fact, a characteristic feature of directed attention is its fleeting nature and the difficulty to maintain it on a task for long periods of time. In this paper, we define lapses of attention as the shift of the focus of thoughts away from the task at hand or environmental demands. The consequences of attentional lapses are very diverse. At the behavioral level, they can result in a lack of responsiveness or sluggish reactions, but they can also result in impulsive responses. Curiously, these behavioral failures can be accompanied by a lack of conscious awareness and the absence of mental activity (mind blanking), or rich, spontaneous mental activity (mind wandering).

It is yet unclear whether these different types of attentional lapses (sluggish vs. impulsive behaviors; mind-blanking vs. mind-wandering) belong to a disparate family of behavioral and phenomenological events, each of them associated with different physiological causes or whether they can be traced back to common underlying physiological causes. Previous models of mind wandering have proposed that mind wandering and mind blanking might arise in distinct neurophysiological states. However, the fact that both sluggish and impulsive responses increase following sleep deprivation and in individuals with attentional deficits implies a common mechanism. Likewise, sleepiness has been associated with both mind wandering and mind blanking despite these two mind states being phenomenologically distinct. Furthermore, investigations of the sleep onset period (hypnagogia) also indicate that subjective experiences resembling mind wandering (focus on internally generated contents) and mind blanking (loss of
awareness) can co-exist at the border between wakefulness and sleep\textsuperscript{17,18}. Interestingly, these studies seem to associate lapses with pressure for sleep, suggesting an involvement of fatigue in the occurrence of lapses.

Indeed, each hour spent awake comes at the cost of mounting sleep pressure. Past research suggests that the need for sleep might only be dissipated by sleep itself\textsuperscript{9} as sleep plays a vital role in neural homeostasis\textsuperscript{20}. When individuals are prevented from sleeping for extended periods of time (as in sleep deprivation studies), a subset of brain regions can start displaying electroencephalographic (EEG) signatures of non-rapid eye-movement (NREM) sleep in the form of sleep-like slow waves (within the delta (1-4 Hz) or theta (4-7 Hz) range), despite individuals being behaviorally and physiologically awake\textsuperscript{21,22}. These sleep-like slow waves within wakefulness are referred to as “local sleep” in contrast with the global whole-brain transition commonly observed at sleep onset\textsuperscript{22–25}.

It has been proposed that local sleep could perturb brain functions and cause behavioral lapses\textsuperscript{22}. In fact, during sleep, slow waves are associated with episodes of widespread neural silencing\textsuperscript{26}, which have been connected to behavioral unresponsiveness and the loss of consciousness\textsuperscript{27,28}. Intracranial studies in humans and rodents showed that likewise, local sleep reduces neuronal firing during waking and is associated with behavioral errors\textsuperscript{21,22}. Local sleep can also be detected in human non-invasive recordings\textsuperscript{29–31} and here again the amount of local sleep recorded in a given brain region correlates with the number of errors performed in a task recruiting this specific brain region\textsuperscript{29,30}. These results strongly suggest that local sleep could explain the behavioral component of attentional lapses\textsuperscript{22}. However, the impact of local sleep on phenomenology is still unclear.

In a recent review, we proposed that local sleep could not only explain the behavioral consequences of attentional lapses, both regarding sluggish and impulsive responses, but also the phenomenological profile of these lapses\textsuperscript{11}. We also argued that local sleep is not an extreme phenomenon, occurring only when individuals are pushed to their limit, but could occur in well-rested individuals\textsuperscript{31} and explain the occurrence of lapses in our everyday lives. To test this framework, we formulate three different hypotheses as follows: (i) Can local sleep predict, at the single trial level, both sluggish and impulsive behaviors in well-rested individuals? (ii) Is local sleep associated with both mind blanking and mind wandering? (iii) Does the location of local
sleep (i.e. which brain regions are affected by a local sleep event) differentiate between sluggishness and impulsivity, mind-blanking and mind-wandering? Through these hypotheses we will test the idea that local sleep could act as a functional switch, transiently perturbing the functioning of a given cortical network. Accordingly, a common physiological event (local sleep) could lead to drastically different outcomes depending on its location within the brain.

To test these hypotheses, we cross-examined the behavioral performance, subjective reports and physiological data from healthy individuals (N=26) performing an undemanding Go/NoGo tasks\(^{32}\) (Figure 1a). We sampled participants’ subjective experience by interrupting them during the task and asking them a series of questions about their mental states prior to the interruption, including whether they were focusing on the task, mind-wandering or mind-blanking (Figure 1b). Finally, we recorded their brain activity using high-density scalp EEG and pupil size as an objective proxy for participants’ level of vigilance\(^{33}\).

**Figure 1. Experimental design and hypotheses.**

*a:* Participants performed both a SART on faces stimuli (NoGo trials: smiling faces) and a SART on digits (NoGo trials: “3”). Face/Digit presentation was continuous (new face/digit every 0.75-1.25s). The Face SART is not shown to comply with bioRxiv policy on the inclusion of photographs.

*b:* Every 30 to 70s, participants were interrupted and instructed to report their state of mind (see
Methods and Supplementary Methods). Most importantly, they were asked to indicate whether they were focusing on the task (task-focused: ON), thinking about nothing (mind-blanking: MB) or thinking about something other than the task (mind-wandering: MW). High-density EEG and pupil size were continuously recorded throughout the task.

Results

Task performance and subjective experience. The Go/NoGo tests (see Methods) require participants’ sustained attention, but our participants declared focusing on the task only in ~48% of the probes (Face Task: 49.4 ± 4.9%; Digit Task: 47.2 ± 5.1%; mean ± Standard Error of the Mean (SEM) across N=26 participants; Figure 2a). In the rest of the time, they declared thinking about something else (Mind Wandering (MW); Face: 38.0 ± 4.3%; Digit: 40.9 ± 4.8%; Figure 2a) or thinking about nothing (Mind Blanking (MB); Face: 12.7 ± 3.0%; Digit: 11.9 ± 2.9%; Figure 2a). These results are well in line with previous findings\textsuperscript{34,35} and highlight the prevalence of attentional lapses. Attentional lapses were also reflected in participants' poor accuracy on the Go/NoGo tests, notably regarding NoGo trials (Face: 35.3 ± 1.3% of false alarms (FA, i.e. errors on NoGo trials); Digit: 33.9 ± 1.3%; Figure 2b).

Next, we focused on the behavioral patterns preceding subjective reports of attentional lapses. Specifically, we examined participants’ behavior 20s before the onset of the probes that led to MW and MB reports (see Methods). To quantify the impact of states of mind (i.e., ON, MW or MB) on behavior, we compared statistical models that either did or did not include states of mind as a predictor of behavior (see Methods and Supplementary Table 1). A significantly better fit by the model incorporating mind states, assessed through a Likelihood Ratio Test, was interpreted as evidence for the influence of mind states. To describe the size and direction of the statistical effects, we report the estimates ($\beta$) of the contrasts of interest (MW vs ON, MB vs ON and MB vs MW) and their 95% confidence-interval (CI). Accordingly, we found a significant effect of mind states on false alarms (model comparison: $\chi^2(2)=115.9, p<10^{-16}$; see Figure 2b), with an increase for both MW and MB compared to ON (MW vs ON: $\beta=0.21$, CI: [0.17, 0.24]; MB vs ON: $\beta=0.17$, CI: [0.12, 0.23]), but similar levels of false alarms for MB and MW (MB vs MW: $\beta=-0.028$, CI: [-0.084, 0.028]). Misses were also modulated across mind states (model comparison: $\chi^2(2)=36.0, p=1.5\times10^{-8}$). Specifically, MW and MB were associated with an increase in misses compared to ON reports (MW vs ON: $\beta=0.011$, CI: [0.005, 0.016]; MB vs ON: $\beta=0.023$, CI: [0.015, 0.032])
and misses were more frequent for MB compared to MW reports (MB vs MW: $\beta=0.013$, CI: [0.005, 0.021]). Finally, mind states were associated with different patterns of reaction times (RT; model comparison: $\chi^2(2)=16.3$, $p=2.9\times10^{-4}$; Figure 2c) with slower reaction times for MB compared to both ON and MW reports (MB vs ON: $\beta=0.019$, CI: [0.009, 0.030]; MB vs MW: $\beta=0.022$, CI: [0.011, 0.032]). Taken together, these results suggest that MW and MB can decrease performance through different ways: MW facilitates impulsivity, evidenced by faster RT and more false alarms, whereas MB facilitates sluggishness, evidenced by slower RT and more misses.
Figure 2. Low arousal is associated with attentional lapses characterized by different behavioral outcomes

**a:** Proportion of mental states reported during probes categorized as task-focused (ON, green), mind-wandering (MW, orange) and mind-blanking (MB, blue) during the tasks with Faces (diamonds for each individual participant; filled surfaces for smoothed density plot) and Digits (circles and surfaces with horizontal lines). Grey diamonds and circles show the average across participants.  

**b:** Proportion of false alarms in the 20s preceding ON, MW and MB reports. The markers’ size is proportional to the number of reports for each participant (same for **c-e**). Grey diamonds and circles show the average across participants, weighted by the number of reports (same for **d** and **e**).  

**c:** Distribution of reaction times (RT) for Go Trials (left: Face; right: Digit) in the 20s preceding ON, MW and MB reports.  

**d:** Vigilance scores (subjective ratings provided during probes) associated with ON, MW and MB reports.  

**e:** Discretized pupil size (see Methods) in the 20s preceding ON, MW and MB reports. In **b-e**, stars show the level of significance of the effect of mind-states (Likelihood Ratio Test, see Methods; ***: p<0.005).

**Vigilance.** Although MW and MB differ according to their phenomenological definition and associated behaviors, both states seem to occur in a similar context of low vigilance and sleepiness. We quantified this by examining the correlation between participants’ vigilance ratings and each mind state (comparison between models including or not the information about mind-states: $\chi^2(2) = 144.8$, p<10$^{-16}$; Figure 2d and Supplementary Table 1). Participants reported lower vigilance ratings for both MW and MB compared to ON (MW vs ON: $\beta$=-0.39, CI: [-0.40, -0.37] and MB vs ON: $\beta$=-0.53, CI: [-0.55, -0.50]). Vigilance ratings were even lower for MB compared to MW (MB vs MW: $\beta$=-0.13, CI: [-0.24, -0.02]). We then examined a classical objective proxy for vigilance: pupil size$^{33,36}$. Pupil size prior to probes (Figure 2e, N=25 participants here, see Methods) was significantly modulated across mind-states (model comparison: $\chi^2(2) = 18.0$, p=1.210$^{-4}$) with MW and MB associated with smaller pupils than ON probes (MW vs ON: $\beta$=-0.29, CI: [-0.43, -0.15]; MB vs ON: $\beta$=-0.22, CI: [-0.45, -0.003]). Pupil size did not differ between MW and MB (MB vs MW: $\beta$=0.065, CI: [-0.16, 0.29]).

**Local Sleep.** Critically, we examined the core hypothesis of our framework$^{11}$: whether apparently distinct families of attentional lapses can be coherently explained by the occurrence of local sleep and its spatio-temporal characteristics. To do so, we first detected the occurrence of local sleep in each EEG electrode using an established approach for detecting slow waves in wakefulness and sleep (see$^{30,31,37}$ and Methods). Operationally, we defined local sleep as the occurrence of large-
amplitude waves within the delta ([1-4] Hz) range. Both the temporal profile and topographical distributions of local sleep detected during the tasks (Figure 3a-b) resemble the slow waves observed in NREM sleep\textsuperscript{37,38}. This is not trivial as our detection algorithm did not select this specific temporal profile or topographical distributions.

![Figure 3. Mind-Wandering, Mind-Blanking and Local Sleep](https://doi.org/10.1101/2020.06.23.166991)

\textbf{Figure 3. Mind-Wandering, Mind-Blanking and Local Sleep}

\textbf{a}: Average waveform of the slow-waves detected over electrode Cz during the behavioral tasks (red, left; \(N=26\) participants). The average waveform of slow waves detected during sleep (blue, right) extracted from another dataset (see Supplementary Methods) is shown for comparison. \textbf{b}:
Scalp topographies of wake slow-waves properties (left: temporal density; middle: peak-to-peak amplitude; right: downward slope; see Methods) averaged across participants (N=26). c: Scalp topographies for slow-waves Density (left), Amplitude (middle) and Slope (right) for the different mind-states (ON, MW and MB).

Next, we checked whether local sleep indexes participants’ level of sleepiness. For this analysis, we quantified the amount and properties of local sleep across the entire scalp by averaging the temporal density of local sleep (number of local sleep events per minute) as well as local sleep amplitude and slope (see Methods) across all 63 electrodes. We verified that each of these variables extracted prior to probe onset ([-20, 0]s) was negatively correlated with the subsequent vigilance ratings obtained in each probe (model comparison between models with or without local sleep density, amplitude or slope: density: \( \chi^2(1)=13.1, p=3.9\times10^{-4}, \beta=-0.074, \text{CI: } [-0.114, -0.034] \); amplitude: \( \chi^2(1)=33.1, p=8.5\times10^{-9}, \beta=-0.023, \text{CI: } [-0.031, -0.015] \); slope: \( \chi^2(1)=82.1, p<10^{-16}, \beta=-2.5\times10^{-3}, \text{CI: } [-3.1\times10^{-3}, -2.0\times10^{-3}] \)).

In addition to vigilance ratings, local sleep also preceded subsequent reports of attentional lapses. Topographical maps show that MW and MB reports seem preceded by larger and steeper slow waves than ON reports (Figure 3c). This was confirmed by a mixed-effect model analysis focusing on local sleep events before each probe and across all electrodes. We observed more local sleep in MW and MB compared to ON reports (comparison between models including or not the information about mind-states to explain slow-wave density: \( \chi^2(2)=31.9, p=1.2\times10^{-7}, \beta=0.048, \text{CI: } [0.023, 0.073] \); MW vs ON: \( \beta=0.097, \text{CI: } [0.060, 0.133] \)) as well as steeper waves (model comparison: \( \chi^2(2)=79.2, p<10^{-16}, \beta=0.78, \text{CI: } [0.60, 0.97] \); MW vs ON: \( \beta=0.20, \text{CI: } [-0.25, 0.29] \)).

To further understand the association between local sleep and attentional lapses, we examined the influence of local sleep on participants' behavior at the single-trial level. To do so, for each trial and electrode, we marked the presence or absence of local sleep events between stimulus onset and offset (see Methods) and used this as a (binary) predictor of RT, misses and false alarms (Figure 4a). This analysis revealed spatially-specific effects of local sleep on distinct behavioral outcomes. Namely, local sleep in frontal electrodes co-occurred with faster reaction times while
local sleep in posterior electrodes co-occurred with slower reaction times ($p_{\text{cluster}}<0.005$, Bonferroni corrected cluster threshold; see Figure 4a). Likewise, frontal local sleep was associated with more false alarms (a marker of impulsivity) while posterior local sleep with more misses (a marker of sluggishness). We obtained a similar spatial correlation pattern when considering mind states: local sleep amplitude and slope was larger for MW compared to ON reports in frontal electrodes only whereas local sleep amplitude was larger for MB compared to ON reports in posterior channels only ($p_{\text{cluster}}<0.05$, Bonferroni corrected cluster threshold; Figure 4b).

Figure 4. Local sleep impacts behavioral performance and subjective reports in a region-specific fashion

a: Single-trial analysis of the effects of local sleep (defined as the presence or absence of local sleep events during stimulus presentation for a given electrode and at the single-trial level) and behavioral outcomes. Mixed-Effects Models were used to quantify the impact of local sleep occurrence on reaction times (left), false alarms (middle) and misses (right) at the single-trial level. Topographies show the scalp distribution of the associated t-values. b: Similar approach as in a, showing the impact of MW vs. ON on local sleep amplitude (left) and slope (middle) and of MB vs. ON on local sleep slope (right). a-b: Red dots denote significant clusters of electrodes ($p_{\text{cluster}}<0.05$, see Methods).

Decision modeling. Finally, we implemented an influential model of two-alternative forced-choice (2AFC) decision making: the diffusion decision model (DDM)\textsuperscript{39}. The DDM decomposes
full reaction-time distributions and choice proportions into latent cognitive processes that are thought to underlie participants’ decisions in 2AFC tasks (see Methods and Supplementary Figure 1). We used this modeling approach to examine how local sleep impacts the different cognitive processes leading to participants’ responses, with a particular focus being the test of our core hypothesis: frontal local sleep disrupts the cognitive mechanisms underlying executive control leading to impulsivity, while posterior local sleep slows down the integration of sensory inputs leading to sluggishness. A hierarchical Bayesian approach was used to fit the DDM to the reaction times obtained in the Go/NoGo tests\(^{40}\) so that each parameter (Supplementary Figure 1) was free to vary by participant, task and local sleep occurrence (present vs. absent). The estimated parameters comprised the rate of evidence accumulation for Go and NoGo responses (i.e. drift rate for Go trials \(v_{\text{Go}}\) and NoGo trials \(v_{\text{NoGo}}\)), the amount of evidence needed to reach a decision (i.e. the decision threshold \(a\)), the decision bias \((z)\) and the non-decision time \((t)\). A drift bias \((v_{\text{Bias}})\) was also computed by taking the difference between the Go and NoGo drift rates (see Methods), where greater values indicate stronger dynamic bias toward the Go choice bound. Simulations confirmed this hierarchical DDM can successfully predict the observed data (Supplementary Figure 2). We report here the differences in the parameters’ estimates in the presence or absence of local sleep (Figure 5).

The scalp topographies of the effect of local sleep on DDM parameters indicate both general and electrode-specific effects. As general (electrode-non-specific) effects (Figure 5d-f), we found first that local sleep was associated with a reduction in decision threshold \((a;\) Figure 5d), consistent with the idea that local sleep facilitates impulsive responses. Second, local sleep was also associated with longer non-decision times \((t;\) Figure 5e), implying that local sleep can slow down neural processes underlying stimulus encoding and/or motor preparation. Finally, local sleep was correlated with an increase in the starting point of the decision process (prior bias \(z;\) Figure 5f), implying shifts in the decision process towards Go responses.

As regional (electrode-specific) effects, we observed contrasting results between posterior and frontal local sleep (Fig 5a-c). Local sleep within posterior electrodes was associated with a reduction of \(v_{\text{Go}}\) and \(v_{\text{Bias}}\), meaning that evidence accumulation was slower for Go decisions and the dynamic bias of the decision process for Go responses was reduced (Figure 5a,c). Conversely, local sleep within frontal electrodes correlated with a reduction of \(v_{\text{NoGo}}\), indicating that evidence
accumulation was slower for NoGo Decisions (Figure 5b). This provides an explanation of the region-specific behavioral results reported in Figure 4, whereby posterior local sleep was associated with sluggish responses and increased misses while frontal local sleep was associated with faster, impulsive responses and more false alarms. Indeed, slower evidence accumulation in favor of Go responses would lead to slower reaction times or even misses, whereas slower evidence accumulation for NoGo responses would lead to faster responses and possibly false alarms.

Figure 5. Global and local effects of local sleep on sub-components of decision-making

Reaction Times in the Go/NoGo tasks were modelled according to a Hierarchical Drift Diffusion Model. a-f: Topographical maps of the effect of local sleep (i.e. whether or not a local sleep event was detected for each trial and for a specific electrode) on the parameters of decision-making: $v_{Go}$ (a), $v_{NoGo}$ (b), $v_{Bias}$ (c), $a$ (d), $t$ (e), $z$ (f). The effect of local sleep was estimated with LMEs (see Methods) and topographies show the scalp distribution of the associated t-values. Red dots denote significant clusters of electrodes ($p_{\text{cluster}}<0.05$, Bonferroni-corrected, see Methods).

These results suggest that local sleep could represent both a general index of fatigue as well as a mechanism underlying specific consequences of sleepiness. In a region-nonspecific manner, local sleep could thus reflect the global “sleepiness” of the brain (reflected in $t$, $a$ and $z$ for global decision parameters) while in a region-specific manner, local sleep could reflect the disruption of specific cognitive processes carried out by the affected brain regions. Taken together, we interpret
these as strong evidence to support the idea that local sleep is a compelling physiological phenomenon, which precedes and co-occurs with subjective and behavioral aspects of attentional lapses. In addition, mechanisms of local sleep are spatio-temporally specific enough to explain distinctive components of attentional lapses in a unified and quantitative manner.

Discussion

According to both in-lab and real-life studies, humans spend up to half of their waking lives not paying attention to their environment or the task-at-hand\(^{34,35}\). However, despite the ubiquitousness of attentional lapses, it is unclear why they occur so frequently in the first place. Part of the difficulty in identifying the neural correlates of attentional lapses could be due to their intrinsic diversity as well as the lack of adequate taxonomy for these lapses\(^{7,8}\). Rather than shying away from this diversity, we embraced this difficult challenge to clarify the neural mechanisms underlying attentional lapses by linking three different levels of explanation: behavior, phenomenology and physiology. Based on our results, we argue that a large subset of attentional lapses could be simply explained by “local sleep”. We show that local sleep can reflect global states of the brain (Figure 5d-f) and, like pupil size or subjective ratings of fatigue (Figure 2d-e), index the build-up of a pressure for sleep. However, local sleep is also specific enough in both time and space (Figure 4 & 5a-c) to account for the occurrence of different types of attentional lapses.

Indeed, recent findings have revisited the classical view of sleep as an all-or-nothing phenomenon\(^{22-24,41,42}\). Although sleep is orchestrated at the scale of the entire brain, some of the key neural mechanisms underlying sleep are implemented and regulated at the level of local cortical circuits\(^{24,25}\). Consequently, when the pressure for sleep increases, an awake brain can start displaying local sleep-like patterns of activity such as delta or theta waves\(^{21,22,29,43}\). These bouts of local sleep are both time-dependent (i.e. increase with time spent awake) and use-dependent (i.e. depend on the level of activation of a given brain region)\(^{29,30}\). The occurrence of local sleep has been linked to perturbations of information processing and task-related errors or attentional lapses in animal and human intracranial data\(^{21,22}\). A similar relationship between local sleep and behavioral errors has been evidenced when detecting local sleep in non-invasive scalp recordings of sleep-deprived\(^{29,30}\) or well-rested\(^{31}\) individuals.
Our findings expand this previous literature by showing that within the same individual, local sleep temporally precedes (at a single-probe level) different types of attentional lapses. In particular, we observed that sluggish responses (slow responses and misses) defined at the behavioral level tended to be associated with mind-blanking at the phenomenological level (Figure 2b-c) and that both were associated with an increase in local sleep over posterior electrodes (Figure 3-4). Conversely, impulsive responses (fast responses and false alarms) were associated with mind-wandering (Figure 2b-c) and with an increase in local sleep over frontal electrodes (Figure 3-4). These results imply a tight relationship between local sleep and behavioral errors: only local sleep events occurring at the right time (i.e. during stimulus presentation) and in the right place (i.e. in the brain regions involved in the task) would be predictive of distinct behavioral and phenomenological aspects of attentional lapses.

Our results are largely consistent with previous findings on the neural correlates of attentional lapses. Most of these studies focused on mind wandering, although often defined as any mental state that is not on task (i.e. MW+MB here). fMRI studies showed that mind wandering in this sense was associated with the activation of the Default Mode Network (DMN). Interestingly, a phasic activation of the DMN in a resting state fMRI paradigm has been suggested to reflect a state of low alertness and possibly local sleep. EEG correlates of mind wandering are less clear. A seminal study reported a reduction of alpha and/or beta oscillations during mind wandering whereas others have reported an increase. However, alpha oscillations are not an unambiguous marker of sleepiness, as they only transiently increase with sleepiness. Alpha power is low when participants are both fully alert or, on the contrary, approaching sleep onset. Thus, the divergent results obtained regarding mind wandering and alpha oscillations could be explained by a shift in participants’ baseline level of fatigue. Importantly, these results are not contradictory to our work (and others) which shows the association between mind wandering and sleepiness.

Previous results on attentional lapses obtained with pupillometry are similarly inconclusive. While most studies found a dampening of stimulus-locked increases in pupil size during mind wandering, results diverge for baseline pupil size, which has either been reported as increasing or decreasing. When distinguished from mind wandering, mind blanking has also been associated with a reduced pupil size compared to task-focused states. Our results largely align with the latter results, with both mind wandering and mind blanking being
characterized by a decrease in pre-probe pupil size (Figure 2e) which goes together with low vigilance ratings (Figure 2d) and local sleep (Figure 3-4). The complex pattern of results reported in the literature regarding pupil size and mind-wandering could be explained by the fact that pupil size does not index only arousal but it also correlates with motivation, cognitive load, etc. In contrast, the sleep-like nature of local sleep (Figure 3) makes it an unambiguous marker of sleepiness. Furthermore, local sleep is a multivariate variable, defined across space and can therefore indicate how brain regions respond differently to sleep pressure. In this study, we scratched only the surface of this rich source of information by showing a fronto-posterior contrast (Figure 4 and 5). Further investigations, including source localization or simultaneous recording of EEG and fMRI, promise a deeper understanding of the mechanisms underlying attentional lapses.

Beyond attentional lapses, our results could inform how different brain regions participate in shaping the stream of consciousness. Local sleep, considered as a spontaneous perturbation of local cortical networks, could indicate the functions fulfilled by these networks. For example, we observed that local sleep in frontal regions was associated with false alarms (Figure 4a), which aligns well with the role of frontal cortices in executive functions and response inhibition. Conversely, local sleep in the back of the brain was associated with misses (Fig 4a), which is consistent with the involvement of parietal cortices in sensorimotor integration. At the phenomenological level, frontal local sleep was associated with mind wandering, whereas posterior local sleep was associated with mind blanking. Thus our results could speak to the debate on the respective involvement of frontal and posterior cortices in supporting different conscious states. Our results suggest that a perturbation of frontal cortices leads to unconstrained thoughts (mind wandering) rather than the loss of awareness, but that awareness decreases when posterior regions go momentarily offline, a pattern similar to the neural correlates of dreaming during sleep or spontaneous thoughts during wakeful rest. However, frontal and posterior local sleep do not only differ by their location but also in terms of spatial expanse: local sleep in frontal electrodes appears more focal, whereas local sleep in posterior electrodes is more widespread (Supplementary Figure 3). Thus, the loss of awareness reported during mind blanking and associated with posterior local sleep could be due to the fact that posterior local sleep might actually involve a broader fronto-parietal network. This is in line with theories attributing an essential role of fronto-parietal connections in the emergence of conscious representations.
In conclusion, we show here that attentional lapses occurring in the context of an undemanding task are accompanied by local sleep, even when participants are well-rested. Furthermore, the location of local sleep events is predictive of certain behavioral and phenomenological properties of these lapses. Thus, we propose local sleep as a more mechanistic and proximate cause to explain attentional lapses. When our minds go somewhere else or nowhere, it is possible that it is because part of our brain is already snoozing.
Methods

Participants. Thirty-two (N=32) healthy adults were recruited and participated in this study. Six individuals were not included in our analyses because of technical issues during recordings or an abnormal quality of physiological recordings assessed through a post-hoc visual inspection of the data. The remaining 26 participants (age: 29.8 ± 4.1 years, mean ± standard-deviation; 10 females) were included in all analyses except for one individual for whom we do not have eye-tracking data.

Experimental Design and Stimuli. Participants were seated in a dim-light room with their chin resting on a support at approximately 60cm from a computer screen. All task instructions and stimuli were displayed and button responses were collected via the Psychtoolbox toolbox extension⁷⁰ for Matlab (Mathworks, Natick, MA, USA).

The experimental design consisted of two modified Sustained Attention to Response Tasks (SARTs)³² in which participants were instructed to pay attention to a series of pictures of human faces in the Face SART blocks or digits in the Digit SART blocks. The order of Face or Digit blocks was pseudo-randomized for each participant. Each block lasted approximately 12 to 15 minutes. Participants were allowed to rest between blocks. Participants performed 3 Face SART blocks and 3 Digit SART blocks for a total duration of 103 ± 19.7 minutes (mean ± standard-deviation) from beginning to end. Each type of the Face and Digit SART was preceded by a brief training session (27 trials) on each SART. During this SART training session, feedback on the proportion of correct trials and average reaction times was provided to participants. Participants were encouraged to prioritize accuracy over speed.

Each face or digit was presented for a duration of 750 to 1250ms (random uniform jitter). Face stimuli were extracted from the Radboud Face Database⁷¹ and consisted of 8 faces (4 females) with a neutral facial expression and one smiling female face. Digits from 1 to 9 were displayed with a fixed font size. For the Face SART, participants were instructed to press a button for all neutral faces (Go trials) but to avoid pressing the response button for the smiling face (NoGo trials). The order of faces was pseudo-randomized throughout the entire task (i.e. we permuted the presentation order every 9 stimuli and we did not present twice the same stimuli in a row). For the Digit SART, participants were instructed to press a button for all digits except the digit “3” (NoGo trials), with the order of the digits pseudo-randomized as well.
During the SART, we stopped the presentation of stimuli at random times (every 30 to 70s, random uniform jitter) with a sound and the word “STOP” displayed on the screen. These interruptions allowed to probe the mental state of the participants with a series of 8 questions (including 1 conditional question; see Supplementary Methods). In particular, we instructed participants to report their attentional focus “just before the interruption”. Participants had to select one of the four following options: (1) “task-focused” (i.e. focusing on the task, ON), (2) “off-task” (i.e. focusing on something other than the task, which we define here as mind wandering MW), (3) “mind blanking” (i.e. focusing on nothing), (4) “don’t remember”. As the 4th option accounted for only 1.1% of all probes and since previous studies do not always distinguish between these options (e.g. 72), we collapsed the 3rd and 4th options as mind-blanking (MB) in all analyses. We also instructed participants to rate their level of vigilance, reflecting “over the past few trials”, with a 4-point scale (Figure 2d; from 1=“Extremely Sleepy” to 4=“Extremely Alert”). Each of the 12-15 min SART blocks included 10 interruptions (in total, 30 interruptions for each SART task). Participants were informed of the presence of interruptions and the nature of each question before starting the experiment. The mind-state categories (ON, MW and MB) were also explained to participants orally and in writing.

**Physiological Recordings and Preprocessing.** High-density scalp electroencephalography (EEG) was recorded using an EasyCap (63 active electrodes) connected to a BrainAmp system (Brain Products GmbH). A ground electrode was placed frontally (Fpz in the 10-20 system). Electrodes were referenced online to a frontal electrode (AFz). Additional electrodes were placed above and below the left and right canthi respectively to record ocular movements (electrooculogram, EOG). Two electrodes were placed over the deltoid muscles to record electrocardiographic (ECG) activity. EEG, EOG and EMG were sampled at 500Hz. Eye-movements and pupil size on one eye were recorded with an EyeLink 1000 system (SR Research) with a sampling frequency of 1000Hz. The eye-tracker was calibrated at the start of each recording using the EyeLink acquisition software.

The EEG signal was analyzed in Matlab with a combination of the SPM12, EEGLab73 and Fieldtrip74 toolboxes. The raw EEG signal was first high-pass filtered above 0.1 Hz using a two-pass 5th-order Butterworth filter. A notch filter was then applied (stop-band: [45, 55] Hz, 4th-order Butterworth filter) to remove line noise. Electrodes that were visually identified as noisy...
throughout the recording were interpolated using neighboring electrodes. Finally, the continuous EEG data was segmented according to probe onsets on a 64s window ([-32, 32] s relative to the probe onset); the average voltage over the entire window (64s) was then removed for each electrode and probe.

Pupil size was analyzed with custom functions in Matlab and corrected for the occurrence of blinks (see 33 and Supplementary Methods). Pupil size was averaged over the stimulus presentation window for each trial (window length: 0.75 to 1.25s). Pupil size values in Figure 2e were computed by averaging the pupil size in all trials within 20s preceding the probe onset and then by discretizing them into 5 bins across all probes for each participant and task to normalize pupil size across participants 33.

**Behavioral Analyses.** Go trials were considered incorrect (Miss) if no response was recorded between stimulus onset and the next stimulus onset. Conversely, NoGo trials were considered incorrect (false alarm) if a response was recorded between stimulus onset and the next stimulus onset. Reaction times were computed from the onset of the stimulus presentation. Trials with reaction times shorter than 300ms were excluded from all analyses (so not considered correct or incorrect). For analyses of behavior prior to probes, we analyzed only the trials within 20s from probes’ onset (Figure 2). For analyses of behavior at the trial level (Figure 4-5), we examined trials within 30s of probes’ onset (minimum inter-probe interval).

**Local sleep.** The detection of local sleep events was based on previous algorithms devised to automatically detect slow waves during NREM sleep 30,37. First, the preprocessed EEG signal was re-referenced to the average of the left and right mastoid electrodes (TP9 and TP10) to match the established guidelines for sleep recordings 75. Then, the signal was down-sampled to 128Hz and band-pass filtered within the delta band. A type-2 Chebyshev filter was used to reach an attenuation of at least 25 dB in the stop-band ([0.05, 7.5] Hz) but less than 3 dB in the pass-band ([0.5, 5] Hz). All waves were detected by locating the negative peaks in the filtered signal. For each wave, the following parameters were extracted: start and end point (defined as zero-crossing respectively prior the negative peak of the wave and following its positive peak), negative peak amplitude and position in time, positive peak amplitude and position in time, peak-to-peak amplitude, downward (from start to negative peak) and upward (from negative to positive peak) slopes.
Slow waves in sleep typically have a larger negative peak compared to their positive peak (Figure 3a) and are predominantly observed over fronto-central channels\textsuperscript{37,38}. This contrasts with artefacts in the EEG signal caused by blinks, which typically have a large positive component and are more frontally distributed. Thus, to reduce the false detection of these artefacts as candidate slow waves, we excluded waves with a positive peak above 75µV. We also excluded waves within 1s of large-amplitude events (>150µV of absolute amplitude). Finally, we discarded all waves that were shorter than 143ms in duration (corresponding to a frequency > 7Hz). We then selected the waves with the highest absolute peak-to-peak amplitude (top 10% computed for each electrode independently) as local sleep slow waves.

**Hierarchical Drift Diffusion Modeling.** Hierarchical Bayesian Drift Diffusion Modeling (HDDM) was used to extend our analysis beyond simple behavioral metrics and examine the impact of local sleep on the sub-processes of decision making. The DDM is a sequential-sampling model of 2AFC decision making that can be considered an extension of signal detection theory into the time-domain, accounting for full reaction time distributions as well as choice behavior\textsuperscript{39}. The HDDM package\textsuperscript{40} in Python 2.8 was used to fit the drift-diffusion model to the SART data. DDM parameters were estimated using a hierarchical Bayesian method that uses Markov-chain Monte Carlo (MCMC) sampling to generate full posterior distributions of model parameters. The following DDM parameters were estimated: the drift rate for Go trials ($v_{Go}$), the drift rate for NoGo trials ($v_{NoGo}$), the decision threshold ($a$), the decision bias ($z$) and the non-decision time ($t$). Drift bias ($v_{Bias}$) was computed by taking the difference between the absolute values of $v_{Go}$ (positive) and $v_{NoGo}$ (negative), where greater values indicate stronger $v_{Go}$ drift bias (Supplementary Figure 1). To examine whether the model could reproduce key patterns in the SART data, posterior predictive checks were undertaken by simulating 100 datasets from the joint posteriors of model parameters and comparing these to the observed data\textsuperscript{76} (Supplementary Figure 3).

To estimate HDDM parameters 8,000 samples from the posterior were generated with MCMC methods and the initial 2,000 were discarded as burn-in to reduce autocorrelation. HDDM models were fit to the data so as to examine the influence of local sleep (event present vs. absent; Figure 5). To do so we considered each EEG electrode separately. For a given electrode, a trial was flagged as being associated with local sleep if the onset of a local sleep event (slow wave) was detected for this electrode during stimulus presentation (i.e. between stimulus onset and offset).
Parameters were also free to vary by task (Digit vs. Face). We included trials within 30s of probe onset to maximize the number of available trials for trial-by-trial analysis. From the estimated models, we extracted the subject-level point-estimates of parameters as the mean of each individual's posterior distribution for a given task (Face and Digit) and mind-state (ON, MW or MB) or local sleep (present or absent) combination.

**Statistical Analyses.** Statistics were performed using Linear Mixed-Effects modeling (LMEs). In all models, subject identity was coded as a categorical random effect. The task type (Digit or Face SART) was used as a categorical fixed effect in all analyses. Several fixed effects were independently tested in our different analyses: mind-states (categorical variable: ON, MW and MB; Figure 2-4) or local sleep (binary variable: present/absent, Figure 4). LMEs were run to predict different variables of interest: behavioral variables (misses, false alarms, reaction times) or physiological variables (pupil size, presence or properties of local sleep slow waves). We also used LMEs to estimate the effect of mind states or local sleep on the point-estimates derived from the HDDM models (Figure 5). Model comparisons were performed using a Likelihood Ratio Test to estimate the influence of multi-level categorical variables such as mind states. In practice, we compared a model including mind-states (along possibly other random and fixed effects) with a model not including mind-states as a predictor. All models and model comparisons are described in Supplementary Table 1. In the Results section, we report the Likelihood Ratio Test as $\chi^2$(df), where $\chi^2$ is the Likelihood Ratio Test statistic and df the degrees of freedom\textsuperscript{77,78}. When several model comparisons were performed for the same analysis using the Likelihood Ratio Test, a Bonferroni correction was applied to the statistical threshold. To indicate the magnitude and direction of the effects, we report the estimates ($\beta$) and confidence interval (CI) for the contrasts of interest (MW vs. ON, MB vs ON, MB vs MW). All models performed are described in Supplementary Table 1. For topographical maps, clusters were identified using a cluster-permutation approach (cluster alpha: 0.01; Monte-Carlo p-value threshold: 0.05 with a Bonferroni correction in the case of multiple comparisons; see Supplementary Methods for details).
Acknowledgments

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Author Contributions

References


Supplementary Methods

Participants. Prior to their participation in the protocol, all 26 participants but one filled in online surveys on Qualtrics (N=25). They reported normal levels of sleepiness (Epworth Sleepiness Scale: 14.6 ± 4.7; mean ± standard-deviation) and mind wandering (Mind Wandering Questionnaire\textsuperscript{1}: 3.6 ± 0.91) in their everyday lives.

Experimental Design and Stimuli. Face stimuli were divided in two parts vertically (half-left and half-right faces) which were flickered on the screen at different frequencies (12 and 15 Hz, counterbalanced across participants). Similarly, the digits were inserted in a Kanizsa illusory square (Fig. 1a) whose right and left parts also flickered at different frequencies (12 and 15 Hz). This flickering was introduced to elicit Steady State Visual Evoked Potentials (SSVEPs) in the EEG signal. The detailed analysis of this aspect of our dataset will be reported elsewhere. As the flickering occurred at a high rate, participants did not report a negative effect on their ability to perform the SART.

Experience Sampling. Following task interruptions (probes), participants were asked to answer a series of 8 questions in the following fixed order: (1) 'Were you looking at the screen?' (response: yes / no); (2) 'Where was your attention focus?' (response: on-task / off-task / blank / don't remember); (3) 'What distracted your attention from the task?' (response: Something in the room / personal / about the task); (4) 'How aware were you of your focus?' (response: from 1, I was fully aware, to 4, I was not aware at all); (5) 'Was your state of mind intentional?' (response: from 1, entirely intentional, to 4, entirely unintentional); (6) 'How engaging were your thoughts?' (response: from 1, not engaging, to 4, very engaging); (7) 'How well do you think you have been performing?' (response: from 1, not good, to 4, very good); (8) 'How alert have you been?' (response: extremely alert / alert / sleepy / extremely sleepy). Question 3 was displayed only if participants answered off-task in question 1. In this report, we focus only on questions (2) and (8).

Physiological Recordings and Preprocessing. The raw pupil size was corrected for the occurrence of blinks as in 2. The timings of blinks were obtained through the EyeLink acquisition software. For each of these blinks, the pupil size was corrected by linearly interpolating the average signal preceding the blink onset ([0.1, 0.2]s) and following the blink offset ([0.1, 0.2]s). The corrected signal was then low-pass filtered below 6Hz (two-pass Butterworth filter at the 4\textsuperscript{th} order). Finally,
for blinks longer than 2s, data points between -0.1s prior to blink onset and 0.1s after blink onset were considered missing.

**Local Sleep.** In sleep, according to established guidelines, only waves with peak-to-peak amplitude exceeding 75µV are defined as slow waves. In wakefulness, previous studies relied on a relative rather than absolute threshold. Here, we defined as slow-waves the waves with absolute peak-to-peak amplitude within the top 10% of all the waves detected for a given EEG electrode and a given participant. On average, the detection threshold was 30 µV (average across N=26 participants and across all electrodes). Figure 3a shows the average waveform of the slow waves detected on electrode Cz as well as the average waveform of waves detected during sleep recording in another published dataset (N=15 participants). To compute the average waveform of sleep slow waves, we applied the same algorithm to epochs of 20s scored as NREM2 and NREM3. Only slow waves with peak-to-peak amplitude over 75µV were considered.

**Drift Diffusion Modeling.** The Drift Diffusion Model (DDM) proposes that a decision variable noisily accumulates evidence from a starting point (z) with drift rate (v) towards one of two boundaries that represent choice alternatives (i.e. ‘Go’ or ‘NoGo’; see Supplementary Figure 1). The decision threshold (a) is the distance between the two boundaries and represents the amount of evidence that must be accumulated before a decision is made. Once the decision variable crosses a decision boundary, a response is made. The DDM captures extra-decisional components, including stimulus encoding, response preparation and execution with the non-decision time parameter (t). Five parameters were fitted using a DDM approach: the starting point (z), drift rates for Go and NoGo responses (vGo and vNoGo), the decision threshold (a), the non-decision time parameter (t). From the drift rates, we also extracted the drift rate bias (vBias). Model selection was done using the Deviance Information Criteria (DIC), which assess goodness of fit while accounting for model complexity in hierarchical models. Posterior predictive checks confirmed that the Go/No-Go DDM was able to reproduce the behaviour of our participants in our task. We simulated behaviour according to the DDM based on 100 draws from the posterior distributions for parameters. The model captured the key patterns of our behavioural data (Supplementary Figure 2; based on the Fz model), including a close matching of observed and predicted No-Go choice proportions as well as Go RT distributions.
Statistics. A cluster-permutation approach (derived from ⁹) was applied to identify significant clusters in topographical maps. Significant clusters were defined as neighboring electrodes with a p-value below a threshold (called “cluster alpha”) of 0.01. For each cluster, we computed the sum of the t-values for all the electrodes belonging to the cluster (which we will refer to as the “cluster statistics”). We then created permuted datasets by permuting the labels of the predictor within each subject, each task and each electrode (N=1,000 permutations). For each of these permuted datasets, we also identified and retrieved the significant clusters and their cluster statistics. However, for each permutation, we retained only the cluster with the maximal absolute cluster statistics. Finally, for each real cluster of the real dataset, we compared their cluster statistics to the distribution of maximal cluster statistics obtained for the permuted datasets for positive and negative clusters separately. A Monte-Carlo p-value was derived from this comparison (p<0.05 means that a negative cluster has a cluster statistics below the 5⁰ percentile of the permuted distribution and that a positive cluster has a cluster statistics above the 95⁰ percentile of the permuted distribution). In cases where several cluster-permutations were performed in the same analysis (Fig. 5 and 6), we corrected the Monte-Carlo p-values of the real clusters with the Bonferroni method.
Supplementary References:


### Supplementary Table 1. Summary of Linear Mixed-Effects Models

<table>
<thead>
<tr>
<th>Figure</th>
<th>Predicted Variable X</th>
<th>Level</th>
<th>Predictor Of Interest</th>
<th>Model 0</th>
<th>Model 1</th>
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<tbody>
<tr>
<td>2b</td>
<td>false alarms, misses, reaction times</td>
<td>Probe</td>
<td>Mind-State (MS)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
<tr>
<td>2d</td>
<td>Vigilance Scores, Pupil Size</td>
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<td>Mind-State (MS)</td>
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<td>Subject)</td>
</tr>
<tr>
<td>N/A</td>
<td>Vigilance Scores</td>
<td>Probe</td>
<td>SW Density (SWD; average across electrodes)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
<tr>
<td>N/A</td>
<td>Vigilance Scores</td>
<td>Probe</td>
<td>SW Amplitude (SWA; average across electrodes)</td>
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<td>Subject)</td>
</tr>
<tr>
<td>N/A</td>
<td>Vigilance Scores</td>
<td>Probe</td>
<td>SW Slope (SWS; average across electrodes)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
<tr>
<td>3c</td>
<td>Slow Wave Density (SWD); Amplitude (SWA); Slope (SWS)</td>
<td>Probe</td>
<td>Mind-State (MS)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
<tr>
<td>4a*</td>
<td>false alarms, misses, reaction times</td>
<td>Trial</td>
<td>Local Sleep (LS; per electrode)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
<tr>
<td>4b*</td>
<td>Slow Wave Amplitude (SWA); Slope (SWS)</td>
<td>Probe</td>
<td>Local Sleep (LS; per electrode)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
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<tr>
<td>5*</td>
<td>a, t, z, vGo, vNoGo, vBias</td>
<td>Subject</td>
<td>Local Sleep (LS; per electrode)</td>
<td>X ~ 1 + Task + (1</td>
<td>Subject)</td>
</tr>
</tbody>
</table>

*: Analyses corrected for multiple comparison (see Methods and Supplementary Methods).
Supplementary Figure 1. Hierarchical Drift Diffusion Modelling

**a:** The Go/NoGo tasks were modelled according to the Drift Diffusion Model (DDM, see Methods and Supplementary Methods). The following parameters were fitted: threshold ($a$), non-decision time or NDT ($t$), bias ($z$), drift rate for Go trials ($v_{GO}$), drift rate for NoGo trials ($v_{NOGO}$) and drift bias (abs($v_{GO}$)-abs($v_{NOGO}$)). The figure shows a graphical representation of these parameters. Note that here, drift rates for NoGo trials are negative. **b-c:** Graphical representation of decision processes using the parameters obtained by the DDM for trials with (LS+) or without (LS-) local sleep. Local sleep was defined as the presence of slow waves on electrodes FCz (b; frontal) and Oz (c; posterior). Note the reduction in decision threshold, drift rates and bias associated with local sleep but the increase in NDT.
Supplementary Figure 2. Hierarchical DDM Fit to Behavioral Data

Posterior predictive checks of Go/No-Go DDM fit to the behavioural data. Observed data (black bars) are plotted underneath model-predicted RT distributions and No-Go choice proportions (red lines). Positive distribution represents the normalised frequency of reaction times (RT) from Go responses. Negative bin at RT=-1 represents the proportion of No-Go responses.
Supplementary Figure 3. Spatial expanse of local sleep events

Four seeds electrodes were selected along the scalp midline from the front (Fz) to back (Oz). For each local sleep event (slow wave) detected in these seed electrodes, we computed the probability that local sleep was also observed in the other channels. The average co-occurrence probability averaged across participants (N=26) is shown for each seed electrode. Note that slow waves detected over Fz tend to co-occur with other local sleep events in a limited number of neighboring channels whereas occipital local sleep (Oz) tend to co-occur with local sleep events in both frontal and posterior electrodes (more widespread).