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Hippocampal memory consolidation during sleep: a comparison of mammals and birds

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Abstract

The transition from wakefulness to sleep is marked by pronounced changes in brain activity. The brain rhythms that characterize the two main types of mammalian sleep, slow-wave sleep (SWS) and rapid eye movement (REM) sleep, are thought to be involved in the functions of sleep. In particular, recent theories suggest that the synchronous slow-oscillation of neocortical neuronal membrane potentials, the defining feature of SWS, is involved in processing information acquired during wakefulness. According to the Standard Model of memory consolidation, during wakefulness the hippocampus receives input from neocortical regions involved in the initial encoding of an experience and binds this information into a coherent memory trace that is then transferred to the neocortex during SWS where it is stored and integrated within preexisting memory traces. Evidence suggests that this process selectively involves direct connections from the hippocampus to the prefrontal cortex (PFC), a multimodal, high-order association region implicated in coordinating the storage and recall of remote memories in the neocortex. The slow-oscillation is thought to orchestrate the transfer of information from the hippocampus by temporally coupling hippocampal sharp-wave/ripples (SWRs) and thalamocortical spindles. SWRs are synchronous bursts of hippocampal activity, during which waking neuronal firing patterns are reactivated in the hippocampus and neocortex in a coordinated manner. Thalamocortical spindles are brief 7–14 Hz oscillations that may facilitate the encoding of information reactivated during SWRs. By temporally coupling the readout of information from the hippocampus with conditions conducive to encoding in the neocortex, the slow-oscillation is thought to mediate the transfer of information from the hippocampus to the neocortex. Although several lines of evidence are consistent with this function for mammalian SWS, it is unclear whether SWS serves a similar function in birds, the only taxonomic group other than mammals to exhibit SWS and REM sleep. Based on our review of research on avian sleep, neuroanatomy, and memory, although involved in some forms of memory consolidation, avian sleep does not appear to be involved in transferring hippocampal memories to other brain regions. Despite exhibiting the slow-oscillation, SWRs and spindles have not been found in birds. Moreover, although birds independently evolved a brain region – the caudolateral nidopallium (NCL) – involved in performing high-order cognitive functions similar to those performed by the PFC, direct connections between the NCL and hippocampus have not been found in birds, and evidence for the transfer of information from the hippocampus to the NCL or other extra-hippocampal regions is lacking. Although based on the absence of evidence for various traits, collectively, these findings suggest that unlike mammalian SWS, avian SWS may not be involved in transferring memories from the hippocampus. Furthermore, it suggests that the slow-oscillation, the defining feature of mammalian and avian

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SWS, may serve a more general function independent of that related to coordinating the transfer of information from the hippocampus to the PFC in mammals. Given that SWS is homeostatically regulated (a process intimately related to the slow-oscillation) in mammals and birds, functional hypotheses linked to this process may apply to both taxonomic groups.

Keywords

slow-wave sleep; rapid eye movement sleep; homeostasis; sharp-wave ripple; theta; spindle; neostriatum caudolaterale; prefrontal cortex; hippocampus; long-term memory

I. INTRODUCTION

A prominent model of mammalian hippocampal memory consolidation proposes that the hippocampus temporarily stores recently acquired information until it can be integrated into preexisting memory traces in the neocortex for long-term storage (Marr, 1970, 1971; Buzsáki, 1989; McClelland, McNaughton & O'Reilly, 1995; Squire & Alvarez, 1995). The gradual transfer of memories to the neocortex is thought to occur primarily during a sleep state referred to as slow-wave sleep (SWS) in non-human mammals or non-rapid eye movement sleep (non-REM sleep; i.e. stages 2–4) in humans. The transfer is thought to occur *via* the coordinated reactivation of information during temporally-coupled neocortical and hippocampal electrophysiological rhythms (reviewed in Mölle & Born, 2009; Walker, 2009; Diekelmann & Born, 2010; O'Neill *et al*., 2010). Although aspects of this memory transfer hypothesis remain unresolved and under active investigation, several converging lines of evidence are consistent with this functional explanation for mammalian SWS. It is unclear, however, whether the transfer of information from the hippocampus to the neocortex is a mammal-specific function of SWS or a more general aspect of this state. Birds, as the only non-mammalian taxonomic group that exhibits SWS (and REM sleep) provide a unique opportunity to distinguish between these alternatives. The hippocampal memory transfer hypothesis has not been evaluated previously within the context of available work on avian sleep, neuroanatomy, and memory consolidation.

In this review, we summarize the similarities between avian and mammalian sleep, and thereby establish birds as a useful taxonomic group in which to elucidate the functions of mammalian SWS and REM sleep. We then review the aspects of sleep that appear to differ between mammals and birds, and evaluate their implications for understanding the functions of sleep in mammals. Despite exhibiting mammalian-like SWS and REM sleep, the available evidence suggests that several neocortical and hippocampal rhythms present in mammals may be missing in birds. Interestingly, in mammals each of these rhythms has been implicated in transferring information from the hippocampus to the prefrontal cortex (PFC) during SWS (reviewed in Mölle & Born, 2009; Diekelmann & Born, 2010). Consequently, we examine the available evidence for a similar transfer of memories in birds from the hippocampus to the caudolateral nidopallium (NCL, former neostriatum caudolaterale), a region proposed to be analogous to the PFC (Güntürkün, 2005*b*). As with the absence of these rhythms, the available data do not provide conclusive evidence for the transfer of memories from the hippocampus to the NCL or other brain regions in birds. Collectively, the apparent absence of brain rhythms implicated in transferring hippocampal memories and the absence of evidence for such a memory transfer in birds are both consistent with the finding that unlike the mammalian PFC, direct connections between the hippocampus and NCL have not been found in birds. Finally, we discuss the implications of this potential difference in brain organization for understanding the functions of sleep. Specifically, we suggest that the rhythms apparently missing in birds may be involved in a mammal-specific function of SWS, whereas rhythms shared by birds and mammals may be

involved in a more general function of this state. Notably, the defining feature of mammalian and avian SWS – the synchronous slow-oscillation of neuronal membrane potentials – may be involved in a homeostatically regulated process that maintains optimal brain performance.

The proposed hypothesis for the differences between sleep and hippocampal memory consolidation in mammals and birds is intended to serve as a framework for future comparative research. We fully acknowledge that the hypothesis hinges on the absence of evidence for a variety of traits, which certainly is not proof of their absence. Although each of the points that form the framework are based on multiple studies from multiple research groups, it is certainly possible that some or all of the mammal-like traits previously undetected in birds will in fact be described in the future. However, the fact that the traits reported to be missing in birds all relate to a specific mammalian memory consolidation system compelled us to propose that there may be a fundamental difference between how mammals and birds process hippocampal memories. By reviewing this body of research and providing the first conceptual framework for integrating the available evidence, we hope to initiate further discussions, research, and insight into the evolution and functions of sleep.

II. AVIAN HIPPOCAMPAL FORMATION AND 'PREFRONTAL CORTEX'

Table 1 provides a list of abbreviations used in the following text.

(1) Hippocampal formation

Given that the reported differences between avian and mammalian sleep reviewed herein may relate to differences in the neuroanatomy and neurophysiology of the hippocampus and associated structures, a brief overview of these structures is in order. Historically, the avian hippocampal formation (HF) has included the hippocampus medioventrally and the area parahippocampalis (APH) dorsolaterally (Karten & Hodos, 1967). Developmental gene expression studies have confirmed that the mammalian and avian HF arise from the same embryonic neural tissue, and therefore are homologous as a field (Smith-Fernandez *et al*., 1998; Puelles *et al*., 2000; Puelles, 2001; Medina & Abellán, 2009). However, the avian HF does not resemble the three-layered mammalian HF (Fig. 1A). Except for a cell-dense V-shaped structure at the most medioventral portion of the avian HF (Fig. 1B), layers and subregions are not readily identifiable in Nissl stains. Although a variety of approaches (e.g. histochemistry for neuropeptides and neurotransmitters, tract tracing, and electrophysiology) have been used to characterize the subregions of the HF, it remains unclear which subregions are homologous in mammals and birds (reviewed in Atoji & Wild, 2006).

In a recent systematic analysis of the avian HF, Atoji & Wild (2004) proposed the following subregion nomenclature and tentative homology with mammalian HF subregions (Fig. 2). The V-shaped region is composed of intrinsic neurons, and therefore may be homologous with the mammalian dentate gyrus. Collectively, the dorsomedial (DM) and dorsolateral (DL) regions comprise the area previously referred to as the APH. The V-shaped region is reciprocally connected with the DM, which may therefore be homologous with the mammalian Ammon's horn (cornu ammonis, CA), although it is unclear which, if any, subdivision of the DM corresponds to CA1, CA2, or CA3. Moreover, unlike the CA regions, but like the subiculum, the DM projects to the hypothalamus. Similar to Ammon's horn and the subiculum, the DM also projects to the lateral and medial septum. Consequently, the DM has features of both Ammon's horn and the subiculum. As with the mammalian hippocampus, the DL and DM project to the lateral and medial septum. Unlike mammals, however, previous studies suggest that projections from the medial septum to the HF are sparse or absent in birds (Krayniak & Siegel, 1978; Atoji & Wild, 2004; Montagnese *et al*., 2004). The DM receives projections from the DL, which may be homologous with the

mammalian entorhinal cortex, a main route through which high-order multimodal associational information from the neocortex enters and leaves the mammalian hippocampus (Lavenex & Amaral, 2000). Unlike the entorhinal cortex, however, the DL only receives direct olfactory information and high-order visual information; projections from high-order multimodal association areas, such as the nidopallium and mesopallium, to the avian HF have not been reported. Similarly, although the DL and the dorsolateral corticoid area (CDL; Fig. 1C, D) – a region reciprocally connected to the DL and DM – project reciprocally to high-order visual areas, unlike the mammalian HF, projections to other pallial regions analogous to the neocortex are sparse or absent altogether (Atoji & Wild, 2005). Collectively, this suggests that the HF is involved in processing different types of information in birds and mammals.

In contrast to the interpretation of Atoji & Wild (2004, 2006), other researchers have come to different conclusions regarding homology between mammalian and avian HF subregions. For instance, Kahn *et al*. (2003) suggest that the V-shaped region corresponds to Ammon's horn, the dorsal part of the DM to the dentate gyrus, and the ventral part of the DM to the subiculum. A circuit that bears some resemblance to the tri-synaptic feed-forward circuit in the mammalian hippocampus has been described in pigeons (Hough, Pang & Bingman, 2002; Kahn *et al*., 2003). However, given the absence of a clear mammal-like, zinc-rich mossy fibre bundle (Faber *et al*., 1989; Montagnese, Krebs & Meyer, 1996), and conflicting interpretations over which regions reflect the dentate gyrus and Ammon's horn (Székely & Krebs, 1996; Kahn *et al*., 2003; Atoji & Wild, 2006), it remains unclear whether information flows through the avian and mammalian HF in a similar manner. Finally, based on gene expression patterns, Puelles *et al*. (2007) propose an alternative description of the HF that includes a region lateral to the CDL that may be homologous to the mammalian entorhinal cortex (see also Suárez *et al*., 2006). While keeping these issues in mind, from here on we rely on the nomenclature of Atoji & Wild (2006).

(2) Caudolateral nidopallium (NCL) – the avian 'prefrontal cortex'

Divac and coworkers were the first to propose that the avian equivalent of the PFC is the NCL (Mogensen & Divac, 1982, 1993; Divac, Mogensen & Björklund, 1985). Since then a growing body of research has accumulated supporting the notion that the NCL and PFC are analogous structures involved in performing high-order cognitive processes (Reiner, 1986; Güntürkün, 1997; Hartmann & Güntürkün, 1998; Braun *et al*., 1999; Kalt, Diekamp & Güntürkün, 1999; Diekamp, Kalt & Güntürkün, 2002; Rose & Colombo, 2005; Milmine, Rose & Colombo, 2008; reviewed in Güntürkün, 2005*a*,*b*; Kirsch, Güntürkün & Rose, 2008; Rose, Güntürkün & Kirsch, 2009). Although the PFC and NCL both develop from the pallium (dorsal portion of the neural tube), they originate from different pallial sectors, and therefore reflect analogous structures (see Section VII). The PFC and NCL also receive input from different, but perhaps functionally similar, thalamic nuclei (Güntürkün, 1997; Kröner & Güntürkün, 1999; Butler *et al*., 2005; Csillag & Montagnese, 2005; Güntürkün, 2005*a*,*b*). Moreover, unlike the mammalian PFC, which is reciprocally connected to the thalamus (Krettek & Price, 1977; Divac & Passingham, 1980; Giguere & Goldman-Rakic, 1988), the avian NCL does not appear to project to the thalamus (Kröner & Güntürkün, 1999; Csillag & Montagnese, 2005; Butler, 2008). Despite these differences, like the PFC, the NCL is a high-order multimodal association area reciprocally connected to secondary sensory regions for processing visual, auditory, somatosensory and trigeminal information (Fig. 3; Leutgeb *et al*., 1996; Metzger, Jiang & Braun, 1998; Braun *et al*., 1999; Kröner & Güntürkün, 1999). In addition, the NCL is reciprocally connected to other multimodal association regions in the nidopallium and mesopallium (Metzger *et al*., 1998; Kröner & Güntürkün, 1999). Finally, similar to the PFC, the NCL also projects to the somatic and limbic striatum, as well as to motor output structures. As a result, like the PFC, the NCL is a

convergence zone for integrating multimodal sensory information and orchestrating behavioural output.

In addition to processing multimodal sensory information, the NCL also appears to be involved in working memory and executive control, high-order cognitive functions similar to those performed by the PFC. In both the PFC and NCL, working memory – the ability to retain and manipulate a representation of an absent physical stimulus across a delay – is dependent upon the release of dopamine in the NCL from projections from the midbrain ventral tegmental area and the substantia nigra (Divac *et al*., 1985; Wynne & Güntürkün, 1995; Metzger *et al*., 1996; Güntürkün & Durstewitz, 2001; Güntürkün, 2005*a*,*b*). The PFC and NCL are also involved in executive control, the ability to plan one's behaviour to obtain a goal (Lissek, Diekamp & Güntürkün, 2002; Kalenscher, Diekamp & Güntürkün, 2003; Güntürkün, 2005*a*,*b*; Kalenscher *et al*., 2005). For instance, neuronal recordings have shown that the NCL is involved in determining what should be remembered and what should be forgotten, a form of executive control fundamental to working memory (Rose & Colombo, 2005). Although these studies suggest that the PFC and NCL are analogous structures, additional studies are needed to characterize fully the extent of this similarity, and assess the potential contribution of other avian brain structures to the performance of PFC-like functions (Emery & Clayton, 2005).

The available neuroanatomical, biochemical, electrophysiological, and behavioural evidence suggests that the mammalian PFC and avian NCL may perform similar functions. However, tract-tracing studies suggest that interactions between the NCL and HF may differ from those between the mammalian PFC and HF (Leutgeb *et al*., 1996; Kröner & Güntürkün, 1999; Atoji *et al*., 2002; Atoji & Wild, 2006; see also Patzke *et al*., 2009). In mammals, the HF interacts closely with the PFC (Simons & Spiers, 2003; Jung *et al*., 2008) *via* monosynaptic excitatory projections from CA1 hippocampal neurons (Swanson, 1981; Jay & Witter, 1991; Thierry *et al*., 2000). In addition, the PFC is reciprocally connected to the hippocampus *via* the entorhinal cortex (Simons & Spiers, 2003), a main structure involved in the transfer of multimodal associational information between the hippocampus and neocortex (Lavenex & Amaral, 2000). In contrast to the mammalian PFC, however, direct projections from the hippocampus to the NCL have not been detected in birds (Leutgeb *et al*., 1996; Kröner & Güntürkün, 1999; Atoji *et al*., 2002; Atoji & Wild, 2006). In addition, projections from the DL – the avian equivalent of the mammalian entorhinal cortex according to Atoji & Wild (2006) – to the NCL have not been found (Leutgeb *et al*., 1996; Kröner & Güntürkün, 1999; Atoji *et al*., 2002; Atoji & Wild, 2006). Also, direct projections have not been detected between the avian entorhinal cortex, as defined by Puelles *et al*. (2007; see also Suárez *et al*., 2006; Abellán *et al*., 2009), and the NCL (Leutgeb *et al*., 1996; Kröner & Güntürkün, 1999). Other hippocampal projection regions, such as the DM, also do not appear to project to the NCL. Moreover, although the DL and DM regions are each reciprocally connected to the CDL (Figs 1C,D, 2), an area that is in some respects (but not all) similar to the mammalian cingulate cortex, unlike the cingulate cortex, which projects to the PFC, projections from the CDL to the NCL have not been found in birds (Kröner & Güntürkün, 1999; Atoji & Wild, 2005). Thus, in contrast to the mammalian hippocampus and PFC, the avian hippocampus and NCL do not appear to be connected to one another. Assuming that this does not reflect a failure to detect such projections, the available studies suggest that, in contrast to the mammalian hippocampus, the avian hippocampus does not receive highly associational multimodal information, and does not have direct connections for conveying the information that it does receive to the NCL. This apparent difference in brain organization may explain some of the reported differences in sleep between mammals and birds. Before delving into the divergent aspects of mammalian and avian sleep, however, we first review the aspects of sleep that these groups have in common.

III. AVIAN AND MAMMALIAN SLEEP

(1) Convergent aspects

Although birds are nested within Reptilia, avian sleep patterns are more similar to those of their distant mammalian relatives, than they are to crocodilians – their closest living relatives – or any other group of reptiles. The electroencephalogram (EEG) patterns observed during wakefulness, SWS, and REM sleep in birds are remarkably similar to those in mammals, even though neurons in the avian pallium – the developmental and functional homologue of the mammalian neocortex – are arranged in a largely nuclear, rather than a cortical (laminar) manner (Jarvis *et al*., 2005; Medina & Abellán, 2009). As in mammals, during SWS, the avian EEG (recorded from the dorsal surface of the pallium) is characterized by prominent high-amplitude, slow-waves, typically quantified as slow-wave activity (SWA; 0.5 – 4 Hz power density). Also as in mammals, the EEG during avian REM sleep shows a low-amplitude, high-frequency, or activated, pattern similar to that occurring during wakefulness. In mammals, slow-waves reflect the synchronous slow-oscillations (<1 Hz) of neocortical neuronal membrane potentials between a hyperpolarized 'down-state' with no action potentials and a depolarized 'up-state' with action potentials occurring at a rate comparable to wakefulness (Steriade, Nuñez & Amzica, 1993; Steriade, 2006; Crunelli & Hughes, 2010; Chauvette, Volgushev & Timofeev, in press). Apparently, similar slow-oscillations occurring in the pallium are the neurophysiological basis for EEG slow-waves in sleeping birds (Reiner, Stern & Wilson, 2001). The mammalian slow-oscillation is synchronized *via* corticocortical projections in a manner resulting in highly synchronous slow network oscillations detectable in the EEG (Amzica & Steriade, 1995; Sanchez-Vives & McCormick, 2000; Timofeev *et al.*, 2000; Hill & Tononi, 2005; Vyazovskiy *et al*., 2009*b*). In humans, slow-oscillations are most prominent in medial regions of the PFC (Massimini *et al*., 2004; Dang-Vu *et al*., 2008; Murphy *et al*., 2009). In addition, during SWS, regional cerebral blood flow (a correlate of neuronal activity) in the medial PFC (mPFC) is reduced when compared to wakefulness (Maquet *et al*., 2005) and inversely correlated with SWA (Dang-Vu *et al*., 2005). Slow-oscillations propagate across the neocortex as a traveling wave, typically in a frontal to posterior direction (Massimini *et al*., 2004; Volgushev *et al*., 2006; Mohajerani *et al*., 2010; Leemburg *et al.,* in press). Although the slow-oscillation has a frequency of <1 Hz, interactions between multiple traveling waves may contribute to the higher frequency SWA that charcterizes stages 3 and 4 sleep (Riedner *et al*., 2007). In mammals, SWA increases and decreases as a function of time spent awake and asleep, respectively, suggesting that SWA reflects homeostatically regulated processes closely tied to the function of SWS (Borbély & Achermann, 2005; Tononi & Cirelli, 2006; Vyazovskiy *et al*., 2009*b*). We recently demonstrated that SWS-related SWA is also homeostatically regulated in birds (Fig. 4; Martinez-Gonzalez, Lesku & Rattenborg, 2008; see also Jones *et al*., 2008*b*), suggesting that homeostasis is a fundamental aspect of SWS (reviewed in Rattenborg, Martinez-Gonzalez & Lesku, 2009).

The absence of high-amplitude slow-waves in sleeping reptiles similar to those observed in sleeping mammals and birds may reflect differences in cortical cytoarchitecture (Rattenborg, 2006, 2007). Notably, although absolute measures of corticocortical connectivity are missing, the three-layered reptilian dorsal cortex appears to lack layers with extensive corticocortical connectivity comparable to layers II and III of the six-layered mammalian neocortex (Medina & Reiner, 2000). Moreover, although the avian pallium is arranged in a largely non-laminar manner, it has been suggested that birds independently evolved neurons with extensive palliopallial interconnectivity, comparable to those in neocortical layers II and III (Medina & Reiner, 2000; Medina & Abellán, 2009). Given the role of corticocortical connections in synchronizing the slow-oscillation in mammals, this convergence in interconnectivity may explain, in part, why only mammals and birds show high-amplitude slow-waves during sleep (Rattenborg, 2006; see also Seelke & Blumberg, 2010). Although it

is certainly possible that SWS-related SWA is simply an epiphenomenon of heavily interconnected brains, the resulting slow network synchrony may also serve a function not found in animals lacking this trait. For instance, given that SWS, heavily interconnected brains, and the ability to perform complex cognitive processes (Emery & Clayton, 2005; Jarvis *et al*., 2005; Kirsch *et al*., 2008) coevolved independently in mammals and birds, the slow, synchronous network oscillation may play a role in maintaining the ability to perform complex cognition in animals with such brains (Rattenborg *et al*., 2009).

(2) Divergent aspects

Despite the marked similarities between avian and mammalian SWS, there also appear to be some potentially important differences. Interestingly, most of these differences relate to interacting hippocampal and neocortical rhythms implicated in the transfer of memories from the hippocampus to the neocortex in mammals.

(a) Thalamocortical spindles—A hallmark of mammalian SWS (human non-REM sleep) is thalamocortical spindles, intermittent brief bursts of 7 – 14 Hz oscillations detectable in the EEG. Although spindle oscillations originate in the thalamic reticular nucleus (TRN), the neocortical slow-oscillation organizes TRN spindle activity *via* collaterals from neocortical projections to other thalamic nuclei (Steriade, 2006). Specifically, during the up-state of the neocortical slow-oscillation, the otherwise asynchronous activity of spindle pacemaker neurons is synchronized by this neocortical input (Contreras, Destexhe & Steriade, 1997). This synchronized activity is relayed to other thalamic nuclei and then back to the neocortex, resulting in spindles occurring during the up-state of the slow-oscillation (Destexhe, Contreras & Steriade, 1999; Steriade, 1999, 2006; Mölle *et al*., 2002, 2006, 2009; Clemens *et al*., 2007; Csercsa *et al*., 2010; Le Van Quyen *et al*., 2010).

Although the slow-oscillation organizes spindles, the exact relationship may be more complex. In humans, spindles occur throughout non-REM sleep, but are most frequent during stage 2 sleep occurring at the start and end of an episode of non-REM sleep. Slow-oscillations also occur throughout non-REM sleep (e.g. stage 2 K-complexes followed by a spindle reflect one cycle of the slow-oscillation; Amzica & Steriade, 1998), but are most prevalent during stages 3 and 4 (i.e. human 'delta sleep' or 'SWS'). During the transition from stage 2 to 3–4 sleep, it is thought that corticothalamic volleys cause the TRN to hyperpolarize thalamocortical neurons to a point where they generate bursts occurring at 1 – 4 Hz, which are relayed back to the neocortex where they contribute to the high level of SWA that characterizes stages 3 and 4 sleep (Steriade, 2006). In addition, or alternatively, as discussed above (see Section III.1), interactions between multiple traveling waves may contribute to the high level of SWA (Riedner *et al*., 2007). Thus, although spindles and slow-oscillations increase as humans enter stage 2 sleep, an inverse relationship between spindle activity and SWA exists during stages 3–4 (Dijk, Hayes & Czeisler, 1993). Despite this complex relationship, spindles occur during the up-state of the slow-oscillation regardless of whether it occurs during stage 2 or 3–4 sleep (Mölle *et al*., 2009). A final issue regarding spindles is that in contrast to humans, a recent study in rats suggests that spindle activity occurring during SWS is positively correlated with SWA (Yasenkov & Deboer, 2010). The reasons for this apparent difference remain unclear. Given these potential differences between rats and humans, we will specify below the exact non-REM sleep state under consideration when discussing results from humans. In rats and birds, non-REM sleep is usually not divided into substates, and therefore will simply be referred to as SWS.

Although the avian pallium generates slow-oscillations and resulting high-amplitude slow-waves during SWS, thalamocortical (or thalamopallial) spindles have not been observed in

epidural EEG recordings from any bird species (e.g. Ookawa & Gotoh, 1965; Ookawa & Kadono, 1966; Hishikawa, Cramer & Kuhlo, 1969; Ookawa, 1972; Van Twyver & Allison, 1972; Walker & Berger, 1972; Šušić & Kovačević, 1973; Dewasmes *et al*., 1985; Ayala-Guerrero, Pérez & Calderón, 1988; Ayala-Guerrero & Vasconcelos-Dueñas, 1988; Tobler & Borbély, 1988; Ayala-Guerrero, 1989; Zepelin, Hartzer & Pendergast, 1998; Ayala-Guerrero, Mexicano & Ramos, 2003; Szymczak, Helb & Kaiser, 1993; Rattenborg *et al*., 2004; Low *et al*., 2008, Martinez-Gonzalez *et al*., 2008). In addition, spindles were not observed in depth recordings from the hyperpallium of tawny owls (*Strix aluco*; Šušić & Kovačević, 1973) or from various telencephalic regions in chickens (*Gallus gallus domesticus*; Ookawa, 1967). Preliminary reports of spindles in birds proved to be artifacts originating from intermittent high-frequency oscillations of the eyes that occur during both SWS and wakefulness in birds (Paulson, 1964; Pettigrew, Wallman & Wildsoet, 1990; Zepelin *et al*., 1998; Hunter *et al*., 2000; Coenen, San Miguel & McKeegan, 2005). Consistent with the absence of reports of spindles during normal sleep, drugs that augment spindling in mammals, such as barbiturates, do not induce spindling in birds, despite having a similar somnogenic effect (Key & Marley, 1962; Ookawa & Kadono, 1968; Ookawa, 1972). Finally, although thalamic nuclei involved in song production show bursts of activity that influence activity in song-related pallial regions in anaesthetized zebra finches (*Taeniopygia guttata*), these bursts do not occur in a rhythmic pattern comparable to that occurring during mammalian thalamocortical spindles (Hahnloser *et al*., 2008).

The reason for the lack of pallial spindles in recordings of avian sleep is unclear largely due to the lack of research on the topic. Although dorsal thalamic nuclei receive reciprocal projections from the hyperpallium in birds (Karten *et al*., 1973; Miceli *et al*., 1987, 2008; Watanabe, 1987; Wild & Williams, 2000), these projections are apparently less extensive than comparable projections in mammals (Jones, 2007; Butler, 2008). As in mammals, the avian TRN projects to other thalamic nuclei (Wild, 1989; Mpodozis *et al*., 1996; Miceli *et al*., 2008), but it is not known whether the TRN receives collateral input from palliothalamic projections comparable to that necessary for synchronizing spindle activity in the mammalian TRN (Butler, 2008). Moreover, it is not known whether neurons in the avian TRN are even capable of generating spindle oscillations. Although neurophysiological properties (e.g. low-threshold Ca^{2+} spike and time-dependent, hyperpolarization-activated inward rectification) involved in the generation of mammalian spindles (Llinás & Steriade, 2006) have been described *in vitro* in the medial nucleus of the dorsolateral thalamus of zebra finches, this nucleus is involved in the pallio-striatopallido-thalamic loop, rather than the thalamo-cortical loop involved in mammalian spindles (Luo & Perkel, 1999), and spindling has not been demonstrated in this circuit *in vivo*. Clearly, additional work is needed to determine whether thalamic nuclei in birds generate spindle oscillations during SWS functionally similar to those occurring in mammals.

Even if thalamic spindles are identified, the absence of spindles in pallial EEG recordings seems to be a fundamental difference between mammalian and avian SWS. Differences in thalamocortical connections (as discussed above) might prevent the transmission of thalamic spindles to the avian pallium. Alternatively, if thalamic spindle activity is relayed to the pallium, it might not induce neuronal synchrony detectable in the avian EEG; perhaps a mammal-like laminar neocortex is necessary for spindle oscillations to manifest in EEG recordings. Although this explanation for the absence of pallial spindles can not be ruled out, it is worth noting that despite lacking a laminar organization, synchronous neuronal activity occurring at frequencies below and above that of mammalian spindles is readily detectable in pallial EEG recordings of the avian brain (Low *et al*., 2008; Vyssotski *et al*., 2009).

(b) Hippocampal sharp-wave/ripples—During mammalian SWS and to a lesser extent quiet wakefulness, grooming, and feeding, CA3 neurons in the hippocampus initiate intermittent highly synchronous bursts of activity that cause high-amplitude (e.g. 2 mV), sharp-wave field potentials in the dentritic layer and 100 – 200 Hz 'ripples' in the pyramidal layer of CA1 (Fig. 5; Buzsáki, 1986,1989;Sirota & Buzsáki, 2005;Ulanovsky & Moss, 2007). In contrast to mammals, such hippocampal sharp-wave/ripples (SWRs) have not been reported in birds during SWS (Ookawa & Gotoh, 1965;van Twyver & Allison, 1972;Sugihara & Gotoh, 1973;Fuchs *et al*., 2006;Martinez-Gonzalez *et al*., 2008,Dos Santos *et al*., 2009). Instead, the avian hippocampus tends to show only slow-waves similar to those observed in other pallial regions, albeit with a lower amplitude during SWS (Sugihara & Gotoh, 1973). In addition, SWRs were not reported in local field potential recordings from multiple hippocampal regions in pigeons during quiet wakefulness (Siegel, Nitz & Bingman, 2000). It seems unlikely that mammal-like SWRs simply evaded detection in pigeons because they are highly synchronous events in rats, resulting in field potentials two to three times greater than the hippocampal theta rhythm (Buzsáki, 1989). Even if future studies detect similar network events in birds occurring on a smaller, local scale that have simply evaded detection in previous studies, this difference in the scale of network synchrony may still reflect functional differences between mammalian and avian hippocampal activity, especially given that it is the highly synchronous nature of SWRs that is thought to influence activity in the neocortex during SWS (Buzsáki, 1989;Buzsáki & Chrobak, 2005).

(c) Hippocampal theta—Although the primary focus of this review is on SWS, reported differences between mammalian and avian REM sleep involve hippocampal rhythms mechanistically and perhaps functionally related to SWRs. Consequently, REM sleep also has bearing on our understanding of the differences between mammalian and avian SWS.

In mammals, a prominent theta (6 – 12 Hz) rhythm occurs in the hippocampus during spatial navigation while awake and during REM sleep (Buzsáki, 2002; Sirota & Buzsáki, 2005; Cornwell *et al*., 2008). In some mammals, theta may also occur in response to sensory feedback in the absence of movement (Ulanovsky & Moss, 2007). The origin of the hippocampal theta rhythm remains debated. Early studies suggested that the medial septum-diagonal band of Broca (MSDB) is necessary for the generation of the hippocampal theta rhythm, as lesioning the MSDB or severing its reciprocal connections with the hippocampus disrupts this rhythm (Green & Arduini, 1954; Petsche, Stumpf & Gogolak, 1962; Winson, 1978; Givens & Olton, 1990; Shirvalkar, Rapp & Shapiro, 2010). By contrast, a recent *in vitro* study suggests that the isolated hippocampus also generates a theta rhythm (Goutagny, Jackson & Williams, 2009). Nonetheless, projections from the MSDB to the hippocampus and feedback from the hippocampus to the MSDB may both be involved in synchronizing theta oscillators in the medial septum and hippocampus (Sirota & Buzsáki, 2005; Colgin & Moser, 2009). Indeed, bursts of neuronal activity in the medial septum occurring at the frequency of theta preceed hippocampal theta activity in anaesthetized rats, suggesting that the medial septum serves as a pacemaker for the hippocampal theta rhythm (Hangya *et al*., 2009). Moreover, medial septum lesions that disrupt the hippocampal theta rhythm also disrupt spatial working memory, suggesting that the contribution of the medial septum to the hippocampal theta rhythm is essential for processing spatial information (Winson,1978; Givens & Olton, 1990; McNaughton, Ruan & Woodnorth, 2006).

In addition to the differences in hippocampal activity observed during mammalian and avian SWS, hippocampal activity also appears to differ during REM sleep. Although birds exhibit EEG activation and other features characteristic of mammalian REM sleep (e.g. rapid eye movements, twitching, reduced muscle tone, and reduced thermoregulation), a hippocampal theta rhythm has not been observed during avian REM sleep (Berger & Walker, 1972; van Twyver & Allison, 1972; Sugihara & Gotoh, 1973; Šušić & Kovačević, 1973; Ookawa,

2004; Fuchs *et al*., 2006; Martinez-Gonzalez *et al*., 2008; Dos Santos *et al*., 2009). In addition, most studies did not find a hippocampal theta rhythm during wakefulness in birds (van Twyver & Allison, 1972; Sugihara & Gotoh, 1973; Dos Santos *et al*., 2009), apparently including flying pigeons navigating home (supplemetal material in Vyssotski *et al*., 2009). The exception was a study that reported a 4 – 5 Hz theta rhythm during wakefulness in multiple regions of the HF in pigeons (Siegel *et al*., 2000). It is unclear, however, whether these results reflect the same phenomenon as the hippocampal theta rhythm in mammals. Unlike the theta rhythm observed in rats that typically occurs while an animal is moving, the theta rhythm in pigeons often occurred when the birds were still (Siegel *et al*. 2000; see also Siegel, Nitz & Bingman, 2002; Bingman *et al*., 2005), a time when the mammalian hippocampus generates SWRs. This observation and the fact that the theta rhythm reported in pigeons is slower than that in mammals, suggest that this slow activity may reflect a drowsy state, rather than a theta rhythm comparable to that observed in mammals. Indeed, the pigeon hippocampus is reciprocally connected to the adjacent hyperpallium (Atoji & Wild, 2006), a primary visual area that starts to oscillate in the 1 – 5 Hz range shortly after the bird becomes immobile, and before it closes its eyes (Tobler & Borbély, 1988; Martinez-Gonzalez *et al*., 2008). Consequently, the theta rhythm observed in pigeons during quiet wakefulness may have originated in the hyperpallium during a drowsy state, rather than in the hippocampus. Also unlike rats, the theta rhythm in pigeons occurred during feeding. We have also observed this phenomenon in EEG recordings from the hyperpallium in a variety of birds. However, concurrent accelerometer recordings have shown that each wave is associated with a head movement (N.C. Rattenborg, unpublished data; see also Fuchs, 2006). Consequently, this activity may reflect an artifact related to movement of the brain, or bursting of neurons in the hyperpallium resulting from concurrent visual saccades (Yang, Yang & Wang, 2008). Future studies need to rule out each of these potential sources of theta activity before concluding that the avian hippocampus generates a theta rhythm comparable to that observed in mammals. Finally, Siegel *et al*. (2000) did not report whether a theta rhythm occurred during REM sleep in their pigeons, as would be expected if the theta reported during wakefulness reflects a phenomenon comparable to that observed in mammals. Perhaps consistent with the absence of a theta rhythm during wakefulness and REM sleep in other studies of pigeons and chickens, projections from the medial septum to the hippocampus, a pathway implicated in the genesis of the hippocampal theta rhythm in mammals (Dragoi *et al*., 1999; Gerashchenko, Salin-Pascual & Shiromani, 2001), appear to be sparse or absent in both species (Krayniak & Siegel, 1978; Atoji & Wild, 2004; Montagnese *et al*., 2004; reviewed in Atoji & Wild, 2006). The apparent absence of SWRs in birds is also consistent with this interpretation given that mammalian SWRs occurring during SWS and quiet wakefulness, and theta rhythms occurring during active wakefulness and REM sleep are thought to reflect functionally coupled, reciprocal modes of hippocampal processing that rely on the hippocampal-septal-hippocampal loop for their coordination (Buzsáki, 1989; Dragoi *et al*., 1999).

(d) Hippocampal place cells—In addition to the equivocal evidence for a hippocampal theta rhythm during avian wakefulness and the apparent absence of theta during REM sleep, mammals and birds also appear to differ in how hippocampal neurons respond to environmental input. In mammals, during initial passage through an environment, CA1 pyramidal cells establish a preference for selectively firing when the animal is in a specific part of the environment (O'Keefe & Dostrovsky, 1971). As an animal moves through the responsive field (i.e. 'place field') of such 'place cells', the cell initially fires during a specific phase of the theta rhythm, but with each successive theta cycle fires at progressively earlier phases (O'Keefe & Recce, 1993; Mehta, Lee & Wilson, 2002; Huxter *et al*., 2008). Such 'phase procession' and the finding that place cells with overlapping place fields fire in succession as an animal moves from one place field to another are thought to provide the

animal with information about its location in space. As discussed later, this sequence of place cell firing is reactivated during SWRs occurring during subsequent SWS, a phenomenon that may reflect the transfer of hippocampal information to the neocortex (Ji & Wilson, 2007).

Interestingly, mammal-like place cells have not been found in pigeons (Kahn & Bingman, 2009). Although some hippocampal cells show transient preferences for firing when a pigeon is at a place with a known food reward (Hough & Bingman, 2004, 2008; Siegel, Nitz & Bingman, 2005, 2006), place fields are unstable when rewards are distributed randomly in space (Kahn *et al*., 2008; Kahn & Bingman, 2009), a pattern unlike that observed in rats where place cells can stably signal the animal's position in the absence of a reward (Lever *et al*., 2002). The apparent absence of cells that signal a pigeon's position independent of reward and equivocal evidence for theta rhythms during wakefulness and REM sleep, two interrelated phenomena involved in processing spatial information in mammals, suggests that there may be fundamental differences in how mammals and birds encode spatial information.

An important caveat, however, is that attempts to identify place cells in birds have been restricted to pigeons walking in an arena. Certainly, it is possible that place cells in birds only respond to rapidly changing spatial scenes as occur during flight. Recent technological advancements that permit hippocampal unit recordings from homing pigeons flying in the wild promise to reveal whether place cells are involved in processing spatial information during flight (Vyssotski *et al*., 2006, 2009).

IV. MODELS OF MAMMALIAN MEMORY CONSOLIDATION

Studies of memory deficits in patients with brain lesions have influenced profoundly the formulation of hippocampal memory consolidation models. Although patients with lesions to the hippocampus and associated temporal lobe structures (Lavenex & Amaral, 2000) are unable to recall events that occurred shortly before the brain damage, recall improves as the age of the memory increases (Scoville & Milner, 1957; Teng & Squire, 1999; Eichenbaum, 2000; Bayley, Hopkins & Squire, 2006; Smith & Squire, 2009). Descriptions of such temporally graded retrograde amnesia led to the hypothesis that the HF serves as a temporary memory storage site that holds information until it can be gradually incorporated within existing neocortical networks over time (Marr, 1971; Buzsáki, 1989; McClelland *et al*., 1995; Squire & Alvarez, 1995; Frankland & Bontempi, 2005). According to this Standard Model of memory consolidation, progressively older memories are retained following HF lesions because they have moved from the HF to the neocortex. McClelland *et al*. (1995) proposed that a brain region, such as the HF, that rapidly encodes the essential associations that compose an experience, is needed to avoid the interference with preexisting long-term memories that would occur if an experience were rapidly encoded in the neocortex. According to this model of memory consolidation, during wakefulness, the HF receives input from the various neocortical regions involved in the initial encoding of the experience and binds this information into a coherent memory trace available for recall (Morris *et al*., 2003; Eichenbaum, 2004), and later reactivation or 'replay' in offline states, such as quiet wakefulness and SWS. During SWS, the coordinated reactivation of the memory trace in the HF and neocortex is thought to lead to a gradual strengthening of corticocortical connections between the neocortical modules involved in the memory trace and associated preexisting neocortical memory traces without interfering with the latter (e.g. Takashima *et al*., 2009). Finally, as the neocortical representation of the memory becomes progressively strengthened and integrated, the hippocampal involvement in the recall of the memory progressively declines. Interestingly, the progressive decline in hippocampal dependence is temporally associated with the recruitment and functional integration of new

neurons into the hippocampal network, a process which may contribute to the erasure of the hippocampal copy of the memory after it has been transferred to the neocortex, thereby enabling the hippocampus to acquire new information (Kitamura *et al*., 2009). Collectively, the Standard Model of memory consolidation both provides a solution to the problem of interference and accounts for the temporally graded nature of hippocampal involvement in memory recall.

Recent studies suggest that the PFC may play an integral role in the transfer, storage, and recall of memories initially stored in the HF (Fig. 6; Frankland & Bontempi, 2004;Wiltgen *et al*., 2004;Jung *et al*., 2008;Takehara-Nishiuchi & McNaughton, 2008). Several studies in rodents (Bontempi *et al*., 1999;Frankland *et al*., 2004;Maviel *et al*., 2004) and humans (Takashima *et al*., 2006;Gais *et al*., 2007; see also discussion in Takashima *et al*., 2009) have shown that the HF is activated during recall of recently formed memories, whereas the mPFC is activated during recall of remote memories. In addition, mPFC lesions impair recall of remote memories more than recent memories, whereas HF lesions impair recall of recent memories more than remote memories (Takehara, Kawahara & Kirino, 2003;Takehara-Nishiuchi *et al*., 2006). Similarly, blocking *N*-methyl-D-aspartate (NMDA) receptor activity or protein synthesis in the ventromedial PFC disrupts long-term, but not short-term memory (Akirav & Maroun, 2006). Moreover, in mice, dendritic spine growth following contextual fear learning increased first in the hippocampus and then weeks later in the mPFC, with the latter being dependent on the former, as expected if the memory was first encoded in the hippocampus and then transferred to the mPFC (Restivo *et al*., 2009). Finally, the mPFC may also inhibit activity in the hippocampus during recall of remote memories, thereby avoiding the re-encoding of remote information in the hippocampus (Fig. 6B;Bontempi *et al*., 1999;Frankland *et al*., 2004;Maviel *et al*., 2004; reviewed in Frankland & Bontempi, 2005).

Collectively, these findings have led to the suggestion that the mPFC is actively involved in storing and recalling long-term memories of information initially temporarily stored in the HF (Bontempi *et al*., 1999; Frankland & Bontempi, 2005; Jung *et al*., 2008). Indeed, given that the PFC may be involved in determining what memories should be recalled from other neocortical regions in a particular context (Tomita *et al*., 1999), it seems necessary that the PFC participates in the transfer of memories from the HF to long-term storage sites in the neocortex. In this manner, the PFC could also store a high-order representation of the memory in an index that could be used later to facilitate the recall of remote context-relevant information stored in other neocortical regions (Jung *et al*., 2008). Although the exact role of the PFC in establishing and recalling remote memories is still being determined, recent experimental evidence suggests that the high-order hippocampal representation of a memory may be transferred *via* direct projections from hippocampal CA1 neurons to the mPFC, which then orchestrates the transfer of the memory from the hippocampus to the neocortex *via* its influence over the intervening entorhinal and perirhinal cortices (Pelletier, Apergis & Paré, 2004; Paz, Bauer & Paré, 2007, 2009).

Before proceeding with a review of the sleep-related mechanisms thought to be involved in transferring memories from the hippocampus to the neocortex, it is necessary to acknowledge that the Standard Model of memory consolidation remains controversial. Notably, it has been argued that the temporally graded retrograde amnesia observed following hippocampal lesions in some, but not all, studies (reviewed in Winocur, Moscovitch & Bontempi, 2010) does not necessarily indicate that older memories are preserved because they were transferred out of the hippocampus before the lesions occurred. Instead, according to the Multiple Trace Theory of memory consolidation, it is thought that as a result of repeated reactivations of memories, multiple, widely distributed traces of a memory are formed over time within the hippocampus (Nadel & Moscovitch, 1997, 2001;

Moscovitch *et al*., 2006). As a result, if the hippocampal lesion is incomplete, there is a greater chance that old memories will have been copied into the remaining hippocampal network than new memories, a scenario that may account for the temporally graded retrograde amnesia observed following incomplete hippocampal lesions. Moreover, unlike the Standard Model, which does not distinguish between types of hippocampal memory, the Multiple Trace Model states that this explanation applies specifically to vivid, detailed episodic and autobiographical memories in humans and context-dependent and spatial memories in non-human animals, memories that are thought always to depend on the hippocampus. During recall of such hippocampal-dependent memories, the hippocampal memory trace is thought to serve as an index that binds the related neocortical components of a memory into a detailed recollection of the event, in a manner similar to that proposed for short-term memories according to the Standard Model.

Although the Multiple Trace Theory proposes that the initial, detailed, context-dependent representation of an event remains in the hippocampus, some hippocampal memories may be reproduced in the neocortex in a transformed form (reviewed in Winocur *et al*., 2010). Specifically, over time hippocampal memories may be reproduced, but in a schematic and generic, context-free version that simply retains the gist of the initial memory (Nadel & Moscovitch, 1997; Rosenbaum, Winocur & Moscovitch, 2001; Wiltgen *et al*., 2010). Hippocampal-neocortical interactions are thought gradually to form this new version of the memory within the neocortex (Winocur *et al*., 2005, Winocur, Moscovitch & Sekeres, 2007). According to this model, temporally graded retrograde amnesia following hippocampal lesions is thought to reflect, in part, the fact that the formation of the context-free memory in the neocortex is a gradual process, much in the same way that the transfer of complete hippocampal memories to the neocortex is thought to be a gradual process, according to the Standard Model.

A critical analysis of the relative merits and weaknesses of each memory consolidation model is beyond the scope of this review. Although this controversy remains unresolved (see Frankland & Bontempi, 2005; Moscovitch *et al*., 2006; Winocur *et al*., 2010), in the following discussion we focus on the Standard Model of memory consolidation because the interacting hippocampal and neocortical rhythms thought to be involved in transferring memories between the hippocampus and neocortex have usually been interpreted within the context of this model. Nonetheless, it is conceivable that the gradual formation in the neocortex of a schematic, context-free version of detailed, context-dependent hippocampal memories relies on the same interacting brain rhythms occurring during sleep.

Finally, in addition to being implicated in consolidating hippocampal memories, sleep also appears to play a role in consolidating memories of how to do things (i.e. procedural memory), such as typing on a keyboard (reviewed in Stickgold & Walker, 2007; Diekelmann & Born, 2010). However, this type of memory does not appear to involve the hippocampus and therefore is not the main focus of this review.

V. SLEEP-RELATED MEMORY CONSOLIDATION

(1) Slow-wave sleep

Consistent with the Standard Model of memory consolidation, during mammalian SWS, electrophysiological rhythms in the hippocampus and neocortex interact in a manner that may support the transfer of information from the hippocampus to the neocortex. As discussed above (see Section III.2*b*), CA3 neurons in the hippocampus initiate SWRs (Buzsáki, 1986; Sirota & Buzsáki, 2005). Although the SWRs can occur in the absence of neocortical input, the neocortical slow-oscillation, relayed *via* the entorhinal cortex, influences the timing of hippocampal SWRs such that they typically occur during the up-

state of the slow-oscillation, in close temporal association with neocortical spindles (Siapas & Wilson, 1998; Sirota *et al*., 2003; Battaglia, Sutherland & McNaughton, 2004; Isomura *et al*., 2006; Mölle *et al*., 2006, 2009; Clemens *et al*., 2007; Ji & Wilson, 2007; see also Dang-Vu *et al*., 2008; Wagner *et al*., 2010). On a fine temporal scale, individual ripple events tend to follow individual spindle waves, suggesting that neocortical input biases the occurrence of ripples to a specific phase of the spindle oscillation (Sirota *et al*., 2003; Isomura *et al*., 2006). In turn, hippocampal ripples may exert a feedback action on neocortical spindles, as spindling in the PFC increases following ripples (Mölle *et al*., 2009; Wierzynski *et al*., 2009). This feedback loop may provide the requisite communication needed for the transfer of information from the hippocampus to the neocortex. Although this relationship between hippocampal and neocortical activity has been observed in multiple neocortical areas, it may be particularly pronounced between the hippocampus and the mPFC (Gais *et al*., 2007; Mölle *et al*., 2009; Peyrache *et al*., 2009; Wierzynski *et al*., 2009; reviewed in Diekelmann & Born, 2010), the neocortical region where slow-oscillations are most prominent (Murphy *et al*., 2009). In support of this hypothesis, in addition to being reciprocally connected to the hippocampus *via* the entorhinal cortex and associated temporal lobe structures, as is much of the neocortex (Lavenex & Amaral, 2000), unlike the rest of the neocortex (Burwell & Witter, 2002) the mPFC receives monosynaptic excitatory projections from CA1 hippocampal neurons (Swanson, 1981; Jay & Witter, 1991; Thierry *et al*., 2000). Importantly, the mPFC exhibits long-term potentiation in response to stimulation of CA1 neurons at a frequency similar to that of ripples (Laroche, Jay & Thierry, 1990; Jay, Burette & Laroche, 1995; reviewed in Laroche, Davis & Jay, 2000; Jung *et al*., 2008), a requisite for the transfer of information.

In addition to the temporal coupling of hippocampal SWRs and neocortical slow-oscillations and spindles, neuronal recordings suggest that information may be transferred between the hippocampus and neocortex during this coordinated activity. Siapas & Wilson (1998) showed that firing of hippocampal neurons during SWRs preceded the firing of neurons in the mPFC, as would be expected if information flows from the hippocampus to the mPFC during SWRs (see also Peyrache *et al*., 2009; Wierzynski *et al*., 2009; Mölle & Born, 2009; Benchenane *et al*., 2010). Moreover, several studies have shown that the pattern of neuronal firing that occurred during prior active wakefulness is reactivated during SWS in both the hippocampus (Wilson & McNaughton, 1994; Skaggs & McNaughton, 1996; Kudrimoti, Barnes & McNaughton, 1999; Nádasdy *et al*., 1999; Hirase *et al*., 2001; Lee & Wilson, 2002; Pennartz *et al*., 2002; see also Pavlides & Winson, 1989) and neocortex (Hoffman & McNaughton, 2002), including the mPFC (Euston, Tatsuno & McNaughton, 2007; Peyrache *et al*., 2009; Johnson *et al*., 2010). Reactivation occurs preferentially during SWRs (Wilson & McNaughton, 1994; Kudrimoti *et al*., 1999; O'Neill, Senior & Csicsvari, 2006; Cheng & Frank, 2008), highly synchronous bursts of activity thought to be ideally suited for driving the output of information from the hippocampus to the neocortex (Sirota & Buzsáki, 2005; see also Wierzynski *et al*., 2009). Importantly, in studies that simultaneously recorded from the hippocampus and neocortex, the replay of the same waking neuronal firing sequence was coordinated between both brain regions (Ji & Wilson, 2007; see also Qin *et al*., 1997). Notably, in a recent study, Peyrache *et al*. (2009) demonstrated that once rats learned a new rule, SWS-related hippocampal SWRs became correlated with the reactivation of rule-related waking neuronal firing patterns in the mPFC, suggesting a specific link between such coordinated activity and learning. In a follow-up study, Benchenane *et al*. (2010) found that theta coherence between the hippocampus and mPFC during wakefulness increased when rats performing a spatial task learned a new rule. During subsequent SWS, neuronal assemblies activated during such periods of high coherence were reactived following hippocampal SWRs. Importantly, in both studies reactivation in the mPFC occurred approximately 30 – 60 ms after SWRs, a finding consistent with other studies showing that SWS-related SWRs can drive neuronal activity in the mPFC (Wierzynski *et al*., 2009),

presumably *via* the direct excitatory projection from CA1 to the mPFC. Thus, although neocortical slow-oscillations can influence the occurrence of SWRs, the resulting SWRs may in turn influence reactivation in the mPFC. Finally, recent studies have also shown that disrupting SWRs impairs memory consolidation, possibly through disrupting the initial stages of memory consolidation in the hippocampus or interfering with the transfer of information to the neocortex (Girardeau *et al*., 2009; Nakashiba *et al*., 2009; Ego-Stengel & Wilson, 2010).

Although it is clear that coordinated reactivation occurs between the hippocampus and neocortex during SWS, it remains unclear how this phenomenon is involved in strengthening neocortical memories. It has been proposed that neocortical spindling occurring concurrently with ripple-related hippocampal-neocortical reactivation might strengthen the neocortical memory trace through inducing conditions favourable for long-term potentiation (Contreras *et al*., 1997; Siapas & Wilson, 1998; Sejnowski & Destexhe, 2000; Sirota *et al*., 2003; Steriade & Timofeev, 2003; Frankland & Bontempi, 2005; Rosanova & Ulrich, 2005; Sirota & Buzsáki, 2005; Destexhe *et al*., 2007; Marshall & Born, 2007). Consistent with a role in memory consolidation, in rats, both ripple and spindle activity increased during SWS occurring immediately after performance of hippocampal-dependent learning tasks (Eschenko *et al*., 2008; Mölle *et al*., 2009; see also Johnson *et al*., 2010), and, importantly, the increase in the density of both ripples and spindles (Eschenko *et al*., 2006; Ramadan, Eschenko & Sara, 2009) correlated with post-sleep improvements in task performance. In humans, stage 2 sleep spindles also increased following performance of hippocampal-dependent task (word pair learning), and as in rats this increase correlated with improved performance (Gais *et al*., 2002; Schabus *et al*., 2004; Schmidt *et al*., 2006). In a similar study of humans that examined the temporal relationship between spindles and the slow oscillation, the increase in spindles occurred primarily during the up-state of slow-oscillations occurring during sleep stages 2–4 (Mölle *et al*., 2009). Increases in stage 2 sleep spindles occurring after training on procedural learning tasks also correlate with improved performance (Nishida & Walker, 2007; Tamaki *et al*., 2008; see also Fogel & Smith, 2006; Rasch *et al*., 2009). Finally, slow (0.75 Hz) transcranial direct current stimulation of the PFC during early non-REM sleep in humans increased slow-oscillations and spindling in the PFC, as well as performance on a hippocampal-dependent task (Marshall *et al*., 2006; see also Massimini *et al*., 2007), possibly *via* slow-oscillations in the PFC driving greater hippocampal transfer.

However, a positive relationship between spindles and memory consolidation has not been found in all studies. Sleep-related enhancements in performance on a visual discrimination task correlated positively with non-REM sleep (stages 2–4) SWA, but negatively with power in the spindle range, in both an experiemntal group who received acoustic stimulation to reduce SWA and an unstimulated control group (Aeschbach, Cutler & Ronda, 2008). Similarly, performance decrements on a visuomotor task following deprivation of slow-waves by acoustic stimulation correlated with changes in SWA, rather than spindles (Landsness *et al*., 2009). Interestingly, both studies implicate SWA in enhancing performance on these tasks, rather than spindles as suggested above for hippocampal memories. However, this difference does not appear to be related to differences in the memory systems activated in the respective studies, because a recent study also found positive and negative relationships between stage 2-related SWA and spindles, respectively, and performance on a hippocampal-dependent spatial navigation task in humans (Wamsley *et al*., 2010). The reasons for these contradictory results remain unclear. Although the evidence implicating spindles in memory consolidation remains equivocal, much of the available data on the relationship between hippocampal and other neocortical rhythms are consistent with the hypothesis that these rhythms are involved in transferring information from temporary stores in the hippocampus to the neocortex. Moreover, emerging data

suggest that this transfer may selectively involve direct projections from the hippocampus to the mPFC.

Finally, in addition to the studies focused on brain rhythms implicated in memory transfer, functional magnetic resonance imaging (fMRI) studies in humans also suggest that non-REM sleep plays a role in processing information in the HF and transferring it to the PFC. For instance, hippocampal activation during non-REM sleep (primarily stages 3–4) occurring after training on a hippocampal-dependent task correlates with post-sleep performance improvements on the task (Peigneux *et al*., 2004). In another study, hippocampal activation induced during stages 3–4 sleep by presenting an odour previously presented during training on a hippocampal-dependent task, increased post-sleep performance on the task, an effect that may have been mediated by the reactivation of task-related memories in the hippocampus (Rasch *et al*., 2007). In both studies, hippocampal activation may reflect the processing of information within the hippocampus and/or transfer of information out of the hippocampus. Other studies suggest that sleep after training on hippocampal-dependent tasks may be necessary for initiating consolidation processes ultimately leading to memory transfer from the HF to the PFC. Takashima *et al*. (2006) found that subjects who engaged in more stage 3–4 sleep during a nap following training on a hippocampal-dependent task, showed less hippocampal activation during post-nap successful recall. This result is consistent with the notion that memories were transferred out of the hippocampus during post-training stage 3–4 sleep. Furthermore, during successful recall, hippocampal activation decreased and mPFC activation increased over the next 90 days. In another fMRI study, Gais *et al*. (2007) showed that a period of post-learning sleep enhances hippocampal activation during recall two days later. In addition, post-learning sleep increased functional connectivity between the hippocampus and mPFC at this time point. Interestingly, six months later, activation of the mPFC during recall depended on post-learning sleep occurring six months earlier. Similarly, Sterpenich *et al*. (2007) found that activation of the hippocampus and mPFC was higher during successful recall of pictures 72 h post-encoding in subjects who were allowed to sleep following encoding when compared to subjects that were sleep-deprived on the first post-encoding night. Activation of the hippocampus and mPFC in the sleep group suggests that post-encoding sleep is involved in initiating the process of transferring memories from the hippocampus to the mPFC. Collectively, these imaging studies in humans, and the studies on sleep-related brain rhythms outlined above are consistent with the notion that sleep occurring after learning is involved in transferring information initially stored in the hippocampus to long-term stores in the PFC.

(2) Rapid eye movement sleep

In addition to SWS, hippocampal activity occurring during mammalian REM sleep may also be involved in processing memories. Current theories postulate that the REM-sleep-related theta rhythm is involved in memory consolidation in mammals (Dragoi *et al*., 1999; Poe *et al*., 2000; Stickgold & Walker, 2007; Montgomery, Sirota & Buzsáki, 2008; Nishida *et al*., 2009). Poe *et al*. (2000) provide experimental evidence consistent with the notion that the replay of waking neuronal activity at specific phases of the theta rhythm during REM sleep may be involved in strengthening recent memories and erasing old memories in the hippocampus. In another study, Montgomery *et al*. (2008) found that the characteristics of the REM-sleep-related theta rhythm differ between wakefulness, and phasic (periods with eye movements and twitching) and tonic (periods without eye movements and twitching) REM sleep, possibly reflecting different modes of information processing. Hippocampal theta and gamma synchrony in the dentate gyrus/CA3 area during tonic REM sleep was more pronounced when compared to wakefulness while running. In contrast to tonic REM sleep, during phasic REM sleep, synchrony between theta and gamma increased among the

dentate gyrus, CA3, and CA1 areas. The authors suggest that these changes may reflect alternations between an internal mode of hippocampal processing during tonic REM sleep and a synchronous output mode for communicating with neocortical targets during phasic REM sleep.

Several studies indicate that the theta rhythm is involved in coordinating activity between the hippocampus and mPFC during wakefulness and REM sleep. During wakefulness, mPFC neurons exhibit phase precession relative to the hippocampal theta rhythm (Jones & Wilson, 2005*a*), suggesting a functional link between these structures. Sirota *et al*. (2008) demonstrated that gamma oscillations in the mPFC and other neocortical areas were phase locked with the hippocampal theta rhythm during wakefulness and REM sleep. During wakefulness, phase locking of neuronal firing between the hippocampus and mPFC (Siapas, Lubenov & Wilson, 2005) increased during performance of a spatial task, suggesting that the coordinated activity between the hippocampus and mPFC is involved in spatial working memory (Jones & Wilson, 2005*b*; see also Hyman *et al*., 2005; Wang & Cai, 2006). Similarly, in a genetic mouse model of schizophrenia, reduced hippocampal – mPFC theta synchrony was associated with reduced performance on a spatial working memory task (Sigurdsson *et al*., 2010). Recently, Benchenane *et al*. (2010) found that hippocampal – mPFC theta coherence during wakefulness (apparently driven by the hippocampus) increased and the phase relationship changed in response to learning new rules on a spatial task. During REM sleep, Popa *et al*. (2010) found that the degree of theta coherence between the hippocampus, mPFC, and basolateral amygdala following Pavlovian fear conditioning influenced fear memory in rats. Collectively, these studies suggest that the hippocampal theta rhythm may have a role in processing information within the hippocampus and coordinating processing between the hippocampus and mPFC, as well as other structures, during wakefulness and REM sleep. Whether such coordinated activity is functionally different during wakefulness and REM sleep is unknown.

(3) Reactivation during wakefulness

Hippocampal SWRs occurring during SWS and theta rhythms occurring during REM sleep are thought to be involved in processing hippocampal memories. However, both of these phenomena also occur during wakefulness – SWRs during quiet wakefulness and theta rhythms during active wakefulness. This raises the question as to whether such activity occurring during sleep is functionally different from that occurring during wakefulness. Indeed, several studies have shown that reactivation of place cell firing sequences occurs during SWRs observed during brief pauses in exploring rats, as well as during SWS (Foster & Wilson, 2006; Jackson, Johnson & Redish, 2006; O'Neill *et al*., 2006; Csicsvari *et al*., 2007; Diba & Buzsáki, 2007; Davidson, Kloosterman & Wilson, 2009; Karlsson & Frank, 2009; Axmacher *et al*., 2009; Gupta *et al*., 2010). Reactivation during such pauses in behaviour can occur in a forward order, as in sleep, as well as in reverse order (Csicsvari *et al*., 2007; Diba & Buzsáki, 2007; Davidson *et al*., 2009; Foster & Wilson, 2006; Gupta *et al*., 2010). In addition, a recent study of rats suggests that place cells also fire during SWRs occurring during wakefulness in a sequence corresponding to available, but not previously traveled paths, suggesting that SWRs may also be involved in the learning and maintenance of a cognitive map (Gupta *et al*., 2010). These findings question whether SWS is simply an offline state particularly conducive to, but not necessary for SWR-mediated memory consolidation in mammals. However, given that SWS-related SWRs occur in conjunction with neocortical slow-oscillations and thalamocortical spindles (unlike SWRs occurring during wakefulness), quantitative or qualitative differences may exist. Consistent with this scenario, Peyrache *et al*. (2009) report that reactivation in the mPFC is associated with SWRs occurring during SWS, but not wakefulness. Dupret *et al*. (2010) also found that SWR-related CA1 place cell reactivation occurring during SWS, but not during extended

periods of quiet wakefulness, after training on a spatial memory task correlated with subsequent performance on the task. Interestingly, SWRs occurring during brief (<2.4 s) pauses in exploratory behaviour during training also correlated with subsequent performance. Although additional research is needed, collectively these studies suggest that SWR-related hippocampal reactivation occurring during pauses in exploratory behaviour may be involved in the early phases of memory consolidation within the hippocampus, whereas that occurring during SWS (but not extended periods of quiet wakefulness) may be involved in coordinating processing between the hippocampus and neocortex.

VI. AVIAN MEMORY TRANSFER?

Like mammals, at least some birds have the capacity to form long-term memories for large amounts of information (Balda & Kamil, 1992; Mettke-Hofmann & Gwinner, 2003; Fagot & Cook, 2006). The following section summarizes the evidence for hippocampal involvement in various types of avian learning and memory, including spatial memory, imprinting (filial and sexual), song learning, and passive avoidance learning (reviewed in Sherry, 2006; Chiandetti *et al*., 2007). Although it is evident that the avian hippocampus is involved in processing spatial memories, conclusive evidence for the transfer of this information to extra-hippocampal regions for long-term storage is lacking. The absence of such evidence is consistent with the apparent absence of the neural pathway and rhythms implicated in this process in mammals.

(1) Spatial memory

(a) Homing in pigeons—Homing pigeons are well known for their ability to find their way home when displaced from their loft. The mechanisms that pigeons use to home depend in part on whether the birds are released from familiar or unfamiliar sites. Debate persists over the type of information that pigeons use to home from distant, unfamiliar sites, but it may include olfactory and geophysical (magnetic and sun) information (reviewed in Wallraff, 2005; Gagliardo *et al*., 2009; Jorge, Marques & Phillips, 2009; Mehlhorn & Rehkämper, 2009; Wiltschko & Wiltschko, 2010). In contrast to homing from unfamiliar sites, recent experiments using global positioning system (GPS) devices have shown that when homing across familiar areas near their loft, pigeons navigate *via* visual landmarks (Biro, Meade & Guilford, 2004; Lipp *et al*., 2004; Gagliardo *et al*., 2007, 2009). Although the HF is not necessary for navigating from distant, unfamiliar sites, several studies suggest that it is involved in homing across familiar landscapes. Lesions to the hippocampus and/or APH (see Bingman & Mench, 1990) impair the ability of pigeons to home using familiar landmarks (Bingman *et al*., 1984; Bingman & Mench, 1990; Gagliardo, Ioalé & Bingman, 1999; Gagliardo *et al*., 2009), suggesting that the HF is either involved in storing a relational representation of space or necessary for the readout of such memories from other brain regions.

In addition to lesion studies, the structures involved in homing across familiar landscapes have also been examined by measuring the expression of immediate early genes (IEGs), such as *Zenk* (also known as *Zif-268*, *Erg-1*, *NGFI-A*, and *Krox-24*) and *c-fos*, or their respective protein products, ZENK and FOS. IEGs are expressed in neurons in response to activation, the first step in a cellular cascade involved in plasticity (reviewed in Clayton, 2000). IEG activity has been used extensively to identify brain regions activated during certain behaviours and cognitive tasks. In an IEG experiment, homing across a familiar landscape induced ZENK expression in the APH, but not in the hippocampus (Shimizu *et al*., 2004). The avian APH is thought to serve a similar function as the mammalian entorhinal cortex, the major conduit for information exchange between the hippocampus and neocortex (Atoji & Wild, 2006). Interestingly, the entorhinal cortex is also activated during performance of a spatial task in humans (Aguirre *et al*., 1996; Maguire *et al*., 1998).

Consequently, activation of the APH during homing across a familiar landscape may reflect the readout of relevant spatial memories from the hippocampus (or APH) and/or the processing of new spatial information.

The only region other than the APH that was activated during homing across a familiar landscape was the medial portion of the medial striatum (mMSt) (Shimizu *et al*., 2004), a region homologous with portions of the mammalian striatum (Reiner *et al*., 2004). Interestingly, the MSt appears to be involved in other forms of learning (see Section VI. 4), such as passive avoidance learning in chicks (Rose & Stewart, 1999) and song learning in songbirds (Doupe & Konishi, 1991; Luo, Ding & Perkel, 2001), raising the possibility that components of the memory of the landscape are stored in the mMSt in homing pigeons. Nonetheless, the disruption of homing across familiar landscapes resulting from HF lesions indicates that the mMSt is not sufficient for homing. Moreover, given that the MSt is implicated in the anticipation of reward in chicks (Izawa *et al*., 2003), activation of the mMSt during homing may reflect motivational processes related to getting home, rather than the readout of memories required to home.

Aside from these lesion and IEG studies, little else is known about where pigeons store spatial information related to homing. Notably absent in the IEG study was increased activation in other pallial regions, such as the NCL – the proposed avian analogue of the mammalian PFC – during homing across a familiar landscape (Shimizu *et al*., 2004). Although it is possible that other IEGs may reveal activation in the NCL (or other regions), the ZENK data suggest that unlike the APH, the NCL is not directly involved in processing spatial information during homing across familiar areas. Nonetheless, ventral portions of the NCL and adjacent pyriform cortex may be involved in processing olfactory information used to navigate during homing from unfamiliar sites (Papi & Casini, 1990; Gagliardo & Divac, 1993; Riters & Bingman, 1999; reviewed in Bingman *et al*., 1998). Thus the APH and NCL may process different types of information related to different phases of homing behaviour.

Finally, to our knowledge there are no data to suggest that the HF temporarily stores homing-related information for later transfer to the NCL (or other brain regions) for long-term storage, as proposed in mammals. Perhaps the only information bearing on this topic is the finding that HF lesions occurring five to six weeks after the onset of training to home across a familiar landscape impair homing performance (Bingman *et al*., 1987). This suggests that storage and/or retrieval of information necessary for homing across familiar landscapes is dependent on the HF for up to six weeks. In conjunction with reports of the absence of direct projections between the avian HF and NCL (Leutgeb *et al*., 1996; Kröner & Güntürkün, 1999; Atoji *et al*., 2002; Atoji & Wild, 2006) and the absence of activation in the NCL during homing (Shimizu *et al*., 2004), this finding suggests that homing-related spatial information is not transferred out of the hippocampus for long-term storage in the NCL, at least under the time frame examined. Certainly, additional studies are needed to determine if such a transfer occurs over a longer time scale, and if so, whether it depends on sleep, as suggested for mammals.

(b) Food caching—Birds in the passerine families Paridae (chickadees and tits), Corvidae (jays and crows), and Sittidae (nuthatches) store food in widely distributed locations for periods of hours, days, or even many months (reviewed in Vander Wall, 1990; Balda & Kamil, 1992). Several studies indicate that the hippocampus is involved in storing spatial information needed to retrieve these caches (Krushinskaya, 1966; Sherry & Vaccarino, 1989; Hampton & Shettleworth, 1996*a*,*b*; Shiflett *et al*., 2003; Smulders, 2006). Krushinskaya (1966, as cited in Smulders, 2006) found that Eurasian nutcrackers (*Nucifraga caryocatactes*) with lesions to the dorsal-medial telencephalon, including the hippocampus,

continued to cache and search for food, but were unable to relocate their caches if more than 2 h elapsed from when the food was hidden. This suggests that the hippocampus is involved in forming long-term spatial memories. Interestingly, the lesioned birds were able to relocate food if it was cached only 10 – 20 min earlier. Although this suggests that the hippocampus is not necessary for forming short-term spatial memories, it is nonetheless possible that intact nutcrackers normally rely on the hippocampus to relocate caches over short time spans, but rely on alternative strategies when the hippocampus is lesioned. As in nutcrackers, lesions of the hippocampus in black-capped chickadees (*Poecile atricapillus*) selectively impair the ability to relocate food cached after the lesion using spatial, but not colour information (Sherry & Vaccarino, 1989; Hampton & Shettleworth, 1996*b*). In contrast to the study on nutcrackers, however, chickadees were only tested 3 h after caching. Consequently, it is not known whether lesioned chickadees are also able to recover more recently cached food, as observed in nutcrackers. More recently, Shiflett *et al*. (2003) reversibly inactivated the hippocampus with lidocaine either 15 min or 3 h after black-capped chickadees were trained on a spatial memory test. Whereas hippocampal inactivation occurring 15 min after training impaired performance, inactivation occurring 3 h after training did not affect performance. In contrast to the study on nutcrackers, this experiment suggests that the hippocampus is involved in the early stages of forming spatial memories, and that memories temporarily stored in the hippocampus may have been transferred out of the hippocampus sometime between 15 min and 3 h after training. However, as the authors acknowledge, given that the lesions may have only partially inactivated the hippocampus, it is possible that early stages of memory processing were more susceptible to the lesions than later forms of hippocampal processing. As a result, the spatial memory may have remained in the hippocampus. Consistent with this explanation, retrieval of previously cached food items occurring 24 h after caching is associated with the upregulation of ZENK and FOS in the hippocampus of black-capped chickadees (Smulders & DeVoogd, 2000). This suggests that the hippocampus is involved in storing and/or retrieving spatial memories related to caching for at least 24 h.

Recent studies of black-capped chickadees have examined the role of two receptors implicated in the formation of long-term spatial memories in mammals. Although blocking the NMDA receptor (NMDA-R) in the hippocampus during the learning phase did not affect memory when tested 15 min later, it did interfere with memory when tested 180 min or 24 h later (Shiflett *et al*., 2004*b*). The effect was only evident when the memory task required that the bird incorporate new information within a preexisting spatial memory. This suggests that the NMDA-R in the hippocampus is involved in modifying long-term spatial memories. However, as noted by Shiflett *et al*. (2004*b*), it is unclear whether this process involves the interaction between the hippocampus and other telencephalic structures or synaptic changes occurring only within the hippocampus. In another study of chickadees, Shiflett *et al*. (2004*a*) showed that cannabinoid receptors (CB1-R) in the hippocampus are also involved in forming long-term spatial memories. Chickadees that received infusions of the CB1-R antagonist on the dorsal surface of the hippocampus just before training on a spatial memory task made fewer errors during retrieval 72 h later. Although this counterintuitive result suggests that normal activation of the CB1-R has an adverse effect on long-term memory, a subsequent experiment showed that blockade of the CB1-R results in previously acquired spatial information interfering with the subsequent acquisition of additional spatial information, a potentially significant problem for birds that sequentially cache and retrieve food items from different locations. Shiflett *et al*. (2004*a*) thus suggest that the CB1-R in the hippocampus may modulate long-term memory formation in a manner that prevents proactive interference. As with the study on the role of hippocampal NMDA-R in long-term memory formation, it is unclear whether the hippocampal CB1-R influences the formation of long-term memories within the hippocampus, or the manner in which the hippocampus

interacts with other telencephalic regions during the formation of potential extra-hippocampal long-term memories.

(c) Spatial memory in finches—As in pigeons and food-caching birds, a recent study suggests that the retrieval of spatial memories also depends on the hippocampus in zebra finches (Watanabe & Bischof, 2004; see also Patel, Clayton & Krebs, 1997; Bailey, Wade & SaIdanha, 2009). In finches that had their hippocampus lesioned after completing four days of training on a spatial task, performance dropped to pretraining levels when retested two days later. By contrast, control finches that were anaesthetized, but not lesioned, continued to perform at the high level attained prior to the lesion. Whether such memories are encoded exclusively within the hippocampus or simply depend on the hippocampus for their readout from other telencephalic regions is unknown.

Collectively, the available data on processing spatial memories in homing pigeons, food-caching birds, and zebra finches do not provide evidence for the transfer of hippocampal information to extra-hippocampal structures for long-term storage, as proposed in mammals.

(2) Imprinting

(a) Filial imprinting—Shortly after hatching, young precocial birds approach conspicuous objects in their environment. When tested later, they show a preference for the objects with which they have become familiar. Under natural conditions, such imprinting results in young birds following their parents (Lorenz, 1937). In the laboratory, the neural mechanisms underlying this form of learning have been examined under controlled conditions by exposing chicks (*Gallus gallus domesticus*) to visual and acoustic imprinting stimuli (reviewed in Horn, 2004). Given that the hippocampus has not been implicated in auditory filial imprinting (Long *et al*., 2002; Bock *et al*., 2005; Thode *et al*., 2005), the following section focuses on filial imprinting to visual stimuli.

Visual imprinting in chicks leads to increased activation, as measured by immunocytochemistry for FOS, in the hippocampus and intermediate and medial mesopallium (IMM), raising the possibility that components of imprinting-related memories could be temporarily stored in the hippocampus and then transferred to the IMM and other sites for long-term storage. Although the hippocampus projects to the IMM in chicks (Bradley, Davies & Horn, 1985), several lines of evidence argue against the hippocampus serving as a temporary memory store during imprinting. Although the number of immunopositive neurons for FOS increases rapidly in the hippocampus and IMM during imprinting, only in the IMM is it correlated with learning (McCabe & Horn, 1994; Suge & McCabe, 2004). Furthermore, although neuronal responsiveness to visual stimulation increases in the hippocampus and IMM following exposure to an imprinting stimulus, only in the IMM is it specifically related to this stimulus (Brown & Horn, 1994; Nicol, Brown & Horn, 1995, 1998). Given these findings, Suge & McCabe (2004) concluded that the hippocampus does not store information specific to filial imprinting; hippocampal activation may simply reflect the parallel encoding of information related to the spatial aspects of the experience. Finally, Nakamori *et al*. (2010) recently demonstrated that a neural circuit between the core region of the hyperpallium densocellulare (HDCo), periventricular region of the hyperpallium densocellular (HDPe), and IMM is specifically involved in visual imprinting in chicks. Importantly, projections to the hippocampus from the HDCo or projections from the hippocampus to the HDCo were not detected, suggesting that the hippocampus is not directly involved in this imprinting circuit.

Despite the lack of evidence for the transfer of imprinting memories from the hippocampus to the IMM, lesion studies indicate that initial memories formed in the IMM during imprinting are transferred to another site. Although memory of the imprinting stimulus is

dependent on the left and right IMM until 4 – 6 h after training, neither structure is required after this time window (Cipolla-Neto, Horn & McCabe, 1982; Honey *et al*., 1995). This suggests that the essential aspects of the imprinting memory are transferred to a long-term storage site other than the IMM, although the exact location of this site (referred to as S') is unknown. Interestingly, although correlates of long-term storage, such as increased postsynaptic densities (Bradley, Horn & Bateson, 1981; Horn, Bradley & McCabe, 1985) and NMDA-type glutamate receptors (McCabe & Horn, 1988, 1991; Horn, 2004), are only observed in the left IMM, the right IMM is required for the formation of the long-term memory in S' (Cipolla-Neto *et al*., 1982). Given that the transfer of the memory from the right IMM to S' takes place over a period of several hours, it is possible that it depends on sleep occurring during this period, as suggested for the transfer of memories from temporary stores in the hippocampus to long-term stores in the neocortex in mammals. Although the potential role of sleep in this transfer has not been investigated, as discussed later (see Section VII.2*a*), sleep nonetheless appears to play a role in imprinting.

(b) Sexual imprinting—In addition to filial imprinting, the hippocampus has been implicated in sexual imprinting, a two-stage process whereby young birds learn about prospective sexual partners (Immelmann *et al*., 1991; Bischof, 2003; Sadananda & Bischof, 2004). The first phase involves acquiring species-specific information from the young bird's mother, father, and siblings. Information acquired during this initial phase is later refined and consolidated during the second phase when sexually mature birds have their first courtship encounter, resulting in an irreversible preference. A variety of methods have been used to measure regional brain activation in response to sexual imprinting, including ^{14}C-2-deoxyglucose autoradiography and immunocytochemistry or *in situ* hybridization for the expression of IEGs *c-fos* and *Zenk* or the presence of their protein products. Although the different methods have revealed different results in some cases (see Lieshoff, Grosse-Ophoff & Bischof, 2004), regions showing increased activation in male zebra finches in response to their first exposure to a female include the hyperpallium accessorium/mesopallium dorsale (HAMD), arcopallium-nidopallium caudale (ANC), medial nido-mesopallium (MNM), and lateral nido-mesopallium (LNM) (Bischof & Herrmann, 1986, 1988; Sadananda & Bischof, 2002, 2006; Lieshoff *et al*., 2004). Sexual imprinting is associated with a reversible increase in spine densities of neurons in HAMD and ANC, and an irreversible decrease in spine density in MNM and LNM (Bischof & Rollenhagen, 1999), changes that may result from long-term potentiation in HAMD and ANC, and long-term depression in MNM and LNM (Rollenhagen & Bischof, 1998). The decrease in spine density in MNM and LNM seems to be specifically related to the consolidation of imprinting memories, whereas the increase in spine density in HAMD and ANC may reflect activation in response to the complexity of the social environment, in general (Rollenhagen & Bischof, 2000; Lieshoff & Bischoff, 2003; Bischof, 2003).

In male finches, the hippocampus is also activated in response to the first exposure to a female (Sadananda & Bischof, 2004). Although the co-activation of the hippocampus and the other regions might indicate that components of the imprinting memory are first processed in the hippocampus and then transferred to other regions (Sadananda & Bischof, 2004), the hippocampus may simply be involved in processing the spatial aspects of the experience, as suggested by work on filial imprinting and song learning (see below). It is unknown whether the memory of the spatial aspects of the sexual imprinting experience is transferred to other regions or remains in the hippocampus.

(3) Song learning

Song learning and production in songbirds involves a distinct network of forebrain nuclei not found in non-songbirds (Gahr, 2000; reviewed in Bolhuis & Gahr, 2006). In young

males, song learning involves memorizing the song of an adult male tutor, which later serves as the template towards which the bird adjusts its own song during development. Several converging lines of evidence indicate that the tutor song is stored (at least in part) in the caudal part of the medial nidopallium (NCM), an auditory association region of the avian pallium, perhaps homologous to the mammalian auditory association cortex (reviewed in Bolhuis & Gahr, 2006; Pinaud & Terleph, 2008). In male zebra finches, although neuronal activation increases in the NCM and the caudal part of the medial mesopallium (CMM) in response to hearing the song of a conspecific, it only correlates with the fidelity of song imitation in the NCM (Bolhuis *et al*., 2000, 2001; Terpstra, Bolhuis & den Boer-Visser, 2004). Similarly, in an electrophysiological study of adult male finches, the degree to which individual neurons in the NCM responded selectively to the tutor song heard earlier during development also correlated with the fidelity of song imitation (Phan, Pytte & Vicario, 2006). Moreover, lesions to the NCM performed in adult male finches impaired their ability to recognize their tutor's song, but not their ability to imitate this song (Gobes & Bolhuis, 2007). Collectively, these studies suggest that the NCM is a necessary neural substrate for long-term memory of the tutor song.

Interestingly, although the NCM is not connected directly to the hippocampus (Bailey *et al*., 2009), some studies have found increased expression of IEGs in the hippocampus following exposure to the song (Bailey, Rosebush & Wade, 2002; Bailey & Wade, 2003, 2005) or call (Vignal, Mathevon & Mottin, 2008; Gobes *et al*., 2009) of a conspecific, raising the possibility that the hippocampus is also involved in the perception of vocalizations and song learning. However, this response has not been found in all studies, and the reasons for the discrepancies remain unresolved (reviewed in Bailey & Wade, 2005; Bolhuis & Gahr, 2006; Bailey *et al*., 2009). For instance, Terpstra *et al*. (2004) found low numbers of immunopositive neurons for ZENK in the hippocampus of adult male finches exposed to the tutor's song, the bird's own song, or that of a novel conspecific. Moreover, unlike the NCM following exposure to the tutor song, where ZENK levels were higher and correlated with the degree of similarity between the bird's song and that of its tutor, a similar relationship was not found in the hippocampus. Furthermore, a recent lesion study suggests that the hippocampus is not directly involved in song learning. Bailey *et al*. (2009) examined the impact of hippocampal lesions on song development and spatial memory in zebra finches. As in previous studies, hippocampal lesions impaired performance on a spatial memory task (Patel *et al*., 1997; Watanabe & Bischof, 2004). However, the birds with hippocampal lesions showed no decrement in various measures of song learning. Combined, these results suggest that the hippocampus is not involved in song learning *per se*. Instead, the activation of the hippocampus observed in some studies may reflect the parallel processing of spatial information specific to the context of those studies (Kruse, Stripling & Clayton, 2004).

(4) Passive avoidance learning

The passive avoidance learning (PAL) task takes advantage of the fact that one-day-old chicks readily peck at and learn about small conspicuous objects in their environment (reviewed in Rose, 2000). In this task, chicks are presented with a coloured bead that has been dipped in methylanthranilate, a noxious-tasting liquid. Chicks that peck at this bead show behavioural signs of disgust (e.g. backing away, head shaking, and bill wiping), and will avoid similarly coloured beads dipped in water after just one experience with the distasteful bead. This task has proved to be a powerful tool for investigating the molecular, structural, and electrophysiological components of learning.

Several studies have investigated the role of the hippocampus and other structures in PAL. In a study of chicks, left, but not right hippocampal lesions performed prior to training impaired subsequent performance on the passive avoidance task. However, bilateral lesions occurring 1 h after acquisition did not affect PAL when the chicks were tested 21 h after the

lesion, possibly because the memory no longer depended on the hippocampus (Sandi, Rose & Patterson, 1992). Although this suggests that the left hippocampus may temporarily (< 1 h) store information related to PAL, it is also possible that it simply serves as a relay for some essential task-related information, and not as a temporary storage site *per se*. Interestingly, although hippocampal lesions occurring 1 h after PAL do not impair performance, synaptic density is reduced in the right hippocampus 6 and 24 h following training (Nikolakopoulou, Davies & Stewart, 2006*a*; see also Ünal *et al*., 2002). As noted by Nikolakopoulou *et al*. (2006*a*), it is unclear whether these changes in the hippocampus are related to learning or stress associated with the task. Along these lines, using bromo-2-deoxyuridine (BrdU), Nikolakopoulou *et al*. (2006*b*) found reduced cell proliferation in the dorsal hippocampus and APH 24 h after PAL. Given that cortisol concenntration was elevated in the hippocampus 20 min after training in this study, the reduction in cell proliferation may also be attributable to stress (Nikolakopoulou *et al*., 2006*b*). Regardless, the finding that chicks that received hippocampal lesions 1 h after training showed PAL when tested 21 h later (Sandi *et al*., 1992), suggests that the changes in synaptic density and cell proliferation observed in the hippocampus are not specifically related to forming memories of the aversive stimulus.

Instead, an extensive body of research indicates that PAL primarily involves the intermediate and medial mesopallium (IMM) and medial striatum (MSt) (reviewed in Rose & Stewart, 1999; Rose, 2000). PAL induces a cascade of molecular events (reviewed in Rose, 2000) that result in increased spine density in the left, and to a lesser extent, right IMM (Patel & Stewart, 1988) and the left and right MSt (Stewart, Csillag & Rose, 1987; Lowndes & Stewart, 1994) 24 h later. Recent studies have also shown increased BrdU labeling for new neurons in the IMM, MSt, and olfactory tubercle nine days after PAL (Dermon *et al*., 2002). These molecular and structural changes seem to be specifically related to PAL, as disrupting the molecular cascade at various points results in amnesia for the previously aversive stimulus (Rose, 2000). Patel, Rose & Stewart (1988) also showed that the increase in spine density in the left IMM is related to learning the passive avoidance task. Finally, lesion studies have shown that these structures are involved in various phases of PAL (Rose, 2000). Although these studies suggest that the memory trace may “flow” first from the left IMM to the right IMM and then from the right IMM to the left and right MSt (reviewed in Rose, 1991), subsequent investigations suggest that the process may be even more complex with different components of the memory becoming distributed in the left IMM and MSt (Patterson & Rose, 1992; Rose, 2000; see also Barber *et al*., 1999). Whether sleep is involved in this system level of memory consolidation is unknown.

(5) Section summary

Collectively, the available studies do not provide conclusive evidence for the transfer of memories from short-term stores in the hippocampus to extra-hippocampal long-term stores in birds. Although the hippocampus is clearly involved in forming spatial memories, conspicuously missing from this body of research is evidence showing the progressive disengagement of the hippocampus and engagement of the NCL (or other extra-hippocampal regions) during recall of progressively older memories, as shown for the mammalian hippocampus and PFC (Bontempi *et al*., 1999; Frankland *et al*., 2004; Maviel *et al*., 2004; Takashima *et al*., 2006; Gais *et al*., 2007; Kitamura *et al*., 2009). The limited data on hippocampal memory processing in birds are consistent with the apparent absence of sleep-related brain rhythms implicated in the transfer of memories from the hippocampus to the neocortex in mammals, as well as the apparent absence of a hippocampal – ‘PFC’ pathway.

VII. SLOW-WAVE SLEEP FUNCTIONS IN MAMMALS AND BIRDS

(1) Shared function?

Interestingly, the absence of evidence in birds for (1) SWRs and spindles, rhythms implicated in transferring hippocampal memories to the PFC in mammals, (2) connections between the HF and NCL, and (3) the transfer of memories from the HF to the NCL (or other extra-hippocampal sites) for long-term storage, may have direct bearing on our understanding of the function of mammalian SWS. If birds exhibited SWRs and spindles without a pathway for transferring hippocampal memories to the NCL, or evidence for the transfer of hippocampal memories, but no SWRs or spindles, this would pose a challenge to the hypothesis that these rhythms are involved in coordinating the transfer of hippocampal information to the PFC for long-term storage in mammals (Frankland & Bontempi, 2005; Takashima *et al*., 2006; Euston *et al*., 2007; Gais *et al*., 2007; Mölle *et al*., 2009). Instead, the apparent differences between birds and mammals in hippocampal – 'PFC' interactions and some SWS-related brain rhythms, lend comparative support to this functional hypothesis for mammalian SWS; lacking a pathway for transferring hippocampal memories to the NCL, birds have no need for SWRs and spindles. Nonetheless, it also suggests that such rhythms reflect mammal-specific aspects of SWS whereas sleep traits shared by birds and mammals may reflect more fundamental aspects of SWS. Notably, the defining feature of mammalian and avian SWS – the synchronous slow-oscillation of neuronal membrane potentials – may serve a function independent from that related to coordinating the transfer of information from the hippocampus to the neocortex in mammals (Fig. 7). Given that SWS-related SWA is homeostatically regulated in both mammals and birds (Tobler, 2005; Rattenborg *et al*., 2009), this process is likely tied to a fundamental function of SWS (Benington, 2000).

The 'synaptic homeostasis hypothesis' provides a mechanistic and functional explanation for SWS homeostasis in mammals and birds. According to this hypothesis, the slow-oscillation maintains synaptic weight (strength and number) in the neocortex at an adaptive level, by reducing synaptic weight accumulated during prior wakefulness (Tononi & Cirelli, 2003, 2006; Massimini, Tononi & Huber, 2009). During wakefulness, engagement with the environment is thought to cause long-term potentiation and a concomitant increase in synaptic strength and number throughout the neocortex (Vyazovskiy *et al*., 2008; Liu *et al*., 2010). If left unchecked, this process would saturate the neocortex with synapses, and thereby impede the ability to acquire new information. The increase in the strength and number of synapses would also increase the amount of energy required to operate the neocortex. During SWS, however, the slow synchronous firing of neocortical neurons (enhanced by the increase in synaptic strength that accumulated during wakefulness) is thought to cause long-term depression and synaptic down-scaling (i.e. a proportional decrease in synaptic weight), thereby preserving the relative strength of synapses, conserving energy, and preparing the neocortex for the acquisition of additional information. Synaptic down-scaling is also thought to cause enhancements in performance by removing weak, newly formed synapses, and thereby increasing the signal-to-noise ratio in neural circuits preferentially potentiated during prior wakefulness (Hill, Tononi & Ghilardi, 2008).

In addition to being supported by several lines of evidence in mammals (Huber *et al*., 2004, 2006; Tononi & Cirelli, 2006; Faraguna *et al*., 2008; Vyazovskiy *et al*., 2008, 2009*a*,*b*; Hanlon *et al*., 2009; Landsness *et al*., 2009; Liu *et al*., 2010; but see Aton *et al*., 2009; Greene & Frank, in press) and emerging evidence in birds (Jones *et al*., 2008*a*; Rattenborg *et al*., 2009; see also Rauske *et al*., 2010), a strength of the synaptic homeostasis hypothesis is that it provides an explicit mechanism that accounts for the homeostatic regulation of SWS-related SWA in mammals and birds. Specifically, as a result of synaptic potentiation occurring during wakefulness, the level of corticocortical (or palliopallial) connectivity is

thought to be the highest at the end of an extended period of wakefulness. As a result, neurons alternate between up- and down-states most synchronously during early SWS, resulting in the highest SWA (Vyazovskiy *et al*., 2009*b*). As sleep continues, synaptic down-scaling, mediated by the slow-oscillation, reduces corticocortical connectivity, neuronal synchrony, SWA, and thereby the efficacy of further down-scaling in a self-limiting manner.

Given that the slow-oscillation and associated EEG SWA have only been described in sleeping mammals and birds, this mechanism for down-scaling may have evolved independently in these groups (Rattenborg *et al*., 2009). Nonetheless, synaptic down-scaling may also occur during sleep in animals apparently lacking slow-oscillations *via* other, as yet unidentified mechanisms. For instance, although synchronous, slow-oscillations have not been detected during sleep in fruit flies (*Drosophila melanogaster*) (Nitz *et al*., 2002; van Swinderen & Andretic, 2003), two recent studies suggest that down-scaling occurs during sleep in *Drosophila melanogaster* (Donlea, Ramanan & Shaw, 2009; Gilestro, Tononi & Cirelli, 2009). Assuming that slow-oscillations have not simply evaded detection in *Drosophila melanogaster* and other non-mammalian/avian taxa, this finding raises the question of why mammals and birds should need a different mechanism to achieve down-scaling. Given that mammals and birds independently evolved large brains capable of complex cognition (Jarvis *et al*., 2005; Rattenborg *et al*., 2009), perhaps such brains require a different mechanism to achieve synaptic down-scaling than that exhibited by *Drosophila melanogaster*. Alternatively, or in addition, components of the slow-oscillation, such as the up-state, with its wake-like pattern of neuronal activity may be involved in other sleep-related functions.

(2) Avian sleep-dependent memory consolidation?

Our review of the available evidence suggests that avian sleep is not involved in transferring memories from short-term stores in the hippocampus to long-term stores in the NCL, or other telencephalic regions. Nonetheless, this does not necessarily mean that sleep is not involved in processing memories in birds. Indeed, as reviewed below (see Sections VII. 2*a*,*b*), sleep has been implicated in two types of avian developmental learning – filial imprinting and song learning – neither of which involves the hippocampus (see Sections VI. 2*a* and 3). In addition, a recent study in European starlings (*Sturnus vulgaris*) suggests that sleep may also play a role in processing memories in adult birds.

(a) Filial imprinting—Sleep appears to be involved in at least some aspects of processing imprinting memories. Horn, Nicol & Brown (2001) tracked neurons in the left and right IMM over a period of 25 h, and found that the proportion of neurons that responded selectively to a visual imprinting stimulus (IS) increased markedly between 8 and 25 h following the first exposure to the IS. Given this temporal pattern, they speculated that sleep occurring during this time might have played a role in increasing responsiveness to the IS. Subsequently, Jackson et al. (2008) assessed the impact of sleep on the number of neurons in the IMM that responded to a compound visual/auditory IS, by sleep-depriving chicks for 7.5 h starting either immediately following training or 7.5 h after training; chicks in the first group were allowed to rest during the second 7.5 h and chicks in the second group were allowed to rest during the first 7.5 h. Responsiveness to the IS was assessed after completing the 15-h post-training period. If sleep occurring immediately after training is involved in processing imprinting memories, chicks that were allowed to rest first should show more neurons responsive to the IS when compared to those that were kept awake first. As predicted, chicks that were allowed to rest first showed significantly more IS-responsive neurons, compared to those in which the opportunity to rest was delayed by 7.5 h. Although the amount of time spent in wakefulness, SWS and REM sleep was not quantified directly during the deprivation or rest periods, in both groups of chicks the EEG recorded from the

IMM showed greater low-frequency (0 – 6 Hz) power during the rest period when compared to the sleep-deprived period. If such EEG activity in the IMM is associated with SWS, as is the case for recordings from the dorsal surface of the avian pallium, then these results suggest that SWS was more prevalent during the rest periods, and therefore may have played a role in processing imprinting memories. However, given that REM sleep also increases following imprinting in chicks (Solodkin, Cardona & Corsi-Cabrera, 1985), it is also possible that post-training REM sleep contributed to the increase in neuronal responsiveness to the IS. Although the precise role played by each sleep state and associated EEG activity remains unclear, this study nonetheless suggests that sleep is involved in processing imprinting memories, and thereby establishes the imprinting paradigm as a powerful method for revealing the link between sleep and memory (Stickgold, 2008). Given the link between SWS and the transfer of hippocampal memories to the neocortex in mammals, it will be interesting to determine whether SWS plays a similar role in transferring imprinting memories between the right IMM and S' (see Section VI.2*a*). Furthermore, if SWS is involved in establishing memories in S', it would be interesting to know if similar mechanisms, such as coordinated replay occurs between the right IMM and S' during sleep. Unfortunately, uncertainty over the location of S' currently precludes this approach.

(b) Song learning—While the hippocampus does not appear to be directly involved in song learning, sleep nonetheless seems to be involved in this process (reviewed in Margoliash & Schmidt, in press). Following exposure to the song of a tutor, juvenile male zebra finches gradually learn to produce a similar song. During song development, the complexity of the song varies daily, declining across the night when finches sleep the most, and increasing during the day in response to singing (Derégnaucourt *et al*., 2005; Crandall *et al*., 2007; Day *et al*., 2009; Shank & Margoliash, 2009). Although this sleep-related decline in song complexity seemingly suggests that sleep is detrimental to song learning, the extent of this daily sleep-related degradation and wake-related restoration in song complexity actually predicts how well the finch ultimately mimics the song of its tutor.

Although the mechanisms through which sleep facilitates song learning remain unclear, activity in several nuclei has been implicated in this process. Spontaneous *Zenk* expression occurring during sleep in the lateral NCM, a region involved in storing the memory of the tutor song (see Section VI.3), correlates with song learning in juvenile finches (Gobes, Zanderbergen & Bolhuis, 2010). In adult male zebra finches, neuronal firing patterns that occurred during singing are reactivated during subsequent sleep in the robust nucleus of the arcopallium (RA), the motor output nucleus involved in controlling song production (Dave & Margoliash, 2000). Reactivation may also occur in the HVC (proper name), the nucleus in the song motor circuit that projects to nucleus RA (see Gobes *et al*., 2010). In adult birds, reactivation in nucleus RA may carry sensory information that could be used to maintain an accurate rendition of the tutor song (Margoliash & Schmidt, in press; see also Rauske *et al*., 2010). In juvenile finches, nucleus RA activity during SWS and REM sleep increases following the first exposure to the tutor song, even before the bird has had a chance to sing (Shank & Margoliash, 2009). This activation may carry sensory information about the tutor song that initiates plastic changes contributing to song learning (Margoliash & Schmidt, in press).

(c) Sleep-dependent memory consolidation in adult birds—In a behavioural study, Brawn, Nusbaum & Margoliash (2010) examined performance on an auditory discrimination task in adult starlings following retention intervals primarily containing either sleep or wakefulness. When tested following a period of daytime wakefulness, mean performance on the task showed a small, non-significant, decrease. By contrast, when tested following periods that encompassed the night, the main sleep period in starlings (Szymczak, 1985), performance increased significantly. The increase in performance following a night

of sleep did not appear to reflect inherent circadian influences on auditory discrimination, as time of day did not influence performance during training. Although sleep was not recorded electrophysiologically in this study, the results nonetheless suggest that, as in mammals, sleep is involved in memory consolidation in adult birds. The mechanisms and brain regions involved in this process are unknown.

(3) Sleep-dependent processing of hippocampal memories in birds?

The finding that birds apparently lack sleep-related rhythms implicated in the transfer of information from the hippocampus to the neocortex in mammals does not necessarily mean that avian sleep has no role in processing hippocampal-dependent memories. Indeed, albeit not involving the hippocampus (see Section VII.2*a*), sleep appears to be involved in the consolidation of imprinting memories in chicks, song learning in finches, and auditory discrimination in starlings. Consequently, it is conceivable that sleep also plays a role in processing hippocampal information in birds. For instance, if neuronal replay occurs within the hippocampus during avian sleep, it may strengthen hippocampal memories, even if those memories are not transferred out of the hippocampus. Sleep may also facilitate hippocampal memories through other mechanisms that do not involve replay or the transfer of memories to other regions. For instance, a common problem for mammals and birds may be that the hippocampus becomes saturated during prolonged wakefulness, thereby impeding its ability to encode additional information (Yoo *et al*., 2007; see also Van der Werf *et al*., 2009). According to the Standard Model of memory consolidation, the transfer of memories to the neocortex during SWS solves this problem for mammals (Lubenov & Siapas, 2008). However, if it is true that memories remain in the avian hippocampus, saturation may be a significant problem for birds, especially those known for their hippocampal-dependent spatial abilities, such as food-caching birds, which remember the location of hundreds to thousands of hidden food items (Roth, Rattenborg & Pravosudov, 2010). As in the neocortex, it is conceivable that sleep-related synaptic down-scaling rectifies this problem in the hippocampus as well (Huber, 2007; Axmacher *et al*., 2009).

VIII. EVOLUTION OF SLEEP-RELATED HIPPOCAMPAL ACTIVITY

The apparent differences between hippocampal activity in mammals and birds raise the question as to which state is ancestral among amniotes. Interestingly, the limited data available from reptiles suggest that the common amniote ancestor to mammals and reptiles (including birds) exhibited sleep-related hippocampal activity in some respects similar to that observed in mammals (reviewed in Hartse, 1994; Rattenborg 2007). During sleep, and to a lesser extent wakefulness, the reptilian medial cortex – the developmental homologue of the mammalian and avian hippocampus – generates distinct high-amplitude, sharp-waves that share several characteristics in common with mammalian hippocampal sharp-waves. The sharp-waves in both groups increase following sleep deprivation and respond similarly to pharmacological agents (Hartse & Rechtschaffen, 1982; Gaztelu García-Austt & Bullock, 1991; Hartse, 1994; Lorenzo, Macadar & Velluti, 1999; Lorenzo & Velluti, 2004; Rattenborg, 2007). Unlike mammalian sharp-waves, however, reptilian sharp-waves do not occur in conjunction with slow-oscillations (as measured by the EEG) in the dorsal cortex, an area thought to be homologous to neocortical primary visual and somatosensory areas (Medina & Reiner, 2000; Medina & Abellán, 2009). Instead, sharp-waves originating in the medial cortex propagate to the dorsal cortex (Gaztelu *et al*., 1991; Lorenzo *et al*., 1999), where they are the dominant EEG feature during sleep. Given this and other differences (e.g. ripples have not been described in reptiles), it is unclear whether reptilian sharp-waves are involved in the same functions as those proposed for sharp-waves in mammals. For instance, although the medial cortex has been implicated in processing spatial information in reptiles and fish (Rodríguez *et al*., 2002), to our knowledge it is unknown whether place cells exist in the reptilian medial cortex, let alone whether such cells replay their waking firing patterns

during sharp-waves in sleep. Moreover, a mammal-like theta rhythm has not been detected in the medial cortex of reptiles (Gaztelu *et al*., 1991). Despite these gaps in our knowledge of comparative hippocampal neurophysiology, it is nonetheless possible that reptilian-like sharp-waves were present in the common ancestor to extant amniotes (reptiles, birds, and mammals), and served as the neural substrate from which components of SWRs evolved in mammals. If this scenario is correct, then instead of elaborating on this initial state, as did mammals, birds apparently dispensed with hippocampal sharp-waves, and evolved other means of processing spatial information in the hippocampus.

The reported differences in hippocampal activity and connectivity observed between mammals and birds may be related to the fact the PFC and NCL are analogous structures that originate from different pallial regions. In amniotes, developmental gene expression studies have shown that the pallium develops as four distinct highly conserved embryonic fields: medial, dorsal, lateral, and ventral pallia (Medina & Abellán, 2009). The medial pallial field gives rise to the mammalian and avian hippocampus, and the reptilian medial cortex, structures involved in processing spatial information. In mammals, the PFC and other neocortical regions medial to the temporal sulcus (i.e. superior cortex), or perhaps the entire neocortex, including the temporal neocortex (Aboitiz, Morales & Montiel, 2003), develop from the dorsal pallial field, which also gives rise to the three-layered dorsal cortex in reptiles and the pseudolayered hyperpallium (or *Wulst*) in birds (Medina & Reiner, 2000). As with the medial pallium, although the derivatives of the dorsal pallium are homologous as a developmental field and, in part, perform similar functions (i.e. processing visual and somatosensory information), the dorsal pallium develops into distinct structures in each group. In mammals, the dorsal pallium expands radially and tangentially into the large, six-layered neocortex, whereas in birds it develops into the pseudolayered hyperpallium with some convergent mammal-like traits not found in reptiles, such as neurons involved in corticocortical (or palliopallial) interconnectivity (Medina & Reiner, 2000; Suárez *et al*., 2006; Medina & Abellán, 2009). Although the hyperpallium, dorsal cortex and superior cortex, including the PFC, evolved from the dorsal pallium – the pallial region immediately lateral to the medial pallium (hippocampus) – the NCL evolved from the ventral pallium, the pallial region most distant from the medial pallium (Puelles *et al*., 2007; Abellán *et al*., 2009). In sauropsids (reptiles and birds), the ventral and lateral pallia form a largely nuclear structure called the dorsoventricular ridge (DVR), a controversial brain region that may be homologous to portions of the mammalian temporal cortex (Karten, 1997; Reiner, 1993; Butler, 1994), the pallial amygdala (Bruce & Neary, 1995), the pallial claustroamygdalar complex (Striedter, 1997; Puelles *et al*., 2000; Martínez-García, Martínez-Marcos & Lanuza (2002), or a combination of the temporal cortex and claustroamygdalar complex as a developmental field (Molnár & Butler, 2002; reviewed in Yamamoto & Reiner, 2007; Aboitiz & Montiel, 2007; Puelles *et al*., 2007; Medina & Abellán, 2009; Abellán *et al*., 2009; Wang, Brzozowska-Prechtl & Karten, 2010). In birds, the DVR is divided into the mesopallium and nidopallium, which originate from the lateral and ventral pallia, respectively. Although it is unclear which components of the reptilian DVR are homologous to the avian nidopallium and mesopallium, in both groups, the DVR is composed of primary and secondary sensory regions and association regions (Martínez-García & Lanuza, 2009). Interestingly, few if any connections have been detected between the reptilian DVR and medial cortex (Ulinski, 1983, 1990; Hoogland & Vermeulen-Vanderzee, 1993; Ten Donkelaar, 1998; Manns & Eichenbaum, 2007), a pattern similar to that observed between the NCL and hippocampus. Thus, unlike the PFC, the NCL apparently evolved from a brain region without direct connectivity to the hippocampus. This may explain, in part, why mammals and birds seem to differ in the manner in which the hippocampus interacts with other pallial regions during both wakefulness and sleep.

Why did a 'PFC'-like structure evolve in different pallial regions in mammals and birds? This may be related to the type of brain that birds inherited from their ancestors. The dorsal cortex of extant reptiles does not appear to include a region similar to the mammalian PFC (Reiner, 1986). However, the DVR is developed in all extant reptiles, albeit to a lesser degree than in birds, suggesting that expansion of the DVR (rather than the cortex, as in mammals) apparently started in sauropsids before birds evolved from theropod dinosaurs. As a result, instead of developing a new PFC-like structure in the dorsal pallium, birds may have elaborated on the preexisting DVR. Consistent with this scenario, fossil edocasts of the avian cranial cavity indicate that for unknown reasons, the avian dorsal pallium (hyperpallium, or *Wulst*) expanded during the Cenozoic, well after the expansion of the DVR in the Mesozoic (Milner & Walsh, 2009), and therefore perhaps after the associated evolution of the PFC-like NCL in the DVR. Given that the DVR may have already been dedicated to performing PFC-like functions, presumably there was no need for the later expansion of the avian dorsal pallium to include the development of a PFC-like structure (but see Wild & Williams, 2000; Shimizu, Patton & Husband, 2010). Even though the avian dorsal pallium did not evolve into a PFC-like structure, the avian hippocampus (medial pallium) nonetheless retained its close association with the dorsal pallium, as evidenced by the extensive reciprocal connections between the hippocampus and hyperpallium. If correct, this evolutionary scenario undoubtedly contributed to the apparent differences between how birds and mammals process and store information.

Other factors may have simultaneously favoured the resulting avian brain organization. For instance, disconnected from a mammal-like role in communicating with a PFC-like structure, the avian hippocampus may have been free to follow a somewhat different evolutionary trajectory from that followed in mammals; perhaps one more suited for processing spatial information during flight (Treves *et al*., 2008). Regardless of the answers to these questions, perhaps the most interesting finding is that, despite the differences between mammals and birds in hippocampal – 'PFC' interactions, the two groups independently evolved remarkably similar cognitive abilities (Emery & Clayton, 2005; Jarvis *et al*., 2005; Kirsch *et al*., 2008) and otherwise similar sleep states.

IX. AVIAN HIPPOCAMPAL AND 'PREFRONTAL CORTEX' PROCESSING DURING WAKEFULNESS

In mammals, it is thought that the HF rapidly encodes and temporarily stores highly associational information until it can be transferred to the neocortex during SWS, thereby avoiding the problem of interference that would occur if new information were rapidly encoded directly within preexisting neocortical memories (McClelland *et al*., 1995). The mammalian hippocampus receives highly processed unimodal and multimodal information from widespread neocortical regions through its connections with the entorhinal, perirhinal, and parahippocampal/postrhinal cortices (Lavenex & Amaral, 2000). Although the avian HF is interconnected with the visual hyperpallium, the developmental homologue of the neocortex, unlike the neocortex, the hyperpallium is not involved in forming high-level multimodal associations. Instead, the NCL and other parts of the DVR, such as the mesopallium, perform this function in birds. Interestingly, as with the NCL, the mesopallium does not appear to project directly to the HF or CDL (Atoji *et al*., 2002; Atoji & Wild, 2005). Thus, unlike the mammalian HF, the avian HF does not appear to receive highly associational multimodal information. Consistent with this apparent difference in connectivity, unlike mammals (Bunsey & Eichenbaum, 1996), performance on highly associational tasks, such as transitive inference, does not depend on the HF in birds (Strasser, Ehrlinger & Bingman, 2004). Consequently, the avian HF does not appear to be ideally suited for serving as a temporary storage site for highly associational information.

If the avian HF is not involved in processing and temporarily storing highly associational information, then perhaps these functions are handled by another brain region. As the recipient of multimodal secondary sensory information, the NCL is seemingly the prime candidate. However, interference may still be a problem. In mammals, it is thought that the PFC stores an index of memories dispersed throughout the neocortex that can be used to recall context-relevant information (Jung *et al*., 2008). Presumably, this index is gradually transferred from the HF to the PFC through the repeated reactivation of a similar index temporarily stored in the hippocampus. Given that the avian NCL is involved in several functions similar to those performed by the PFC (working memory and executive control), the NCL may also require an index to recall context-relevant memories. But if the NCL performs this function and/or serves as a long-term storage site itself, then, assuming that the avian brain is faced with the same interference problem that the HF is thought to resolve in mammals (McClelland *et al*., 1995), how do birds rapidly encode new associational information in the NCL without interfering with preexisting memories? Interference could be avoided if certain subregions of the NCL are involved in rapidly encoding information during wakefulness, while others are involved in integrating this information into preexisting memory traces, perhaps during offline states, such as quiet wakefulness or sleep. Alternatively, birds may have evolved an as yet unidentified mechanism to circumvent the interference problem altogether.

X. CONCLUSIONS

1. Despite exhibiting mammal-like homeostatically regulated SWS (Rattenborg *et al*., 2009), a hippocampus, and a brain region analogous to the PFC, birds appear to lack the neural pathway and brain rhythms implicated in transferring information from the hippocampus to the PFC during mammalian SWS. In conjunction with the lack of conclusive evidence for the transfer of information from the hippocampus to extra-hippocampal regions, this suggests that the transfer of memories from the hippocampus to the PFC reflects a mammal-specific function of SWS. Moreover, it suggests that the slow-oscillation, the defining feature of SWS shared by mammals and birds, may serve a more fundamental function than that related to transferring hippocampal memories in mammals.
2. The slow-oscillation may serve a function independent from that related to its influence over hippocampal rhythms and neocortical spindling in mammals. For instance, given that SWS-related SWA, the EEG correlate of the slow-oscillation, is homeostatically regulated in birds and mammals, functional hypotheses that explicitly account for this process, such as the ‘synaptic homeostasis hypothesis’ (Tononi & Cirelli, 2006), may apply to both taxonomic groups (Rattenborg *et al*., 2009).
3. Determining if and how functional theories linked to SWS homeostasis in mammals and birds integrate with the proposed mammal-specific function of transferring memories from the hippocampus to the PFC is a significant challenge for future sleep research, especially given that the slow-oscillation is thought to cause long-term depression (according to the synaptic homeostasis hypothesis) and long-term potentiation (*via* its coordinating influence over hippocampal ripples and thalamocortical spindles) in the same network (Tononi & Cirelli, 2006; Diekelman & Born, 2010).
4. In mammals, SWRs occurring during quiet wakefulness and SWS, and the theta rhythm occurring during active wakefulness and REM sleep are thought to reflect complimentary, reciprocal modes of hippocampal processing. Interestingly, in addition to apparently lacking SWRs during quiet wakefulness and SWS, birds also

apparently lack a hippocampal theta rhythm during active wakefulness and REM sleep. Given that both modes of hippocampal activity are seemingly missing in birds, the manner in which the avian hippocampus processes information may be fundamentally different from that in mammals during wakefulness, SWS, and REM sleep.

5. The apparent absence of a mammal-like theta rhythm during avian REM sleep suggests that brain activation, the shared feature of mammalian and avian REM sleep, may be the most fundamental feature of this state, much in the same way as the slow-oscillation may be the most fundamental feature of mammalian and avian SWS. Brain activation may play a role in memory consolidation through reactivating information acquired during wakefulness (Pavlides & Winson, 1989; Maquet *et al*., 2000; Louie & Wilson, 2001; Riberio *et al*., 2002; Peigneux *et al*., 2003) and/or processing changes in memory traces initiated during prior SWS (Riberio *et al*., 2002, 2007; Tononi & Cirelli, 2003; Diekelmann & Born, 2010).

6. Ultimately, teasing apart the relative contributions of the diverse sleep-related brain rhythms to memory consolidation and other proposed sleep functions is a promising approach for revealing sleep mechanisms and functions in mammals, as well as more general aspects of sleep. As illustrated in this review, research on birds may be particularly informative because they seem to have independently evolved several, but, importantly, not all aspects of mammalian SWS and REM sleep. The dissociation between certain sleep states and certain brain rhythms observed in birds may prove to be particularly informative when attempting to isolate the functions of these rhythms in mammals.

7. Finally, we would like to emphasize that the apparent absence of a mammal-like hippocampal – 'PFC' dialogue during avian sleep and wakefulness should not be viewed as indicating that the bird brain is somehow 'rudimentary' when compared to the mammalian brain. Although future studies may reveal differences in avian and mammalian cognition linked to the differences in hippocampal - 'PFC' interactions discussed herein, the available evidence instead suggests that birds independently evolved a brain capable of performing complex cognitive tasks similar to those performed by the mammalian brain. This suggests that complex cognition can be achieved *via* alternative types of brain organization (Jarvis *et al*., 2005; Kirsch *et al*., 2008). In this respect, in addition to informing our understanding of sleep, comparative studies of birds and mammals may reveal overriding principles of brain organization that mediate complex cognition.

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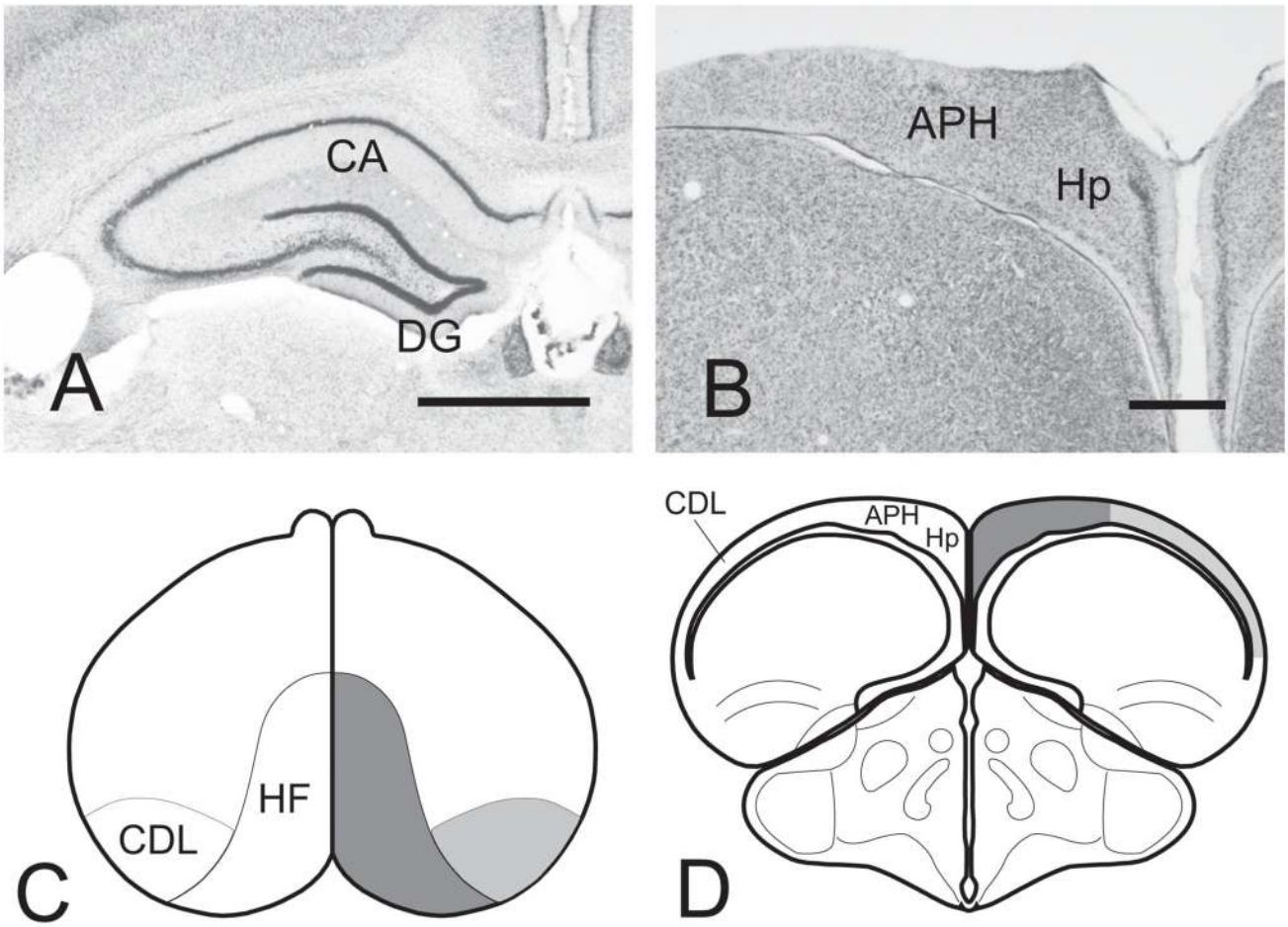

Fig. 1.

(A, B) Histological comparison of the hippocampal formation between the rat and pigeon. (A) Layers of the dentage gyrus (DG) and cornu ammonis (CA) are conspicuous in the rat. (B) The V-shaped layer is the only readily apparent structure in the pigeon hippocampus (Hp). APH: area parahippocampalis. (C, D) Location and extent of the pigeon hippocampal formation (HF, dark gray) and dorsolateral corticoid area (CDL, light grey). (C) Dorsal view. (D) Transverse section. Scale bars in A, B = 1 mm. Reproduced with permission from Atoji & Wild (2007).

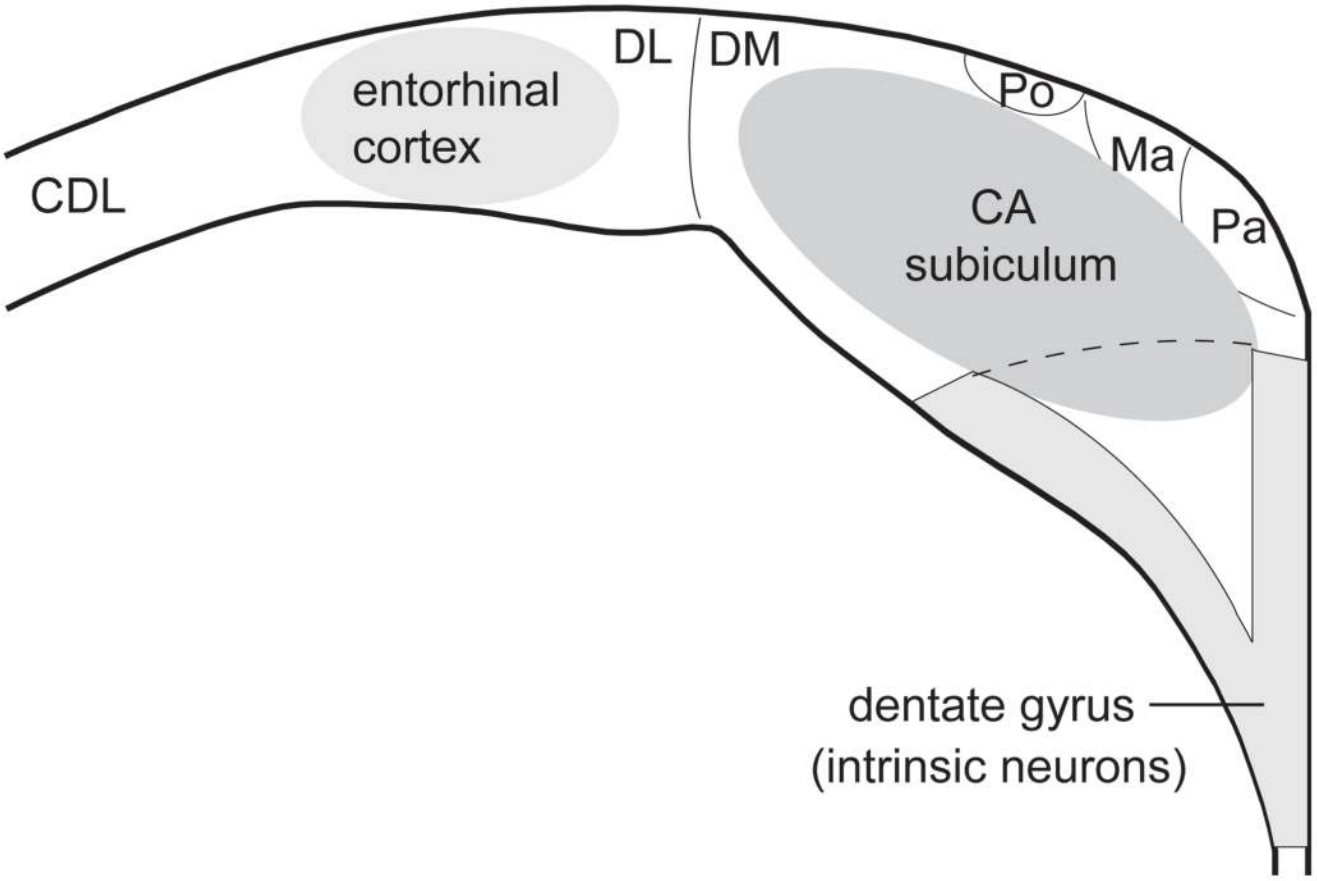

Fig. 2.

Hypothesized homology of hippocampal formation (HF) subregions between mammals and birds. The medioventral V-shaped layer (shaded light grey) is comparable to the mammalian dentate gyrus, the dorsomedial region (DM) to cornu ammonis (CA) and the subiculum, and the dorsolateral region (DL) to the entorhinal cortex. Other histologically identified regions include the magnocellular region (Ma), the parvocellular region (Pa), and the cell-poor region (Po). CDL: dorsolateral corticoid area. Reproduced with permission from Atoji & Wild (2007).

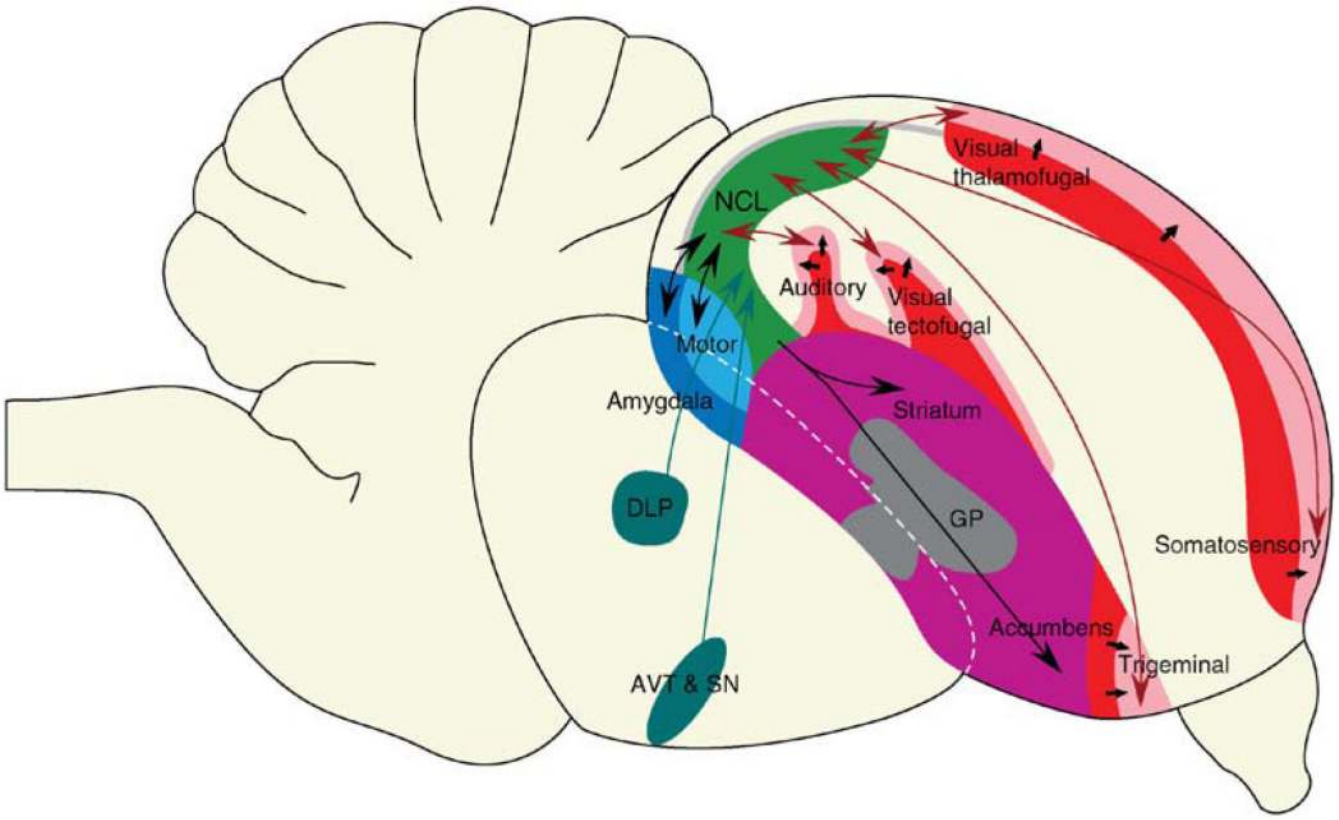

Fig. 3.

Afferent and efferent connections of the caudolateral nidopallium (NCL). Primary sensory areas are depicted in red, secondary and tertiary areas in pink. The primary sensory areas project to secondary and tertiary structures (small black arrows), which have reciprocal connections with the NCL (red arrows). The visual thalamofugal and tectofugal systems correspond to the geniculocortical and colliculo-pulvino-extrastriate systems of mammals, respectively. The area labeled "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). GP: globus pallidus. Reproduced with permission from Springer Science+Business Media: *Encyclopedia of Neuroscience,* Evolution of association pallial areas: in birds, 2009, pp. 1215–1219, Figure 2, Rose, Güntürkün & Kirsch.

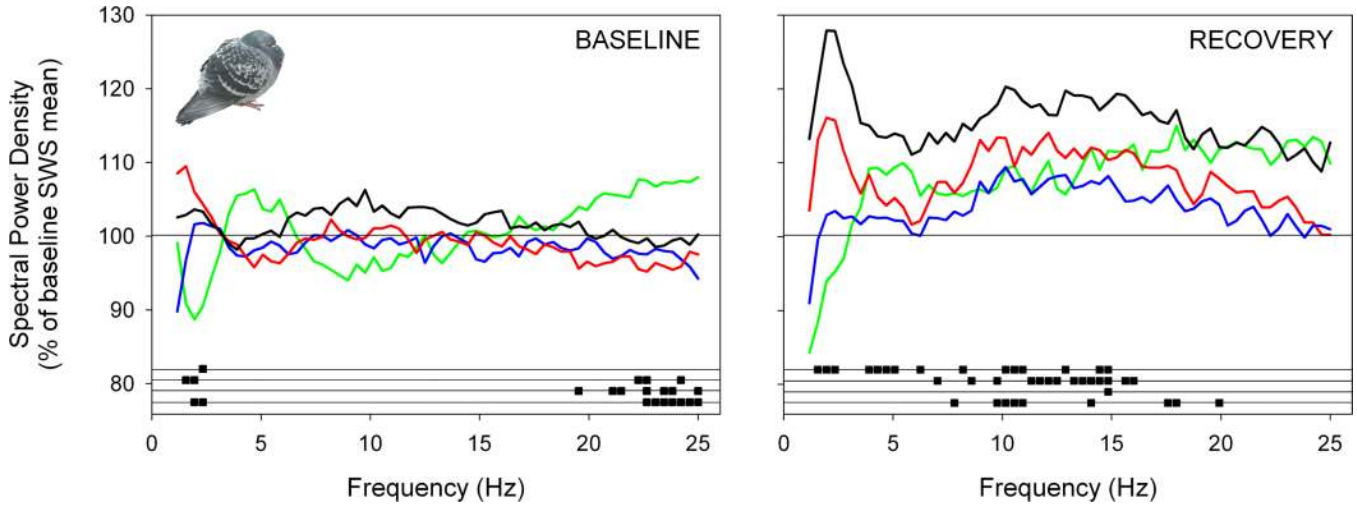

Fig. 4.

Mammalian-like slow-wave sleep (SWS) homeostasis in the pigeon (*Columba livia*). Spectral power density during SWS for the four 3-h quarters (black, first; red, second; blue, third; green, fourth) of the baseline night and the recovery night following 8 h of daytime sleep deprivation. The data presented is from bipolar electroencephalograms of the right anterior pallium. Black squares at the bottom of each plot reflect statistical significance ($P < 0.05$) for the first quarter (top row) to the fourth quarter (bottom row). Significance in the baseline plot reflects the comparison of power density at a specific frequency bin for that quarter of the baseline night to the all-night baseline SWS mean. In the recovery plot, significance reflects the comparison of power density at a specific frequency bin for that quarter of the recovery night to the corresponding frequency bin and quarter of the baseline night. Power increased most markedly in the low-frequency range (< 5.0 Hz) during the first quarter of the recovery night and progressively declined thereafter, a response indicative of mammalian-like SWS homeostasis. Modified from Martinez-Gonzalez *et al*. (2008). Pigeon image: Niels Rattenborg.

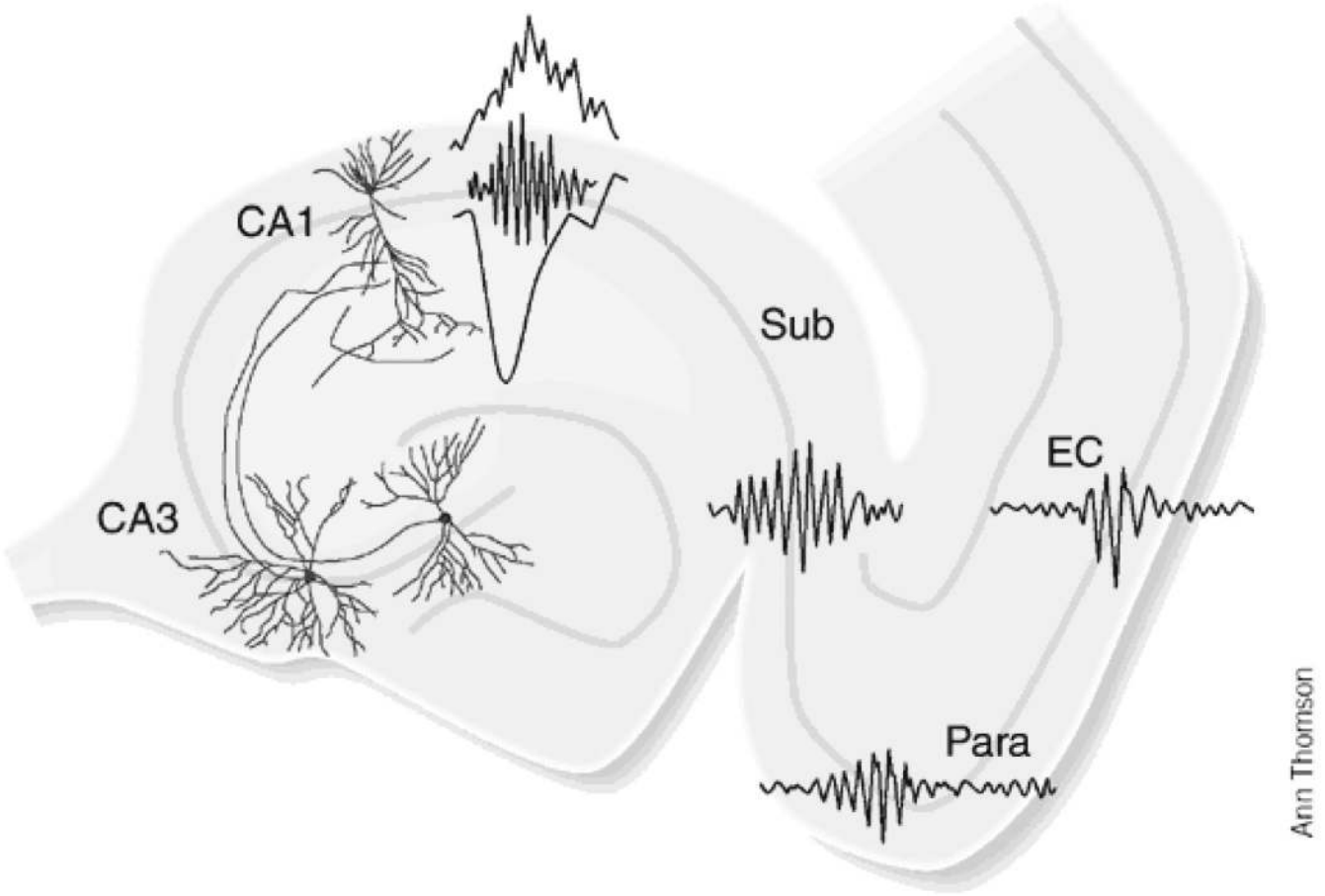

Fig. 5.

Mammalian hippocampal formation showing sharp-wave/ripple complexes (SWRs). Self-organized bursts of activity in the hippocampal CA3 region produces a sharp-wave field potential in the dendritic layer of CA1 and a short-lived fast-frequency field oscillation (200 Hz ripple) within the stratum pyramidale, as well as a phase-related discharge of the neurons. Hippocampal output, in turn, produces similar SWRs in the subiculum (Sub), parasubiculum (Para) and deep layers of the entorhinal cortex (EC). Although SWRs occur spontaneously during quiet wakefulness, neocortical input biases their timing to the up-state of the neocortical slow-oscillation during slow-wave sleep. Reprinted by permission from Macmillan Publishers Ltd: Nature Neuroscience, Buzsáki & Chrobak, 2005.

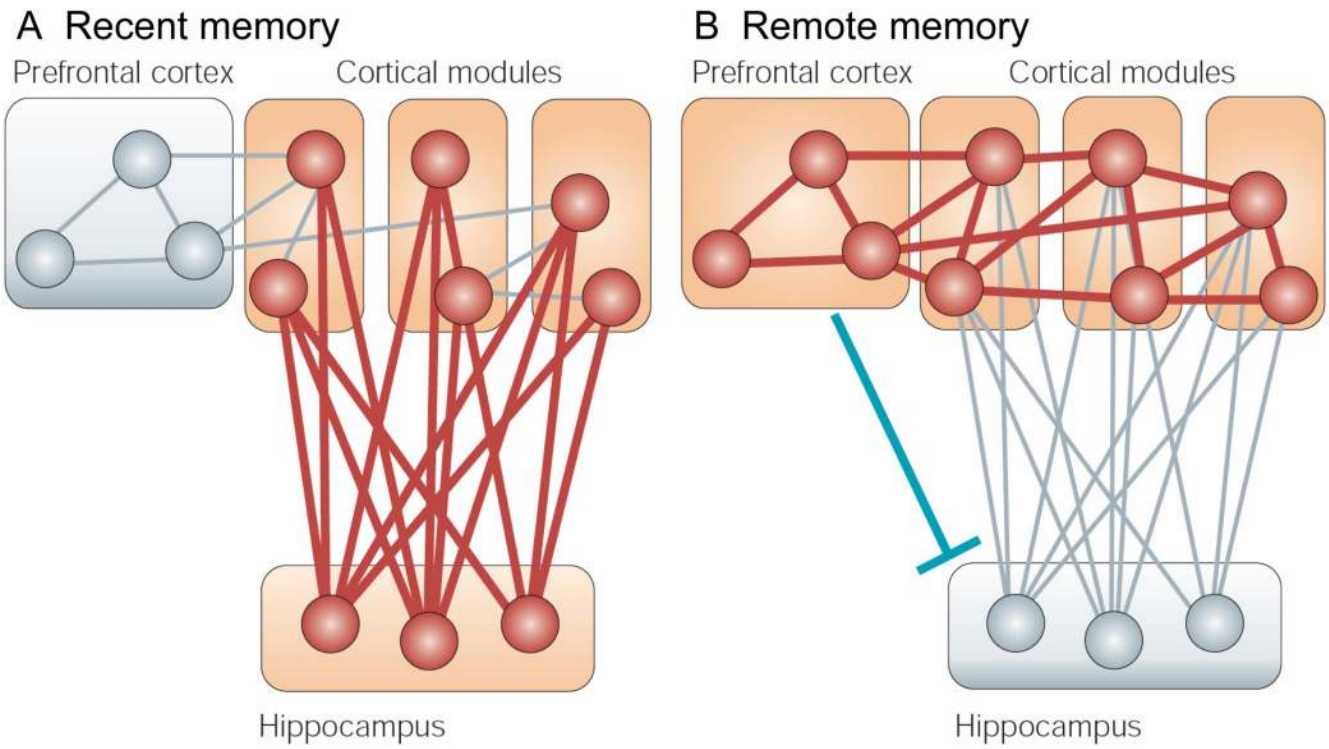

Fig. 6.

Proposed roles of the prefrontal cortex (PFC) in the formation and recall of remote memories. Initially, memories are encoded in hippocampal–neocortical networks (A, thick lines). At this early time point, the hippocampus is crucial for integrating information from distributed cortical modules, each representing individual components of a memory. However, over time direct projections from the hippocampus are thought to transfer a high-order representation of the memory to the PFC (B), which then uses this information to facilitate the transfer of information from the hippocampus to the neocortex, *via* the entorhinal and perirhinal cortices. As initially proposed for the hippocampus, the PFC may also use this version of the memory to strengthen the connections between the distributed cortical modules involved in the memory (thick lines), and to integrate the memory within related preexisting memories. Later, the PFC may also use this memory to identify and recall context-relevant information from remote memory stores. Finally, during recall of remote memories, the PFC appears to inhibit hippocampal activity (blue line), thereby preventing the encoding of redundant information. Reprinted by permission from Macmillan Publishers Ltd: Nature Reviews Neuroscience, Frankland & Bontempi, 2005.

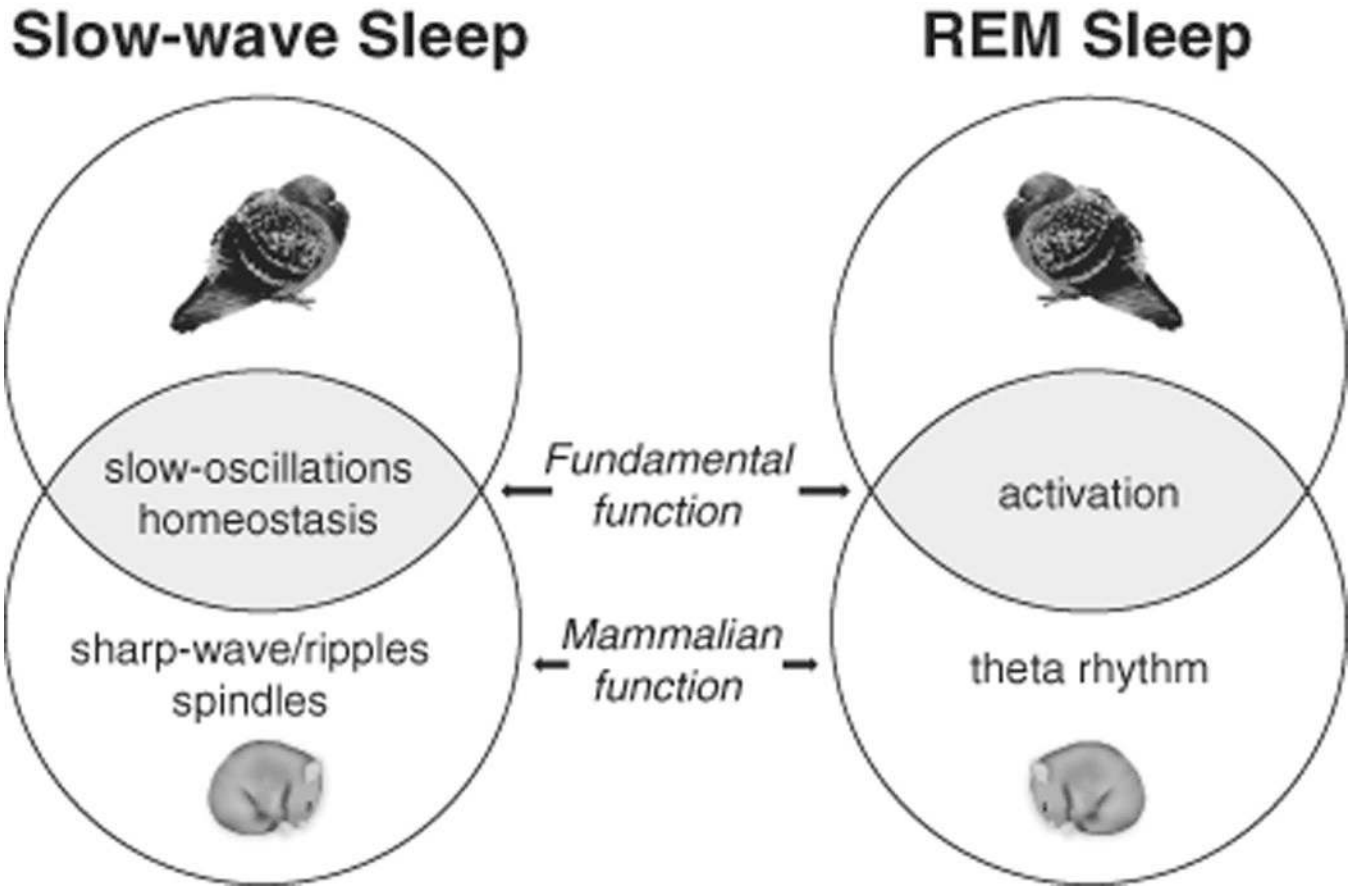

Fig. 7.

Functional implications of the similarities and differences between mammalian and avian sleep-related brain rhythms. REM: rapid eye movement. Rhythms shared by mammals and birds (grey overlap between circles) are likely to be involved in a fundamental function of each sleep state, whereas rhythms occurring only in mammals are most likely involved in mammal-specific functions. Images: pigeon (Niels Rattenborg), rat reprinted by permission from Macmillan Publishers Ltd: Nature Neuroscience, Mehta, 2007.

Table 1

Definitions of abbreviations used in the text.

ANC	Arcopallium-nidopallium caudale
APH	Area parahippocampalis
AVT	Area ventralis tegmentalis
BrdU	Bromo-2-deoxyuridine
CA	*Cornu Ammonis* (Ammon's horn) of the mammalian hippocampus
CB1-R	Cannabinoid receptor
CDL	Dorsolateral corticoid area
CMM	Caudal part of the medial mesopallium
DG	Dentate gyrus
DL	Dorsolateral region of the hippocampal formation
DLP	Nucleus dorsolateralis posterior thalami
DM	Dorsomedial region of the hippocampal formation
DVR	Dorsoventricular ridge
EC	Entorhinal cortex
EEG	Electroencephalogram
fMRI	Functional magnetic resonance imaging
FOS	Protein product of the immediate early gene *c-fos*
GP	Globus pallidus
HAMD	Hyperpallium accessorium/mesopallium dorsale
HDCo	Core region of the hyperpallium densocellulare
HDPe	Periventricular region of the hyperpallium densocellulare
HF	Hippocampal formation
Hp	Hippocampus
IEG	Immediate early gene
IMM	Intermediate and medial mesopallium
IS	Imprinting stimulus
LNM	Lateral nido-mesopallium
Ma	Magnocellular region of the hippocampal formation
mMSt	Medial portion of the medial striatum
MNM	Medial nido-mesopallium
mPFC	Medial prefrontal cortex
MSDB	Medial septum-diagonal band of Broca
MSt	Medial striatum
NCL	Caudolateral nidopallium
NCM	Caudal part of the medial nidopallium
NMDA	*N*-methyl-D-aspartate
NMDA-R	*N*-methyl-D-aspartate receptor
Pa	Parvocellular region of the hippocampal formation
PAL	Passive avoidance learning
Para	Parasubiculum

PFC	Prefrontal cortex
Po	Cell-poor region of the hippocampal formation
RA	Robust nucleus of the arcopallium
REM	Rapid eye movement
SN	Substantia nigra
Sub	Subiculum
SWA	Slow-wave activity
SWRs	Sharp-wave/ripples
SWS	Slow-wave sleep
TRN	Thalamic reticular nucleus