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Review

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

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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

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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).

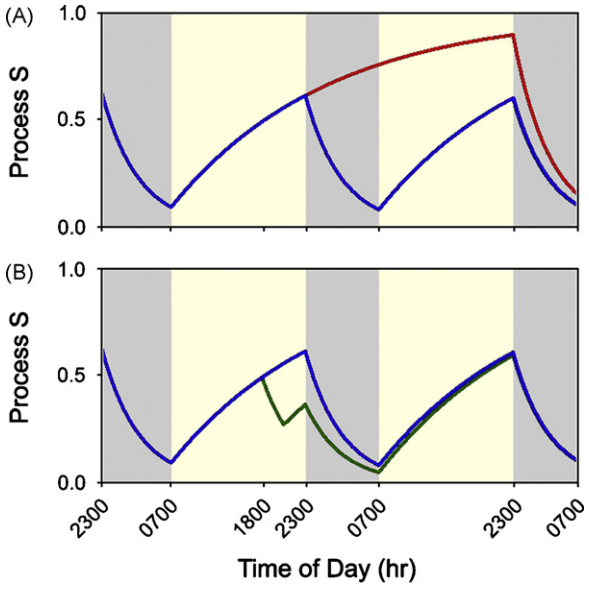

Fig. 1. Model of homeostatically regulated Process S derived from the time course of electroencephalogram slow-wave activity (SWA) in a diurnal mammal. Across a normal 24-h period (blue lines in (A) and (B)). Process S is thought to accumulate as a saturating exponential function of time spent awake 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