J. Sleep Res. (2008) 17, 140–153 Sleep in animals

doi: 10.1111/j.1365-2869.2008.00636.x

Increased EEG spectral power density during sleep following short-term sleep deprivation in pigeons (*Columba livia*): evidence for avian sleep homeostasis

DOLORES MARTINEZ-GONZALEZ, JOHN A. LESKU and NIELS C. RATTENBORG

Max Planck Institute for Ornithology, Seewiesen, Germany

Accepted in revised form 11 December 2007; received 7 September 2007

SUMMARY Birds provide a unique opportunity to evaluate current theories for the function of sleep. Like mammalian sleep, avian sleep is composed of two states, slow-wave sleep (SWS) and rapid eye-movement (REM) sleep that apparently evolved independently in mammals and birds. Despite this resemblance, however, it has been unclear whether avian SWS shows a compensatory response to sleep loss (i.e., homeostatic regulation), a fundamental aspect of mammalian sleep potentially linked to the function of SWS. Here, we prevented pigeons (*Columba livia*) from taking their normal naps during the last 8 h of the day. Although time spent in SWS did not change significantly following short-term sleep deprivation, electroencephalogram (EEG) slow-wave activity (SWA; i.e., 0.78–2.34 Hz power density) during SWS increased significantly during the first 3 h of the recovery night when compared with the undisturbed night, and progressively declined thereafter in a manner comparable to that observed in similarly sleep-deprived mammals. SWA was also elevated during REM sleep on the recovery night, a response that might reflect increased SWS pressure and the concomitant 'spill-over' of SWS-related EEG activity into short episodes of REM sleep. As in rodents, power density during SWS also increased in higher frequencies (9–25 Hz) in response to short-term sleep deprivation. Finally, time spent in REM sleep increased following sleep deprivation. The mammalian-like increase in EEG spectral power density across both low and high frequencies, and the increase in time spent in REM sleep following sleep deprivation suggest that some aspects of avian and mammalian sleep are regulated in a similar manner.

KEYWORDS bird, evolution, pallium, phylogeny, sleep function, slow-wave activity

INTRODUCTION

As in mammals, birds exhibit slow-wave sleep (SWS) and rapid eye-movement (REM) sleep (reviewed in Campbell and Tobler, 1984; Rattenborg and Amlaner, 2002). In both taxonomic groups, the electroencephalogram (EEG) during SWS is characterized by high-amplitude, low-frequency (≤ 4.0 Hz) activity, whereas the EEG during REM sleep resembles the low-amplitude, high-frequency activity of wakefulness. In contrast, reptiles do not show high-amplitude, low-frequency EEG activity during sleep, indicating that birds and mammals independently evolved forebrain structures necessary for generating the EEG patterns that characterize mammalian and avian SWS (Rattenborg, 2006). In this respect, comparative studies of sleep in birds and mammals provide a unique opportunity to investigate the still disputed function(s) of sleep (Rechtschaffen, 1998; Siegel, 2005; Lesku *et al*., 2006; Roth *et al*., 2006; Tononi and Cirelli, 2006; Lima and Rattenborg, 2007; Rattenborg *et al.,* 2007; Rial *et al*., 2007; Stickgold and Walker, 2007; Lesku *et al*., 2008).

Despite the gross similarities in the EEG correlates of SWS between mammals and birds, SWS might be regulated

Correspondence: Niels C. Rattenborg, PhD, Max Planck Institute for Ornithology – Seewiesen, Sleep & Flight Group, Eberhard-Gwinner-Strasse, 82139 Seewiesen, Germany. Tel.: +49 (0)8157 932 279; fax: +49 (0)8157 932 344; e-mail: rattenborg@orn.mpg.de

differently in each taxonomic group. In mammals that engage in extended periods of wakefulness, EEG slow-wave activity (SWA; approximately 0.5–4.5 Hz power density) is highest during the first bout of SWS and progressively declines thereafter (reviewed in Borbély and Achermann, 2005). Extending wakefulness beyond that normally experienced on a daily basis increases subsequent SWA further. In humans, taking a nap in the day reduces SWA during non-REM sleep at night (Werth *et al*., 1996). This relationship between time spent awake and SWS-related SWA has been found in every mammalian species investigated (reviewed in Tobler, 2005). The link between prior time awake and SWA, as well as the positive correlation between arousal threshold and SWA (Frederickson and Rechtschaffen, 1978; Neckelmann and Ursin, 1993), suggest that SWS is homeostatically regulated with SWA reflecting the intensity of SWS and presumed SWS-related functions (reviewed in Borbély and Achermann, 2005; see also Huber *et al*., 2004).

The homeostatic regulation of SWS is a fundamental aspect of mammalian sleep that forms the foundation for current theories proposed for the function of SWS (e.g., Krueger and Obál, 1993, 2003; Benington and Frank, 2003; Tononi and Cirelli, 2003, 2006). Given that SWS homeostasis is likely to be directly linked to the function of SWS (Benington, 2000), an obvious question is: *Do birds, which show similar sleep-related EEG activity, also show an increase in SWA following sleep deprivation?* The only EEG-based studies that examined the effect of extending wakefulness in birds did not detect an increase in SWS-related SWA (0.75–4.5 Hz power density: Tobler and Borbély, 1988; 0.75–4.0 Hz power density: Berger and Phillips, 1994) following long-term (≥ 24 h) sleep deprivation in pigeons (*Columba livia*). The apparent absence of a compensatory response to sleep deprivation suggested that the avian forebrain lacks the neural cytoarchitecture necessary for SWS homeostasis, as present in the mammalian neocortex (Zepelin *et al*., 2005). Although the avian pallium and pallial mammalian neocortex are derived from homologous embryonic neural tissue, and function in a similar manner during wakefulness (Emery and Clayton, 2004; Jarvis *et al*., 2004), the avian pallium is arranged in a nuclear manner that lacks the true laminar organization of the neocortex (Medina and Reiner, 2000; Jarvis *et al*., 2004; Reiner, 2005), a possible requisite for the EEG expression of SWS homeostasis.

The apparent absence of a compensatory response to sleep loss in birds would seem to challenge the taxonomic applicability of functional sleep theories that hinge on SWS homeostasis, or at least suggest that SWA is regulated differently in birds, and therefore may be associated with different functions (see Roth *et al*., 2006). Nevertheless, it may be premature to conclude that SWS is regulated differently in mammals and birds (Tobler, 2005). In rodents, the increase in SWA observed following short-term sleep deprivation, is no longer evident following long-term sleep deprivation (Rechtschaffen *et al*., 1999). This pattern is most evident in the Syrian hamster (*Mesocricetus auratus*) where SWA (0.25–4.0 Hz) increased following 3 h, but not 24 h of sleep deprivation (Tobler and Jaggi, 1987). Given this relationship between the duration of sleep deprivation and subsequent SWA in hamsters, a shorter (i.e., < 24 h), presumably more ecologically realistic, period of sleep deprivation might induce a compensatory increase in SWA during recovery SWS in birds as well. To determine whether pigeons show a compensatory increase in SWA following short-term sleep deprivation, we compared SWS-related SWA during a normal night to that occurring immediately following 8 h of sleep deprivation. To detect any regional differences in recovery sleep similar to that observed in mammals (Vyazovskiy *et al*., 2002), we recorded the EEG from the dorsal surface of anterior, medial, and posterior pallial regions of each cerebral hemisphere. We expected SWA during recovery SWS at night to (1) initially increase above baseline levels and (2) to progressively decline across the recovery night following sleep deprivation. A preliminary report of our findings was previously published in abstract form (Rattenborg and Martinez-Gonzalez, 2007).

METHODS

General protocol

The EEG was recorded from pigeons during two consecutive 24-h periods under a 12 : 12 light–dark photoperiod with lights on at 07:00 and off at 19:00 h. The first 24-h period started at 07:00 and served as a baseline. Starting at 11:00 on the second day, the pigeons were kept awake for 8 h during the day, half of which is normally spent asleep (see section Results). At lights out the birds were allowed to sleep undisturbed. The experiment ended at the end of the second night.

Animals and housing

Five adult tippler pigeons (*C. livia*; three females; two males) were purchased from a local breeder. Each bird was housed in an individual wooden enclosure (length = 79 cm, width = 60 cm, height = 60 cm). A rectangular opening on the enclosure door covered with wire mesh provided ventilation. The back wall was made of white translucent Plexiglas. Fluorescent lights placed behind the upper third of the Plexiglas wall and room light entering through the opening in the cage door provided light in the daytime (400–500 lux measured at head level in the center of the enclosure). An infrared-sensitive camera was placed in each corner of the enclosure, and an infrared (940 nm) light source hung from the center of the ceiling provided luminance for the cameras at night. The floor of the enclosure was covered with paper and an inverted ceramic dish placed in the center served as a perch. The birds were given mixed grain pigeon feed, grit and water *ad libitum*. Each bird was able to hear, but not able to see the other bird in the recording room. The temperature was maintained between 23.5 °C and 25.5 °C. The experiments were approved by the Government of Upper Bavaria and adhere to the NIH standards for using animals in research.

EEG electrode implantation

To detect potential regional differences in the response to sleep deprivation, six EEG electrodes were placed over each cerebral hemisphere using standard stereotaxic techniques. In brief, after establishing a suitable surgical anesthetic plane using isoflurane (1.5–2.0% vaporized in 1.0 LPM O_2) 14 holes (0.5 mm diameter) were drilled through the exposed cranium to the level of the dura. The 12 holes for the EEG electrodes were arranged in three rows (i.e., anterior, medial and posterior pallium) with four holes per row. The anterior row was positioned at AP + 13.0 mm, the medial row at AP + 9.25 mm and the posterior row at AP + 4.75 mm (Karten and Hodos, 1967). Within a row, holes were drilled 2.0 and 6.0 mm lateral (i.e., L 2.0 and L 6.0) of the midline on each side. In the anterior and medial rows, the medial electrodes were positioned over the hyperpallium apicale (L 2.0) and the lateral electrodes (L 6.0) were positioned over the mesopallium (see Reiner, 2005 for new avian brain nomenclature). In the posterior row, the medial electrodes (L 2.0) were positioned over the area parahippocampalis and the lateral electrodes (L 6.0) were positioned over the area corticoidea dorsolateralis, thin contiguous structures overlying the nidopallium caudale and separated from it by a narrow ventricular space. An additional hole for the reference electrode was placed over the center of the cerebellum, and a hole for the ground electrode was placed 2 mm anterior of the anterior (L 2.0) hole on the right hemisphere. All electrodes were made from gold-plated pins (0.5 mm diameter) with rounded tips. Each electrode was placed on the dura and glued in position using cyanoacrylic adhesive and wired to a connector that was mounted on the head with Paladur® dental acrylic (Heraeus Kulzer, Hanau, Germany, http://www.heraeus-kulzer.com). The birds were connected to the recording cable after at least 5 days of post-operative recovery in the recording enclosure. The recording cable was connected to a swivel (Plastics One®, Inc., Roanoke, VA, USA, http://www.plastics1.com) mounted on the ceiling of the enclosure. Baseline recordings started after at least 1 week of adaptation to the recording cable.

EEG recordings

Each EEG signal was referenced to the cerebellum and digitally recorded at 200 Hz using commercially available amplifiers (Embla® A10) and software (Embla®, Somnologica Science v. 3.3.1, Broomfield, CO, USA, http://www.embla.com). The low-cut finite impulse response (FIR) filter was set at 0.5 Hz (−6 dB at 0.5 Hz and 0 dB at 0.78 Hz) and the high-cut anti-aliasing FIR filter was set at 100 Hz (−20 dB at 100 Hz and 0 dB at 80 Hz). The Embla® amplifiers automatically apply the anti-ailiasing filter after first sampling the data at 2000 Hz (see http://www.embla.com/support/knowbase/show.asp?ID = 172). The data are then down-sampled to 200 Hz. A 50 Hz notch filter eliminated potential electrical interference. No additional filtering was applied after data acquisition. Bipolar EEG recordings were created offline for the anterior (AP + 13.0, L 6.0–L 2.0), medial (AP + 9.25, L 6.0–L 2.0) and posterior (AP + 4.75, L 6.0–L 2.0) pallia of each hemisphere and used for sleep staging and spectral power density analysis. Monopolar recordings with each pallial electrode referenced to the cerebellum showed results similar to these bipolar derivations (data not shown), as did a bipolar derivation (AP + 4.5, L 2.0–AP + 13.0, L 2.0) that most closely approximated that used in an earlier sleep deprivation experiment on pigeons (AP + 6.0, L 2.0–AP + 13.0, L 2.0; Tobler and Borbély, 1988).

Sleep deprivation

As in previous short-term sleep deprivation studies in mammals, the pigeons were kept awake using the 'gentle handling' technique. Whenever high-amplitude, low-frequency activity appeared in any EEG recording, we stimulated the pigeon by tapping or moving the floor of their enclosure, making a noise or, towards the end of the deprivation, gently touching the bird.

Sleep staging

The state of the pigeons was visually determined for each 4-s epoch using a combination of EEG and video recordings. Three states were scored: wakefulness, SWS and REM sleep. Wakefulness was characterized by relatively low-amplitude, high-frequency EEG activity. SWS was scored when more than half of an epoch showed low-frequency activity with an amplitude approximately twice that of alert wakefulness. In each case, the onset of scored SWS typically corresponded with the onset of sleep behavior (e.g., immobility, head drawn into the chest and one or both eyes closed). Because pigeons show interhemispheric asymmetries in low-frequency activity during SWS (Rattenborg *et al.*, 2001), SWS was scored whenever at least one EEG recording met SWS criteria. In practice, as in a previous study (Rattenborg *et al.*, 2001), both hemispheres usually showed some level of SWS-related EEG activity simultaneously. REM sleep was characterized by periods of EEG activation (> 2 s) occurring in association with bilateral eye closure and behavioral signs of reduced muscle tone (e.g., head dropping, swaying, and sliding of the wings off the side of the body). Finally, we calculated the duration of each episode of each sleep state by summing the number of consecutive epochs scored as the same state.

Spectral power density analysis

Fast Fourier transforms were performed on EEG recordings to calculate power density in 0.39 Hz bins between 0.78 and 25 Hz during each 4-s scoring epoch (Embla®, Broomfield, CO, USA, http://www.embla.com). EEG artifacts were visually detected and omitted from the analysis. Most artifacts were associated with gross movements during wakefulness (e.g., preening, feeding, walking). Because the pigeons were rarely awake and motionless, the majority (> 80%) of the

EEG during wakefulness was contaminated with artifacts. Consequently, wakefulness was not included in the spectral analysis. In contrast to wakefulness, on average >95% of the sleeping epochs were artifact free on the baseline and recovery nights and therefore included in the spectral analysis (98.67 ± 0.69% (mean ± SEM) and 95.41 ± 2.25% during baseline and recovery SWS, respectively; 97.75 ± 0.99% and 97.73 ± 0.80% during baseline and recovery REM sleep, respectively). For each sleep state and frequency bin, we expressed the average spectral power density for each quarter of the baseline and recovery nights as a percent of the entire baseline night average for that state.

A previous study in pigeons found that SWA during SWS was lower when the birds had at least one eye open (Tobler and Borbély, 1988). To detect potential changes in spectral power density during recovery SWS related to changes in the proportion of SWS occurring with eyes open or closed, the state of each eye was determined at the start of each minute during the first 3 h of the baseline and recovery nights, when changes in spectral power density were expected to be the greatest. For each eye, we calculated the proportion of corresponding epochs scored as SWS during which that eye was open or closed.

Statistics

We used two-way or three-way repeated-measures analysis of variance (rmANOVA) and two-tailed paired *t*-tests when comparing various aspects of wakefulness and sleep between the baseline and recovery conditions. In instances where the overall rmANOVA model was significant, we conducted paired *t*-tests to identify specific comparisons for which significance was reached. To characterize the time course of SWA on the baseline and recovery nights, one-way rmANOVAS were conducted on the data for each night separately. Variables were transformed (when necessary) to meet the assumption of normality of residuals. All statistical analyses were conduced in either Systat 10 (SPSS 2000) or SPSS 15 (SPSS 2006).

RESULTS

Efficacy of sleep deprivation

The gentle handling technique was successful in reducing sleep (Fig. 1). During the 8-h period of sleep deprivation, the proportion of time spent awake (Fig. 1a) increased significantly relative to baseline (baseline, 48.74 ± 1.43% versus deprivation, 91.58 ± 3.32%; $F = 183.043$; df = 1,60; $P < 0.001$). Likewise, the time spent in SWS (Fig. 1b) was significantly reduced (baseline, 45.19 ± 1.82% versus deprivation, 8.42 ± 3.32%; $F = 160.562$; df = 1,60; $P < 0.001$) and REM sleep (Fig. 1c) did not occur at all during this period (baseline, 6.07 ± 0.86% versus deprivation, 0.00 ± 0.00%; $F = 134.640$; df = 1,60; $P < 0.001$). The small amount of residual SWS during sleep deprivation occurred in brief episodes lasting 2–4 s, the time required to detect sleep and stimulate the bird.

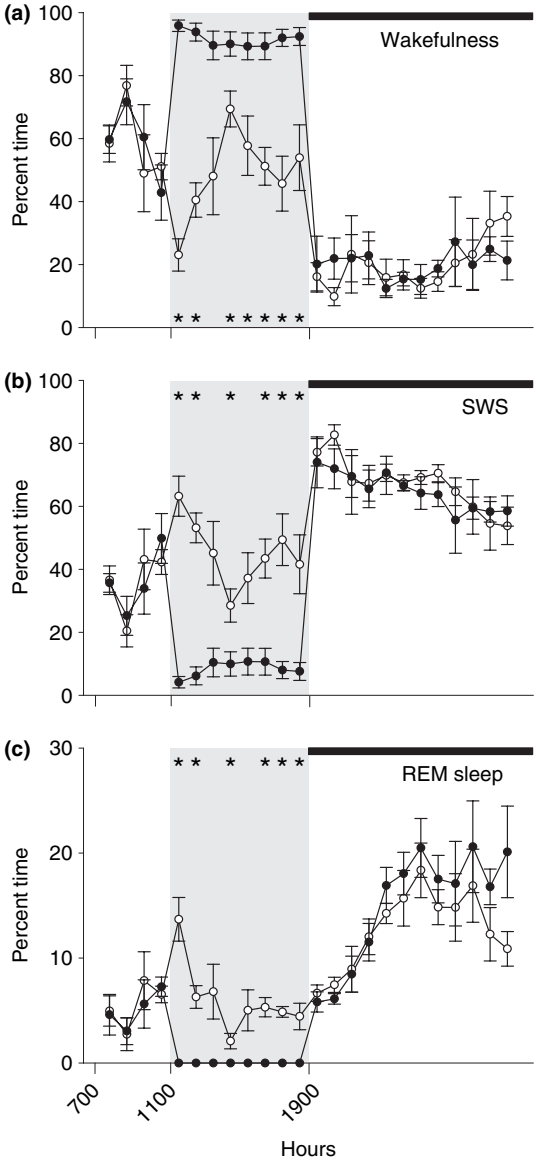

Figure 1. Effect of sleep deprivation on time spent in wakefulness (a), slow-wave sleep (b) and rapid eye-movement (REM) sleep (c). The percent time (mean ± SEM) spent in each state for each hour of the first and second 24-h periods is plotted at the middle of each hour. The first 24-h period served as an undisturbed baseline (open circles). The second 24-h period (filled circles) is divided into an additional 4-h period of baseline prior to the start of sleep deprivation (07:00–11:00 h), 8 h of sleep deprivation (11:00–19:00 h) shaded in grey, and 12 h of recovery following sleep deprivation (19:00–07:00 h). The black bar at the top, right of each plot indicates night. Statistical differences ($P < 0.05$, two-tailed, paired *t*-test) between the baseline and recovery nights are indicated by an asterisk. Although the rmANOVA revealed an effect of treatment on time spent in REM sleep at night, none of the hourly *post hoc* comparisons were significantly different between the baseline and recovery nights.

Baseline and recovery sleep architecture

During the 12-h night of recovery, the time spent awake did not differ significantly from the baseline night (Fig. 1a; baseline, 20.17 ± 4.28% versus recovery, 20.21 ± 2.36%;

$F < 0.001$, df = 1,92, $P = 0.988$), nor did the time spent in SWS (Fig. 1b; baseline, 67.08 ± 2.95% versus recovery, 64.84 ± 1.68%; $F = 0.943$, df = 1,92, $P = 0.334$). In contrast to SWS, time spent in REM sleep increased following sleep deprivation (baseline, 12.75 ± 1.64% versus recovery, 14.95 ± 1.79%; $F = 9.190$, df = 1,92, $P = 0.003$; Fig. 1c).

Sleep duration

Figure 2 shows the duration of SWS (a) and REM sleep (b) episodes across the baseline and recovery nights. The duration of episodes of SWS decreased ($F = 17.021$, df = 11,92, $P < 0.001$) and the duration of REM sleep episodes increased ($F = 21.337$, df = 11,92, $P < 0.001$) across both nights. However, the duration of both SWS and REM sleep episodes decreased on the recovery night when compared with the baseline night ($F = 18.569$, df = 1,92, $P < 0.001$ and $F = 4.303$, df = 1,92, $P = 0.041$, respectively). This seems to reflect more frequent switching between SWS and REM sleep on the recovery night, rather than shorter episodes of sleep (SWS and REM sleep combined), as the duration of sleep episodes (Fig. 2c) was not different between the baseline and recovery nights ($F = 0.005$, df = 1,92, $P = 0.942$). For the statistical tests on sleep state episode duration, the data were log transformed to meet the assumption of normality of residuals.

Spectral power density

Slow-wave sleep

Figure 3a shows spectral power density (0.78–25 Hz) during SWS on the baseline night for each region of the pallium. In general, spectral power density below 2.5 Hz was highest during the first *or* second quarter of the night and lowest during the last in all regions except the left and right posterior pallia. The relative power density of high frequencies (approximately 5–25 Hz) were lowest during the first and highest during the last quarter of the baseline night in the posterior pallia. Components of this pattern were also evident to a lesser extent in the medial and anterior pallia (Fig. 3a).

Figure 3b shows spectral power density (0.78–25 Hz) during SWS on the recovery night for each region of the pallium. Although the amount of time spent in SWS did not increase during recovery (Fig. 1b), power density was significantly affected by prior sleep deprivation. Notably, power density below 2.5 Hz was greatest during the first quarter of the night and then decreased across each successive quarter in the left and right, anterior and medial pallia. In the left and right anterior pallia, this effect extended out to 5.08 Hz. In addition to the increase in low frequencies, high-frequency (approximately, 9–25 Hz) power density also increased significantly during the first quarter in the left anterior pallia, and to varying degrees across all quarters of the recovery night in the right anterior and left medial pallia (approximately, 8–15 Hz).

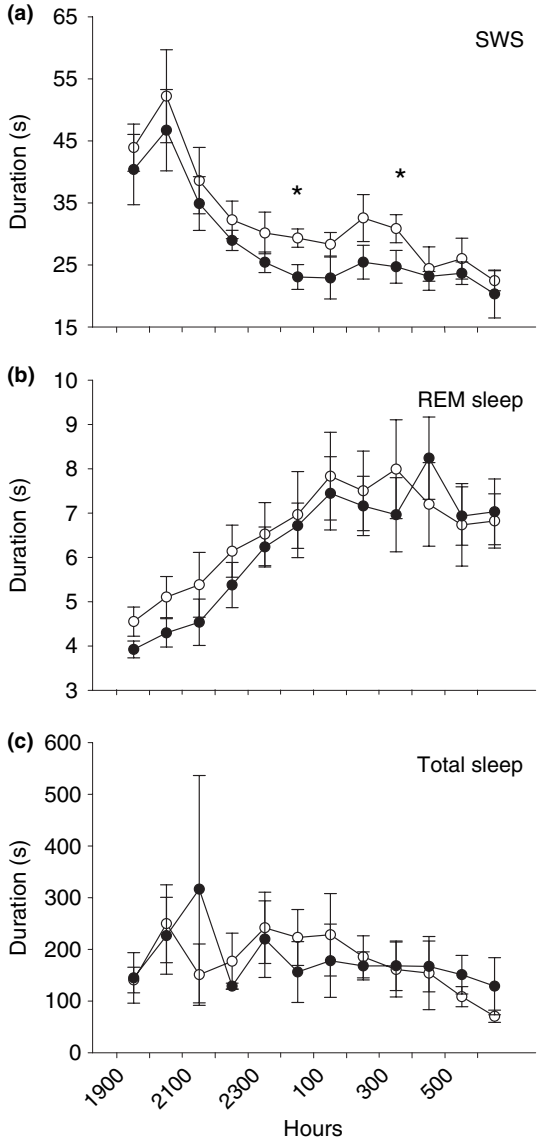

Figure 2. The effect of sleep deprivation on the duration of sleep episodes. The duration of slow-wave sleep (SWS) (a), rapid eye-movement (REM) sleep (b) and total sleep (SWS and REM sleep combined) (c) episodes across each hour of the baseline (open circles) and recovery (filled circles) nights. The mean (±SEM) duration is plotted in seconds at the middle of each hour. Data underlying this figure were log transformed for analysis, but is presented here untransformed so that the units are more easily interpretable. Statistical differences ($P < 0.05$, two-tailed, paired *t*-test) between the baseline and recovery nights are indicated by an asterisk. Although the rmANOVA revealed an effect of treatment on the duration of SWS and REM sleep episodes at night, none of the hourly *post hoc* comparisons were significantly different between the baseline and recovery nights for REM sleep.

The posterior pallia showed a response different from the other regions. Power density in the left posterior pallium was largely unaffected by prior sleep deprivation, whereas the right posterior pallium showed a pronounced increase in mean power density below 2.5 Hz. Unlike the anterior and medial pallia where the results were largely consistent across all birds,

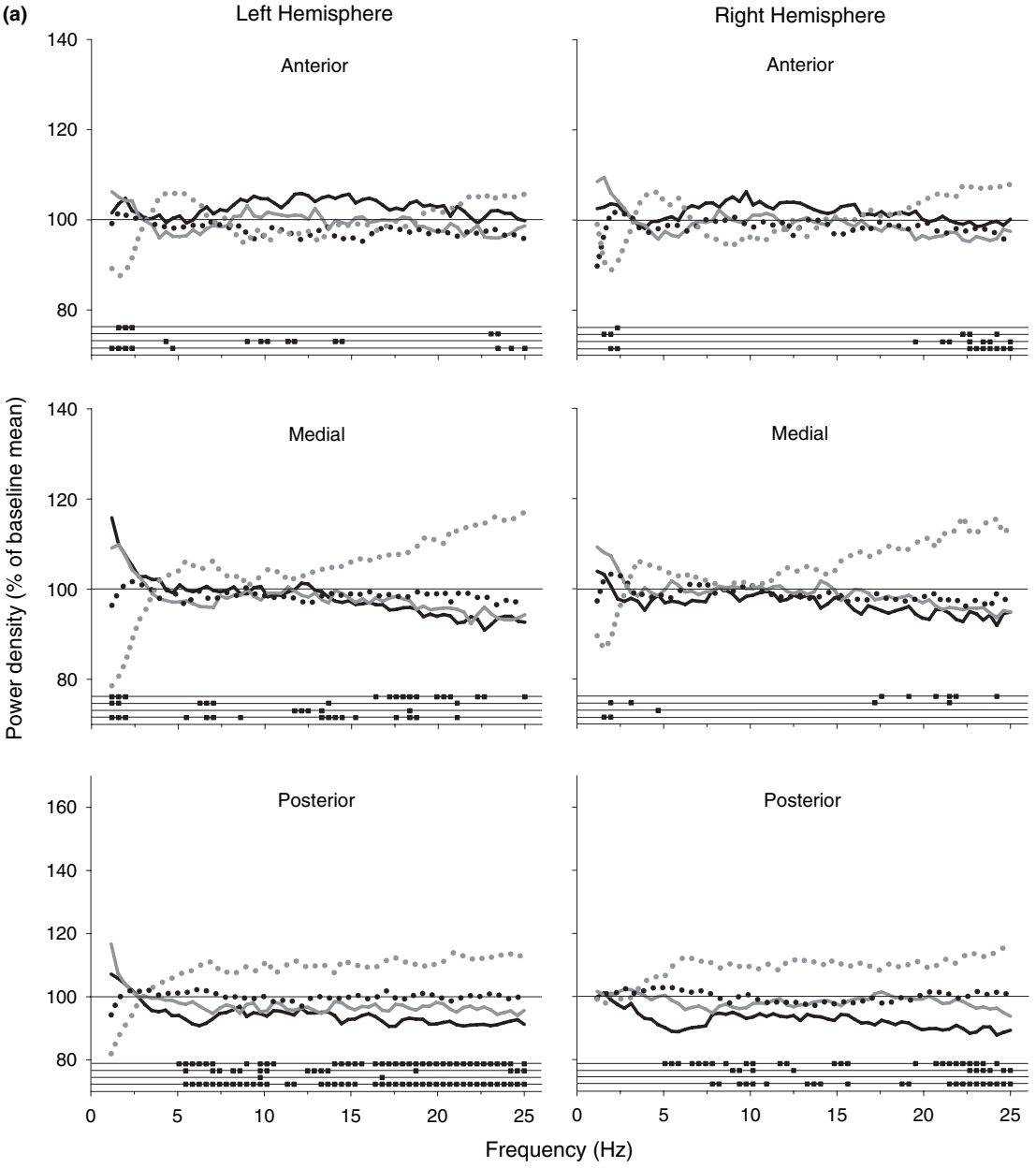

Figure 3. EEG spectral power density (0.78–25 Hz) during slow-wave sleep (SWS) on the baseline (a) and recovery (b) nights. The power density for each quarter (1st, solid black line; 2nd, solid grey line; 3rd, dotted black line; 4th, dotted grey line) of each night is expressed as a percent of the *entire baseline night* SWS mean (i.e., the 100% line) for each frequency bin and brain region (left and right, anterior, medial and posterior pallia) in each pigeon. The mean percent is plotted at the end of each frequency bin. For the baseline night, values for each quarter and frequency bin were compared to the baseline night average. Significant differences ($P < 0.05$, two-tailed paired *t*-test after significant rmANOVA) are indicated by filled squares on the lines at the bottom of each plot; statistical data for the first through fourth quarters are presented on the first (top) through fourth (bottom) lines, respectively. For the recovery night, values for each quarter and frequency bin were compared to the corresponding *quarter of the baseline night*, with significant differences similarly indicated at the bottom of each plot.

however, the increase in low-frequency power in the right posterior pallium was present in only three birds; the remaining two showed a much smaller response, and as a result, although the increase in *mean* power density was greatest in this region (note the different scale on the plots for the posterior pallia), it only reached statistical significance for the first frequency bin (0.78–1.17 Hz) during the first quarter of the recovery night.

Figure 4 summarizes the changes in SWA (i.e., 0.78–2.34 Hz) during SWS across the baseline and recovery nights for the left and right, anterior and medial pallia. Sleep deprivation significantly increased SWA during recovery in the left and right anterior pallia (left, $F = 40.959$, df = 1,28, $P < 0.001$; right, $F = 4.424$, df = 1,28, $P = 0.045$) and the left and right medial pallia (left, $F = 9.170$, df = 1,28, $P = 0.005$; right, $F = 6.604$, df = 1,28, $P = 0.016$). SWS-related SWA during

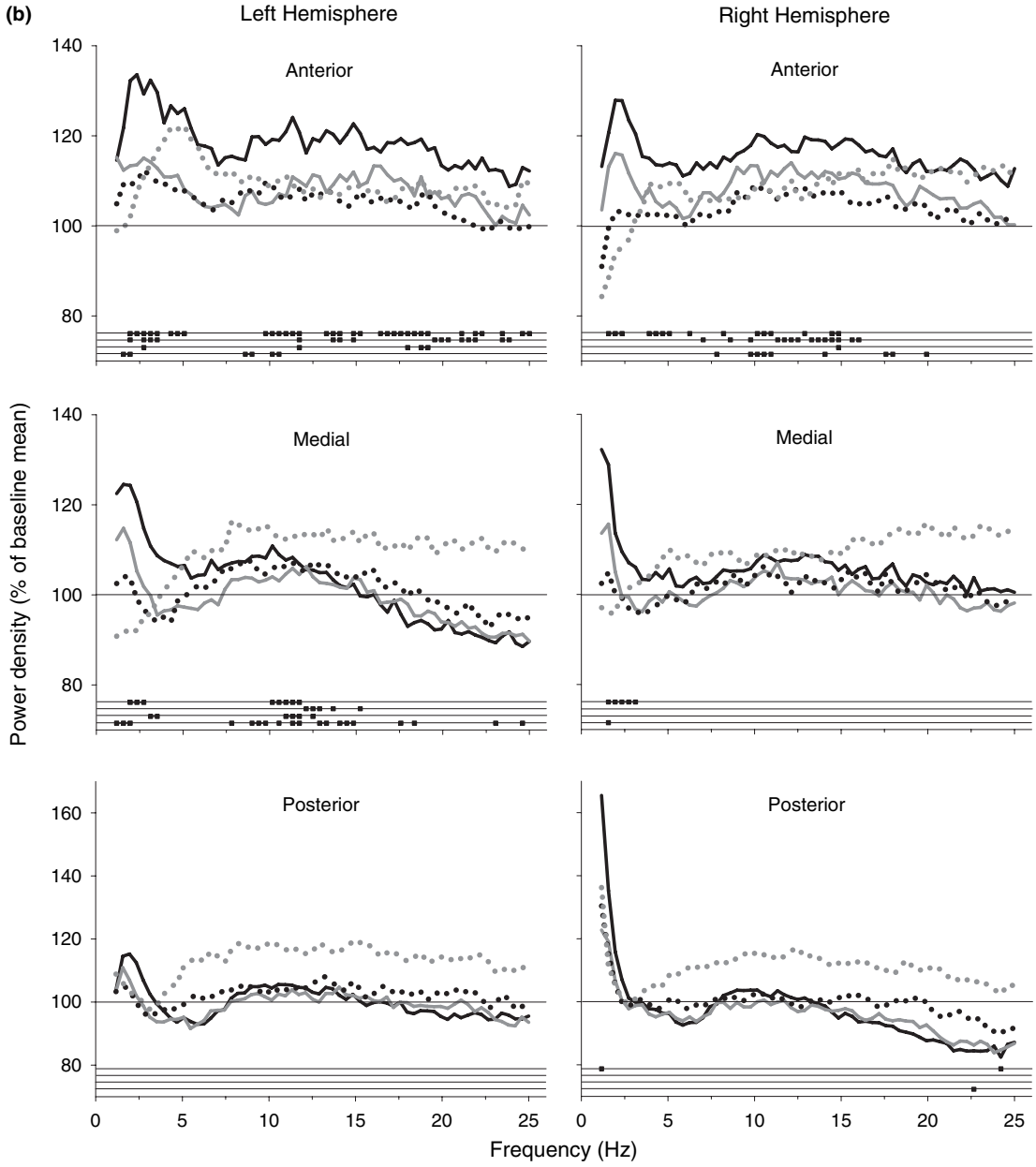

Figure 3. (*Continued*).

the first quarter was significantly greater during recovery when compared with baseline for the left ($t = 4.704$, df = 4, $P = 0.009$) and right ($t = 4.897$, df = 4, $P = 0.008$) anterior pallia, and the right medial pallium ($t = 3.496$, df = 4, $P = 0.025$); the left medial pallium showed a similar trend ($t = 2.475$, df = 4, $P = 0.069$) (Fig. 4). SWA was also greater during the last quarter in the left anterior and medial pallia (anterior, $t = 3.536$, df = 4, $P = 0.024$; medial, $t = 5.916$, df = 4, $P = 0.004$), and the right medial pallium showed a similar trend ($t = 2.693$, df = 4, $P = 0.055$). None of the *post hoc* tests for the other quarters were significant. One-way rmANOVAs showed that SWA decreased in all regions across the recovery night (left anterior, $F = 8.587$, df = 3,12, $P = 0.003$; left medial, $F = 12.348$, df = 3,12, $P < 0.001$; right anterior, $F = 19.032$, df = 3,12, $P < 0.001$; right medial, $F = 5.993$, df = 3,12, $P = 0.010$). One-way rmANOVAs also revealed an effect of time on the baseline night for all regions except the right anterior pallium (left anterior, $F = 11.971$, df = 3,12, $P < 0.001$; left medial, $F = 37.995$, df = 3,12, $P < 0.001$; right anterior, $F = 2.050$, df = 3,12, $P = 0.161$; right medial, $F = 4.882$, df = 3,12, $P = 0.019$). *Post-hoc* t-tests for the baseline night revealed a significant increase in SWA from the baseline night mean during the first quarter for the left anterior ($t = 6.377$, df = 4, $P = 0.004$) and medial ($t = 3.477$, df = 4, $P = 0.026$) pallia, a significant increase during the second quarter in the left medial pallium ($t = 2.870$, df = 4, $P = 0.046$), and a significant decrease during the last quarter in the left anterior ($t = -4.320$, df = 4, $P = 0.013$) and medial

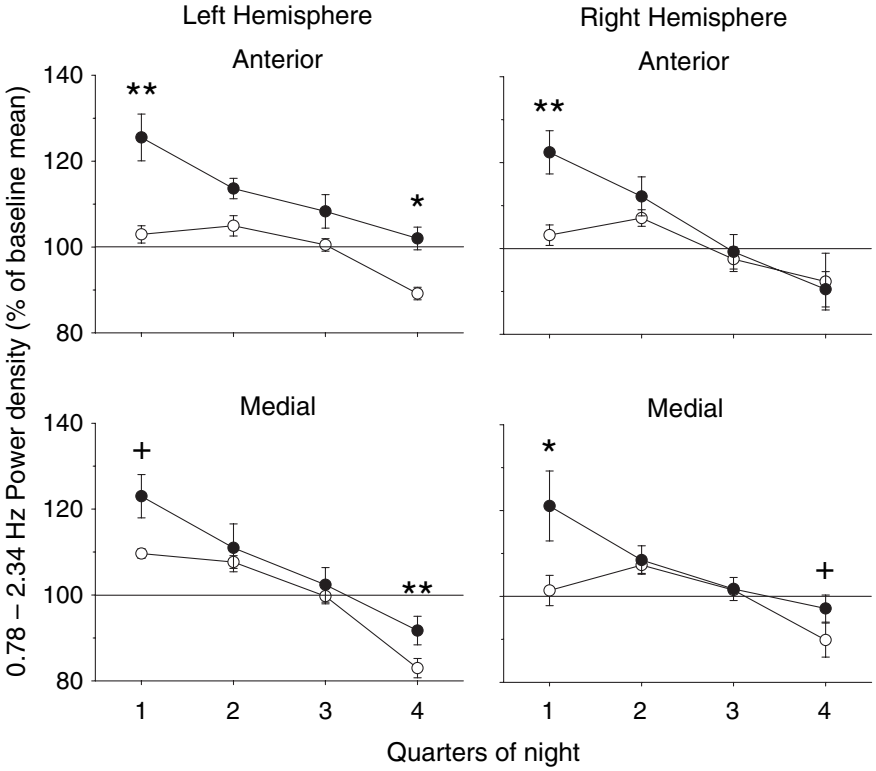

Figure 4. Slow-wave activity (0.78–2.34 Hz power density) during slow-wave sleep on the baseline and recovery nights. Slow-wave activity during each quarter of the baseline (open circles) and recovery (filled circles) nights is expressed as a percent (mean ± SEM) of the entire baseline night average for the left and right, anterior and medial pallia. Statistical differences between the baseline and recovery nights are indicated as follows: $^{**}P < 0.01$; $^{*}P < 0.05$; $^{+}P < 0.07$ (two-tailed, paired *t*-test after significant rmANOVA).

($t = -3.307$, df = 4, $P = 0.030$) pallia. The right medial pallium also showed a trend for higher SWA during the second quarter ($t = 2.693$, df = 4, $P = 0.056$) and lower SWA during the last quarter of the night ($t = -2.677$, df = 4, $P = 0.056$).

The increase in SWA during the first quarter of the recovery night, when the increase in SWA was the greatest, was similar across the left and right, anterior and medial pallia. Specifically, comparisons between the left and right anterior pallia, left and right medial pallia, left anterior and left medial pallia, and right anterior and right medial pallia were all non-significant ($P > 0.27$).

Finally, eye-closure during SWS occurring in the first quarter of the night (when the increase in SWA was the greatest) did not change between the baseline and recovery nights. During SWS, the left eye was closed 77.53 ± 8.77% of the time on the baseline night and 77.37 ± 7.64% of the time on the recovery night ($t = -0.013$, df = 4, $P = 0.990$), and the right eye was closed 86.72 ± 8.50% of the time on the baseline night and 78.85 ± 10.67% on the recovery night ($t = -1.409$, df = 3, $P = 0.254$). The comparison for the right eye was based on only four birds because one bird obscured this eye on both nights.

REM sleep

Figure 5 shows power density (0.78–25 Hz) during REM sleep for each pallial region and quarter of the baseline (a) and recovery (b) nights. For both nights, power density for each frequency bin is expressed as a percent of the entire baseline night average during REM sleep for that specific bin and, thus reflects relative changes in power density during REM sleep. In general, the patterns evident in SWS were also present in REM sleep, particularly in the left anterior and medial pallia. Figure 6 shows the difference in power density during SWS and REM sleep for the first quarter of each night. Although the changes in relative power density during REM sleep and SWS were comparable (Figs 3 and 5), power density, expressed as a percent of average across all frequency bins during SWS occurring during the first quarter of the baseline night, was greater during SWS than REM sleep on both nights for frequencies up to approximately 15–22 Hz.

DISCUSSION

To our knowledge this is the first evidence for an increase in EEG spectral power density following sleep deprivation in birds. As in rodents (rats: Borbély *et al.*, 1984; Syrian hamsters: Tobler and Jaggi, 1987; mice: Huber *et al.*, 2000), short-term (8 h) sleep deprivation caused a significant increase in power density in both low and high frequencies during recovery SWS. Notably, in the left and right, anterior and medial pallia, SWA (i.e., 0.78–2.34 Hz) during SWS increased above baseline levels during the first quarter of the recovery night, and progressively decreased across the night in a manner comparable to that observed in similarly sleep-deprived mammals. In contrast to these results, Tobler and Borbély (1988) did not detect an increase in SWA (0.75–4.5 Hz) following 24 h of sleep deprivation in pigeons. This discrepancy is not attributable to the different frequency bands used

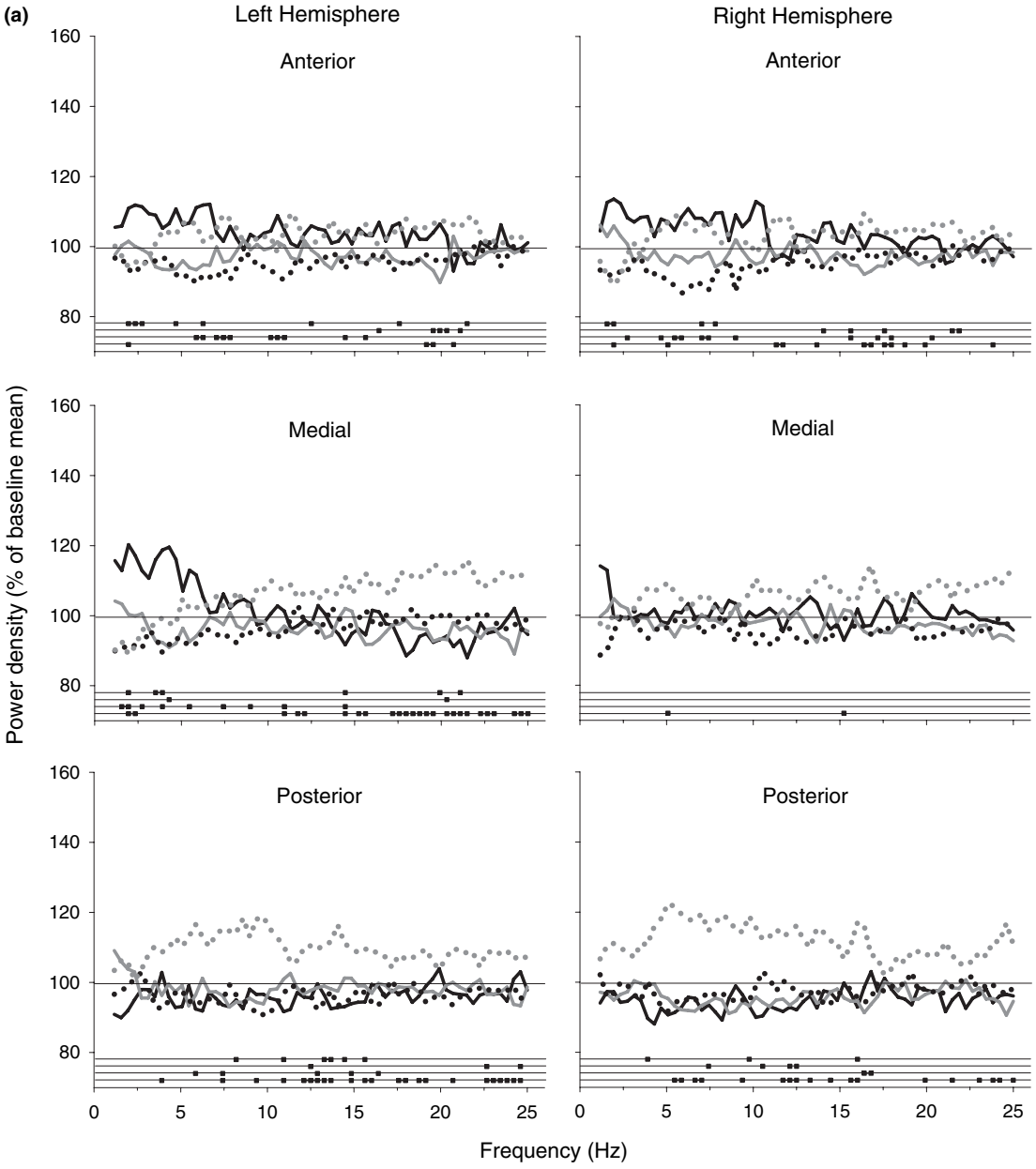

Figure 5. EEG spectral power density (0.78–25 Hz) during REM sleep on the baseline (a) and recovery (b) nights. The power density for each quarter (1st, solid black line; 2nd, solid grey line; 3rd, dotted black line; 4th, dotted grey line) of each night is expressed as a percent of the *entire baseline night* REM sleep mean (i.e., the 100% line) for each frequency bin and brain region (left and right, anterior, medial and posterior pallia) in each pigeon. The mean percent is plotted at the end of each frequency bin. For the baseline night, values for each quarter and frequency bin were compared to the baseline night average. Significant differences ($P < 0.05$, two-tailed paired *t*-test after significant rmANOVA) are indicated by filled squares on the lines at the bottom of each plot; statistical data for the first through fourth quarters are presented on the first (top) through fourth (bottom) lines, respectively. For the recovery night, values for each quarter and frequency bin were compared to the corresponding *quarter of the baseline night*, with significant differences similarly indicated at the bottom of each plot.

to characterize SWA in the respective studies because the increase in low-frequency power density in the left and right anterior pallia extended out to 5.08 Hz, thereby encompassing the 0.75–4.5 Hz band. Instead, sleep deprivation experiments in rodents suggest that this difference might be due to the duration of sleep deprivation used in the earlier study. In Syrian hamsters, SWS-related SWA (0.25–4.0 Hz) increased markedly following 3 h, but not 24 h of sleep deprivation (Tobler and Jaggi, 1987; see also Rechtschaffen *et al*., 1999 for a similar pattern in rats). The similarly divergent responses to short- and long-term sleep deprivation in pigeons and Syrian hamsters suggests that differences in the duration of sleep deprivation may explain why an increase in SWA was observed in our study and not in the earlier study. Nevertheless, additional experiments that directly compare the effects of 8 and 24 h of sleep deprivation are needed to rule out other

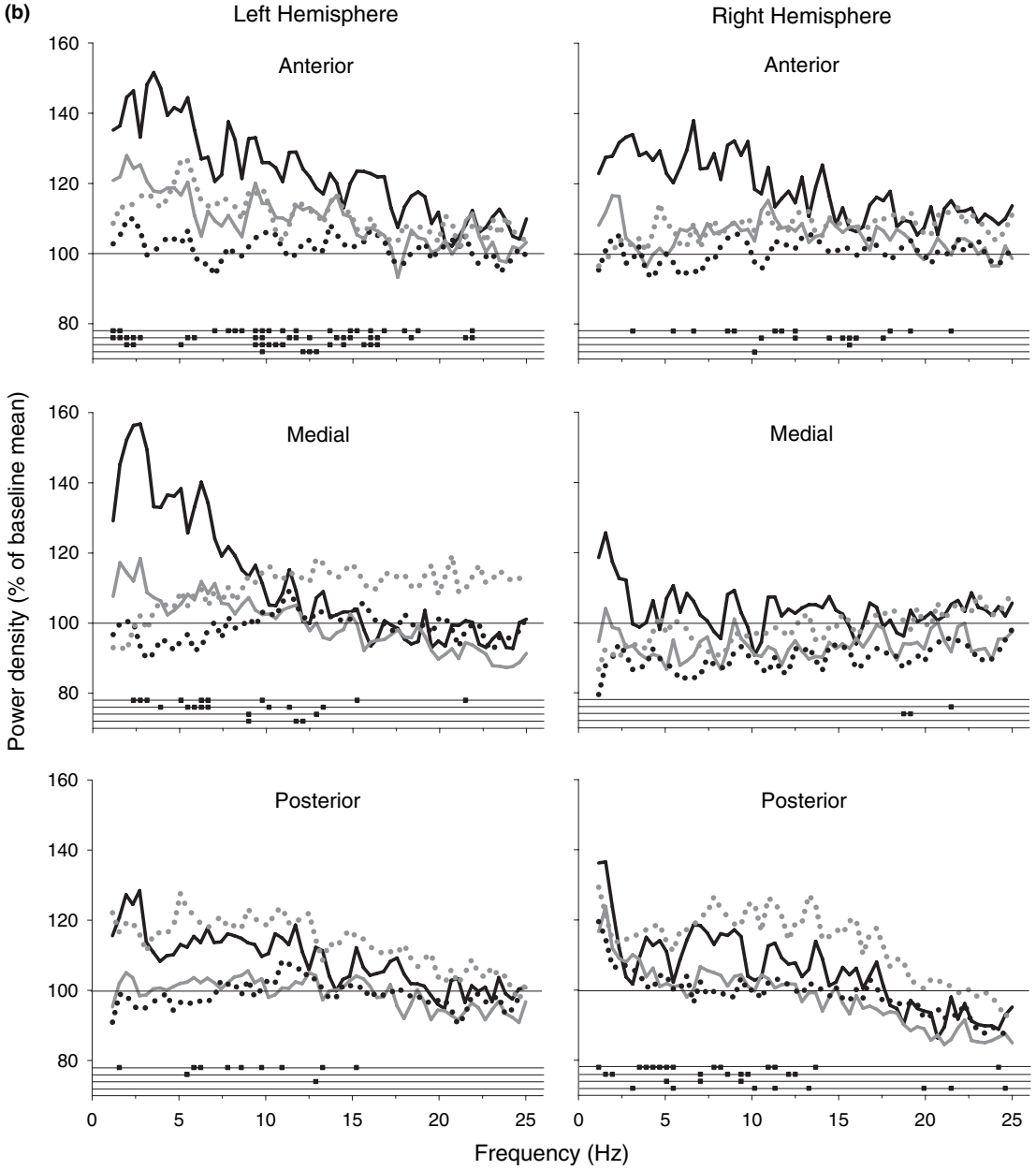

Figure 5. (*Continued*)

factors (e.g., pigeon strain, electrode placement, efficacy of the deprivation procedure) that might have contributed to the different responses to short- and long-term sleep deprivation in pigeons.

Our results have direct bearing on a previous report of sleep suppression in pigeons. Berger and Phillips (1994) reported that constant light (LL) reduced sleep in pigeons to $< 5\%$ of the recording time for periods lasting several weeks without causing signs of elevated sleep pressure during LL, such as increased drowsiness, SWA (0.75–4.0 Hz power density between 50 and 200 μV^2/Hz), or aspects of the 'sleep deprivation syndrome' (i.e., debilitated appearance) observed in rats subjected to long-term sleep deprivation via the disk-over-water method (Rechtschaffen and Bergmann, 2002). Moreover, time spent asleep and SWA did not increase above baseline levels during the first 24 h after the birds ($n = 2$ for the analysis of SWA) were switched from LL to constant darkness (DD; i.e., < 3 lux, red incandescent bulb). Based on these findings, Berger and Phillips (1994) concluded that pigeons do not show a mammalian-like compensatory rebound in SWA following sleep deprivation. In the present study, however, pigeons spent 42.1% of the 12-h light phase in SWS, an amount similar to the 37.7% reported by Tobler and Borbély (1988). Differences in light levels do not seem to explain the contradictory results because the light level used in our study (400–500 lux) was greater, and presumably more alerting, than that used in Berger and Phillips' study (200 lux). Although other factors cannot be ruled out, some of the

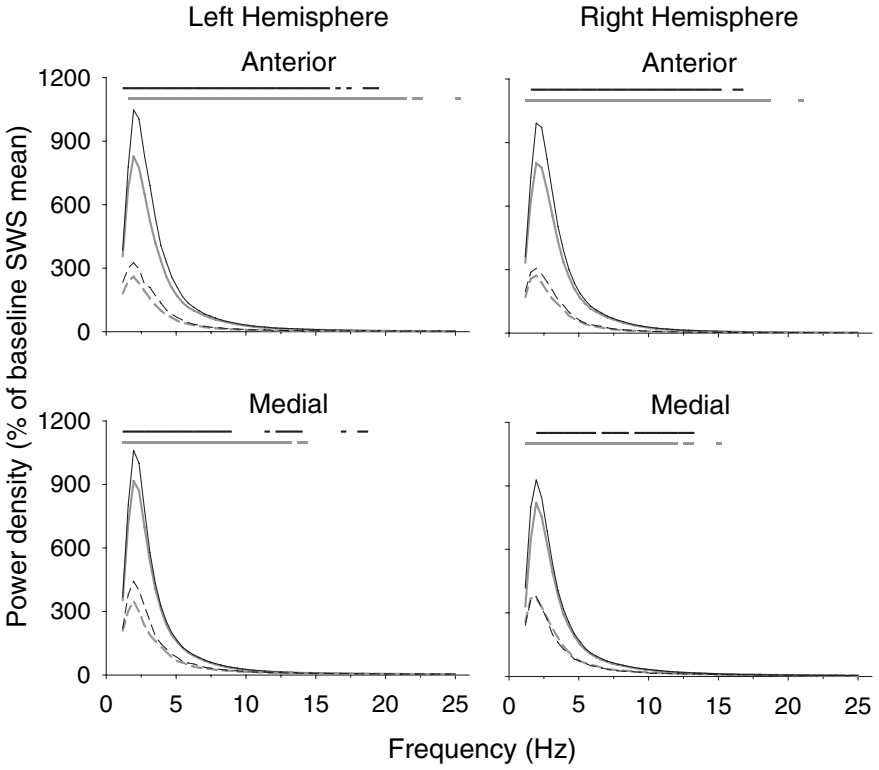

Figure 6. EEG power density (0.78–25 Hz) during SWS (solid lines) and REM sleep (dashed lines) during the first quarter of the baseline (grey lines) and recovery (black lines) nights for the left and right, anterior and medial pallia. To reduce variability between individuals, the power density for each frequency bin and state was calculated as a percent of power density averaged across all frequencies (0.78–25 Hz) for each bird during SWS occurring during the first quarter of the baseline night. The mean percent for all birds is plotted at the end of each frequency bin. Significant differences ($P < 0.05$, two-tailed paired *t*-test after significant rmANOVA) between baseline SWS and baseline REM sleep, and recovery SWS and recovery REM sleep are indicated by the grey and black bars, respectively, at the top of each plot.

differences seem to rest in the definition of sleep used in the respective studies. Whereas Berger and Phillips included a drowsy category, we followed Tobler and Borbély's (1988) approach and included states presumably comparable to Berger and Phillips' drowsiness in the calculation of SWS time. Even with drowsiness included in SWS time, however, their pigeons only engaged in SWS 28.3% of the time during the light phase when housed under a 12 : 12 light–dark (LD) photoperiod, and 26.8% of the time when exposed to LL. As with the scoring of SWS, the remaining difference may reflect a stricter threshold for scoring drowsiness.

The apparent differences in the scoring of sleep in pigeons may be reflected in the analysis of SWA under LL. Although the time spent in drowsiness and SWS during LL was similar to that during the light phase under LD, SWA (averaged across all states and expressed as a percent of the 24-h average under LD) actually increased from 87.2% during the light phase of LD to 94.5% under LL. Consequently, although time spent in drowsiness and SWS did not increase during LL, SWA did increase (albeit non-significantly given the small sample size) when compared to the light phase of LD, presumably during either drowsiness or wakefulness. Furthermore, if the occurrence of SWA reflects homeostatically regulated SWS-related processes, regardless of the state in which it occurs (Borbély *et al.*, 1984; Finelli *et al.*, 2000; Vyazovskiy and Tobler, 2005), then, given the small decrease (5.5%) in SWA during LL when compared with the 24-h LD average, it seems unlikely that LL would cause an increase in SWA following the transition to DD. Consequently, LL may not be an effective means of inducing significant SWA deprivation in pigeons. In contrast, our results demonstrate that pigeons compensate for the loss of SWA and sleep (as defined herein) during the daytime, by increasing SWA during recovery sleep at night, and therefore are consistent with the notion that SWA occurring in the light reflects homeostatically regulated sleep processes.

The increase in SWA following short-term sleep deprivation and the progressive decline across the recovery night in pigeons is consistent with earlier studies describing the time course of SWA during avian sleep. A decline in SWA across the night suggestive of SWS homeostasis has been described in domestic hens (*Gallus domesticus*; number of 2.5–5.0 Hz, high-amplitude waves; van Luijtelaar *et al.*, 1987), European blackbirds (*Turdus merula*; 0.5–4.0 Hz power density; Szymczak *et al.*, 1996), non-migrating white-crowned sparrows (*Zonotrichia leucophrys gambelii*; 1.5–2.5 Hz power density; Rattenborg *et al.*, 2004) and non-migrating Swainson's thrushes (*Catharus ustulatus*; 1.5–4.0 Hz power density; Fuchs, 2006). In contrast, and perhaps surprisingly given the increase in SWA observed following sleep deprivation, our pigeons did not show a pronounced decline in SWA during SWS on the baseline night. Nevertheless, a significant effect of time was present with SWA being highest during the first *or* second quarter of the baseline night and lowest during the last. It remains unclear, however, why SWA was not consistently highest during the first 3 h of the baseline night in this and previous studies of pigeons (Tobler and Borbély, 1988; Berger and Phillips, 1994).

In contrast to the left and right, anterior and medial pallia, where the effect of short-term sleep deprivation on SWA was evident and largely similar, the results for the posterior pallia

were inconclusive. Whereas the left hemisphere did not show a significant increase in any frequency bin during the first quarter of the recovery night, the right posterior pallium showed a large increase in mean SWA. Due to marked variability between birds, however, this increase was only significant for the 0.78–1.17 Hz frequency bin. Consequently, this finding should be interpreted with caution pending replication with a larger number of birds.

As with SWS, when compared to the baseline night, spectral power density increased across a broad range of frequencies during REM sleep on the recovery night, albeit the absolute magnitude of this effect was clearly lower in REM sleep than SWS. Notably, power density increased most markedly below approximately 5 Hz. Although this finding suggests that the increase in SWA during recovery sleep is not specific to SWS, it may also reflect greater 'spill-over' of SWS-related EEG activity into REM sleep on the recovery night resulting from increased SWS pressure. Indeed, early in the baseline night when episodes of REM sleep were shortest (4.5–5.5 s), the EEG often did not achieve the fully activated pattern characteristic of longer episodes of REM sleep occurring later in the night (see Fuchs (2006) for a similar pattern in thrushes). As a result, low-frequency power density during REM sleep was greatest during the first quarter of the baseline night. This pattern was even more pronounced on the recovery night due to (1) the increase in SWA during SWS occurring early in the recovery night, and (2) the shortening of REM sleep episodes during the recovery night. A similar increase in SWA during REM sleep following 24 h of sleep deprivation in humans (Borbély *et al*., 1981), rats (Franken *et al*., 1991), and rabbits (Tobler *et al*., 1990) was also attributed to the spill-over (Tobler *et al*., 1990) of SWS-related SWA into REM sleep resulting from increased SWS pressure. Nevertheless, it should be noted that SWA during REM sleep did not increase in rats (Tobler and Borbély, 1990) or hamsters (Deboer *et al*., 1994) following shorter (i.e., 3–6 h) periods of sleep deprivation more comparable to that employed in our study. This difference might reflect a greater propensity for SWS-related SWA to spill-over into REM sleep in pigeons, due to the short duration of REM sleep episodes, especially on the recovery night. Alternatively, it may reflect a fundamental difference between mammals and pigeons in the way the EEG during REM sleep responds to sleep deprivation.

During the baseline night high-frequency (5–25 Hz) power density during SWS was lowest during the first and highest during the last quarter of the night in the posterior pallium. This pattern was also partially evident in the medial and anterior pallia. A similar increase in high frequencies during SWS occurring towards the end of the night has been observed previously in pigeons (Tobler and Borbély, 1988). Nocturnal Syrian hamsters (Tobler and Jaggi, 1987), Djungarian hamsters (*Phodopus sungorus*: Deboer *et al*., 1994) and rats (Trachsel *et al*., 1988) also show an increase in high-frequency activity toward the end of the main sleep period, although rabbits show the opposite pattern (Tobler *et al*., 1990). As demonstrated in rodents, high-frequency power density in pigeons may be modulated by circadian changes in brain temperature (reviewed in Deboer, 1998).

In addition to SWA, high-frequency (approximately 9–25 Hz) power density during SWS also increased following sleep deprivation in the left anterior pallium. Increases in high-frequency (8–15 Hz) power density were also evident to varying degrees across the night in the right anterior and left medial pallia. The increase in SWA and high-frequency power density were separated by a distinct dip in the magnitude of the increase around 6–8 Hz. Interestingly, sleep-deprived rats (Borbély *et al*., 1984), Syrian hamsters (Tobler and Jaggi, 1987), rabbits (Tobler *et al*., 1990) and mice (Huber *et al*., 2000) show a similar increase in high-frequency EEG activity, including the dip around 6–8 Hz. As suggested for rats and hamsters, the increase in high-frequency EEG activity following sleep deprivation may reflect residual or 'covert' activation stemming from the deprivation procedure occurring concurrently with SWA during recovery SWS (Borbély *et al*., 1984; Tobler and Jaggi, 1987). Although the cause or functional significance of the increase in high-frequency power density following sleep deprivation remains unclear, its presence in pigeons, rodents, and rabbits nonetheless further supports the suggestion that the avian pallium and mammalian neocortex respond similarly to sleep loss.

In addition to EEG spectral power density, sleep architecture was also affected by short-term sleep deprivation. Although time spent in SWS did not change during recovery, REM sleep showed a small, yet significant increase. Tobler and Borbély (1988) also observed an increase in REM sleep following 24 h of sleep deprivation in pigeons (see also Newman *et al*., 2008). Thus, a mammalian-like increase in REM sleep following sleep deprivation is a consistent finding in studies of pigeons.

Although time spent in REM sleep increased during recovery, the duration of REM sleep episodes actually decreased significantly during the recovery night when compared with the baseline night. A similar decrease in the duration of SWS episodes also occurred during recovery. Given that the duration of sleep (SWS and REM sleep combined) episodes was not affected by sleep deprivation, the reduction in the duration of SWS and REM sleep episodes apparently reflects more frequent switching between SWS and REM sleep during recovery. Interestingly, the decrease in the duration of SWS episodes suggests that the increase in SWA in pigeons was not mediated through an increase in SWS continuity, as observed in mammals where increased SWA is usually associated with decreased sleep fragmentation following sleep deprivation (Franken *et al*., 1991; Huber *et al*., 2000; Vyazovskiy *et al*., 2007).

Perspectives

Finally, the increase in SWA observed during sleep in the avian pallium following short-term sleep deprivation suggests that the mammalian neocortex is not necessary for the expression of a compensatory response in EEG activity. Perhaps this is

not surprising given that neurons in the avian pallium exhibit the slow-oscillations (Reiner *et al*., 2001) and connectivity (Rattenborg, 2006) necessary to generate SWA in the first place. The ability to increase SWA in response to sleep deprivation in the avian pallium may involve cytoarchitecture and mechanisms similar to those involved in mammalian SWS homeostasis (Gvilia *et al*., 2000; Obál and Krueger, 2003; Tononi and Cirelli, 2006; Bourgin *et al*., 2007; Huber *et al*., 2007; Yasuda *et al*., 2007). Assuming that this is the case, it remains unclear whether these mechanisms and associated functions evolved through common descent from a stem amniote (the common ancestor to extant reptiles, birds, and mammals) that exhibited a similar precursor state, or independently in the respective ancestors of mammals and birds.

Although the apparent absence of SWA in the three-layered dorsal cortex of sleeping reptiles supports an independent origin for SWA in mammals and birds, some degree of homology may nonetheless exist between reptilian sleep and mammalian SWS. Specifically, sleeping reptiles show hippocampal spikes similar to those observed during SWS in mammals (reviewed in Hartse, 1994; Rattenborg, 2006, 2007). Moreover, as in mammals, hippocampal spiking increases following sleep deprivation in reptiles (reviewed in Hartse, 1994). Thus at least one of the electrophysiological correlates of SWS and its homeostatic regulation are apparently present in reptiles. This suggests that the reptilian dorsal cortex may also exhibit neuronal activity (i.e., slow-oscillations) similar to that which generates SWA in mammals (Steriade, 2006). Even if neurons in the dorsal cortex are shown to exhibit slow-oscillations (intracellular recordings are needed to resolve this issue), this activity may be largely asynchronous between neurons due to low corticocortical connectivity (Rattenborg, 2006). In this regard, the synchronous neuronal activity that gives rise to SWA in mammals and birds may be an emergent property of their large, heavily interconnected pallia (i.e., the avian hyperpallium and the mammalian neocortex). Interestingly, this emergent property may perform emergent functions, not necessarily found in reptiles and other animals. Such functions may support their large, heavily interconnected brains and associated capacity to perform complex cognitive processes (Medina and Reiner, 2000; Emery and Clayton, 2004; Jarvis *et al*., 2005; Rattenborg, 2006). Furthermore, functions associated with this emergent property may complement other sleep-related cellular processes that predate the evolution of mammals and birds (Cirelli *et al*., 2005; Cirelli, 2006). Clearly, additional studies are needed to clarify the evolutionary history of the functions of sleep and the mechanisms underlying its regulation in vertebrates and invertebrates.

ACKNOWLEDGEMENTS

We thank Martina Oltrogge and Hans Hacker for assistance with the pigeons, and Sylvia Kuhn for determining the genetic sex of the pigeons. This research was supported by the Max Planck Society.

REFERENCES

Benington, J. H. Sleep homeostasis and the function of sleep. *Sleep*, 2000, 23: 959–966.

Benington, J. H. and Frank, M. G. Cellular and molecular connections between sleep and synaptic plasticity. *Prog. Neurobiol.*, 2003, 69: 71–101.

Berger, R. J. and Phillips, N. H. Constant light suppresses sleep and circadian rhythms in pigeons without consequent sleep rebound in darkness. *Am. J. Physiol.*, 1994, 267: R945–R952.

Borbély, A. A. and Achermann, P. Sleep homeostasis and models of sleep regulation. In: M. H. Kryger, T. Roth and W. C. Dement (Eds) *Principles and Practice of Sleep Medicine, 4th edn*. Elsevier Saunders, Philadelphia, 2005: 405–417.

Borbély, A. A., Baumann, F., Brandeis, D., Strauch, I. and Lehmann, D. Sleep deprivation: effect on sleep stages and EEG power density in man. *Electroencephalogr. Clin. Neurophysiol.*, 1981, 51: 483–495.

Borbély, A. A., Tobler, I. and Hanagasioglu, M. Effect of sleep deprivation on sleep and EEG power spectra in the rat. *Behav. Brain Res.*, 1984, 14: 171–182.

Bourgin, P., Fabre, V., Huitron-Resendiz, S., Henriksen, S. J., Prospero-Garcia, O., Criado, J. R. and de Lecea, L. Cortistatin promotes and negatively correlates with slow-wave sleep. *Eur. J. Neurosci.*, 2007, 26: 729–738.

Campbell, S. S. and Tobler, I. Animal sleep: a review of sleep duration across phylogeny. *Neurosci. Biobehav. Rev.*, 1984, 8: 269–300.

Cirelli, C. Cellular consequences of sleep deprivation in the brain. *Sleep Med. Rev.*, 2006, 10: 307–321.

Cirelli, C., LaVaute, T. M. and Tononi, G. Sleep and wakefulness modulate gene expression in Drosophila. *J. Neurochem.*, 2005, 94: 1411–1419.

Deboer, T. Brain temperature dependent changes in the electroencephalogram power spectrum of humans and animals. *J. Sleep Res.*, 1998, 7: 254–262.

Deboer, T., Franken, P. and Tobler, I. Sleep and cortical temperature in the Djungarian hamster under baseline conditions and after sleep deprivation. *J. Comp. Physiol. [A]*, 1994, 174: 145–155.

Emery, N. J. and Clayton, N. S. The mentality of crows: convergent evolution of intelligence in corvids and apes. *Science*, 2004, 306: 1903–1907.

Finelli, L. A., Baumann, H., Borbély, A. A. and Achermann, P. Dual electroencephalogram markers of human sleep homeostasis: correlation between theta activity in waking and slow-wave activity in sleep. *Neurosci.*, 2000, 101: 523–529.

Franken, P., Dijk, D. J., Tobler, I. and Borbely, A. A. Sleep deprivation in rats: effects on EEG power spectra, vigilance states, and cortical temperature. *Am. J. Physiol.*, 1991, 261: R198–R208.

Frederickson, C. J. and Rechtschaffen, A. Effects of sleep deprivation on awakening thresholds and sensory evoked potentials in the rat. *Sleep*, 1978, 1: 69–82.

Fuchs, T. Brain-behavior adaptations to sleep loss in the nocturnally migrating Swainson's thrush (*Catharus ustulatus*). PhD dissertation. Bowling Green State University, 2006.

Gvilia, I., Xu, F., McGinty, D. and Szymusiak, R. Homeostatic regulation of sleep: a role for preoptic area neurons. *J. Neurosci.*, 2000, 26: 9426–9433.

Hartse, K. M. Sleep in insects and nonmammalian vertebrates. In: M. H. Kryger, T. Roth and W. C. Dement (Eds) *Principles and Practice of Sleep Medicine, 2nd edn*. W. B. Saunders, Philadelphia, 1994: 95–104.

Huber, R., Deboer, T. and Tobler, I. Effects of sleep deprivation on sleep and sleep EEG in three mouse strains: empirical data and simulations. *Brain Res.*, 2000, 857: 8–19.

Huber, R., Ghilardi, M. F., Massimini, M. and Tononi, G. Local sleep and learning. *Nature*, 2004, 430: 78–81.

Huber, R., Tononi, G. and Cirelli, C. Exploratory behavior, cortical BDNF expression, and sleep homeostasis. *Sleep*, 2007, 30: 129–139.

Jarvis, E. D., Gunturkun, O., Bruce, L., Csillag, A., Karten, H., Kuenzel, W., Medina, L., Paxinos, G., Perkel, D. J., Shimizu, T., Striedter, G., Wild, J. M., Ball, G. F., Dugas-Ford, J., Durand, S. E., Hough, G. E., Husband, S., Kubikova, L., Lee, D. W., Mello, C. V., Powers, A., Siang, C., Smulders, T. V., Wada, K., White, S. A., Yamamoto, K., Yu, J., Reiner, A. and Butler, A. B. Avian Brain Nomenclature Consortium. Avian brains and a new understanding of vertebrate brain evolution. *Nat. Rev. Neurosci.*, 2005, 6: 151–159.

Karten, H. J. and Hodos, W. *A Stereotaxic Atlas of the Brain of the Pigeon, (Columba livia)*. The Johns Hopkins Press, Balimore, 1967.

Krueger, J. M. and Obál, F., Jr. A neuronal group theory of sleep function. *J. Sleep Res.*, 1993, 2: 63–69.

Krueger, J. M. and Obál, F., Jr. Sleep function. *Front. Biosci.*, 2003, 8: d511–d519.

Lesku, J. A., Roth, T. C., 2nd, Amlaner, C. J. and Lima, S. L. A phylogenetic analysis of sleep architecture in mammals: the integration of anatomy, physiology, and ecology. *Am. Nat.*, 2006, 168: 441–453.

Lesku, J. A., Roth, T. C., Rattenborg, N. C., Amlaner, C. J. and Lima, S. L. Phylogenetics and the correlates of mammalian sleep: a reappraisal. *Sleep Med. Rev.*, 2008, 12, in press. doi:10.1016/j.smrv.2007.10.003.

Lima, S. L. and Rattenborg, N. C. A behavioural shutdown can make sleeping safer: a strategic perspective on the function of sleep. *Anim. Behav.*, 2007, 74: 189–197.

van Luijtelaar, E. L., van der Grinten, C. P., Blokhuis, H. J. and Coenen, A. M. Sleep in the domestic hen (*Gallus domesticus*). *Physiol. Behav.*, 1987, 41: 409–414.

Medina, L. and Reiner, A. Do birds possess homologues of mammalian primary visual, somatosensory and motor cortices? *Trends Neurosci.*, 2000, 23: 1–12.

Neckelmann, D. and Ursin, R. Sleep stages and EEG power spectrum in relation to acoustical stimulus arousal threshold in the rat. *Sleep*, 1993, 16: 467–477.

Newman, S. M., Paletz, E. M., Rattenborg, N. C., Obermeyer, W. H. and Benca, R. M. Sleep deprivation in the pigeon (*Columba livia*) using the disk-over-water method. *Physiol. Behav.*, 2008, 93: 50–58.

Obál, F., Jr and Krueger, J. M. Biochemical regulation of non-rapid-eye-movement sleep. *Front. Biosci.*, 2003, 8: d520–d550.

Rattenborg, N. C. Evolution of slow-wave sleep and palliopallial connectivity in mammals and birds: a hypothesis. *Brain Res. Bull.*, 2006, 69: 20–29.

Rattenborg, N. C. Response to commentary on evolution of slow-wave sleep and palliopallial connectivity in mammals and birds: a hypothesis. *Brain Res. Bull.*, 2007, 72: 187–193.

Rattenborg, N. C. and Amlaner, C. J. Phylogeny of sleep. In: T. Lee-Chiong, M. Sateia and M. Carskadon (Eds) *Sleep Medicine*. Hanley and Belfus, Philadelphia, 2002: 7–22.

Rattenborg, N. and Martinez-Gonzalez, D. Avian slow-wave sleep homeostasis. *Sleep*, 2007, 30: A127.

Rattenborg, N. C., Lesku, J. A., Martinez-Gonzalez, D. and Lima, S. L. The non-trivial functions of sleep. *Sleep Med. Rev.*, 2007, 11: 405–409.

Rattenborg, N. C., Lima, S. L. and Amlaner, C. J. Unilateral eye closure and interhemispheric EEG asymmetry during sleep in the pigeon (*Columba livia*). *Brain Behav. Evol.*, 2001, 58: 323–332.

Rattenborg, N. C., Mandt, B. H., Obermeyer, W. H., Winsauer, P. J., Huber, R., Wikelski, M. and Benca, R. M. Migratory sleeplessness in the white-crowned sparrow *(Zonotrichia leucophrys gambelii)*. *PLoS Biol.*, 2004, 2: 924–936.

Rechtschaffen, A. Current perspectives on the function of sleep. *Perspect. Biol. Med.*, 1998, 41: 359–390.

Rechtschaffen, A. and Bergmann, B. M. Sleep deprivation in the rat: an update of the 1989 paper. *Sleep*, 2002, 25: 18–24.

Rechtschaffen, A., Bergmann, B. M., Gilliland, M. A. and Bauer, K. Effects of method, duration, and sleep stage on rebounds from sleep deprivation in the rat. *Sleep*, 1999, 22: 11–31.

Reiner, A. A new avian brain nomenclature: why, how and what. *Brain Res. Bull.*, 2005, 66: 317–331.

Reiner, A., Stern, E. A. and Wilson, C. J. Physiology and morphology of intratelencephalically projecting corticostriatal-type neurons in pigeons as revealed by intracellular recording and cell filling. *Brain Behav. Evol.*, 2001, 58: 101–114.

Rial, R. V., Nicolau, M. C., Gamundi, A., Akaarir, M., Aparicio, S., Garau, C., Tejada, S., Roca, C., Gene, L., Moranta, D. and Esteban, S. Sleep and wakefulness, trivial and non-trivial: which is which? *Sleep Med. Rev.*, 2007, 11: 411–417.

Roth, T. C., II, Lesku, J. A., Amlaner, C. J. and Lima, S. L. A phylogenetic analysis of the correlates of sleep in birds. *J. Sleep Res.*, 2006, 15: 395–402.

Siegel, J. M. Clues to the functions of mammalian sleep. *Nature*, 2005, 437: 1264–1271.

Steriade, M. Grouping of brain rhythms in corticothalamic systems. *Neurosci.*, 2006, 137: 1087–1106.

Stickgold, R. and Walker, M. P. Sleep-dependent memory consolidation and reconsolidation. *Sleep Med.*, 2007, 8: 331–343.

Szymczak, J. T., Kaiser, W., Helb, H. W. and Beszczynska, B. A study of sleep in the European blackbird. *Physiol. Behav.*, 1996, 60: 1115–1120.

Tobler, I. Phylogeny of sleep regulation. In: M. H. Kryger, T. Roth and W. C. Dement (Eds) *Principles and Practice of Sleep Medicine, 4th edn.* Elsevier Saunders, Philadelphia, 2005: 77–90.

Tobler, I. and Borbély, A. A. Sleep and EEG spectra in the pigeon (*Columba livia*) under baseline conditions and after sleep-deprivation. *J. Comp. Physiol. A-Sens. Neur. Behav. Physiol.*, 1988, 163: 729–738.

Tobler, I. and Borbély, A. A. The effect of 3-h and 6-h sleep deprivation on sleep and EEG spectra of the rat. *Behav. Brain Res.*, 1990, 36: 73–78.

Tobler, I. and 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: 449–459.

Tobler, I., Franken, P. and Scherschlicht, R. Sleep and EEG spectra in the rabbit under baseline conditions and following sleep deprivation. *Physiol. Behav.*, 1990, 48: 121–129.

Tononi, G. and Cirelli, C. Sleep and synaptic homeostasis: a hypothesis. *Brain Res. Bull.*, 2003, 62: 143–150.

Tononi, G. and Cirelli, C. Sleep function and synaptic homeostasis. *Sleep Med. Rev.*, 2006, 10: 49–62.

Trachsel, L., Tobler, I. and Borbély, A. A. Electroencephalogram analysis of non-rapid eye movement sleep in rats. *Am. J. Physiol.*, 1988, 255: R27–R37.

Vyazovskiy, V. V. and Tobler, I. Theta activity in the waking EEG is a marker of sleep propensity in the rat. *Brain Res.*, 2005, 1050: 64–71.

Vyazovskiy, V. V., Borbély, A. A. and Tobler, I. Interhemispheric sleep EEG asymmetry in the rat is enhanced by sleep deprivation. *J. Neurophysiol.*, 2002, 88: 2280–2286.

Vyazovskiy, V. V., Achermann, P. and Tobler, I. Sleep homeostasis in the rat in the light and dark period. *Brain Res. Bull.*, 2007, 74: 37–44.

Werth, E., Dijk, D. J., Achermann, P. and Borbély, A. A. Dynamics of the sleep EEG after an early evening nap: experimental data and simulations. *Am. J. Physiol.*, 1996, 271: R501–R510.

Yasuda, K., Churchill, L., Yasuda, T., Blindheim, K., Falter, M. and Krueger, J. M. Unilateral cortical application of interleukin-1beta (IL1beta) induces asymmetry in fos, IL1beta and nerve growth factor immunoreactivity: implications for sleep regulation. *Brain Res.*, 2007, 1131: 44–59.

Zepelin, H., Siegel, J. M. and Tobler, I. Mammalian sleep. In: M. H. Kryger, T. Roth and W. C. Dement (Eds) *Principles and Practice of Sleep Medicine, 4th edn.* Elsevier Saunders, Philadelphia, 2005: 91–100.