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Review

Avian sleep homeostasis: Convergent evolution of complex brains, cognition and sleep functions in mammals and birds

Niels C. Rattenborg *, Dolores Martinez-Gonzalez, John A. Lesku

Max Planck Institute for Ornithology, Seewiesen, Germany

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ABSTRACT

Birds are the only taxonomic group other than mammals that exhibit high-amplitude slow-waves in the electroencephalogram (EEG) during sleep. This defining feature of slow-wave sleep (SWS) apparently evolved independently in mammals and birds, as reptiles do not exhibit similar EEG activity during sleep. In mammals, the level of slow-wave activity (SWA) (low-frequency spectral power density) during SWS increases and decreases as a function of prior time spent awake and asleep, respectively, and therefore reflects homeostatically regulated sleep processes potentially tied to the function of SWS. Although birds also exhibit SWS, previous sleep deprivation studies in birds did not detect a compensatory increase in SWS-related SWA during recovery, as observed in similarly sleep-deprived mammals. This suggested that, unlike mammalian SWS, avian SWS is not homeostatically regulated, and therefore might serve a different function. However, we recently demonstrated that SWA during SWS increases in pigeons following short-term sleep deprivation. Herein we summarize research on avian sleep homeostasis, and cast our evidence for this phenomenon within the context of theories for the function of SWS in mammals. We propose that the convergent evolution of homeostatically regulated SWS in mammals and birds was directly linked to the convergent evolution of large, heavily interconnected brains capable of performing complex cognitive processes in each group. Specifically, as has been proposed for mammals, the interconnectivity that forms the basis of complex cognition in birds may also instantiate slow, synchronous network oscillations during SWS that in turn maintain interconnectivity and cognition at an optimal level.

Contents

* Corresponding author at: Max Planck Institute for Ornithology, Sleep & Flight Group, Eberhard-Gwinner-Strasse, D-82319 Seewiesen, Germany. Tel.: +49 8157 932 279; fax: +49 8157 932 344.
E-mail address: rattenborg@orn.mpg.de (N.C. Rattenborg).

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1. Introduction

The function(s) of sleep remain an unresolved question in neuroscience (Rechtschaffen, 1998; Siegel, 2005; Stickgold, 2005; Tononi and Cirelli, 2006; Lima and Rattenborg, 2007; Rattenborg et al., 2007; Lesku et al., 2008a). The nature of the changes in brain activity that distinguish sleep from wakefulness might provide clues to the purpose for sleep. In mammals and birds, sleep behavior is associated with two distinct brain states, slow-wave sleep (SWS) and rapid eye movement (REM) sleep (Campbell and Tobler, 1984; Rattenborg and Amlaner, 2002; Lesku et al., in press). Whereas the electroencephalogram (EEG) during REM sleep resembles the low-amplitude, high-frequency pattern characteristic of wakefulness, the EEG during SWS shows high-amplitude, slow-waves (0.5–4.5 Hz). In mammals, the amount of slow-wave activity (SWA) (0.5–4.5 Hz power density) during SWS increases and decreases, as a function of prior time spent awake and asleep, respectively, and therefore appears to be homeostatically regulated with the intensity of SWS reflected in the level of SWA (Borbély and Achermann, 2005; Tobler, 2005). The dependence of SWA on prior wakefulness and sleep has been modelled as a homeostatically regulated process (Process S) reflecting sleep need that accumulates as a saturating exponential function during wakefulness and declines exponentially during sleep (Fig. 1; Borbély and Achermann, 2005).

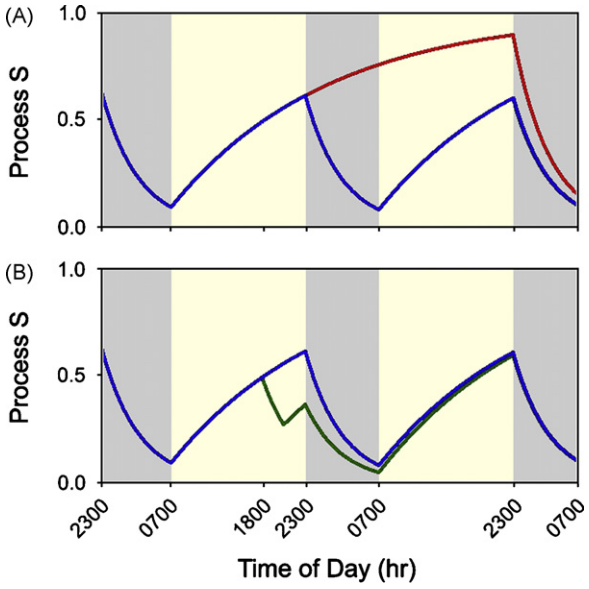


Fig. 1. Model of homeostatically regulated Process S derived from the time course of electroencephalogram slow-wave activity (SWA) in a diurnal mammal. Across a normal 24-h period (blue lines in (A) and (B)). Process S is thought to accumulate as a saturating exponential function of time spent awake during the day (yellow shading), and decline as an exponential function of time spent in SWS at night (gray shading). Staying awake for 40 h (A, red line) causes Process S to accumulate more than normal, as reflected by an increase in SWA during recovery sleep. Conversely, taking a 2-h nap starting at 1800 (B, green line) reduces Process S, as reflected by lower SWA at the start of the subsequent major sleep period. This model of sleep homeostasis has been validated in several species of mammals, and forms the basis for current theories for the function of SWS (modified from Tobler and Achermann, 2007).

The function of SWS is likely to be closely tied to the homeostatic regulation of SWS (Benington, 2000). Accordingly, this phenomenon forms the basis for several theories for the function of mammalian SWS (e.g., Krueger and Obál, 1993, 2003; Benington and Frank, 2003; Tononi and Cirelli, 2003, 2006). Until recently, however, it was unclear whether such theories were applicable to birds. Although birds are the only taxonomic group other than mammals to show unequivocal SWS, previous studies in pigeons (*Columba livia*) did not detect an increase in SWA following sleep deprivation (Tobler and Borbély, 1988), and thereby suggested that birds might lack the neuroanatomy and mechanisms involved in mammalian SWS homeostasis (Zepelin et al., 2005). This view was perhaps reinforced by the belief that the avian telencephalon lacks a cortical structure comparable to the laminar neocortex (Medina and Reiner, 2000), a possible requirement for SWS homeostasis. Indeed, historically the nuclear arrangement of the avian telencephalon contributed to the belief that it was primarily composed of a hypertrophied striatum (Fig. 2A; Jarvis et al., 2005). However, evidence accumulating over the last four decades (Karten, 1969) and culminating with recent developmental gene expression studies (Smith-Fernandez et al., 1998; Puelles et al., 2000) indicate that most of the avian telencephalon is derived from the same pallial embryonic neural tissue that gives rise to the mammalian neocortex, lateral cortex, hippocampus, claustrum and the lateral parts of the amygdala (Fig. 2B; Jarvis et al., 2005), a conclusion consistent with the finding that the avian pallium is capable of orchestrating complex cognitive processes, previously thought to require a neocortex (*reviewed in* Emery and Clayton, 2004; Butler and Hodos, 2005; Butler, 2008; Kirsch et al., 2008). Given this revised view on the developmental and functional homology between the mammalian neocortex and avian pallium, we revisited the possibility of avian sleep homeostasis and found that despite lacking a laminar neocortex, SWS is nonetheless homeostatically regulated in pigeons in a manner similar to that observed in mammals (Martinez-Gonzalez et al., 2008). Consequently, avian SWS may serve a function similar to that in mammals.

In this review, we first summarize previous behavioral and electrophysiological research on avian sleep homeostasis. Although our focus is on SWS homeostasis, we also review the evidence for avian REM sleep homeostasis. We also discuss the implication of sleep homeostasis for birds that remain continuously active for days, weeks, or longer (Rattenborg, 2006a). We then cast our recent evidence for avian SWS homeostasis within the context of current mammalian-based theories for the function of SWS that hinge on SWS homeostasis. We also elaborate upon our recent proposal that the independent evolution of SWS in mammals and birds is directly linked to the independent evolution of large (relative to body mass; Jerison, 2001), heavily interconnected brains (Rattenborg, 2006b, 2007). Notably, given our recent evidence for avian SWS homeostasis, we suggest that the interconnectivity that forms the basis of complex cognition in birds may also instantiate slow, synchronous network oscillations that in turn maintain interconnectivity and cognition at an optimal level, as proposed for mammals (Tononi and Cirelli, 2003, 2006). Throughout we intend to underscore the notion that the independent evolution of homeostatically regulated SWS (and REM sleep) in birds provides a largely untapped opportunity to

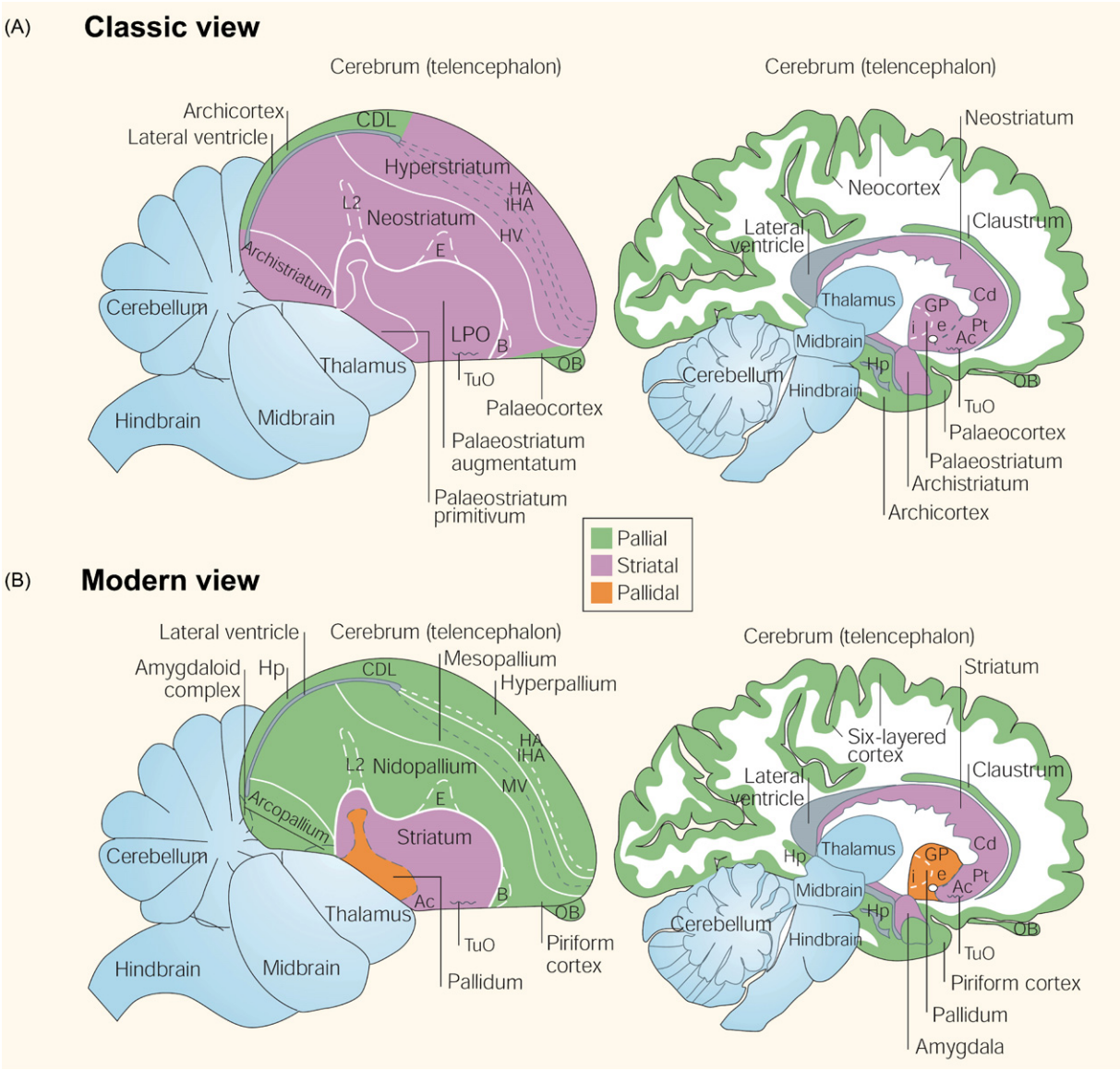


Fig. 2. Avian and mammalian brain relationships. (A) Classic view of avian and mammalian brain relationships (left, songbird; right, human). Although past authors had different opinions about which brain regions are pallium vs. subpallium, we have colored individual brain regions according to the meaning of the names given to those brain regions. Ac, accumbens; B, nucleus basalis; Cd, caudate nucleus; CDL, dorsal lateral corticoid area; E, ectostriatum; GP, globus pallidus (i, internal segment; e, external segment); HA, hyperstriatum accessorium; HV, hyperstriatum ventrale; IHA, interstitial hyperstriatum accessorium; L2, field L2; LPO, lobus parolfactorius; OB, olfactory bulb; Pt, putamen; TuO, olfactory tubercle. (B) Modern consensus view of avian and mammalian brain relationships according to the conclusions of the Avian Brain Nomenclature Forum. Solid white lines are lamina (cell-sparse zones separating brain subdivisions). Large white areas in the human cerebrum are axon pathways called white matter. Dashed gray lines divide regions that differ by cell density or cell size; dashed white lines separate primary sensory neuron populations from adjacent regions. Abbreviations where different from (A): E, entopallium; B, basorostralis; HA, hyperpallium apicale; Hp, hippocampus; IHA, interstitial hyperpallium apicale; MV, mesopallium ventrale. Adapted by permission from Macmillan Publishers Ltd: Nature Reviews Neuroscience, Jarvis et al. (2005).

determine the functions of the electrophysiological correlates of sleep in mammals through revealing overriding principles common to both lineages.

2. Avian sleep homeostasis

2.1. Behavioral evidence

The first evidence for sleep homeostasis in birds came from a study of sleep behavior in barbary doves (*Streptopelia risoria*). Lendrem (1983) measured eye-closure (a correlate of sleep) in groups of doves following 6–36 h of exposure to a ferret, a potential predator. After removing the predator, the doves spent more time with their eyes closed. Moreover, the degree to which eye closure increased was correlated with the duration of prior exposure to the predator. This suggested that the doves compensated for a reduction in eye closure during exposure to the predator by increasing eye closure once the predator was removed. Although this dose-dependent relationship is consistent with sleep homeostasis in general, given the absence of electrophysiological recordings, it was not possible to determine whether the increase in eye closure reflected increased time in SWS or REM sleep, nor whether SWS intensity (as measured by SWA) increased following sleep deprivation. This is also a limitation of subsequent behavioral studies of sleep homeostasis.

Boerema et al. (2003) examined the effects of sleep deprivation on the proportion of behaviorally defined sleep spent with one or both eyes closed in chickens (*Gallus domesticus*). Unlike most

mammals, birds can keep one eye open during SWS (Rattenborg et al., 2000). Unilateral eye closure is associated with an interhemispheric asymmetry in EEG SWA, with the hemisphere contralateral to the open eye showing lower SWA than the hemisphere contralateral to the closed eye. Sleep with both eyes closed is associated with either SWS with high levels of SWA in both hemispheres or REM sleep. Mallard ducks (*Anas platyrhynchos*) are able to increase the proportion of SWS spent with one eye open versus with both eyes closed in response to a perceived increase in the risk of predation (Rattenborg et al., 1999). The preference for engaging in SWS with both eyes closed under safe conditions suggests that SWS with one eye open is less efficient than SWS with both eyes closed, presumably due to the lower level of SWA in the hemisphere contralateral to the open eye. Given these findings, Boerema et al. (2003) predicted that following sleep deprivation, chickens would decrease the time spent with one eye closed and increase the time spent with both eyes closed. The investigators prevented chickens from taking their normal daytime naps during the entire 14-h light phase by walking past or tapping the cage whenever the birds showed signs of sleep behavior. As expected, time spent with one eye closed decreased and time spent with both eyes closed increased during the night following daytime sleep deprivation. These results are consistent with the idea that chickens compensate for daytime sleep loss by allocating more time to sleeping with both eyes closed. However, because REM sleep also typically occurs with both eyes closed, it is unclear whether the increase in bilateral eye closure following sleep deprivation reflects an increase in time spent in REM sleep or SWS with high levels of SWA in both hemispheres.

Recently, Bobbo et al. (2008) examined the effect of sleep deprivation on time spent with one or both eyes closed in 11-day-old female chicken chicks. The chicks were kept awake for 8 h by forcing them to walk continuously on a treadmill. During recovery, the time spent with both eyes closed increased by over 100%. However, unlike the reduction in time spent with one eye closed observed in older chickens following sleep deprivation (Boerema et al., 2003), the time spent with one eye closed, which only encompassed 1% of the behaviorally defined sleep time, did not change in chicks following sleep deprivation. The large increase in sleep with both eyes closed is consistent with the notion that sleep is homeostatically regulated in young chickens; however, given that the birds were forced to walk continuously, exercise may have contributed to the increase in sleep. Moreover, as with the aforementioned behavioral study, it is not possible to determine the extent to which the increase in bilateral eye closure reflected an increase in REM sleep or SWS with high levels of SWA in both hemispheres.

2.2. Electrophysiological evidence

2.2.1. Slow-wave sleep homeostasis during spontaneous sleep

In mammals that engage in extended periods of wakefulness on a daily basis, SWA during SWS is greatest at the start of a major sleep period and progressively declines thereafter (Tobler, 2005). Only a few studies have looked for a similar pattern in birds. In a study of chickens, van Luijtelaar et al. (1987) provided the first description of a change in low-frequency EEG activity across the night in a bird. Rather than using fast Fourier transforms (FFTs) to quantify changes in low-frequency EEG activity, as done in later studies, van Luijtelaar et al. used an automated method to count the number of slow-waves (2.5–5.0 Hz) that exceeded an arbitrary voltage. The number of slow-waves per each 2-h period of the 10-h night declined significantly across the night. Although this decline was suggestive of SWS homeostasis, it might have reflected a decline in the occurrence of SWS across the night, rather than a decline in the amount of high-voltage, slow-waves during SWS.

Subsequently, Tobler and Borbély (1988) used FFTs to examine the time course of SWA in pigeons. Although the pigeons slept more at night, a clear decline in SWA (0.75–4.5 Hz power density) across the night was not evident during sleep. Similarly, Berger and Phillips (1994) also did not detect a decline in SWA (0.75–4.0 Hz power density between 50 and 200 μV^2/Hz) at night in pigeons. In contrast to these studies, however, we recently found a significant effect of time of night on the level of SWA (0.78–2.34 Hz power density) during SWS in pigeons; mean SWA was highest during the first *or* second quarter of the night (depending of the location of the EEG electrodes) and lowest during the last (Martinez-Gonzalez et al., 2008). Although the overall trend was for SWA to decline across the night, it remains unclear why SWA was not consistently highest during the first quarter, as expected if SWS is homeostatically regulated in pigeons.

Although the evidence for SWS homeostasis in pigeons based solely on the spontaneous time course of nocturnal SWA is equivocal, SWA clearly declines across the night in several species of songbirds. In a study of the European blackbird (*Turdus merula*), Szymczak et al. (1996) provided the first evidence for a decline across the night in SWS-related SWA (0.5–4.0 Hz power density) (Fig. 3). SWA calculated for successive 3-h periods of the night declined significantly in all five birds studied, a pattern consistent with SWS homeostasis. However, in contrast to mammals where high levels of SWA are associated with high arousal thresholds (Frederickson and Rechtschaffen, 1978; Neckelmann and Ursin, 1993), blackbirds were *more* likely to respond when natural sounds were presented during SWS occurring early in the night than late in the night. This finding seemingly argues against the idea that the level of SWA reflects sleep intensity in blackbirds, as it does in mammals, and therefore may not reflect homeostatically regulated processes. Nonetheless, because seven types of sounds were each presented 5–7 times across the night, it is also possible that the birds habituated to the sounds over time, and therefore were less likely to respond later in the night. An increase in REM sleep later in the night might have also had a carry over effect on response probabilities during SWS, as previous studies have shown that the arousal threshold is highest during REM sleep in various species of birds (Rojas-Ramírez and Tauber, 1970; Van Twyver and Allison, 1972; Walker and Berger, 1972). Clearly, the relationship between

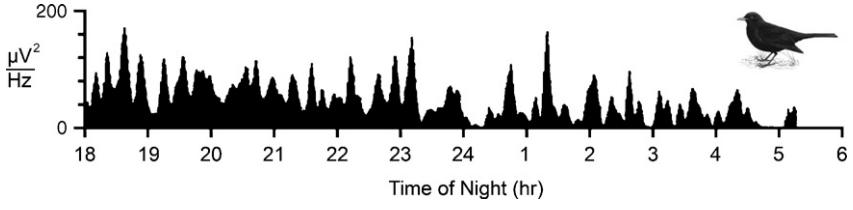


Fig. 3. Decline in slow-wave activity (SWA; 0.5–4.0 Hz power density) during slow-wave sleep occurring across an undisturbed 12-h night in a European blackbird (*Turdus merula*). This study provided the first electrophysiological evidence for the homeostatic regulation of SWA during undisturbed sleep in birds. Reprinted from Szymczak et al. (1996), with permission from Elsevier.

SWS-related SWA and responsiveness to the environment warrants further study in birds.

A decline in low-frequency EEG activity during SWS has been observed recently in two additional species of songbirds. White-crowned sparrows (*Zonotrichia leucophrys gambelii*) in a non-migratory state showed a significant decline across the night in 1.5–2.5 Hz power density during SWS in both cerebral hemispheres (Rattenborg et al., 2004; *see also* Jones et al., 2008a). Swainson's thrushes (*Catharus ustulatus*) in a non-migratory state also showed a decline across the night in 1.5–4.0 Hz power density during SWS (Fuchs, 2006). The progressive decline in SWS-related SWA across the night suggests that these diurnal birds spontaneously accrue a sleep deficit during the day that results in greater SWA early in the night, a pattern consistent with the notion that avian SWS is homeostatically regulated (e.g., Fig. 1). Nonetheless, sleep deprivation experiments are needed to confirm that the decline in SWA occurring across the night reflects a homeostatic process, rather than the influence of a circadian factor on the expression of avian SWA (*see* Section 2.2.2).

The spontaneous sleep patterns of captive white-crowned sparrows were also investigated during the fall migratory season (Rattenborg et al., 2004). During the fall and spring seasons, captive songbirds that would normally be migrating at night in the wild exhibit nocturnal migratory restlessness (*Zugunruhe*) characterized by hopping around the cage and periods of wing whirring (Berthold and Querner, 1988; Berthold et al., 2000). The amount of time spent exhibiting migratory restlessness during each night is correlated with the time that songbirds would fly in the wild, and therefore seems to reflect a natural endogenous drive to migrate (Gwinner, 1986; Berthold and Querner, 1988; Berthold, 1996). As a result of this nocturnal migratory behavior, white-crowned sparrows slept approximately two-thirds less during the fall season when compared to the summer non-migratory season. Perhaps surprisingly, given this marked reduction in nighttime sleep, SWS-related SWA was not consistently elevated during the remaining sleep at night. Although this finding suggested that sleep is not homeostatically regulated in sparrows, at least at this time of year, the birds may have partially compensated for lost SWS by engaging in more daytime drowsiness, a mixed state with characteristics of both wakefulness and SWS (Rattenborg et al., 2004; Jones et al., 2008a; *see also* Fuchs et al., 2006). However, there was no evidence of a compensatory increase in REM sleep. Although REM sleep occurred earlier in the night during the migratory season, the percent of total sleep time spent in REM sleep did not increase as expected if the birds compensated for REM sleep lost during the previous nights of migratory restlessness. Ultimately, sleep deprivation studies are needed to determine whether sleep in songbirds is homeostatically regulated during the migratory seasons.

2.2.2. Slow-wave sleep homeostasis following sleep deprivation

Tobler and Borbély (1988) conducted the first EEG-based avian sleep deprivation study. Pigeons previously housed under a 12:12 light–dark (LD) photoperiod were kept awake for 24 h via gentle handling under constant light. At the end of the sleep deprivation, the birds were returned to the 12:12 LD schedule and allowed to recover starting at lights out. Although the time spent in REM sleep increased significantly, particularly during the first 6 h of recovery, and wakefulness decreased slightly during recovery, the time spent in SWS was unchanged when compared to the baseline night. Importantly, SWA during SWS did not increase above baseline levels during recovery. This negative result tentatively suggested that avian SWS lacks the intensity dimension that characterizes mammalian SWS, and therefore is not homeostatically regulated. However, the authors acknowledged that other durations of sleep deprivation might reveal a compensatory increase in SWS-related SWA in birds (Tobler, 2005). Although evidence for SWS homeostasis was not detected in this study, it nonetheless provided the first evidence for REM sleep homeostasis in a bird (*see also* Section 2.2.3).

A subsequent study used constant light (LL) in an attempt to enforce long-term sleep suppression in pigeons (Berger and Phillips, 1994). Pigeons housed under LL were reported to sleep very little for up to 74 days without exhibiting signs of increased sleep pressure such as increased drowsiness or SWA (0.75–4.0 Hz power density between 50 and 200 μV^2/Hz). Also, the pigeons did not develop signs of the "sleep deprivation syndrome" (e.g., debilitated appearance) exhibited by rats chronically sleep deprived using the disk-over-water method (Rechtschaffen and Bergmann, 2002). Furthermore, when switched to constant darkness (DD) the birds did not show a compensatory increase in time spent in SWS or the amount of SWA during the first 12 h of DD. Although this study seemed to corroborate the previous sleep deprivation study done on pigeons (Tobler and Borbély, 1988), a closer examination of the data suggests that the birds were only minimally deprived of SWA when housed under LL (*see* Martinez-Gonzalez et al., 2008). Although the amount of scored SWS and drowsiness was low and unchanged during LL when compared to the light phase of the previous 12:12 LD schedule, the amount of SWA averaged across all states and expressed as a percent of the 24-h average under the LD photoperiod actually increased from 87.2% during the light phase of LD to 94.5% under LL ($N = 2$ for the spectral analysis). If SWA reflects a homeostatically regulated sleep process, regardless of the state in which it occurs (Borbély et al., 1984; Finelli et al., 2000; Vyazovskiy and Tobler, 2005a), then the increase in SWA during LL above the levels observed during the light phase of the LD photoperiod suggests that the pigeons were compensating for the loss of the SWS that would normally occur during the dark phase of LD. Furthermore, given that this compensatory response under LL limited the overall reduction in SWA to only 5.5% less than that obtained under LD, it is perhaps not surprising that SWA did not increase immediately following the transition to DD. Consequently, if anything, this study provides evidence for, rather than against, the presence of SWS homeostasis in pigeons. Finally, it is worth noting that Jones et al. (2008a) arrived at a similar conclusion regarding this study.

In a follow-up study, Mintz et al. (1998) examined the effect of administering melatonin during the daytime on subsequent SWS-related SWA during the night. Based on their previous finding that a physiological dose of melatonin induces SWS during the daytime in pigeons (Phillips and Berger, 1992), Mintz et al. predicted that if SWS is homeostatically regulated then when compared to a baseline night, SWA during nighttime SWS should be lower following a day with melatonin induced SWS. In contrast to this prediction, however, SWA during SWS at night did not change significantly following daytime melatonin administration. Although this finding suggested that SWS is not homeostatically regulated in pigeons, the authors acknowledged that the daytime dose of melatonin might have persisted into the night with the result that the SWS-inducing effect of melatonin masked the decline in SWA expected if SWA is homeostatically regulated. Measurements of melatonin levels at night are needed to rule out this possibility.

The previous studies on pigeons question the existence of avian SWS homeostasis similar to that found in mammals. We recently re-examined the question of sleep homeostasis in pigeons using a short-term sleep deprivation protocol (Martinez-Gonzalez et al., 2008). Our approach was based on the observation that in some rodents, SWS-related SWA only increases following short periods of sleep deprivation. This pattern is most evident in the Syrian

hamster (*Mesocricetus auratus*) where SWA increased markedly after just 3 h of sleep deprivation and remained elevated through 8 h of recovery, but did not increase during the first 2 h of recovery following 24 h of sleep deprivation (Tobler and Jaggi, 1987; *see also* Rechtschaffen et al., 1999 for a similar pattern in rats, and Section 2.2.3 for a possible mechanism for the divergent responses to short- and long-term sleep deprivation). Consequently, as noted by Tobler (2005), it was conceivable that pigeons would show an increase in SWA during SWS following shorter, presumably more ecologically realistic, periods of sleep deprivation lasting less than 24 h. Pigeons housed under a 12:12 LD photoperiod were kept awake during the last 8 h of the light phase, a time when they often nap (Tobler and Borbély, 1988). The birds were kept awake by gently stimulating them whenever slow-waves appeared in the EEG. At lights out the birds were allowed to sleep undisturbed. When compared to the baseline day, SWS was reduced from 45.2% time to only 8.4% during deprivation, and REM sleep decreased from 6.1% to 0. During recovery, the time spent in SWS did not change significantly relative to baseline, but REM sleep showed a significant increase, particularly later in the night. Importantly, although time spent in SWS did not change, EEG power density during SWS increased across both low and high frequencies (Fig. 4). Notably, SWA (0.78–2.34 Hz power density) during SWS increased significantly during the first quarter of the recovery night and progressively declined thereafter (Fig. 5). This effect was evident in anterior and medial EEG recordings from both cerebral hemispheres. Power density also increased to a lesser extent in higher frequencies (9–25 Hz) following short-term sleep deprivation (Fig. 4), an effect also observed in rabbits (Tobler et al., 1990) and several species of rodent (Borbély et al., 1984; Tobler and Jaggi, 1987; Huber et al., 2000; Lesku et al., 2008b). The presence of an increase in SWA following short-term sleep deprivation suggests that SWS is homeostatically regulated in pigeons. Moreover, as suggested from the study on Syrian hamsters, the difference in the duration of sleep deprivation may explain why SWA increased after 8 h, but not 24 h of sleep deprivation in pigeons, although this needs to be tested directly in the same laboratory using the same methods and pigeon strain.

The increase in SWA following short-term sleep deprivation in pigeons clearly demonstrates that aspects of mammalian sleep regulation are present in birds. Nonetheless, notable differences were also evident between sleep regulation in pigeons and mammals. For instance, although the increase in SWA was most pronounced during SWS, SWA also increased during REM sleep, a response only observed in mammals following longer periods of sleep deprivation (Borbély et al., 1981; Franken et al., 1991; Tobler et al., 1990). This finding appeared to simply reflect the spill-over of SWA into REM sleep resulting from increased SWS pressure and the shortening of REM sleep episodes on the recovery night. Also unlike mammals, the duration of SWS episodes was shorter on the recovery night. In mammals, the increase in SWA is usually associated with an increase in the duration of SWS episodes (Franken et al., 1991; Huber et al., 2000; Vyazovskiy et al., 2007). The shorter duration of SWS and REM sleep episodes on the recovery night reflected more frequent switching between SWS and REM sleep, rather than wakefulness, because the duration of sleep (SWS and REM sleep combined) episodes was not significantly different between the baseline and recovery nights. In this respect, the results from pigeons are interesting because they suggest that SWS-related SWA increased in pigeons even though they switched in and out of SWS more often on the recovery night.

Following our initial description of avian SWS homeostasis in pigeons (Martinez-Gonzalez et al., 2008), Jones et al. (2008a) reported similar results in the white-crowned sparrow. Sparrows in a non-migratory state were deprived of sleep during the first 6 h of the night (10:14 LD photoperiod). Although the time spent in SWS did not change during recovery, as in pigeons, SWS-related SWA (0.5–4.0 Hz power density) increased above baseline levels, and progressively declined thereafter (Fig. 6). Interestingly, sparrows also showed an increase in higher-frequency (10–20 Hz) power density during recovery SWS comparable to that observed in pigeons and some mammals (see above). The importance of this study rests in the fact that it replicates our results from pigeons in a distantly related avian order (Hackett et al., 2008), and thereby suggests that SWS homeostasis may be a general phenomenon in birds, as it is in mammals (Tobler, 2005).

2.2.3. Relationship between slow-wave and rapid eye movement sleep homeostasis

As previously noted, REM sleep increases following both 24 and 8 h of total sleep deprivation in pigeons (Tobler and Borbély, 1988; Martinez-Gonzalez et al., 2008; *see also* Newman et al., 2008). Interestingly, this increase occurred during the first half of the night following 24 h of sleep deprivation, and the last half of the night following 8 h of sleep deprivation. It is tempting to speculate that this difference is related to the presence of an increase in SWA following 8 h, but not 24 h of sleep deprivation. In rats, the increase in SWA following 3–24 h of sleep deprivation (Tobler and Borbély, 1986) is replaced by an immediate and large increase in REM sleep if the deprivation is extended to 96 h or longer (Rechtschaffen et al., 1999). Although the timing of this shift in the type of recovery sleep obviously differs between rats and pigeons, similar mechanisms may nonetheless account for this general pattern. The interpretation of this shift in the type of recovery sleep in rats remains contentious, but may indicate that the restorative function of REM sleep exceeds that of SWS, and therefore REM sleep takes precedence over SWS under these extreme conditions (Rechtschaffen et al., 1999; Rechtschaffen and Bergmann, 1999a,b, 2002). Alternatively, the early REM sleep rebound following long-term sleep deprivation might reflect a homeostatic response to unintended selective REM sleep deprivation resulting from the failure to prevent short bouts of SWS (Benington and Heller, 1999) and the occurrence of SWA in the waking EEG in chronically sleep-deprived animals (Borbély et al., 1984; Tobler et al., 1990; Borbély, 2001). The increase in REM sleep and decrease in SWA during SWS may also be a stress response related to long-term sleep deprivation (Feinberg, 1999; Horne, 2000; Kim et al., 2007). Along these lines, a recent gene expression study in rats showed that in comparison to short-term sleep deprivation (8 h), long-term sleep deprivation (1 week) caused a more pronounced generalized inflammatory and stress response in the brain (Cirelli et al., 2006). Moreover, the expression of plasticity-related genes in the neocortex, such as *BDNF* (brain-derived neurotrophic factor), increased primarily following short-term sleep deprivation (Cirelli et al., 2006). This is interesting because the increase in SWA following short-term sleep deprivation may be mediated by BDNF in rats (Hairston et al., 2004; Huber et al., 2007; Faraguna et al., 2008). Consequently, this difference in gene expression following short- and long-term sleep deprivation may, in part, explain why SWS-related SWA only increases during recovery sleep following short periods of sleep loss in rats (Rechtschaffen et al., 1999). A similar phenomenon may therefore also explain why SWA increased following 8 h (Martinez-Gonzalez et al., 2008), but not 24 h of sleep deprivation in pigeons (Tobler and Borbély, 1988).

2.3. Sleep homeostasis and continuous activity

The presence of SWS homeostasis in pigeons and sparrows, suggest that birds in general may be constrained by the need to

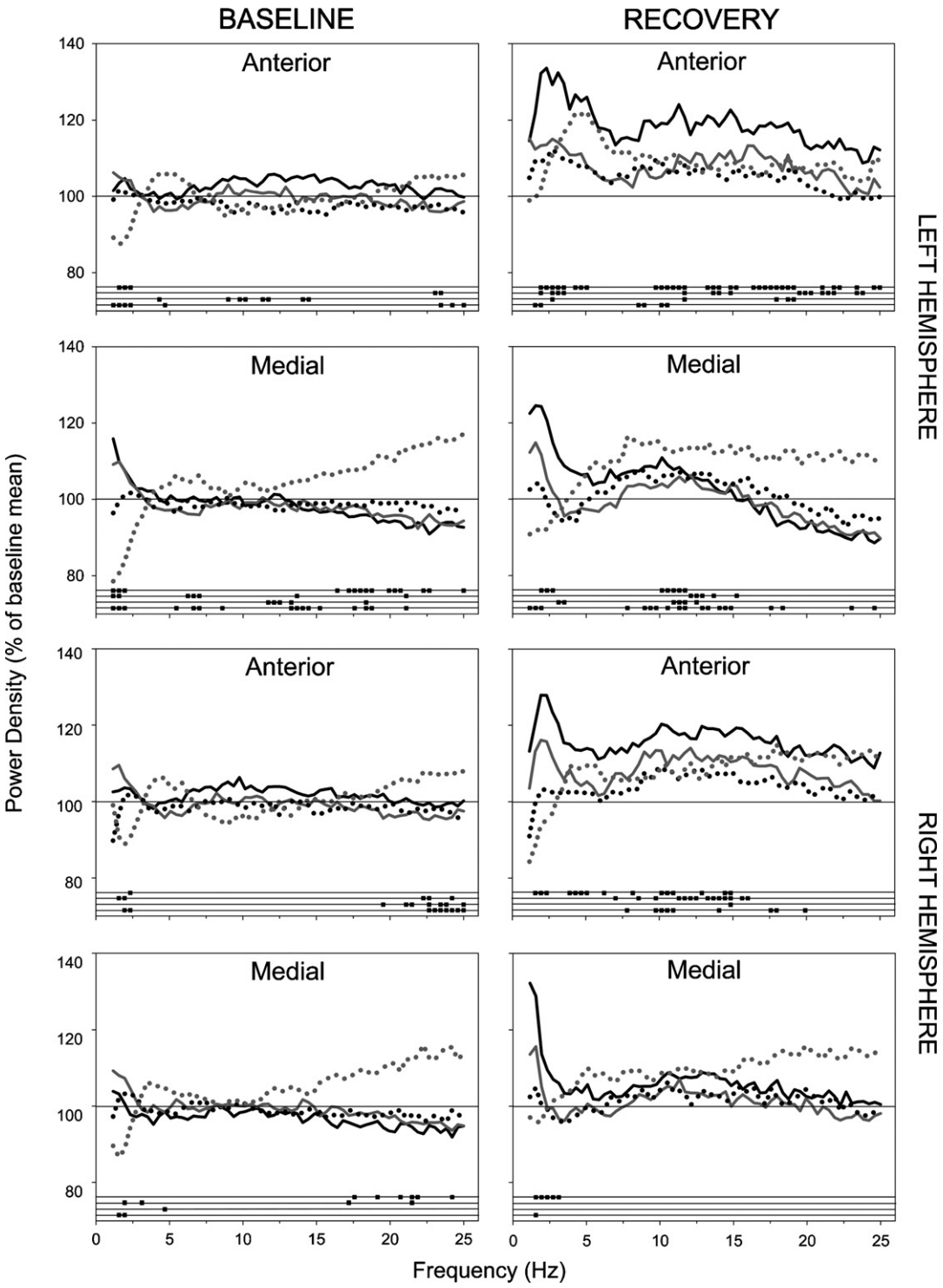


Fig. 4. Electroencephalogram power density (0.78–25 Hz) during slow-wave sleep (SWS) on the baseline (left column) and recovery (right column) nights. The power density for each quarter (first, solid black line; second, solid gray line; third, dotted black line; fourth, dotted gray line) of each night 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 and medial pallia) in each pigeon ($N = 5$). 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 repeated measures ANOVA) are indicated by filled squares on the lines at the bottom of each plot; statistical data for the first through fourth quarters is 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 (modified from Martinez-Gonzalez et al., 2008).

recover lost sleep after periods of wakefulness. However, several species engage in behaviors that are seemingly at odds with the homeostatic regulation of sleep. Notably, some birds engage in non-stop flights lasting several days, weeks, or possibly longer (Rattenborg, 2006a). Moreover, some birds breeding under the continuous light of the artic summer appear to be active throughout much of the 24-h period (Hillman and Young, 1977). Although it is possible that these birds sleep in flight or obtain

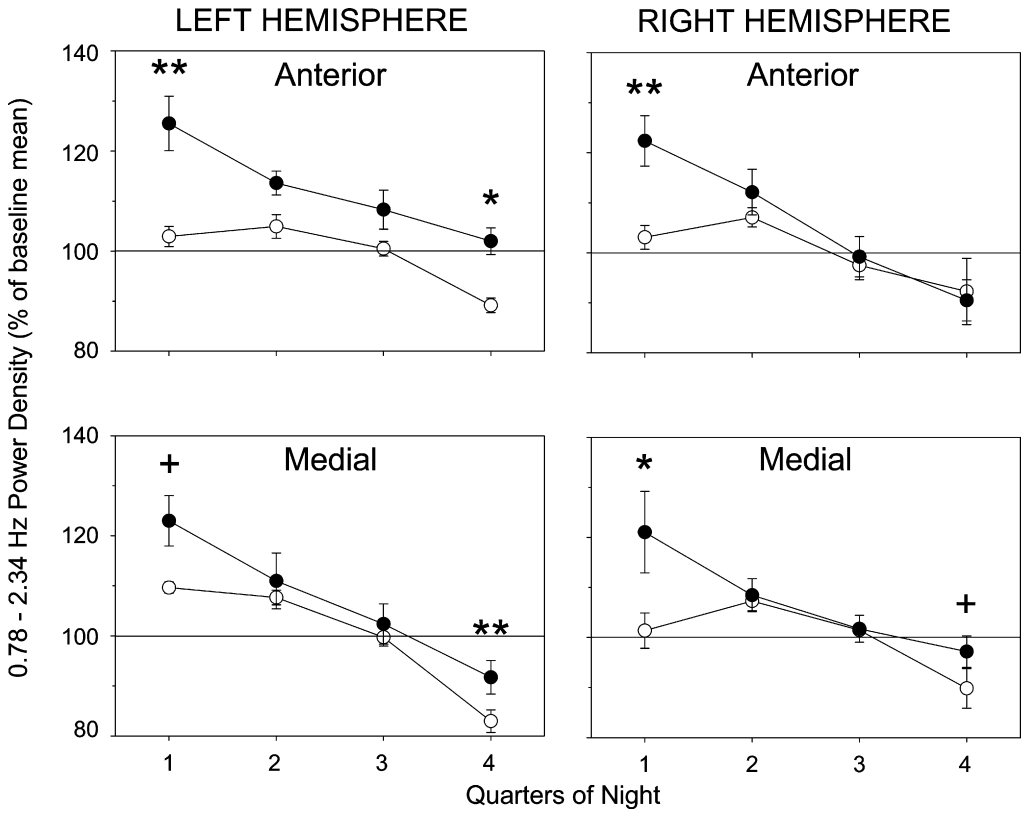


Fig. 5. Slow-wave activity (SWA; 0.78–2.34 Hz power density) during slow-wave sleep for each quarter of the baseline (open circles) and recovery (filled circles) nights. SWA during each quarter of each night is expressed as a percent (mean ± S.E.M.) of the entire baseline night average for the left and 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 repeated measures ANOVA) (modified from Martinez-Gonzalez et al., 2008).

more sleep than suspected during the arctic summer, it is also possible that they have evolved novel neurophysiological mechanisms to temporarily suspend sleep homeostasis at times of the year when the benefits of engaging in other behaviors are greater than the costs of forgoing sleep. If confirmed with electrophysiological recordings, determining how birds achieve such periods of sleeplessness may provide insight into the function of sleep.

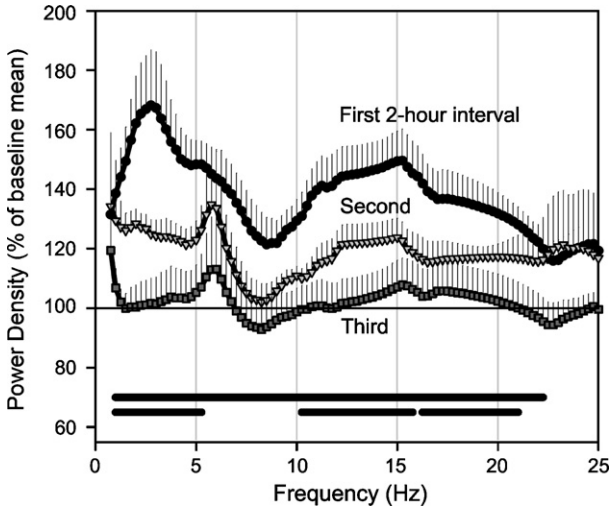


Fig. 6. Time course of slow-wave sleep (SWS) electroencephalogram (EEG) power density during the first three 2-h intervals of recovery following sleep deprivation in white-crowned sparrows. Power for each frequency bin is expressed as a percent (+S.E.M.) of the power in that bin during SWS averaged across the baseline night. The horizontal bars indicate significant differences ($P < 0.05$, paired *t*-test, $N = 6$) between interval 1 (top bar) or interval 2 (bottom bar) and the corresponding hours of the baseline night. The EEG was recorded from the hyperpallium, although the exact derivation (left vs. right, anterior vs. posterior) varied across birds (modified from Jones et al., 2008a).

3. Convergent evolution of heavily interconnected brains, complex cognition and slow-wave sleep in mammals and birds

3.1. Absence of slow-waves in sleeping reptiles and invertebrates

The presence of homeostatically regulated SWA in mammals and birds raises the question whether SWA and associated functions evolved from a similar state in the common ancestor to mammals and birds, or independently in their respective ancestors. Among vertebrates, sleep has been studied most extensively in mammals and birds. This is unfortunate, because studies of poikilothermic vertebrates (reptiles, amphibians, and fish) might provide critical clues to the origin of SWS and REM sleep in mammals and birds. Most studies of sleep in reptiles and amphibians failed to detect high-amplitude EEG slow-waves characteristic of SWS in mammals and birds (*reviewed in* Hartse, 1994). Although some controversy persists (*see* Hartse, 1994; Rial et al., 2007; Rattenborg, 2007), the most consistent EEG correlate of behaviorally defined sleep in reptiles is the appearance of intermittent high-voltage spikes arising from background EEG activity with a voltage similar to or lower than that occurring during quiet wakefulness. In addition to being associated with sleep behavior, including elevated arousal thresholds, reptilian spikes increase following sleep deprivation in chelonian (Flanigan, 1974; Flanigan et al., 1974), iguanid (Flanigan, 1973) and crocodilian reptiles (Flanigan et al., 1973). Early studies suggested that the spikes recorded from the three-layered dorsal cortex of

reptiles were similar to spikes arising from the mammalian hippocampus during SWS (*reviewed in* Hartse, 1994). Notably, both spikes responded similarly to pharmacological manipulations (Hartse and Rechtschaffen, 1982; *reviewed in* Rattenborg, 2007). Indeed, recent studies of turtles *in vitro* (Lorenzo and Velluti, 2004; Lorenzo et al., 1999) and *in vivo* (Gaztelu et al., 1991; Lorenzo et al., 1999) suggest that reptilian sleep-related spikes originate in the medial cortex, the anatomical and functional homologue of the mammalian hippocampus (López et al., 2003), and propagate to the adjacent dorsal cortex. If these spikes reflect the same phenomenon as the spikes recorded during spontaneous sleep, then, at least at the level of the hippocampus, reptilian sleep is similar to mammalian SWS. Despite this similarity, however, the studies on reptiles suggest that sleep-related high-amplitude SWA evolved independently in the respective ancestors of mammals and birds (Rattenborg, 2006b, 2007).

As in reptiles, sleep in invertebrates also seems to lack large-scale, slow, synchronous neuronal activity similar to that occurring during mammalian and avian SWS. Among invertebrates, the electrophysiological correlates of sleep have been examined most extensively in arthropods (van Swinderen, 2007). In a seminal study of sleep in bees (*Apis millifera*), Kaiser and Steiner-Kaiser (1983) provided the first evidence for sleep-related changes in central nervous system activity and responsiveness to sensory stimulation in an invertebrate. At night when bees assumed a characteristic resting posture, optomotor interneurons showed a decrease in spontaneous firing rate and reduced responsiveness to visual stimulation. Importantly, responsiveness to visual stimuli was rapidly restored when the bees were exposed to a stronger stimulus. This study demonstrated that as in vertebrates, sleep in bees is also associated with a change in brain state (i.e., reduced spontaneous firing). Nonetheless, because the pattern of sleep-related neuronal firing or local field potential (LFP) recordings were not reported in this study, it is unclear whether sleep in bees is associated with neuronal or network properties comparable to those occurring during SWS in mammals and birds.

A growing body of evidence indicates that fruit flies (*Drosophila melanogaster*) exhibit a homeostatically regulated sleep state that in many respects is similar to sleep in mammals (Shaw et al., 2000; Hendricks et al., 2000; Huber et al., 2004b). Recently, however, Nitz et al. (2002) recorded LFPs from the brain of awake and sleeping *Drosophila*, but did not find electrophysiological correlates of sleep comparable to those in mammals and birds (*see also* van Swinderen and Andretic, 2003; Hendricks and Sehgal, 2004; Cirelli and Bushey, 2008). Compared to wakefulness, sleep behavior was associated with a decrease in spectral power density across all frequencies in the differential recordings obtained from an electrode in the medial protocerebrum and one in the optic lobe. The higher power during wakefulness was due largely to the occurrence of intermittent, 5–50 ms high-amplitude spikes in the medial protocerebrum arising from low-amplitude background activity. There were no frequency-specific changes during sleep other than the absence of spikes. In this respect, Nitz et al. note that the LFP recordings obtained during sleep resembled those obtained when they experimentally suppressed sodium channel activity. Thus the reduction in LFP power during sleep appears to reflect a general decrease in neuronal activity. In a subsequent study, LFPs recorded between the medial protocerebrum and optic lobe under varying doses of isoflurane gas anesthesia also showed a progressive decline in spectral power density across all frequencies with increasing doses of isoflurane (van Swinderen, 2006). The decrease in power density during sleep and anesthesia in *Drosophila* is opposite to the pattern observed in mammals and birds, where low-frequency power density increases during SWS and light isoflurane anesthesia. Apparently, sleeping *Drosophila* lack large-scale, slow, synchronous neuronal activity (at least in the brain regions examined) comparable to that occurring during mammalian and avian SWS, either because the membrane potentials of individual neurons do not oscillate slowly during sleep, or because the mechanisms required to synchronize slow oscillatory neuronal activity in a manner detectable in LFP recordings are absent.

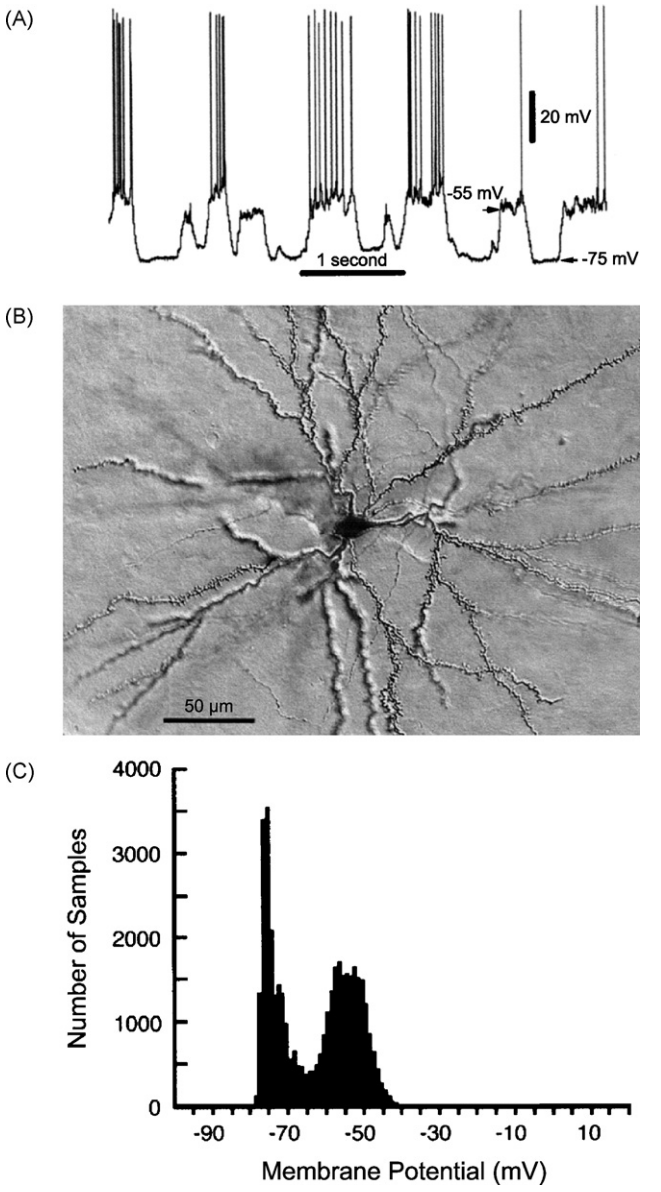
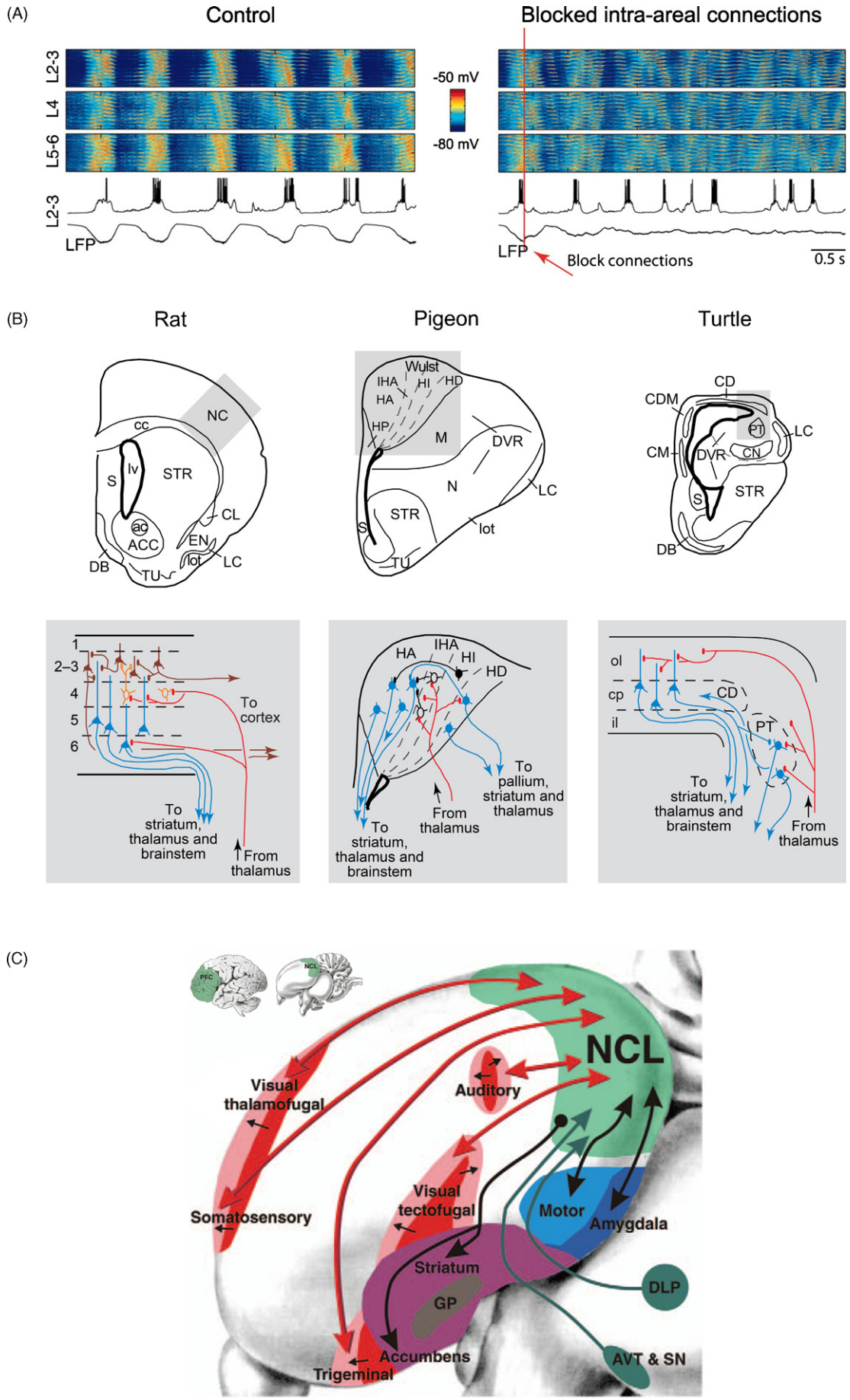


Fig. 7. Spontaneous slow oscillations of the membrane potential in pallial neurons of an anesthetized pigeon. (A) Slow oscillations of the membrane potential between "up-states" (−55 mV) with frequent action potentials and "down-states" (−75 mV) with no action potentials recorded from a neuron (B) in the external pallium. (C) Frequency histogram of membrane potentials showing the tendency of a neuron in the nidopallium caudolateral to be either in the down-state (−75 mV) or up-state (−55 mV) (modified from Reiner et al., 2001, S. Karger AG, Basel).

3.2. Corticocortical connectivity generates slow-waves in mammals

The absence of an increase in SWA during sleep in poikilothermic vertebrates and invertebrates may reflect fundamental differences in the neurophysiology of individual neurons or large-scale network interactions during sleep. Historically, the absence of high-amplitude slow-waves in the dorsal cortex of


(A)
Control
Blocked intra-areal connections
L2-3
L4
L5-6
L2-3
LFP
-50 mV
-80 mV
LFP
Block connections
0.5 s
(B)
Rat
Pigeon
Turtle
NC
cc
lv
STR
S
CL
ac
ACC
EN
lot
LC
DB
TU
Wulst
IHA
HI
HD
HA
HP
M
DVR
N
LC
STR
S
lot
TU
CD
CDM
PT
LC
CM
DVR
CN
STR
S
DB
1
2–3
4
5
6
To cortex
To striatum, thalamus and brainstem
From thalamus
HA
IHA
HI
HD
To pallium, striatum and thalamus
From thalamus
To striatum, thalamus and brainstem
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To striatum, thalamus and brainstem
From thalamus
(C)
PFC
NCL
NCL
Visual thalamofugal
Auditory
Somatosensory
Visual tectofugal
Motor
Amygdala
Striatum
GP
DLP
Accumbens
Trigeminal
AVT & SN

sleeping reptiles was attributed to the absence of a thick, six-layered neocortex responsible for generating EEG slow-waves in mammals (*see* Harste, 1994). Although this hypothesis provided a reasonable explanation at the time, recent advances in our understanding of the genesis of the EEG correlates of SWS in mammals and the evolution of the forebrain in amniotes (i.e., reptiles, birds, and mammals) suggest that cortical thickness *per se* may not explain the absence of slow-waves in reptiles. Instead, a comparatively high degree of interconnectivity in the pallium may be essential for generating the EEG correlates of mammalian and avian SWS (Rattenborg, 2006b, 2007). During anaesthesia and spontaneous SWS, the membrane potentials of mammalian neocortical neurons oscillate slowly (<1 Hz) between a hyperpolarized "down-state" with no action potentials and a depolarised "up-state" with action potentials occurring at a high rate comparable to wakefulness (Steriade, 2006). As shown in Fig. 7, similar slow oscillations have also been observed in pallial neurons of anesthetized pigeons (Reiner et al., 2001). In mammals, corticocortical connections synchronize the slow-oscillation of cortical neurons in a manner that generates high-amplitude slow-waves in the EEG (Amzica and Steriade, 1998; Steriade, 2006). The synchronizing role of corticocortical connections has been shown *in vivo* (Amzica and Steriade, 1995; Timofeev et al., 2000) and *in vitro* (Sanchez-Vives and McCormick, 2000). In humans, slow-oscillations propagate as a travelling wave across the neocortex, presumably via corticocortical connections (Massimini et al., 2004). Furthermore, conditions that increase (e.g., longitudinal Probst bundle formation in mice with congenital callosal dysgenesis; Vyazovskiy and Tobler, 2005b) or decrease (e.g., dark-rearing in cats and mice; Miyamoto et al., 2003) the level of connectivity in specific regions of the neocortex also increase and decrease, respectively, the level of SWA in those regions. Additionally, a recently developed computer model of the thalamocortical network capable of simulating the transition from wakefulness to SWS shows the synchronizing effect that corticocortical connectivity has on SWS-related brain activity (Hill and Tononi, 2005). With the corticocortical connections intact, neurons oscillate in synchrony and therefore generate high-amplitude slow-waves in simulated LFPs (Fig. 8A). In contrast, when the corticocortical connections are removed, the oscillations of individual neurons become less strong and more asynchronous with respect to other neurons, with the result that LFPs show minimal slow-waves. Similarly, reducing synaptic strength in the network (without actually removing connections) also causes the membrane potential oscillations of individual neurons to decrease in amplitude and become more asynchronous with respect to other neurons, resulting in lower amplitude slow-waves in simulated LFPs (Esser et al., 2007). Taken together, the evidence gathered *in vitro*, *in vivo* and *in computo* demonstrates the importance of corticocortical connectivity in generating the large-scale, slow, synchronous oscillations of neuronal membrane potentials that underlie EEG SWA in mammals.

3.3. Limited corticocortical connectivity in the reptilian dorsal cortex

Given the role of corticocortical connections in synchronizing neuronal activity during mammalian SWS, the absence of slow-waves in the three-layered dorsal cortex of sleeping reptiles may be due to the absence of extensive corticocortical connectivity, comparable to that present in the six-layered mammalian neocortex (Rattenborg, 2006b, 2007). Indeed, connectional, neurochemical and developmental evidence indicate that the reptilian dorsal cortex is only homologous to layers I, V, and VI of the mammalian neocortex; reptiles lack layers with extensive corticocortical connectivity equivalent to mammalian supragranular layers II-III (Reiner, 1991, 1993; Butler and Hodos, 2005). As shown in Fig. 8B, projections from the thalamus terminate on the dendritic spines of pyramidal neurons that in turn project primarily to the striatum, thalamus and brainstem (Medina and Reiner, 2000). Interestingly, the mammalian layers with extensive corticocortical connectivity (II–III) originate from a subventricular zone (SVZ) of neuronal proliferation during development (Tarabykin et al., 2001; Noctor et al., 2004; Wu et al., 2005), a neurogenic region not found in turtles (Martínez-Cerdeño et al., 2006; Molnár et al., 2006; Cheung et al., 2007). Although additional studies of other mammalian and reptilian species are needed, this evolutionary innovation may, in part, explain why mammals, but not reptiles, exhibit extensive corticocortical connectivity and SWA during sleep.

3.4. Extensive connectivity in the avian pallium

3.4.1. Hyperpallium

If the degree of corticocortical connectivity explains why SWA does not increase in sleeping reptiles, then birds should also show a comparatively high degree of connectivity in the pallium. Although absolute measures of connectivity are not available, several lines of evidence are consistent with this notion. In pigeons, we found that SWA was greatest during baseline SWS and increased most markedly in response to sleep deprivation in epidural EEG recordings that included the hyperpallium (Martinez-Gonzalez et al., 2008), a primary visual and somatosensory area (Medina and Reiner, 2000; Manger et al., 2002). During development, the hyperpallium originates from the dorsal pallium, the same embryonic pallial tissue that gives rise to the three-layered reptilian dorsal cortex and the portion of the six-layered

Fig. 8. (A) Synchronization of the slow oscillations of simulated neocortical neurons through corticocortical connections. Membrane potential rasters for 100 neurons from layers II–III (L2–3), IV (L4), and V–VI (L5–6). Local field potentials for layers II–III and a representative intracellular trace (V_m) from layers II–III for a duration of 5 s. (Left) The control condition. Note the regular stable oscillation reflected at all levels of the network. (Right) After blockade of horizontal intralaminar connections. Note that the network becomes desynchronized, but individual neurons continue to produce up- and down-states, albeit of shorter duration and reduced amplitude. A similar response occurs when horizontal interlaminar connections are blocked (modified from Hill and Tononi, 2005 and used with permission). (B) Comparison of the dorsal pallium in representative mammalian, avian, and reptilian species. The top row shows transverse cross-sections through the brain. The area shaded in gray is expanded in the bottom row to show network connections. Note the comparatively high degree of palliopallial connectivity in the rat neocortex and the pigeon hyperpallium (or *Wulst*), when compared to the turtle dorsal cortex (see text for details). *Abbreviations*: ac, anterior commisure; ACC, nucleus accumbens; cc, corpus callosum; CD, dorsal cortex; CDM, dorsomedial cortex; CL, claustrum; CM, medial cortex; CN, core nucleus of the DVR; cp, cell plate; DB, diagonal band of Broca; DVR, dorsal ventricular ridge; EN, endopiriform region; HA, hyperpallium apicale; HD, hyperpallium densocellulare; HI, hyperpallium intercalatum; HP, hippocampal complex; IHA, nucleus interstitialis hyperpallii apicalis; il, inner layer; LC, lateral cortex; lot, lateral olfactory tract; lv, lateral ventricle; M, mesopallium; N, nidopallium; NC, neocortex; ol, outer layer; PT, pallial thickening; S, septum; STR; striatum; TU, olfactory tubercle. Reprinted from Medina and Reiner (2000) with permission from Elsevier. (C) Simplified schematic diagram showing some of the connections of the nidopallium caudolateral (NCL). The reciprocal connections with the sensory systems are depicted in red (primary sensory areas) and pink (surrounding secondary and tertiary sensory areas). The primary sensory areas project to secondary and tertiary structures (small black arrows), which then have reciprocal projections to and from the NCL (large red arrows). The visual thalamofugal and tectofugal systems correspond to the geniculocortical and colliculo-pulvino-extrastriate systems of mammals, respectively. The area labelled 'motor' is the arcopallium, which has descending projections to various motor and premotor structures. Thalamic afferents arise from the nucleus dorsolateralis posterior thalami (DLP). Dopaminergic afferents stem from the area ventralis tegmentalis (AVT) and the substantia nigra (SN). *Abbreviations*: GP, globus pallidus. Inset showing a side view of a human (left) and of a pigeon (right) brain with the prefrontal cortex (PFC) and NCL depicted in green. Reprinted from Güntürkün (2005a) with permission from Elsevier.

mammalian neocortex medial to the temporal sulcus (Puelles et al., 2000; Medina and Reiner, 2000). However, unlike the laminar dorsal cortex and neocortex, the hyperpallium is arranged in pseudolayers formed by a series of parallel nuclear structures: the hyperpallium apicale (HA), nucleus interstitialis hyperpallii apicalis (IHA), hyperpallium intercalatum (HI), and hyperpallium densocellulare (HD) (Fig. 8B; Medina and Reiner, 2000). Although the cytoarchitecture of the hyperpallium is unlike the laminar dorsal cortex and neocortex of reptiles and mammals, respectively, the hyperpallium and neocortex are similar from the standpoint of connectivity (Fig. 8B). As with granular layer IV of the neocortex, thalamic projections to the hyperpallium synapse on granular neurons in the nucleus IHA and the lateral portion of the HD (Karten et al., 1973). Along with the HI, the IHA and HD project to HA which in turn sends projections to brainstem, thalamus, striatum, the lateral portion of the nidopallium frontale, and, to a lesser extent, other pallial regions (Shimizu et al., 1995). HI and HD also project to the striatum and pallial area corticoidea dorsolateralis. This comparatively large amount of connectivity in the hyperpallium may be sufficient to explain the presence of SWA in birds.

3.4.2. Dorsal ventricular ridge

Alternatively, SWA may originate from heavily interconnected pallial regions underlying and connected to the hyperpallium. Notably, as shown in Fig. 8B, a pallial region called the dorsal ventricular ridge (DVR) is expanded in birds when compared to reptiles (Butler and Cotterill, 2006; Butler, 2008). The pallial DVR is not readily apparent in mammals and its homology to specific pallial structures in the mammalian brain remains controversial. The DVR may be homologous with the temporal neocortex, or subcortical pallial structures such as the claustrum, endopiriform region, and/or the pallial amygdala (*reviewed in* Reiner et al., 2005; Aboitiz and Montiel, 2007). Regardless of exact homology, however, from the standpoint of interconnectivity and function, the two main regions that form the DVR, the nidopallium and mesopallium, are similar to the neocortex (Tömböl, 1995; Butler, 2008).

The avian nidopallium is a heavily interconnected brain region involved in multimodal sensory integration and learning (Bonke et al., 1979; Wild et al., 1993; Margoliash, 1997; Husband and Shimizu, 1999; Bolhuis and Gahr, 2006). Notably, connectional, neurochemical, electrophysiological, and behavioral evidence from pigeons indicate that the nidopallium caudolateral (NCL) is analogous to the mammalian prefrontal cortex (PFC), even though the NCL and PFC originate from different regions of the embryonic pallium and the NCL lacks lamination (Fig. 8C; Güntürkün, 2005a,b; Kirsch et al., 2008). As with the PFC, the NCL is involved in executive functions, including working memory. Additional complex cognitive abilities comparable to those mediated by the supragranular layers of the mammalian neocortex, and presumably requiring a similar degree of pallial interconnectivity, are also related to the size of the avian nidopallium, in general (Rehkämper and Zilles, 1991; Jarvis et al., 2005; Butler and Cotterill, 2006). For instance, the nidopallium is unusually large in corvids (ravens, crows, and jays), birds with cognitive abilities (e.g., causal reasoning, flexibility, imagination, and prospection) comparable to those exhibited by primates (Emery and Clayton, 2004; Lefebvre et al., 2004). Notably, the manufacture and use of tools (Weir et al., 2002; Hunt and Gray, 2003; Taylor et al., 2007) is correlated with larger relative nidopallium volumes (Lefebvre et al., 2002; *see also* Cnotka et al., 2008). The cognitive abilities of parrots, including rudiments of referential language and abstract categorical reasoning (Pepperberg, 2002), may also be related to the expansion of the nidopallium and mesopallium in this taxon (Iwaniuk and Hurd, 2005). Finally, birds with larger mesopallia are more often observed engaging in innovative feeding behaviors (Timmermans et al., 2000) that may enhance survival in novel environments (Sol et al., 2005a,b).

The independent evolution of complex cognition in birds comparable to that observed in mammals may be linked to the independent evolution of cell types resembling layers II–III in the mammalian neocortex (Karten, 1969, 1991; Butler and Hodos, 2005). Indeed, as with the heavily interconnected supragranular layers (II–III) of the mammalian neocortex, recent studies suggest that chicks (Cheung et al., 2007), but not turtles (Martínez-Cerdeño et al., 2006; Molnár et al., 2006), exhibit a laterally positioned SVZ. Moreover, the SVZ in chicks appears to express similar transcription factors as the mammalian SVZ (Cheung et al., 2007), and therefore may contribute similar types of neurons to the nidopallium and mesopallium. Thus, although birds lack a dorsally positioned SVZ like the one that forms the supragranular layers of the neocortex, they appear to have independently evolved a SVZ in a more lateral position. Given the role of the SVZ in generating corticocortical connectivity in the neocortex, neurons originating in the avian SVZ may serve a similar function. Indeed, when compared to the reptilian DVR, the avian DVR is more elaborate in terms of cytoarchitecture and number of cell groups, differences that may explain the absence of evidence for complex cognition in reptiles and amphibians comparable to that in birds (Butler and Cotterill, 2006).

Given the existing evidence for pallial interconnectivity in the avian DVR and associated cognitive abilities comparable to those mediated by the interconnected supragranular layers of the mammalian neocortex, the DVR is a likely source for SWA in birds. Although EEG SWA has been reported in the avian DVR (Ookawa, 2004), regional differences in the amount of SWA have not been systematically characterized. Moreover, SWS homeostasis has not been measured directly in the DVR. Given that the increase in SWA following sleep deprivation is most pronounced in the heavily interconnected mammalian PFC (Finelli et al., 2001), SWA may also increase most markedly following sleep deprivation in the NCL, the avian analogue of the PFC. Furthermore, slow-waves may originate in the NCL and propagate to other pallial regions, just as they usually originate in the PFC and propagate as a travelling wave to other regions of the neocortex (Massimini et al., 2004).

3.5. Other explanations

Although we have emphasized the role of corticocortical (or palliopallial) connectivity in the genesis of SWS-related SWA in mammals and birds, other differences between the brains of poikilothermic and homeothermic vertebrates may account for the absence of SWA in sleeping reptiles. For instance, although the basic neurochemistry, neuroanatomy, and neurophysiology of the thalamic reticular nucleus (a region involved in the genesis of sleep-related neuronal oscillations in mammals; Steriade, 2006), are similar across mammals, birds, and reptiles (Kenigfest et al., 2005; Llinás and Steriade, 2006), the thalamocorticothalamo circuitry is less elaborate in reptiles (Butler and Cotterill, 2006). Thus, although obvious qualitative differences are not readily apparent at the level of the thalamus, quantitative differences in the thalamocorticothalamo circuitry in conjunction with reduced corticocortical connectivity might account for the absence of SWA in reptiles. Finally, given that glia may amplify the effect of corticocortical connectivity on neuronal synchrony during sleep (Amzica and Steriade, 2000), differences between poikilothermic and homeothermic vertebrates in the type and/or density of glia (Ari and Kálmán, 2008) may also explain why reptiles lack SWS-related SWA.

4. The emergence of slow-wave sleep functions in mammals and birds

Certainly, the absence of SWA in poikilotherms may simply reflect the absence of slow oscillations in the membrane potential of individual neurons during sleep rather than the absence of sufficient connectivity. Intracellular recordings are needed to resolve this issue. If the capacity to oscillate slowly during sleep is absent in poikilotherms then it apparently evolved *de novo* in the respective ancestors of mammals and birds, possibly to perform sleep-dependent functions unique to homeotherms. Alternatively, slow oscillations may occur in poikilotherms, albeit with insufficient connectivity to synchronize this activity. In this case, the large-scale, slow, synchronous network oscillations that characterize mammalian and avian SWS may simply be an epiphenomenon of their heavily interconnected brains without a specific function above and beyond that presumably associated with asynchronous slow oscillations occurring in individual neurons. However, it is also possible that large-scale, slow, synchronous network oscillations are an emergent property that serves an emergent function possibly linked to maintaining adaptive levels of interconnectivity and associated complex cognition in mammals and birds.

Recent theoretical and empirical work suggests that slow-waves are directly involved in the function of SWS (Sejnowski and Destexhe, 2000; Steriade and Timofeev, 2003; Huber et al., 2004a,b; Jha et al., 2005; Marshall et al., 2006). Notably, several theories either implicitly or explicitly link the homeostatic regulation of SWA to sleep-dependent synaptic plasticity (Krueger and Obál, 1993, 2003; Benington, 2000; Benington and Frank, 2003; Tononi and Cirelli, 2003, 2006). However, the precise mechanisms whereby SWA might mediate plasticity remain an area of active research. Although the contribution of other proposed mechanisms cannot be ruled out, the "synaptic homeostasis hypothesis" in particular, is appealing because it not only accounts for the build up of SWA during wakefulness and the decline in SWA during sleep (i.e., Process S), but also provides a specific mechanism whereby the slow network oscillations that define SWS mediate sleep-dependent enhancements in performance (Tononi and Cirelli, 2003, 2006). According to this hypothesis, brain use during wakefulness causes long-term potentiation and a concomitant net increase in the strength and number of synapses, which if left unchecked would saturate, resulting in increased energy demands, decreased space, and an impaired ability to acquire additional information. SWA occurring during sleep is thought to solve this fundamental problem of a plastic nervous system. At sleep onset, levels of SWA are high as a result of synaptic strength accrued during learning while awake. This increase in effective connectivity causes the slow-oscillations of neurons to be more synchronous, and thereby levels of SWA in the EEG to be high. The large-scale, slow oscillations of neuronal networks are then thought to cause long-term depression and synaptic downscaling, a global decrease in synaptic strength. In this manner, slow oscillations reset the overall synaptic strength to an adaptive level that reduces energy expenditure, conserves space, and prepares the brain for a new bout of learning during wakefulness. Moreover, the slow oscillations are thought to increase the signal-to-noise ratio in neuronal networks by eliminating synapses below a minimal threshold. This may explain why performance on certain cognitive tasks increases following sleep (Huber et al., 2004a; Stickgold, 2005). Interestingly, synaptic downscaling is a self-limiting process; as synapses weaken, neurons oscillate less synchronously and consequently induce less downscaling.

Each component of the synaptic homeostasis hypothesis is supported by experimental evidence. Molecular evidence indicates that the level of SWA during sleep is linked to plasticity occurring during prior wakefulness (Cirelli et al., 2004, 2005a,b,c; Cirelli, 2006; Huber et al., 2007; Faraguna et al., 2008; Vyazovskiy et al., 2008). Similarly, electrophysiological evidence suggests that wakefulness is associated with a net increase in neocortical synaptic strength (Vyazovskiy et al., 2008). Moreover, the level of SWA in a neocortical region appears to reflect the level of synaptic strength accumulated in that brain region during prior wakefulness (Huber et al., 2004a; Huber et al., 2006; Huber et al., 2008). The slope of slow-waves, a possible correlate of synaptic strength (Esser et al., 2007), decreases across the night in rats (Vyazovskiy et al., 2008) and humans (Riedner et al., 2007), as expected if a decrease in synaptic strength accounts for the decline in SWA across the night. Genes involved in synaptic depression are preferentially expressed during sleep (Cirelli et al., 2004) and the induction of slow burst firing in neurons in a pattern similar to that occurring during SWS causes long-term depression (Czarnecki et al., 2007). Finally, learning a procedural task causes SWA to increase in the brain region used during training on the task (Huber et al., 2004a). Importantly, the degree to which SWA increases in that region predicts performance enhancements during retesting after sleep, a finding consistent with the notion that slow-waves play an active role in sleep-related learning, possibly through reducing the signal-to-noise ratio in related neural circuits (Hill et al., 2008).

The presence of homeostatically regulated SWA in pigeons (Martinez-Gonzalez et al., 2008) and sparrows (Jones et al., 2008a) raises the possibility that avian SWA is also involved in synaptic homeostasis. Indeed, as in rats (Cirelli et al., 2004; Vyazovskiy et al., 2008), wakefulness and sleep are associated with the expression of genes involved in synaptic potentiation and long-term depression, respectively, in the telencephalon of white-crowned sparrows (Jones et al., 2008b). Moreover, we have recently shown that unilateral visual stimulation during wakefulness, a condition that most likely induces potentiation in the contralateral visual hyperpallium, induces an asymmetric increase in SWA during recovery SWS with SWA being greatest in the visually stimulated hyperpallium (Lesku et al., 2008c). Thus, as in mammals (Huber et al., 2004a; Vyazovskiy and Tobler, 2008), avian SWS homeostasis appears to reflect local brain use (potentiation) during prior wakefulness. Also like mammals, avian sleep may be involved in learning (Dave et al., 1998; Dave and Margoliash, 2000; Derégnaucourt et al., 2005; Margoliash, 2005; Crandall et al., 2007; Hahnloser and Fee, 2007; Jackson et al., 2008), although a direct link between SWA and learning has not been established. Albeit not as extensive as that in mammals, the available evidence is consistent with notion that avian SWS is involved in synaptic homeostasis.

If for the sake of discussion we assume that SWS serves a similar function in mammals and birds, then an obvious question is whether the mechanisms and functions associated with SWA evolved independently in mammals and birds (perhaps in association with the independent evolution of large, heavily interconnected brains cable of performing complex cognitive processes), or were they acquired from a common ancestor with similar sleep. Although future studies may detect slow neuronal activity during sleep in poikilothermic animals, the apparent lack of SWA in sleeping reptiles and *Drosophila* suggests that the network processes that generate large-scale, slow, synchronous activity evolved independently in mammals and birds, and may therefore serve a function unique to homeotherms. By no means does this imply that sleep in homeotherms and poikilotherms is unrelated. Indeed, some sleep mechanisms and functions occur-

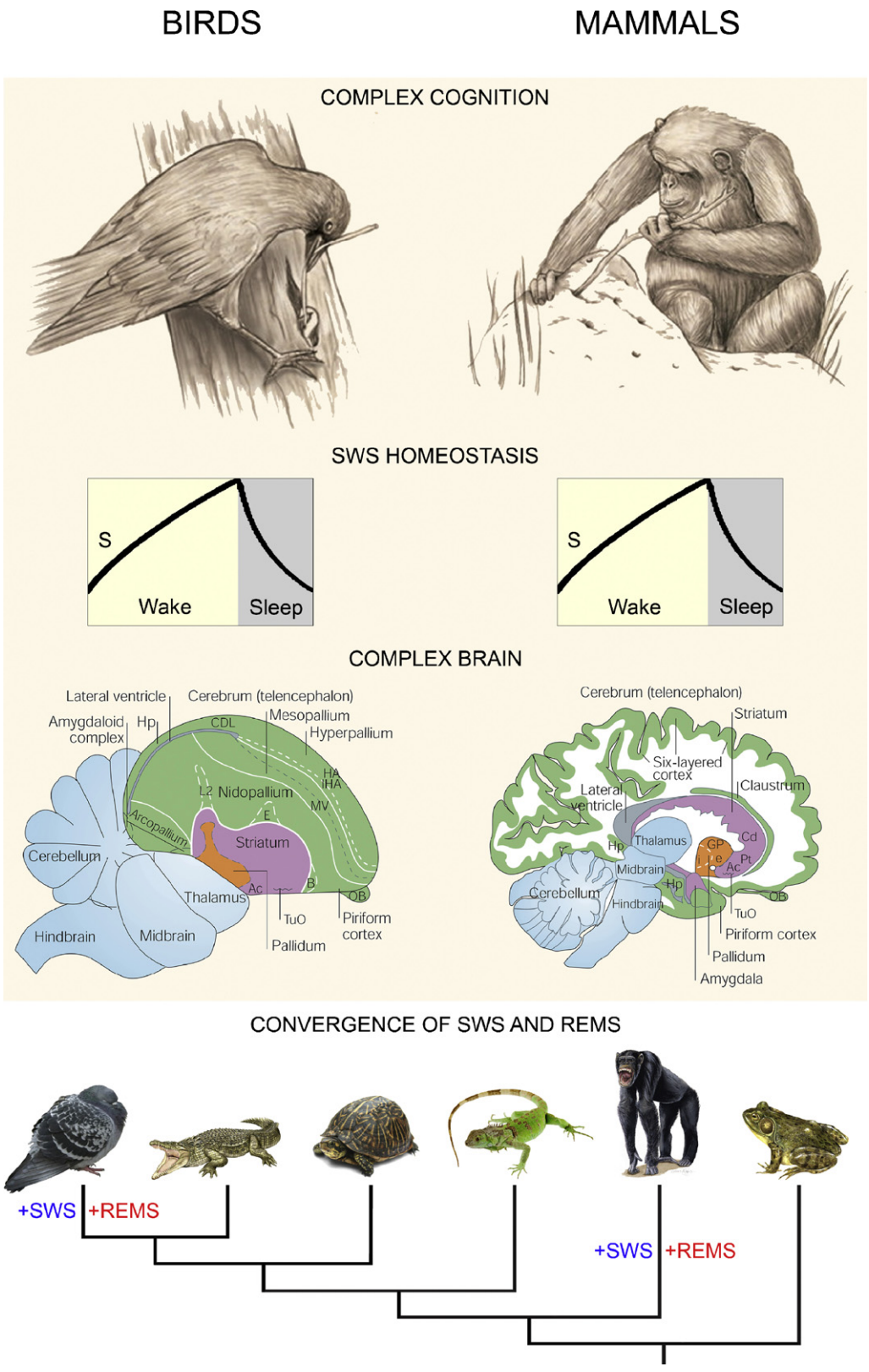

Fig. 9. (Bottom) Convergent evolution of slow-wave sleep (SWS) and rapid eye movement sleep (REMS) in birds and mammals. [Pigeon (*Columba livia*), ©2008, Niels Rattenborg; saltwater crocodile (*Crocodylus porosus*) used with permission courtesy of Encyclopaedia Britannica, Inc., ©2008; Florida box turtle (*Terrapene carolina bauri*), ©2008, Jonathan Zander; green iguana (*Iguana iguana*), C. Martin Harvey, AfriPics.com; chimpanzee (*Pan troglodytes*), ©2008 Jonathan Higgins; bullfrog (*Rana catesbeiana*) used with permission of www.torontozoo.com/adoptapond]. (Shaded panel) The proposed link between the independent evolution of large, heavily interconnected brains, homeostatically regulated SWS, and complex cognition in birds and mammals. Note that the graphs of SWS homeostasis (Process S as in Fig. 1) are simply meant to indicate that evidence exists for SWS homeostasis; the exact kinetics of SWA and Process S have not been determined in birds. The drawings of tool use in a New Caledonian crow (*Corvus moneduloides*) and a chimpanzee by C. Cain are from Emery and Clayton (2004). Reprinted with permission from AAAS. See Fig. 2 caption for brain nomenclature. The brain diagrams were adapted by permission from Macmillan Publishers Ltd: Nature Reviews Neuroscience, Jarvis et al. (2005).

ring at the cellular level may be highly conserved (Mignot, 2008; Allada and Siegel, 2008). For instance, voltage dependent potassium channels appear to play a role in determining sleep duration in *Drosophila* (Cirelli et al., 2005a,b,c; Koh et al., 2008) and mammals (Douglas et al., 2007). Moreover, some of the changes in gene expression occurring between wakefulness and sleep in the brain are similar in *Drosophila* (Cirelli et al., 2005a,b,c; Zimmerman et al., 2006; Naidoo et al., 2007), mammals (Cirelli et al., 2004) and birds (Jones et al., 2008b). As in mammals, and possibly birds, sleep also appears to be involved in plasticity in *Drosophila* (Hendricks et al., 2001; Joiner et al., 2006; Pitman et al., 2006; Ganguly-Fitzgerald et al., 2006; Bushey et al., 2007), even though they apparently lack SWA. Such sleep-dependent plasticity may thus depend on cellular processes that do not require synchronized, slow neuronal activity. A promising area of future research is to determine whether SWA serves a function related to plasticity distinct from, but perhaps complementary to that occurring at the cellular level in animals lacking SWA, or whether it simply reflects the evolution of a new mechanism to achieve the same function in mammals and birds. For instance, although all animals capable of learning seemingly need to perform synaptic downscaling, and may in fact conduct this process during sleep, the demand for downscaling may have been markedly greater in animals with particularly complex brains (Jerison, 2001) and cognition (*reviewed in* Emery and Clayton, 2004; Butler and Hodos, 2005), as found in mammals and birds. As a result, homeotherms may have evolved more efficient mechanisms (i.e., SWA-mediated synaptic depression) than those that may exist in other animals to maintain their heavily interconnected brains and associated cognitive abilities. While this line of reasoning is certainly speculative, it is nonetheless a possibility worth taking into consideration when attempting to integrate research on sleep in poikilotherms and sleep in mammals and birds. Clearly, additional research is needed to determine the extent to which molecular, electrophysiological and functional components of sleep are homologous or analogous in homeotherms and poikilotherms.

5. Conclusions

Until recently, the absence of experimental evidence for SWS homeostasis in birds had been difficult to interpret, especially given the presence of SWA that is otherwise remarkably similar to that in mammals. Indeed, the absence of avian SWS homeostasis posed a significant challenge for functional hypotheses that hinge on SWS homeostasis in mammals, or at least suggested that SWS might perform a different function in birds. However, the recent evidence for SWS homeostasis in birds now indicates that SWS may serve a similar function in mammals and birds (Martinez-Gonzalez et al., 2008). Moreover, given the apparent absence of large-scale, slow network oscillations in the brains of sleeping *Drosophila* and reptiles, this function may have evolved independently in mammals and birds. Specifically, as shown in Fig. 9, we suggest that in conjunction with independently evolving large, heavily interconnected brains capable of performing complex cognitive processes, mammals and birds required a means to maintain this level of connectivity and cognition at an optimal level. Based on evidence primarily from mammals, the function of mammalian and avian SWS may be synaptic downscaling mediated by the slow, synchronous network oscillations that occur during SWS (Tononi and Cirelli, 2003, 2006). Furthermore, we suggest that this function of SWS may have been complimentary to sleep-related cellular processes, including those involved in plasticity, that predate the evolution of mammals and birds. In this respect, synaptic downscaling may not have been the initial function of sleep, but rather a secondary function tailored to the needs of animals with particularly complex brains and cognition. An equally viable alternative, however, is that sleep in both homeotherms and poikilotherms is involved in synaptic downscaling, but birds and mammals required a different, presumably more effective mechanism, to downscale their heavily interconnected brains, than that occurring in other animals. In this scenario, SWA-mediated synaptic downscaling may have been a secondarily evolved mechanism to achieve the same sleep function as present in their predecessors. Distinguishing between these alternatives is a promising avenue for future research into the evolution and functions of sleep.

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References

Aboitiz, F., Montiel, J., 2007. Origin and evolution of the vertebrate telencephalon, with special reference to the mammalian neocortex. Advances in Anatomy Embryology and Cell Biology 194, 1–116.
Allada, R., Siegel, J.M., 2008. Unearthing the phylogenetic roots of sleep. Current Biology 18, R670–R679.
Amzica, F., Steriade, M., 1995. Disconnection of intracortical synaptic linkages disrupts synchronization of a slow oscillation. Journal of Neuroscience 15, 4658–4677.
Amzica, F., Steriade, M., 1998. Electrophysiological correlates of sleep delta waves. Electroencephalography and Clinical Neurophysiolology 107, 69–83.
Amzica, F., Steriade, M., 2000. Neuronal and glial membrane potentials during sleep and paroxysmal oscillations in the neocortex. Journal of Neuroscience 20, 6648–6665.
Ari, C., Kálmán, M., 2008. Evolutionary changes of astroglia in elasmobranchii comparing to amniotes: a study based on three immunohistochemical markers (GFAP, S-100, and glutamine synthetase). Brain, Behavior and Evolution 71, 305–324.
Benington, J.H., 2000. Sleep homeostasis and the function of sleep. Sleep 23, 959–966.
Benington, J.H., Frank, M.G., 2003. Cellular and molecular connections between sleep and synaptic plasticity. Progress in Neurobiology 69, 71–101.
Benington, J.H., Heller, H.C., 1999. Implications of sleep deprivation experiments for our understanding of sleep homeostasis. Sleep 22, 1033–1043.
Berger, R.J., Phillips, N.H., 1994. Constant light suppresses sleep and circadian rhythms in pigeons without consequent sleep rebound in darkness. American Journal of Physiology 267, R945–R952.
Berthold, P., 1996. Control of Bird Migration. Chapman and Hall, London.
Berthold, P., Querner, U., 1988. Was Zugunruhe wirklich ist: Eine quantitative Bestimmung mit Hilfe von Video-Aufnahmen bei Infrarotbeleuchtung. Journal of Ornithology 129, 372–375.
Berthold, P., Fiedler, W., Querner, U., 2000. Migratory restlessness or Zugunruhe in birds: a description based on video recordings under infrared illumination. Journal of Ornithology 141, 285–299.
Bobbo, D., Nelini, C., Mascetti, G.G., 2008. Binocular and monocular/unihemispheric sleep in the domestic chick (*Gallus gallus*) after a moderate sleep deprivation. Experimental Brain Research 185, 421–427.
Boerema, A.S., Riedstra, B., Strijkstra, A.M., 2003. Decrease in monocular sleep after sleep deprivation in the domestic chicken. Behaviour 140, 1415–1420.
Bolhuis, J.J., Gahr, M., 2006. Neural mechanisms of birdsong memory. Nature Reviews Neuroscience 7, 347–357.
Bonke, B.A., Bonke, D., Scheich, H., 1979. Connectivity of the auditory forebrain nuclei in the guinea fowl (*Numida meleagris*). Cell and Tissue Research 200, 101–121.
Butler, A.B., 2008. Evolution of brains, cognition, and consciousness. Brain Research Bulletin 75, 442–449.
Butler, A.B., Cotterill, R.M., 2006. Mammalian and avian neuroanatomy and the question of consciousness in birds. Biological Bulletin 211, 106–127.
Borbély, A.A., 2001. From slow waves to sleep homeostasis: new perspectives. Archives in Italian Biology 139, 53–61.
Borbély, A.A., Achermann, P., 2005. Sleep homeostasis and models of sleep regulation. In: Kryger, M.H., Roth, T., Dement, W.C. (Eds.), Principles and Practice of Sleep Medicine. 4th ed. Elsevier Saunders, Philadelphia, pp. 405–417.
Borbély, A.A., Tobler, I., Hanagasioglu, M., 1984. Effect of sleep deprivation on sleep and EEG power spectra in the rat. Behavioural. Brain Research 14, 171–182.
Borbély, A.A., Baumann, F., Brandeis, D., Strauch, I., Lehmann, D., 1981. Sleep deprivation: effect on sleep stages and EEG power density in man. Electroencephalography and Clinical Neurophysiology 51, 483–495.

Bushey, D., Huber, R., Tononi, G., Cirelli, C., 2007. *Drosophila* hyperkinetic mutants have reduced sleep and impaired memory. Journal of Neuroscience 27, 5384–5393.

Butler, A.B., Hodos, W., 2005. Comparative Vertebrate Neuroanatomy: Evolution and Adaptation, 2nd ed. Wiley-Liss, New York.

Campbell, S.S., Tobler, I., 1984. Animal sleep: a review of sleep duration across phylogeny. Neuroscience and Biobehavioral Reviews 8, 269–300.

Cheung, A.F., Pollen, A.A., Tavare, A., DeProto, J., Molnár, Z., 2007. Comparative aspects of cortical neurogenesis in vertebrates. Journal of Anatomy 211, 164–176.

Cirelli, C., 2006. Cellular consequences of sleep deprivation in the brain. Sleep Medicine Reviews 10, 307–321.

Cirelli, C., Faraguna, U., Tononi, G., 2006. Changes in brain gene expression after long-term sleep deprivation. Journal of Neurochemistry 98, 1632–1645.

Cirelli, C., Gutierrez, C.M., Tononi, G., 2004. Extensive and divergent effects of sleep and wakefulness on brain gene expression. Neuron 41, 35–43.

Cirelli, C., LaVaute, T.M., Tononi, G., 2005a. Sleep and wakefulness modulate gene expression in *Drosophila*. Journal of Neurochemistry 94, 1411–1419.

Cirelli, C., Huber, R., Gopalakrishnan, A., Southard, T.L., Tononi, G., 2005b. Locus ceruleus control of slow-wave homeostasis. Journal of Neuroscience 25, 4503–4511.

Cirelli, C., Bushey, D., Hill, S., Huber, R., Kreber, R., Ganetzky, B., Tononi, G., 2005c. Reduced sleep in *Drosophila* Shaker mutants. Nature 434, 1087–1092.

Cirelli, C., Bushey, D., 2008. Sleep and wakefulness in *Drosophila melanogaster*. Annals of the New York Academy of Sciences 1129, 323–329.

Cnotka, J., Güntürkün, O., Rehkämper, G., Gray, R.D., Hunt, G.R., 2008. Extraordinary large brains in tool-using New Caledonian crows (*Corvus moneduloides*). Neuroscience Letters 433, 241–245.

Crandall, S.R., Adam, M., Kinnischtzke, A.K., Nick, T.A., 2007. HVC neural sleep activity increases with development and parallels nightly changes in song behavior. Journal of Neurophysiology 98, 232–240.

Czarnecki, A., Birtoli, B., Ulrich, D., 2007. Cellular mechanisms of burst firing-mediated long-term depression in rat neocortical pyramidal cells. Journal of Physiology 578, 471–479.

Dave, A.S., Margoliash, D., 2000. Song replay during sleep and computational rules for sensorimotor vocal learning. Science 290, 812–816.

Dave, A.S., Yu, A.C., Margoliash, D., 1998. Behavioral state modulation of auditory activity in a vocal motor system. Science 282, 2250–2254.

Derégnaucourt, S., Mitra, P.P., Fehér, O., Pytte, C., Tchernichovski, O., 2005. How sleep affects the developmental learning of bird song. Nature 433, 710–716.

Douglas, C.L., Vyazovskiy, V., Southard, T., Chiu, S.Y., Messing, A., Tononi, G., Cirelli, C., 2007. Sleep in Kcna2 knockout mice. BMC Biolology 5, 42.

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

Esser, S.K., Hill, S.L., Tononi, G., 2007. Sleep homeostasis and cortical synchronization. I. Modeling the effects of synaptic strength on sleep slow waves. Sleep 30, 1617–1630.

Faraguna, U., Vyazovskiy, V.V., Nelson, A.B., Tononi, G., Cirelli, C., 2008. A causal role for brain-derived neurotrophic factor in the homeostatic regulation of sleep. Journal of Neuroscience 28, 4088–4095.

Feinberg, I., 1999. Delta homeostasis, stress, and sleep deprivation in the rat: a comment on Rechtschaffen et al. Sleep 22, 1021–1030.

Finelli, L.A., Borbély, A.A., Achermann, P., 2001. Functional topography of the human nonREM sleep electroencephalogram. European Journal of Neuroscience 13, 2282–2290.

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

Franken, P., Dijk, D.J., Tobler, I., Borbély, A.A., 1991. Sleep deprivation in rats: effects on EEG power spectra, vigilance states, and cortical temperature. American Journal of Physiology 261, R198–R208.

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

Flanigan, W.F., 1973. Sleep and wakefulness in iguanid lizards, *Ctenosaura pectinata* and *Iguana iguana*. Brain Behavior and Evololution 8, 401–436.

Flanigan, W.F., 1974. Sleep and wakefulness in chelonian reptiles. II. The redfooted tortoise, *Geochelone carbonaria*. Archives in Italian Biology 112, 253–277.

Flanigan, W.F., Wilcox, R.H., Rechtschaffen, A., 1973. The EEG and behavior continuum of the crocodilian, *Caiman sclerops*. Electroencephraphy and Clinical Neurophysiology 34, 521–538.

Flanigan, W.F., Knight, C.P., Hartse, K.M., Rechtschaffen, A., 1974. Sleep and wakefulness in chelonian reptiles. I. The box turtle, *Terrapene carolina*. Archives in Italian Biology 112, 227–252.

Fuchs, T., 2006. Brain-Behavior Adaptations to Sleep Loss in the Nocturnally Migrating Swainson's Thrush (*Catharus ustulatus*). PhD Dissertation. Bowling Green State University.

Fuchs, T., Haney, A., Jechura, T.J., Moore, F.R., Bingman, V.P., 2006. Daytime naps in night-migrating birds: behavioural adaptation to seasonal sleep deprivation in the Swainson's thrush, *Catharus ustulatus*. Animal Behaviour 72, 951–958.

Ganguly-Fitzgerald, I., Donlea, J., Shaw, P.J., 2006. Waking experience affects sleep need in *Drosophila*. Science 313, 1775–1781.

Gaztelu, J.M., García-Austt, E., Bullock, T.H., 1991. Electrocorticograms of hippocampal and dorsal cortex of two reptiles: comparison with possible mammalian homologs. Brain Behavior and Evolution 37, 144–160.

Güntürkün, O., 2005a. The avian 'prefrontal cortex' and cognition. Current Opinion in Neurobiology 15, 686–693.

Güntürkün, O., 2005b. Avian and mammalian "prefrontal cortices": limited degrees of freedom in the evolution of the neural mechanisms of goal-state maintenance. Brain Research Bulletin 66, 311–316.

Gwinner, E., 1986. Circannual rhythms in the control of avian migrations. In: Rosenblatt, J.S. (Ed.), Advances in the Study of Behavior. 1st ed. Academic Press, New York, pp. 191–228.

Hackett, S.J., Kimball, R.T., Reddy, S., Bowie, R.C., Braun, E.L., Braun, M.J., Chojnowski, J.L., Cox, W.A., Han, K.L., Harshman, J., Huddleston, C.J., Marks, B.D., Miglia, K.J., Moore, W.S., Sheldon, F.H., Steadman, D.W., Witt, C.C., Yuri, T., 2008. A phylogenomic study of birds reveals their evolutionary history. Science 320, 1763–1768.

Hahnloser, R.H., Fee, M.S., 2007. Sleep-related spike bursts in HVC are driven by the nucleus interface of the nidopallium. Journal of Neurophysiology 97, 423–435.

Hairston, I.S., Peyron, C., Denning, D.P., Ruby, N.F., Flores, J., Sapolsky, R.M., Heller, H.C., O'Hara, B.F., 2004. Sleep deprivation effects on growth factor expression in neonatal rats: a potential role for BDNF in the mediation of delta power. Journal of Neurophysiology 91, 1586–1595.

Hartse, K.M., 1994. Sleep in insects and nonmammalian vertebrates. In: Kryger, M.H., Roth, T., Dement, W.C. (Eds.), Principles and Practice of Sleep Medicine. 1st ed. Elsevier Saunders, Philadelphia, pp. 95–104.

Hartse, K.M., Rechtschaffen, A., 1982. The effect of amphetamine, nembutal, alpha-methyl-tyrosine, and parachlorophenylalanine on the sleep-related spike activity of the tortoise, *Geochelone carbonaria*, and on the cat ventral hippocampus spike. Brain Behavior and Evolution 2, 199–222.

Hendricks, J.C., Sehgal, A., 2004. Why a fly? Using Drosophila to understand the genetics of circadian rhythms and sleep. Sleep 27, 334–342.

Hendricks, J.C., Finn, S.M., Panckeri, K.A., Chavkin, J., Williams, J.A., Sehgal, A., Pack, A.I., 2000. Rest in *Drosophila* is a sleep-like state. Neuron 25, 129–138.

Hendricks, J.C., Williams, J.A., Panckeri, K., Kirk, D., Tello, M., Yin, J.C., Sehgal, A., 2001. A non-circadian role for cAMP signaling and CREB activity in *Drosophila* rest homeostasis. Nature Neuroscience 4, 1108–1115.

Hill, S., Tononi, G., 2005. Modeling sleep and wakefulness in the thalamocortical system. Journal of Neurophysiology 93, 1671–1698.

Hill, S., Tononi, G., Ghilardi, M.F., 2008. Sleep improves the variability of motor performance. Brain Research Bulletin 76, 605–611.

Hillman, A.K.K., Young, C.M.A., 1977. Observations of breeding-behavior of meadow pipit *Anthus pratensis* under continual daylight. Ibis 119, 206–207.

Horne, J.A., 2000. REM sleep—by default? Neuroscience and Biobehavioral Reviews 24, 777–797.

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

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

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

Huber, R., Hill, S.L., Holladay, C., Biesiadecki, M., Tononi, G., Cirelli, C., 2004b. Sleep homeostasis in *Drosophila melanogaster*. Sleep 27, 628–639.

Huber, R., Ghilardi, M.F., Massimini, M., Ferrarelli, F., Riedner, B.A., Peterson, M.J., Tononi, G., 2006. Arm immobilization causes cortical plastic changes and locally decreases sleep slow wave activity. Nature Neuroscience 9, 1169–1176.

Huber, R., Määttä, S., Esser, S.K., Sarasso, S., Ferrarelli, F., Watson, A., Ferreri, F., Peterson, M.J., Tononi, G., 2008. Measures of cortical plasticity after transcranial paired associative stimulation predict changes in electroencephalogram slow-wave activity during subsequent sleep. Journal of Neuroscience 28, 7911–7918.

Hunt, G.R., Gray, R.D., 2003. Diversification and cumulative evolution in New Caledonian crow tool manufacture. Proceedings: Biological Sciences 270, 867–874.

Husband, S.A., Shimizu, T., 1999. Efferent projections of the ectostriatum in the pigeon (*Columba livia*). Journal of Comparative Neurology 406, 329–345.

Iwaniuk, A.N., Hurd, P.L., 2005. The evolution of cerebrotypes in birds. Brain Behavior and Evolution 65, 215–230.

Jackson, C., McCabe, B.J., Nicol, A.U., Grout, A.S., Brown, M.W., Horn, G., 2008. Dynamics of a memory trace: effects of sleep on consolidation. Current Biology 18, 393–400.

Jarvis, E.D., Güntürkün, 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., Butler, A.B., 2005. Avian Brain Nomenclature Consortium. Avian brains and a new understanding of vertebrate brain evolution. Nature Reviews Neuroscience 6, 151–159.

Jerison, H.J., 2001. The evolution of neural and behavioral complexity. In: Roth, G., Wullimann, M.F. (Eds.), Brain Evolution and Cognition. Wiley, New York, pp. 523–553.

Jha, S.K., Jones, B.E., Coleman, T., Steinmetz, N., Law, C.T., Griffin, G., Hawk, J., Dabbish, N., Kalatsky, V.A., Frank, M.G., 2005. Sleep-dependent plasticity requires cortical activity. Journal of Neuroscience 25, 9266–9274.

Joiner, W.J., Crocker, A., White, B.H., Sehgal, A., 2006. Sleep in *Drosophila* is regulated by adult mushroom bodies. Nature 441, 757–760.

Jones, S.G., Vyazovskiy, V.V., Cirelli, C., Tononi, G., Benca, R.M., 2008a. Homeostatic regulation of sleep in the white-crowned sparrow (Zonotrichia leucophrys gambelii). BMC Neuroscience 9, 47.

Jones, S., Pfister-Genskow, M., Benca, R.M., Cirelli, C., 2008b. Molecular correlates of sleep and wakefulness in the brain of the white-crowned sparrow. Journal of Neurochemistry 105, 46–62.
Kaiser, W., Steiner-Kaiser, J., 1983. Neuronal correlates of sleep, wakefulness and arousal in a diurnal insect. Nature 301, 707–709.
Karten, H.J., 1969. The organization of the avian telencephalon and some speculationson the phylogeny of the amniote telencephalon. In: Pertras, J., Noback, C.R. (Eds.), Comparative and Evolutionary Aspects of the Vertebrate Central Nervous System, vol. 167. The New York Academy of Sciences, pp. 164–179.
Karten, H.J., 1991. Homology and evolutionary origins of the 'neocortex'. Brain Behavior and Evolution 38, 264–272.
Karten, H.J., Hodos, W., Nauta, W.J., Revzin, A.M., 1973. Neural connections of the "visual wulst" of the avian telencephalon. Experimental studies in the piegon (*Columba livia*) and owl (*Speotyto cunicularia*). Journal of Comparative Neurology 150, 253–278.
Kenigfest, N., Belekhova, M., Repérant, J., Rio, J.P., Ward, R., Vesselkin, N., 2005. The turtle thalamic anterior entopeduncular nucleus shares connectional and neurochemical characteristics with the mammalian thalamic reticular nucleus. Journal of Chemical Neuroanatomy 30, 129–143.
Kim, Y., Laposky, A.D., Bergmann, B.M., Turek, F.W., 2007. Repeated sleep restriction in rats leads to homeostatic and allostatic responses during recovery sleep. Proceedings of the National Academy of Sciences, USA 104, 10697–106702.
Kirsch, J.A., Güntürkün, O., Rose, J., 2008. Insight without cortex: lessons from the avian brain. Consciousness and Cognition 17, 475–483.
Koh, K., Joiner, W.J., Wu, M.N., Yue, Z., Smith, C.J., Sehgal, A., 2008. Identification of SLEEPLESS, a sleep-promoting factor. Science 321, 372–376.
Krueger, J.M., Obál Jr., F., 1993. A neuronal group theory of sleep function. Journal of Sleep Research 2, 63–69.
Krueger, J.M., Obál Jr., F., 2003. Sleep function. Frontiers in Bioscience 8, d511–d519.
Lefebvre, L., Nicolakakis, N., Boire, D., 2002. Tools and brains in birds. Behaviour 139, 939–973.
Lefebvre, L., Reader, S.M., Sol, D., 2004. Brains, innovations and evolution in birds and primates. Brain Behavior and Evolution 63, 233–246.
Lendrem, D.W., 1983. Sleeping and vigilance in birds. In: Koella, W.P. (Ed.), Sleep 1982. Karger, Basle, pp. 134–138.
Lesku, J.A., Martinez-Gonzalez, D, Rattenborg, N.C., in press. Sleep and sleep states: phylogeny and ontogeny. In: Squire, L.R. (Ed.), Encyclopedia of Neuroscience. Elsevier Ltd., Oxford.
Lesku, J.A., Roth II, T.C., Rattenborg, N.C., Amlaner, C.J., Lima, S.L., 2008a. Phylogenetics and the correlates of mammalian sleep: a reappraisal. Sleep Medicine Reviews 12, 229–244.
Lesku, J.A., Bark, R.J., Martinez-Gonzalez, D., Rattenborg, N.C., Amlaner, C.J., Lima, S.L., 2008b. Predator-induced plasticity in sleep architecture in wild-caught Norway rats (*Rattus norvegicus*). Behavioural Brain Research 189, 298–305.
Lesku, J.A., Martinez-Gonzalez, D., Wilzeck, C., Rattenborg, N.C., 2008c. Regional slow-wave sleep homeostasis in the pigeon (*Columba livia*). Sleep 31, A111.
Lima, S.L., Rattenborg, N.C., 2007. A behavioural shutdown can make sleeping safer: a strategic perspective on the function of sleep. Animal Behaviour 74, 189–197.
Llinás, R.R., Steriade, M., 2006. Bursting of thalamic neurons and states of vigilance. Journal of Neurophysiology 95, 3297–3308.
López, J.C., Vargas, J.P., Gómez, Y., Salas, C., 2003. Spatial and non-spatial learning in turtles: the role of medial cortex. Behavioural Brain Research 143, 109–120.
Lorenzo, D., Velluti, J.C., 2004. Noradrenaline decreases spike voltage threshold and induces electrographic sharp waves in turtle medial cortex in vitro. Brain Behavior and Evolution 64, 104–114.
Lorenzo, D., Macadar, O., Velluti, J.C., 1999. Origin and propagation of spontaneous electrographic sharp waves in the in vitro turtle brain: a model of neuronal synchronization. Clinical Neurophysiology 110, 1535–1544.
Manger, P.R., Elston, G.N., Pettigrew, J.D., 2002. Multiple maps and activity-dependent representational plasticity in the anterior Wulst of the adult barn owl (*Tyto alba*). European Journal of Neuroscience 16, 743–750.
Margoliash, D., 1997. Functional organization of forebrain pathways for song production and perception. Journal of Neurobiology 33, 671–693.
Margoliash, D., 2005. Song learning and sleep. Nature Neuroscience 8, 546–548.
Marshall, L., Helgadóttir, H., Mölle, M., Born, J., 2006. Boosting slow oscillations during sleep potentiates memory. Nature 444, 610–613.
Martínez-Cerdeño, V., Noctor, S.C., Kriegstein, A.R., 2006. The role of intermediate progenitor cells in the evolutionary expansion of the cerebral cortex. Cerebral Cortex 16 (Suppl. 1), i152–i161.
Martinez-Gonzalez, D., Lesku, J.A., Rattenborg, N.C., 2008. Increased EEG spectral power density during sleep following short-term sleep deprivation in pigeons (*Columba livia*): evidence for avian sleep homeostasis. Journal of Sleep Research 17, 140–153.
Massimini, M., Huber, R., Ferrarelli, F., Hill, S., Tononi, G., 2004. The sleep slow oscillation as a traveling wave. Journal of Neuroscience 24, 6862–6870.
Medina, L., Reiner, A., 2000. Do birds possess homologues of mammalian primary visual, somatosensory and motor cortices? Trends in Neurosciences 23, 1–12.
Mignot, E., 2008. Why we sleep: the temporal organization of recovery. PLoS Biolology 6, e106.
Mintz, E.M., Phillips, N.H., Berger, R.J., 1998. Daytime melatonin infusions induce sleep in pigeons without altering subsequent amounts of nocturnal sleep. Neuroscience Letters 258, 61–64.
Miyamoto, H., Katagiri, H., Hensch, T., 2003. Experience-dependent slow-wave sleep development. Nature Neuroscience 6, 553–554.
Molnár, Z., Métin, C., Stoykova, A., Tarabykin, V., Price, D.J., Francis, F., Meyer, G., Dehay, C., Kennedy, H., 2006. Comparative aspects of cerebral cortical development. European Journal of Neuroscience 23, 921–934.
Naidoo, N., Casiano, V., Cater, J., Zimmerman, J., Pack, A.I., 2007. A role for the molecular chaperone protein BiP/GRP78 in *Drosophila* sleep homeostasis. Sleep 30, 557–565.
Neckelmann, D., Ursin, R., 1993. Sleep stages and EEG power spectrum in relation to acoustical stimulus arousal threshold in the rat. Sleep 16, 467–477.
Newman, S.M., Paletz, E.M., Rattenborg, N.C., Obermeyer, W.H., Benca, R.M., 2008. Sleep deprivation in the pigeon (*Columba livia*) using the disk-over-water method. Physiology and Behavior 93, 50–58.
Nitz, D.A., van Swinderen, B., Tononi, G., Greenspan, R.J., 2002. Electrophysiological correlates of rest and activity in *Drosophila melanogaster*. Current Biology 12, 1934–1940.
Ookawa, T., 2004. The electroencephalogram and sleep in the domestic chicken. Avian and Poultry Biology Reviews 15, 1–8.
Pepperberg, I.M., 2002. In search of king Solomon's ring: cognitive and communicative studies of Grey parrots (*Psittacus erithacus*). Brain Behavior and Evolution 59, 54–67.
Phillips, N.H., Berger, R.J., 1992. Melatonin infusions restore sleep suppressed by continuous bright light in pigeons. Neuroscience Letters 145, 217–220.
Pitman, J.L., McGill, J.J., Keegan, K.P., Allada, R., 2006. A dynamic role for the mushroom bodies in promoting sleep in *Drosophila*. Nature 441, 753–756.
Puelles, L., Kuwana, E., Puelles, E., Bulfone, A., Shimamura, K., Keleher, J., Smiga, S., Rubenstein, J.L., 2000. Pallial and subpallial derivatives in the embryonic chick and mouse telencephalon, traced by the expression of the genes Dlx-2, Emx-1, Nkx-2.1, Pax-6, and Tbr-1. Journal of Comparative Neurology 424, 409–438.
Noctor, S.C., Martínez-Cerdeño, V., Ivic, L., Kriegstein, A.R., 2004. Cortical neurons arise in symmetric and asymmetric division zones and migrate through specific phases. Nature Neuroscience 7, 136–144.
Rattenborg, N.C., 2006a. Do birds sleep in flight? Naturwissenschaften 93, 413–425.
Rattenborg, N.C., 2006b. Evolution of slow-wave sleep and palliopallial connectivity in mammals and birds: a hypothesis. Brain Research Bulletin 69, 20–29.
Rattenborg, N.C., 2007. Response to commentary on evolution of slow-wave sleep and palliopallial connectivity in mammals and birds: a hypothesis. Brain Research Bulletin 72, 187–193.
Rattenborg, N.C., Amlaner, C.J., 2002. Phylogeny of sleep. In: Lee-Chiong, T., Sateia, M., Carskadon, M. (Eds.), Sleep Medicine. Hanley and Belfus, Philadelphia, pp. 7–22.
Rattenborg, N.C., Amlaner, C.J., Lima, S.L., 2000. Behavioral, neurophysiological and evolutionary perspectives on unihemispheric sleep. Neuroscience and Biobehavioral Reviews 24, 817–842.
Rattenborg, N.C., Lima, S.L., Amlaner, C.J., 1999. Facultative control of avian unihemispheric sleep under the risk of predation. Behavioural Brain Research 105, 163–172.
Rattenborg, N.C., Lesku, J.A., Martinez-Gonzalez, D., Lima, S.L., 2007. The non-trivial functions of sleep. Sleep Medicine Reviews 11, 405–409.
Rattenborg, N.C., Mandt, B.H., Obermeyer, W.H., Winsauer, P.J., Huber, R., Wikelski, M., Benca, R.M., 2004. Migratory sleeplessness in the white-crowned sparrow (*Zonotrichia leucophrys gambelii*). PLoS Biology 2, 924–936.
Rechtschaffen, A., 1998. Current perspectives on the function of sleep. Perspectives in Biology and Medicine 41, 359–390.
Rechtschaffen, A., Bergmann, B.M., 1999a. Sleep stage priorities in rebounds from sleep deprivation: a response to Feinberg. Sleep 22, 1025–1030.
Rechtschaffen, A., Bergmann, B.M., 1999b. Sleep rebounds and their implications for sleep stage substrates: a response to Benington and Heller. Sleep 22, 1038–1043.
Rechtschaffen, A., Bergmann, B.M., 2002. Sleep deprivation in the rat: an update of the 1989 paper. Sleep 25, 18–24.
Rechtschaffen, A., Bergmann, B.M., Gilliland, M.A., Bauer, K., 1999. Effects of method, duration, and sleep stage on rebounds from sleep deprivation in the rat. Sleep 22, 11–31.
Rehkämper, G., Zilles, K., 1991. Parallel evolution in mammalian and avian brains: comparative cytoarchitectonic and cytochemical analysis. Cell and Tissue Research 263, 3–28.
Reiner, A., Yamamoto, K., Karten, H.J., 2005. Organization and evolution of the avian forebrain. Anatomical Record Part A 287, 1080–1102.
Reiner, A., 1991. A comparison of neurotransmitter-specific and neuropeptide-specific neuronal cell types present in the dorsal cortex in turtles with those present in the isocortex in mammals: implications for the evolution of isocortex. Brain Behavior Evolution 38, 53–91.
Reiner, A., 1993. Neurotransmitter organization and connections of turtle cortex: implications for the evolution of mammalian isocortex. Comparative Biochemistry and Physiology: Comparative Physiology 104, 735–748.
Reiner, A., Stern, E.A., Wilson, C.J., 2001. Physiology and morphology of intratelencephalically projecting corticostriatal-type neurons in pigeons as revealed by intracellular recording and cell filling. Brain Behavior Evolution 58, 101–114.
Rial, R.V., Nicolau, M.C., Gamundi, A., Akaârir, M., Garau, C., Aparicio, S., Tejada, S., Moranta, D., Gené, L., Esteban, S., 2007. Comments on evolution of sleep and the palliopallial connectivity in mammals and birds. Brain Research Bulletin 72, 183–186.
Riedner, B.A., Vyazovskiy, V.V., Huber, R., Massimini, M., Esser, S., Murphy, M., Tononi, G., 2007. Sleep homeostasis and cortical synchronization. III. A high-density EEG study of sleep slow waves in humans. Sleep 30, 1643–1657.

Rojas-Ramírez, J.A., Tauber, E.S., 1970. Paradoxical sleep in two species of avian predator (Falconiformes). Science 167, 1754–1755.
Sanchez-Vives, M.V., McCormick, D.A., 2000. Cellular and network mechanisms of rhythmic recurrent activity in neocortex. Nature Neuroscience 3, 1027–1034.
Sejnowski, T.J., Destexhe, A., 2000. Why do we sleep? Brain Research 886, 208–223.
Shaw, P.J., Cirelli, C., Greenspan, R.J., Tononi, G., 2000. Correlates of sleep and waking in *Drosophila melanogaster*. Science 287, 1834–1837.
Shimizu, T., Cox, K., Karten, H.J., 1995. Intratelencephalic projections of the visual Wulst in pigeons (*Columba livia*). Journal of Comparative Neurology 359, 551–572.
Siegel, J.M., 2005. Clues to the functions of mammalian sleep. Nature 437, 1264–1271.
Smith-Fernandez, A.S., Pieau, C., Repérant, J., Boncinelli, E., Wassef, M., 1998. Expression of the Emx-1 and Dlx-1 homeobox genes define three molecularly distinct domains in the telencephalon of mouse, chick, turtle and frog embryos: implications for the evolution of telencephalic subdivisions in amniotes. Development 125, 2099–2111.
Sol, D., Lefebvre, L., Rodríguez-Teijeiro, J.D., 2005a. Brain size, innovative propensity and migratory behaviour in temperate Palaearctic birds. Proceedings: Biological Sciences 272, 1433–1441.
Sol, D., Duncan, R.P., Blackburn, T.M., Cassey, P., Lefebvre, L., 2005b. Big brains, enhanced cognition, and response of birds to novel environments. Proceedings of the National Academy of Sciences, USA 102, 5460–5465.
Steriade, M., 2006. Grouping of brain rhythms in corticothalamic systems. Neuroscience 137, 1087–1106.
Steriade, M., Timofeev, I., 2003. Neuronal plasticity in thalamocortical networks during sleep and waking oscillations. Neuron 37, 563–576.
Stickgold, R., 2005. Sleep-dependent memory consolidation. Nature 437, 1272–1278.
Szymczak, J.T., Kaiser, W., Helb, H.W., Beszczynska, B., 1996. A study of sleep in the European blackbird. Physiology and Behavior 60, 1115–1120.
Tarabykin, V., Stoykova, A., Usman, N., Gruss, P., 2001. Cortical upper layer neurons derive from the subventricular zone as indicated by Svet1 gene expression. Development 128, 1983–1993.
Taylor, A.H., Hunt, G.R., Holzhaider, J.C., Gray, R.D., 2007. Spontaneous metatool use by New Caledonian crows. Current Biology 17, 1504–1507.
Timmermans, S., Lefebvre, L., Boire, D., Basu, P., 2000. Relative size of the hyperstriatum ventrale is the best predictor of feeding innovation rate in birds. Brain Behavior and Evolution 56, 196–203.
Timofeev, I., Grenier, F., Bazhenov, M., Sejnowski, T.J., Steriade, M., 2000. Origin of slow cortical oscillations in deafferented cortical slabs. Cerebral Cortex 10, 1185–1199.
Tobler, I., 2005. Phylogeny of sleep regulation. In: Kryger, M.H., Roth, T., Dement, W.C. (Eds.), Principles and Practice of Sleep Medicine. 4th ed. Elsevier Saunders, Philadelphia, pp. 77–90.
Tobler, I., Achermann, P., 2007. Sleep homeostasis. Scholarpedia 2, 2432.
Tobler, I., Borbély, A.A., 1986. Sleep EEG in the rat as a function of prior waking. Electroencephalography and Clinical Neurophysiology 64, 74–76.
Tobler, I., Borbély, A.A., 1988. Sleep and EEG spectra in the pigeon (*Columba livia*) under baseline conditions and after sleep-deprivation. Journal of Comparative Physiology, Part A: Sensory, Neural and Behavioral Physiology 163, 729–738.
Tobler, I., Jaggi, K., 1987. Sleep and EEG spectra in the Syrian hamster (*Mesocricetus auratus*) under baseline conditions and following sleep deprivation. Journal of Comparative Physiology, Part A: Sensory, Neural and Behavioral Physiology 161, 449–459.
Tobler, I., Franken, P., Scherschlicht, R., 1990. Sleep and EEG spectra in the rabbit under baseline conditions and following sleep deprivation. Physiology and Behavior 48, 121–129.
Tömböl, T., 1995. Golgi Structure of Telencephalon of Chicken. Semmelweis University Medical School, Budapest.
Tononi, G., Cirelli, C., 2003. Sleep and synaptic homeostasis: a hypothesis. Brain Research Bulletin 62, 143–150.
Tononi, G., Cirelli, C., 2006. Sleep function and synaptic homeostasis. Sleep Medicine Reviews 10, 49–62.
van Luijtelaar, E.L., van der Grinten, C.P., Blokhuis, H.J., Coenen, A.M., 1987. Sleep in the domestic hen (*Gallus domesticus*). Physiology and Behavior 41, 409–414.
van Swinderen, B., 2006. A succession of anesthetic endpoints in the *Drosophila* brain. Journal of Neurobiology 66, 1195–1211.
van Swinderen, B., 2007. Sleep in invertebrates. In: Kass, J.H., Striedter, G.F., Rubenstein, J.L.R. (Eds.), Evolution of Neurvous Systems: A Comprehensive Reference, vol. 1. Elsevier Academic Press, New York, pp. 451–456.
van Swinderen, B., Andretic, R., 2003. Arousal in Drosophila. Behavioral Processes 64, 133–144.
Van Twyver, H., Allison, T., 1972. A polygraphic and behavioral study of sleep in the pigeon (*Columba livia*). Experimental Neurology 35, 138–153.
Vyazovskiy, V.V., Tobler, I., 2005a. Theta activity in the waking EEG is a marker of sleep propensity in the rat. Brain Research 1050, 64–71.
Vyazovskiy, V.V., Tobler, I., 2005b. Regional differences in NREM sleep slow-wave activity in mice with congenital callosal dysgenesis. Journal of Sleep Research 14, 299–304.
Vyazovskiy, V.V., Tobler, I., 2008. Handedness leads to interhemispheric EEG asymmetry during sleep in the rat. Journal of Neurophysiology 99, 969–975.
Vyazovskiy, V.V., Achermann, P., Tobler, I., 2007. Sleep homeostasis in the rat in the light and dark period. Brain Research Bulletin 74, 37–44.
Vyazovskiy, V.V., Cirelli, C., Pfister-Genskow, M., Faraguna, U., Tononi, G., 2008. Molecular and electrophysiological evidence for net synaptic potentiation in wake and depression in sleep. Nature Neuroscience 11, 200–208.
Walker, J.M., Berger, R.J., 1972. Sleep in the domestic pigeon (*Columba livia*). Behavioral Biology 7, 195–203.
Weir, A.A., Chappell, J., Kacelnik, A., 2002. Shaping of hooks in New Caledonian crows. Science 297, 981.
Wild, J.M., Karten, H.J., Frost, B.J., 1993. Connections of the auditory forebrain in the pigeon (*Columba livia*). Journal of Comparative Neurology 337, 32–62.
Wu, S.X., Goebbels, S., Nakamura, K., Nakamura, K., Kometani, K., Minato, N., Kaneko, T., Nave, K.A., Tamamaki, N., 2005. Pyramidal neurons of upper cortical layers generated by NEX-positive progenitor cells in the subventricular zone. Proceedings of the National Academy of Sciences, USA 102, 17172–17177.
Zepelin, H., Siegel, J.M., Tobler, I., 2005. Mammalian sleep. In: Kryger, M.H., Roth, T., Dement, W.C. (Eds.), Principles and Practice of Sleep Medicine. 4th ed. Elsevier Saunders, Philadelphia, pp. 91–100.
Zimmerman, J.E., Rizzo, W., Shockley, K.R., Raizen, D.M., Naidoo, N., Mackiewicz, M., Churchill, G.A., Pack, A.I., 2006. Multiple mechanisms limit the duration of wakefulness in *Drosophila* brain. Physiological Genomics 27, 337–350.