Research report

Predator-induced plasticity in sleep architecture in wild-caught Norway rats (*Rattus norvegicus*)

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Abstract

Sleep is a prominent behaviour in the lives of animals, but the unresponsiveness that characterizes sleep makes it dangerous. Mammalian sleep is composed of two neurophysiological states: slow wave sleep (SWS) and rapid-eye-movement (REM) sleep. Given that the intensity of stimuli required to induce an arousal to wakefulness is highest during deep SWS or REM sleep, mammals may be most vulnerable during these states. If true, then animals should selectively reduce deep SWS and REM sleep following an increase in the risk of predation. To test this prediction, we simulated a predatory encounter with 10 wild-caught Norway rats (*Rattus norvegicus*), which are perhaps more likely to exhibit natural anti-predator responses than laboratory strains. Immediately following the encounter, rats spent more time awake and less time in SWS and REM sleep. The reduction of SWS was due to the shorter duration of SWS episodes, whereas the reduction of REM sleep was due to a lower number of REM sleep episodes. The onset of SWS and REM sleep was delayed post-encounter by about 20 and 100 min, respectively. The reduction of REM sleep was disproportionately large during the first quarter of the sleep phase, and slow wave activity (SWA) (0.5–4.5 Hz power density) was lower during the first 10 min of SWS post-encounter. An increase in SWA and REM sleep was observed later in the sleep phase, which may reflect sleep homeostasis. These results suggest that aspects of sleep architecture can be adjusted to the prevailing risk of predation.
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1. Introduction

Sleep is a reversible period of relative unresponsiveness to the environment associated with changes in the electroencephalogram (EEG). The EEG of a sleeping mammal alternates between two basic states: slow wave sleep (SWS) and rapid-eye-movement (REM) sleep. SWS-related slow wave activity (SWA, approximately 0.5–4.5 Hz power density) is typically greatest at the start of a sleep period and declines thereafter [1]. Short-term (<24 h) sleep deprivation increases SWA further as a function of prior time spent awake, and the intensity of stimuli required to induce wakefulness (i.e., the arousal threshold) is greater as SWA increases [2], collectively suggesting that SWS is homeostatically regulated with SWA reflecting the intensity of SWS [3,4,1]. Although SWS is most intense at the start of a sleep bout, under certain conditions it may be beneficial to delay the appearance of deep SWS (high SWA) to a later time in response to ecological demands, such as an increase in the risk of predation. However, it is unknown whether animals can modulate the intensity of SWS in this way. As arousal thresholds during REM sleep are also high [5,6], animals might selectively reduce REM sleep as well in response to heightened risk.

An increase in the risk of predation causes changes in other animal behaviours [7–10], and the same may also be true for sleep [11,12]. For example, foraging animals are often less able to detect an approaching predator than those overtly alert [13]; when predation risk increases, such animals reduce their foraging rate in favour of more vigilant behaviours [14]. A sleeping animal is also much less likely to detect an approaching predator than when awake, and some data suggest that predators can target less-vigilant [15,16] and potentially sleeping [17] prey. Indeed, the fact that many animals take measures to reduce

their exposure to predators during sleep (e.g., burrows, grouping, etc.) indicates that they are vulnerable when sleeping. Ducks (*Anas platyrhynchos*) sleeping in riskier environments, such as the edge of a group, engage in more unihemispheric sleep and maintain the contralateral eye to the awake hemisphere open and directed away from the group ([18]; see [19,20] for earlier behavioural work on ducks and doves). In mammals, the only predation-related work is that on sleep in domesticated laboratory rodents (reviewed in [21]). For example, laboratory mice conditioned to associate an auditory tone with an unavoidable foot shock engage in less REM sleep during subsequent sleep bouts [22,23]. However, it is unclear whether the reduction in REM sleep following mild electric shock or fear conditioning in laboratory rodent strains (where anti-predator behaviour may be reduced or absent; e.g., [24]) reflects a natural response to recent predatory encounters.

Here, we examine predator-induced plasticity in sleep architecture in wild-caught Norway rats (*Rattus norvegicus*). We predicted that rats subjected to a higher perceived risk of predation would initially spend more time awake, and when sleep ultimately occurred it would be reduced in REM sleep and deep SWS. Overall, our results show that when faced with heightened risk, rats exhibit more vigilant (less deep) forms of sleep.

2. Materials and methods

2.1. Trapping

Rats were trapped at a farm 8 km west of Terre Haute, Indiana, USA (39.5°N 87.5°W), using standard Tomahawk traps (Tomahawk Live Trap Co.) baited with peanut butter and oats. Traps were checked twice daily, once at sunrise and once at sunset.

2.2. Housing and care

Ten rats (6 males and 4 females, mean weight = 277 g, range 179–361 g) were kept in a room maintained at 25 °C on a 12-h light:12-h dark schedule with lights-on at 06:00 h and off at 18:00 h. Rats were housed individually in stainless steel cages (length = 62 cm, width = 46 cm, height = 37 cm). The front door of each cage was made of transparent Plexiglas. As a human experimenter served as the simulated predator (see Section 2.4 below), it was important to minimize frequent contact with the rats. Thus, a metal chute that delivered food *ad libitum* into the feeding dish without opening the cage door was mounted onto the front door of each cage; a water spout on the back wall delivered water *ad libitum*. Rats were given commercially available rat chow daily at 18:00 h (lights-off) as Norway rats are largely nocturnal [25], see also [26]. Shelter was provided in the form of a plastic sleeping box. A grate on the bottom of the cage allowed urine and feces to fall into a removable stainless steel tray filled with pine chips beneath. Rats were housed for experimentation under an Indiana State University IACUC-approved protocol (#9-15-05:CJA/JL).

2.3. EEG electrode implantation

To record the EEG, two electrodes were implanted over each cerebral hemisphere. Rats were anesthetized using isoflurane (2–4% vaporized in 1.0 LPM O_2). Four holes (3.175 mm) were drilled through the cranium to the level of the dura. The two holes overlying each hemisphere were symmetrical on either side of the sagittal suture. Anterior holes were drilled 2.0 mm anterior of the bregma and 2.5 mm lateral of the sagittal suture (i.e., 2.0A, 2.5L); posterior holes were drilled 5.5 mm posterior of the bregma and 3.0 mm lateral of the sagittal suture (i.e., −5.5P, 3.0L). A Teflon-coated multi-stranded 36-gauge stainless steel wire (Cooner Wire Co.) was inserted into each hole and secured with a stainless

steel screw (Plastics One, Inc.). The screw was then covered with dental acrylic (DuraLay, Reliance Dental Mfg. Co.). Electrode wires terminated at a Dale connector that served as the headplug. Rats were given at least 2 weeks recovery before experimentation.

2.4. Experimental protocol

Before the baseline recording, the recording tether was secured to the headplug and to a commutator (Dragonfly, Inc., model number SL-10). A portion of the top half of the sleeping box was removed to make it possible for a rat to enter the box with the tether attached. The rats were given enough food and water to last until the end of the experimental trial. EEG and video (10 frames/s) were recorded from the rats during two 12 h periods; a wall-mounted video camera was located about 2 m away from the front of the cage. After 2 days of acclimating to the tether, the first 12 h period started at 06:00 h and served as the baseline. At 05:50 h the following day, a researcher entered the (dark) recording room, opened the cage door and chased, but did not touch, the rat around its cage with a gloved hand; illumination (<1 lux at the level of the rat) was provided by a small dimmed flashlight. A rat was chased until it showed an obvious escape response (e.g., running or jumping) (between 0.5 and 2 min). The experimenter then left the recording room just prior to the onset of the normal sleeping phase at 06:00 h (lights-on). EEG and video recordings ended 12 h later at 18:00 h. Rats were run two at a time (in two separate cages) whenever possible.

2.5. EEG recordings

Two bipolar EEG derivations (2.0A, 2.5L − −5.5P, 3.0L) were amplified and filtered (the low cut filter was set at 0.3 Hz and the high cut filter was set at 30 Hz) using a Grass Model 12C2 amplifier (Grass Technologies, Astro-Med, Inc.). The signals were then digitally sampled and stored at 256 samples/s using Grass Gamma 4.7 software.

2.6. State scoring

The state of the rats (wakefulness, SWS, transition sleep, and REM sleep) was scored using synchronized EEG and video recordings for each 30 s epoch of the baseline and post-encounter days (see [27] for details). An epoch was scored as a particular state when that state occupied more than one half of the 30 s epoch. The EEG and behavioural correlates of wakefulness were a high-frequency, low-amplitude pattern often associated with motor activity and active exploration of the cage. SWS was identified as an EEG of low-frequency (<4 Hz), high-amplitude waves accompanied by behavioural quiescence. The EEG of REM sleep consisted of high-frequency and consistently low-amplitude activity with a theta rhythm (7–8 Hz), and occurred only during periods of quiescence with occasional skeletomuscular twitches. REM sleep always appeared either after an episode of SWS or after an episode of a high-frequency, high-amplitude transition between SWS and REM sleep; this state that has been reported previously in several rodent species (see [28]), including laboratory rats [27], and also recently in the ferret (Order: Carnivora, *Mustela putorius furo*, [29]). This transition state was observed predominantly between adjacent episodes of SWS and REM sleep, but sometimes occurred between episodes of SWS and wakefulness (in the absence of REM sleep) and perforating a bout of SWS (see also [30]). Following other studies, we henceforth refer to this state as 'transition sleep'.

2.7. Spectral analysis

To measure potential predator-induced plasticity in the depth of SWS, we calculated SWS-related spectral power density using 5 s fast Fourier transforms (0.5 Hz bins) from the left hemisphere EEG derivation using commercially available software (Embla, Somnologica Science 3.3.1). We illustrated SWA during SWS in terms of time spent in SWS rather than time elapsed since the start of the sleep phase, because rats had significantly less SWS immediately post-encounter (see Section 3). Only artifact-free epochs consisting entirely of SWS were included in the spectral analyses. Spectral power density is presented as a percentage of average power density over the entire baseline day per individual rat.

2.8. Statistical analysis

We compared the time spent in each state – wakefulness, SWS, transition sleep, and REM sleep – between the 12 h baseline day and the 12 h post-encounter day divided into four quarters (06:00–09:00, 09:00–12:00, 12:00–15:00, 15:00–18:00). We made the same baseline–post-encounter comparisons for the duration and number of state episodes. For SWS, we compared the duration of time before the appearance of the first SWS episode (SWS onset latency) and spectral power density between the 2 days. We also compared the percentage of total sleep time allocated to REM sleep, and the duration of time between the first episode of SWS and the first episode of REM sleep (REM sleep onset latency).

For most variables, our full statistical model was a two-way repeated measures analysis of variance (rmANOVA) quantifying the main effects of treatment (baseline day, post-encounter day), time (quarter of day), and the interaction between treatment and time. The main effect of treatment and/or the interaction between treatment and time was almost always significant, so we conducted paired *t*-tests to determine the time point(s) at which significance was reached. Paired *t*-tests were used to analyze sleep onset latencies. We note that the insulation on the recording tether for one rat became compromised at the end of hour seven of the post-encounter day, thus comparisons involving data including and beyond hour eight are based on data for nine rats only. Data were log-transformed (as indicated) when necessary to meet the assumptions of parametric statistical analyses. All statistical analyses were conducted in either Systat 10 (SPSS, Inc.) or SPSS 15.0 (SPSS, Inc.).

3. Results

For all variables measured, being chased by the gloved hand had the greatest effect during the first few hours post-encounter. We quantify these effects on each of the four scored states (wakefulness, SWS, transition sleep, and REM sleep) in turn below.

The amount of time spent awake (Fig. 1a) increased during the first quarter of the post-encounter day ($F_{3,61} = 9.165$, $P < 0.001$ for treatment × time interaction; $t = 5.374$, d.f. = 9, $P < 0.001$ for pairwise comparison of first quarter). The duration of episodes of wakefulness (Fig. 1b) also increased during the first quarter of the post-encounter day ($F_{1,61} = 4.280$, $P = 0.043$ for main effect of treatment; $t = 1.998$, d.f. = 9, $P = 0.077$ for pairwise comparison of first quarter). The number of episodes of wakefulness (Fig. 1c) was not significantly different between the baseline and post-encounter days ($F_{1,61} = 1.686$, $P = 0.199$).

The amount of time spent in SWS (Fig. 2a) decreased during the first quarter of the post-encounter day ($F_{3,61} = 6.547$, $P < 0.001$ for treatment × time interaction; $t = -3.993$, d.f. = 9, $P = 0.003$ for pairwise comparison of first quarter). There was a statistical tendency towards a small increase in the time spent in SWS during the third quarter of the day ($t = 2.208$, d.f. = 8, $P = 0.058$), a pattern exhibited by 8 of the 9 rats. The duration of SWS episodes (Fig. 2b) likewise decreased during the first quarter of the post-encounter day ($F_{3,61} = 6.403$, $P < 0.001$ for treatment × time interaction; $t = -5.751$, d.f. = 9, $P < 0.001$ for pairwise comparison of first quarter). The number of SWS episodes (Fig. 2c) was significantly lower on the post-encounter day ($F_{1,61} = 4.354$, $P = 0.041$ for main effect of treatment), but did not differ significantly at any time point ($P \geq 0.083$ for pairwise comparisons). The onset of SWS was delayed significantly post-encounter (baseline: 3.1 ± 1.1 min vs. post-encounter: 25.4 ± 7.5 min; $t = 2.900$, d.f. = 9, $P = 0.018$).

SWS-related SWA (0.5–4.5 Hz power density; Fig. 3) differed significantly between the baseline and post-encounter days
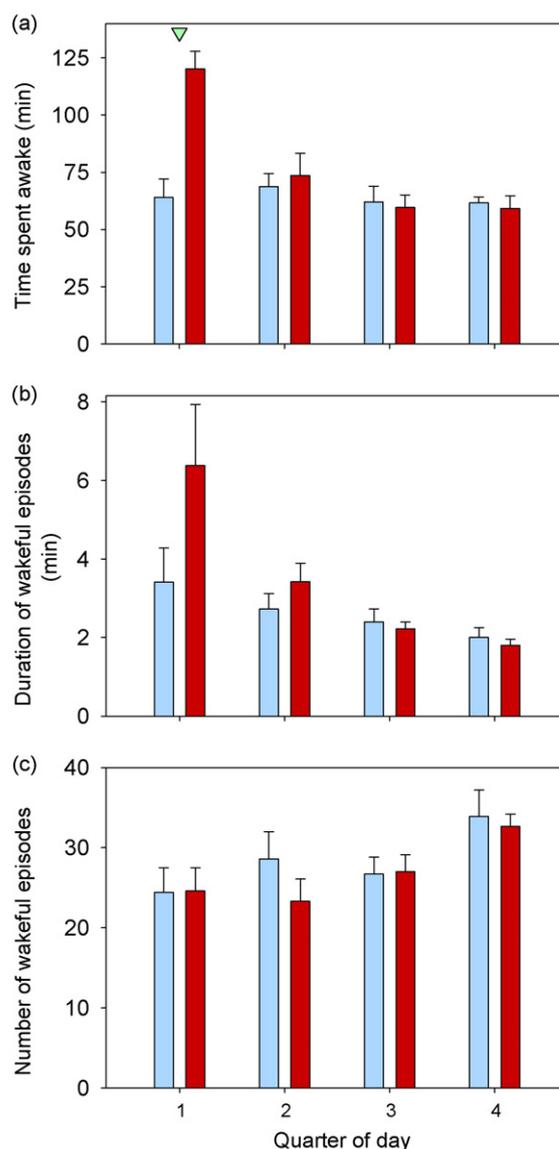

Fig. 1. (a) The time spent in wakefulness over the four quarters of the baseline (blue) and post-encounter (red) days, as well as the (b) duration and (c) number of episodes of wakefulness. Data are presented as means ± S.E. Significance is denoted by triangles at the top of each plot above a given pairwise comparison. Data underlying panel b were log-transformed for analysis, but are presented here untransformed so that the units are more easily interpretable (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).

($F_{1,296} = 11.609$, $P < 0.001$ for main effect of treatment). Power density was significantly lower during the first 10 min of SWS of the post-encounter day ($t = -2.793$, d.f. = 8, $P = 0.024$), an effect that extended into the 2.0–9.5 Hz bandwidth (Fig. 4a). The rats also took significantly longer to accrue the first 10 min of SWS post-encounter (baseline: 15.4 ± 1.9 min vs. post-encounter: 48.4 ± 9.5 min; $t = 3.201$, d.f. = 8, $P = 0.013$) and the post-encounter number of SWS episodes was significantly greater during this period (baseline: 3.78 ± 0.64 vs. post-encounter: 7.89 ± 0.73; $t = 3.786$, d.f. = 8, $P = 0.005$). SWA was generally greater during the post-encounter day once about 30 min of SWS had accrued, but not all differences at a given
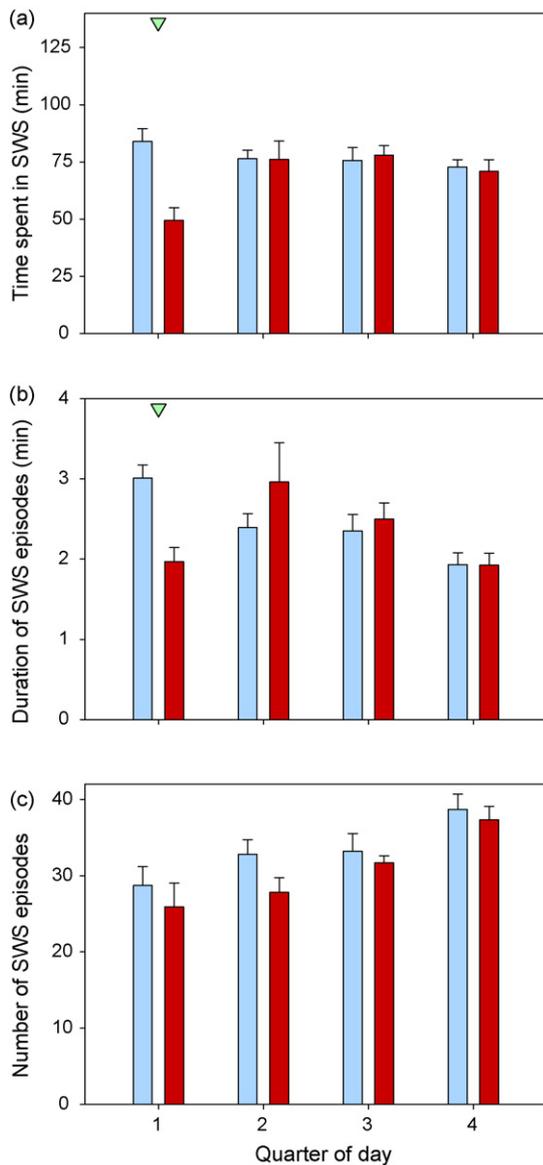
Fig. 2. (a) The time spent in SWS, as well as the (b) duration and (c) number of SWS episodes over the four quarters of the baseline (blue) and post-encounter (red) days. Data are presented as means ± S.E. Significance is denoted by triangles at the top of each plot above a given pairwise comparison. Data underlying panel b were log-transformed for analysis, but are presented here untransformed so that the units are more easily interpretable (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).
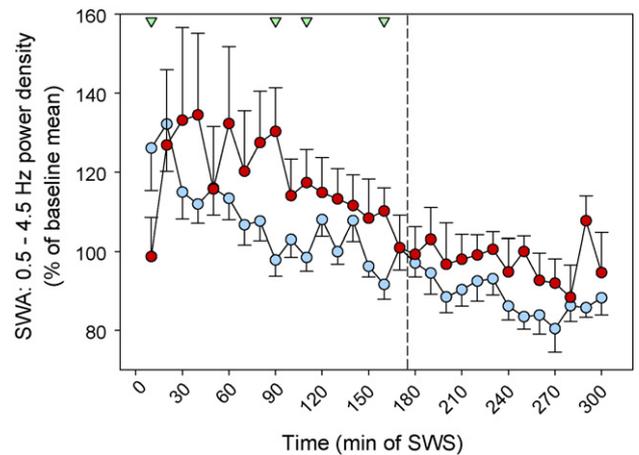

Fig. 3. SWS-related SWA (0.5–4.5 Hz power density) across 10 min time bins of SWS (excluding intervening wakefulness) for the baseline (blue) and post-encounter (red) days. The repeated measures ANOVA was based only on data to the left of the vertical dashed line at 170 min of SWS where $N = 10$ rats; sample sizes to the right side of this line decline quickly (especially post-encounter as most rats did not accrue 300 min of SWS). SWA (mean ± S.E.) is expressed as a percentage of 12 h baseline mean per individual rat. Significance is denoted by triangles at the top of the plot above a given pairwise comparison (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).

time point were significantly different from the baseline (Fig. 3). The post-encounter increase in power density was not specific to the 0.5–4.5 Hz bandwidth. For instance, once 90 min of SWS had accrued (Fig. 4b), the increase of SWS-related spectral power density was significant in both the 2.0–8.0 Hz and 13.0–24.5 Hz bandwidths. At the sixteenth 10 min bin of SWS (Fig. 4c), the low-frequency increase in power density (1.5–7.0 Hz) was still significant. For these spectral power analyses, data from one rat for the first time bin was excluded as it was a statistical outlier (studentized residual = 2.585) that showed a marked increase in SWA following treatment.

The time spent in transition sleep (Fig. 5a) did not differ significantly between the baseline and post-encounter days ($F_{1,61} = 1.557$, $P = 0.217$), nor did the duration of episodes of transition sleep (Fig. 5b; $F_{1,59} = 0.187$, $P = 0.667$). The number of transition sleep episodes (Fig. 5c) decreased significantly during the first quarter of the post-encounter day ($F_{3,61} = 3.426$, $P = 0.023$ for treatment × time interaction; $t = -3.933$, d.f. = 9, $P = 0.003$ for pairwise comparison of first quarter).

The time spent in REM sleep (Fig. 6a) decreased significantly during the first quarter of the post-encounter day ($F_{3,61} = 9.049$, $P < 0.001$ for treatment × time interaction; $t = -10.358$, d.f. = 9, $P < 0.001$ for pairwise comparison of first quarter) and increased significantly during the last quarter ($t = 2.531$, d.f. = 8, $P = 0.035$). The duration of REM sleep episodes (Fig. 6b) was not affected by treatment ($F_{1,58} = 0.026$, $P = 0.874$). The number of REM sleep episodes (Fig. 6c) decreased significantly during the first quarter of the post-encounter day ($F_{3,61} = 4.987$, $P = 0.004$ for treatment × time interaction; $t = -7.378$, d.f. = 9, $P < 0.001$ for pairwise comparison of first quarter). The REM sleep onset latency increased significantly post-encounter (baseline: 13.3 ± 3.4 min vs. post-encounter: 114.8 ± 24.3 min; $t = 4.347$, d.f. = 9, $P = 0.002$). The percentage of total sleep time allocated to REM sleep (%REM sleep; Fig. 7) likewise decreased significantly during the first-quarter of the post-encounter day ($F_{3,61} = 9.017$, $P < 0.001$ for treatment × time interaction; $t = -6.281$, d.f. = 9, $P < 0.001$ for pairwise comparison of first quarter), and tended to be higher during the last quarter ($t = 1.899$, d.f. = 8, $P = 0.094$).

4. Discussion

Rats altered their sleep and waking behaviour after being chased by a simulated predator (gloved hand) just prior to the
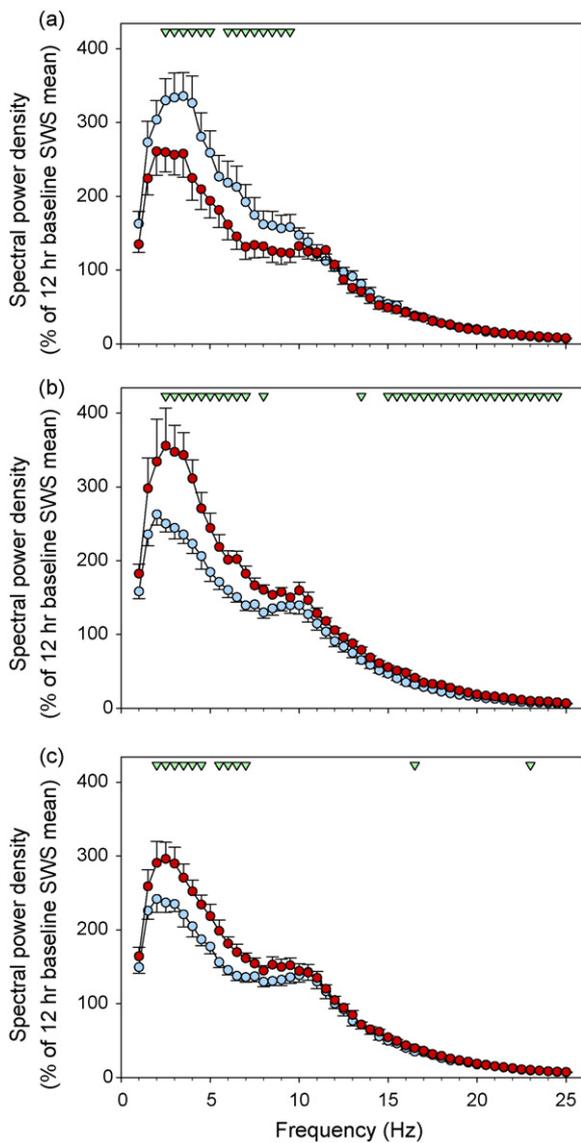
Fig. 4. Spectral power density (0.5–25.0 Hz) for (a) the first 10 min of SWS, (b) the ninth 10 min bin of SWS, and (c) the sixteenth 10 min bin of SWS for the baseline (blue) and post-encounter (red) days. These three time points correspond to significant pairwise comparisons in Fig. 3. Power density (mean ± S.E.) is expressed as a percentage of 12 h baseline SWS mean per individual rat. Significance is denoted by triangles at the top of each plot above a given pairwise comparison (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).
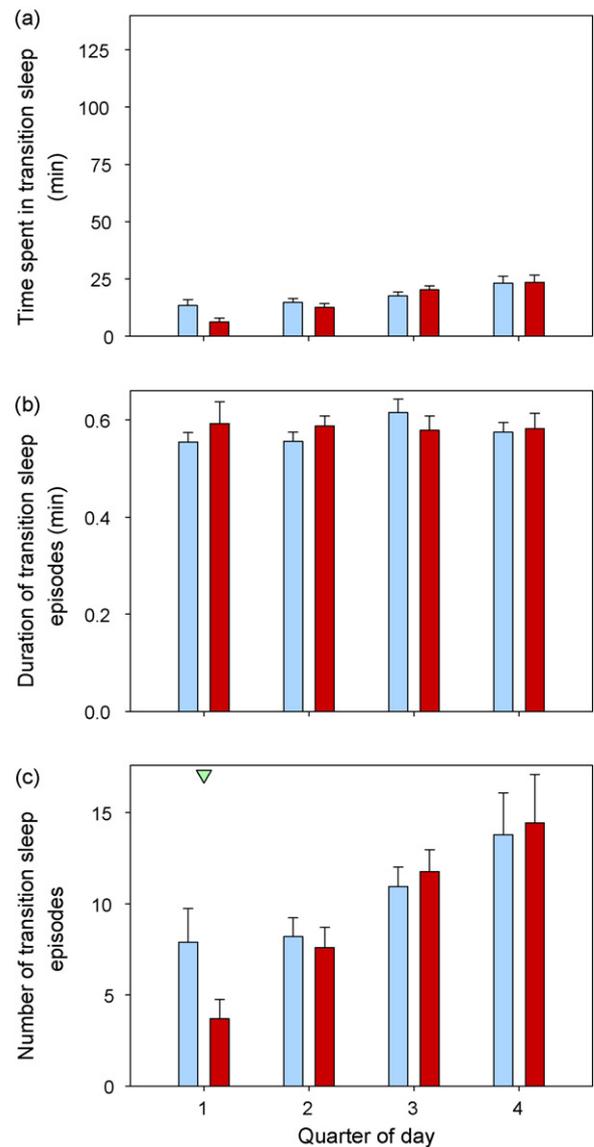

Fig. 5. (a) The time spent in transition sleep over the four quarters of the baseline (blue) and post-encounter (red) days, as well as the (b) duration and (c) number of transition sleep episodes. Data are presented as means ± S.E. Significance is denoted by triangles at the top of each plot above a given pairwise comparison (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).

onset of the light phase of the photoperiod (i.e., the normal sleeping phase for Norway rats). During the first 3 h of the baseline day, rats spent the majority of their time sleeping, but post-encounter, the rats passed most of this time awake. This increase in wakefulness was largely due to an increase in the duration of episodes of wakefulness, rather than an increase in the number of individual wakeful episodes. These basic results suggest that the rats were threatened by the gloved hand and perceived an increase in the risk of predation.

Concurrent with the increase in wakefulness was a reduction in the time spent in SWS. During the first quarter of the day, the rats reduced their time in SWS by 37% following the threatening encounter. The reduction of SWS was due primarily to a decrease

in the duration of SWS episodes, rather than to a decrease in the number of SWS episodes. The appearance of the first SWS episode was delayed by about 20 min post-encounter. In addition to the reduction in the time spent in SWS, SWA decreased during the first portion of SWS following the simulated predatory encounter. This reduction in power density suggests that the intensity or depth of SWS was reduced immediately after the interaction with the putative predator. It is tempting to conclude that rats are able to actively modulate the depth of SWS in response to the increase in the risk of predation. However, increased sleep fragmentation during the first hour following the threatening encounter coupled with the shorter duration of SWS episodes could have resulted in SWS with lower SWA as SWA is lower at the onset of SWS [31]. Such an interpretation
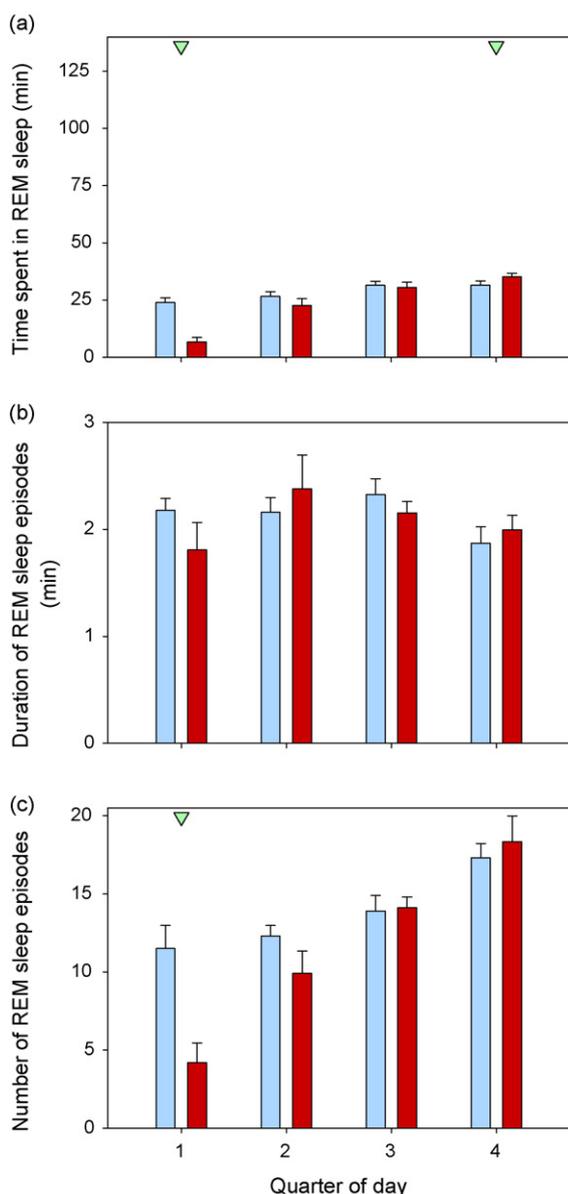
Fig. 6. (a) The time spent in REM sleep over the four quarters of the baseline (blue) and post-encounter (red) days, as well as the (b) duration and (c) number of REM sleep episodes. Data are presented as means ± S.E. Significance is denoted by triangles at the top of each plot above a given pairwise comparison (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).

Fig. 7. The percentage of total sleep time allocated to REM sleep for the baseline (blue) and post-encounter days (red). Data are presented as means ± S.E. Significance is denoted by triangles at the top of the plot above a given pairwise comparison (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article).

does not negate the idea that rats can control the depth of SWS in response to ecological demands; it does, however, suggest that rats do so (in part) indirectly through the fragmentation of SWS. Nonetheless, it is also possible that when faced with a less immediate, but more persistent measure of risk (e.g., sleep sites that differ in their accessibility to predators), rats might remain asleep, and modulate the depth of SWS according to the prevailing level of risk. In any case, further study into the modulation of SWA in response to ecologically relevant demands represents an exciting avenue for future research.

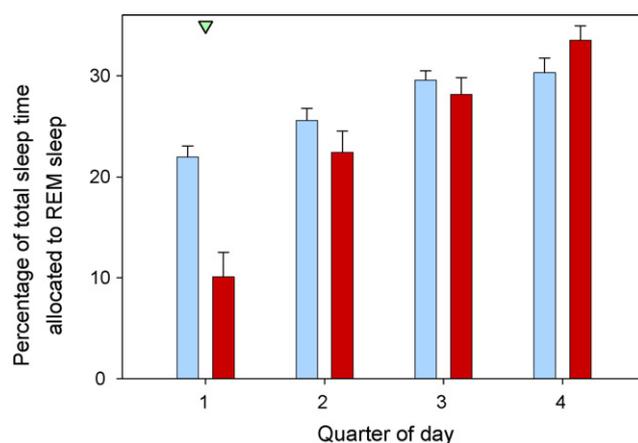SWS-related spectral power density during much of the post-encounter day was *greater* relative to baseline, an effect that was present in higher frequencies (2.0–8.0 Hz and 13.0–24.5 Hz) as well. Interestingly, this bimodal increase in SWS-related low- and high-frequency activity has been observed in the nocturnal Syrian hamster (*Mesocricetus auratus*) following 3 h of total sleep deprivation beginning at lights-on and may reflect covert activation during SWS [32], see also [33]. The low-frequency (1.5–7.0 Hz) rebound remained significant until (at least) 160 min of SWS had accrued. Because the rats were awake twice as much immediately after the brief exposure to the simulated predator, the subsequent increase in SWA could reflect a homeostatic response to increased wakefulness [1,34,33]. In conjunction with sleep loss, the possible change in the quality of the resulting wakefulness could explain the rebound in SWA if the additional wakefulness was more activated and/or if short-term memories of the interaction with the simulated predator required consolidation during subsequent sleep [35]. Indeed, several recent studies have found that exposure to particularly stressful situations can increase SWA during subsequent sleep (e.g., [35–37]), and that this increase is not simply a glucocorticoid mediated stress response [38].

The effects of the threatening encounter on REM sleep were more pronounced than for SWS. The time spent in REM sleep was reduced by 75% for the 3 h immediately following the simulated predatory encounter. The percentage of total sleep time allocated to REM sleep (%REM sleep) during the first quarter of the post-encounter day was also less than half of that of the baseline day, suggesting that REM sleep is a particularly dangerous sleep state that is selectively reduced when faced with an increased risk of predation. Thus, although animals awakened from REM sleep may be better prepared for wakefulness (see [11]), they appear to be more vulnerable beforehand. Furthermore, the onset of REM sleep was delayed by over 100 min following the threatening encounter. Unlike SWS, the reduction in REM sleep was not due to a reduction in the duration of REM sleep episodes, but rather to a decrease in the number of REM sleep episodes. The time spent in REM sleep and %REM sleep increased during the last 3 h of the day, which may reflect REM

sleep homeostasis. Measures of transition sleep were largely unaffected by the simulated predatory encounter.

Other studies have also found REM sleep to be more sensitive to stressors than SWS. A recent comparative analysis found that mammalian species sleeping under higher risks of predation engage in less REM sleep, but not significantly less SWS, than those sleeping in safer locations [12], see also [39]. This phylogenetic pattern might represent an evolutionary strategy to reduce vulnerability during sleep by favouring more vigilant (less deep) forms of sleep. Unfortunately, however, sufficient comparative data are lacking on the depth of SWS, hence that variable could not be included in the Lesku et al. [12] analysis. Recent studies on laboratory mice found that REM sleep was reduced during trials involving unavoidable footshocks preceded by an auditory tone, an effect which persisted for several hours after presentation of the auditory tone in the absence of the footshock [22,23], see also [21]. As in our study, the number of REM sleep episodes and the percentage of total sleep time allocated to REM sleep were also reduced in these mice [22,23]. Interestingly, the magnitude of these reductions was specific to the degree of anxiousness displayed by each laboratory strain, such that more anxious lines showed a stronger sleep-related response [22,23]. Similar findings have been reported in laboratory mice moved to an open (potentially risky) environment [40]. The broad congruence between the results of our study on wild rats and those obtained using laboratory varieties [21] validates the use of laboratory strains in the stress and fear literature.

Perspectives: The fact that rats reduced sleep following the threatening encounter suggests that sleeping is dangerous. Certain states of sleep, such as REM sleep and deep SWS, appear to be more dangerous than others. Interestingly, REM sleep and deeper SWS appear to be particularly important in facilitating enhancements in performance [41–44], suggesting that the vulnerability associated with the deeper forms of sleep is the specific cost for plasticity during wakefulness [44]. An examination of the functional consequences of predator-induced plasticity in sleep architecture on memory processing and plasticity might provide insight into the tradeoff between an ecological demand for anti-predator vigilance and the physiological requirement for sleep.

Acknowledgements

References

[1] Borbély AA, Achermann P. Sleep homeostasis and models of sleep regulation. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th ed. Philadelphia: WB Saunders; 2005. p. 405–17.

[2] Neckelmann D, Ursin R. Sleep stages and EEG power spectrum in relation to acoustical stimulus arousal threshold in the rat. Sleep 1993;16:467–77.

[3] Borbély AA, Tobler I, Hanagasioglu M. Effect of sleep-deprivation on sleep and EEG power spectra in the rat. Behav Brain Res 1984;14:171–82.

[4] Tobler I, Borbély AA. Sleep EEG in the rat as a function of prior waking. Electroencephalogr Clin Neurophysiol 1986;64:74–6.

[5] Dillon RF, Webb WB. Threshold of arousal from activated sleep in the rat. J Comp Physiol Psychol 1965;59:447–9.

[6] Tolaas J. REM sleep and the concept of vigilance. Biol Psychiatry 1978;13:135–48.

[7] Sih A. Predators and prey lifestyles: an evolutionary and ecological overview. In: Kerfoot WC, Sih A, editors. Predation: direct and indirect impacts on aquatic communities. Hanover: University Press of New England; 1987. p. 203–24.

[8] Sih A. Predation risk and the evolutionary ecology of reproductive behavior. J Fish Biol 1994;45:111–30.

[9] Lima SL, Dill LM. Behavioural decisions made under the risk of predation: a review and prospectus. Can J Zool 1990;68:619–40.

[10] Lima SL. Stress and decision making under the risk of predation: recent developments from behavioral, reproductive, and ecological perspectives. Adv Study Behav 1998;27:215–90.

[11] Lima SL, Rattenborg NC, Lesku JA, Amlaner CJ. Sleeping under the risk of predation. Anim Behav 2005;70:723–36.

[12] Lesku JA, Roth TC, Amlaner CJ, Lima SL. A phylogenetic analysis of sleep architecture in mammals: the integration of anatomy, physiology, and ecology. Am Nat 2006;168:441–53.

[13] Lima SL, Bednekoff PA. Temporal variation in danger drives antipredator behavior: the predation risk allocation hypothesis. Am Nat 1999;153:649–59.

[14] Lima SL. Vigilance while feeding and its relation to the risk of predation. J Theor Biol 1987;124:303–16.

[15] FitzGibbon CD. A cost to individuals with reduced vigilance in groups of Thomson's gazelles hunted by cheetahs. Anim Behav 1989;37:508–10.

[16] Krause J, Godin JGJ. Influence of prey foraging posture on flight behavior and predation risk: predators take advantage of unwary prey. Behav Ecol 1996;7:264–71.

[17] Fredriksson GM. Predation on sun bears by reticulated python in East Kalimantan, Indonesian Borneo. Raffles Bull Zool 2005;53:165–8.

[18] Rattenborg NC, Lima SL, Amlaner CJ. Half-awake to the risk of predation. Nature 1999;397:397–8.

[19] Lendrem DW. Sleeping and vigilance in birds. I. Field observations of the mallard (*Anas platyrhynchos*). Anim Behav 1983;31:532–8.

[20] Lendrem DW. Sleeping and vigilance in birds. II. An experimental study of the barbary dove (*Streptopelia risoria*). Anim Behav 1984;32:243–8.

[21] Pawlyk AC, Morrison AR, Ross RJ, Brennan FX. Stress-induced changes in sleep in rodents: models and mechanisms. Neurosci Biobehav Rev 2008;32:99–117.

[22] Sanford LD, Tang XD, Ross RJ, Morrison AR. Influence of shock training and explicit fear-conditioned cues on sleep architecture in mice: strain comparison. Behav Gen 2003;33:43–58.

[23] Sanford LD, Yang LH, Tang XD. Influence of contextual fear on sleep in mice: a strain comparison. Sleep 2003;26:527–40.

[24] Koide T, Moriwaki K, Ikeda K, Niki H, Shiroishi T. Multi-phenotype behavioral characterization of inbred strains derived from wild stocks of *Mus musculus*. Mam Gen 2000;11:664–70.

[25] Nowak RM. Walker's mammals of the world. Baltimore: Johns Hopkins University Press; 1999.

[26] van Twyver H. Sleep patterns of five rodent species. Physiol Behav 1969;4:901–5.

[27] Datta S, Hobson JA. The rat as an experimental model for sleep neurophysiology. Behav Neurosci 2000;114:1239–44.

[28] Tobler I, Deboer T. Sleep in the blind mole rat *Spalax ehrenbergi*. Sleep 2001;24:147–54.

[29] Jha SK, Coleman T, Frank MG. Sleep and sleep regulation in the ferret (*Mustela putorius furo*). Behav Brain Res 2006;172:106–13.

[30] Mandile P, Vescia S, Montagnese P, Romano F, Giuditta A. Characterization of transition sleep episodes in baseline EEG recordings of adult rats. Physiol Behav 1996;60:1435–9.

[31] Trachsel L, Tobler I, Borbély AA. Electroencephalogram analysis of non-rapid eye movement sleep in rats. Am J Physiol 1988;255:R27–37.

[32] Tobler I, Jaggi K. Sleep and EEG spectra in the Syrian hamster (*Mesocricetus auratus*) under baseline conditions and following sleep deprivation. J Comp Physiol A 1987;161:449–59.

[33] Martinez-Gonzalez D, Lesku JA, Rattenborg NC. Increased EEG spectral power density during sleep following short-term sleep deprivation in pigeons (*Columba livia*): evidence for avian sleep homeostasis. J Sleep Res,2008, 17, doi:10.1111/j.1365-2869.2008.00636.x.

[34] Tobler I. Phylogeny of sleep regulation. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th ed. Philadelphia: WB Saunders; 2005. p. 77–90.

[35] Meerlo P, de Bruin EA, Strijkstra AM, Daan S. A social conflict increases EEG slow-wave activity during subsequent sleep. Physiol Behav 2001;73:331–5.

[36] Hellman K, Abel T. Fear conditioning increases NREM sleep. Behav Neurosci 2007;121:310–23.

[37] Tang XD, Yang LH, Sanford LD. Interactions between brief restraint, novelty and footshock stress on subsequent sleep and EEG power in rats. Brain Res 2007;1142:110–8.

[38] Chang FC, Opp MR. Role of corticotropin-releasing hormone in stressor-induced alterations of sleep in rat. Am J Physiol 2002;283:R400–7.

[39] Lesku JA, Roth TC, Rattenborg NC, Amlaner CJ, Lima SL. Phylogenetics and the correlates of mammalian sleep: a reappraisal. Sleep Med Rev, 2008, 12, doi:10.1016/j.smrv.2007.10.003.

[40] Tang XD, Xiao JH, Liu XL, Sanford LD. Strain differences in the influence of open field exposure on sleep in mice. Behav Brain Res 2004;154:137–47.

[41] Gais S, Born J. Declarative memory consolidation: mechanisms acting during human sleep. Learn Mem 2004;11:679–85.

[42] Huber R, Ghilardi MF, Massimini M, Tononi G. Local sleep and learning. Nature 2004;430:78–81.

[43] Stickgold R. Sleep-dependent memory consolidation. Nature 2005;437:1272–8.

[44] Tononi G, Cirelli C. Sleep function and synaptic homeostasis. Sleep Med Rev 2006;10:49–62.