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Review

Sleep and memory in mammals, birds and invertebrates

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ABSTRACT

Sleep supports memory consolidation. Based on studies in mammals, sleep-dependent consolidation has been conceptualized as 'active system consolidation'. During waking, information is encoded into an initial store (hippocampus). During subsequent sleep, some of the newly encoded memories are selected to be reactivated and redistributed toward networks serving as long-term store (e.g., neocortex), whereby memories become transformed into more general, schema-like representations. Here we asked whether sleep in non-mammalian species might play a comparable role for memory. The literature review revealed that sleep produces enhancing effects on memory in all non-mammalian species studied. Furthermore, across species some of the hallmarking features of active system consolidation were identified: Studies of filial imprinting in chicks suggest that a redistribution of imprinting memory toward long-term storage sites occurs during sleep; song learning in birds appears to be driven by reactivations of song representations during sleep; studies of bees demonstrated the selectivity of sleep-dependent consolidation, benefiting extinction but not original classical conditioning. Although overall fragmentary, first evidence in non-mammalian species suggests active system consolidation might be an evolutionary conserved function of sleep.

Contents

Abbreviations: SWS, slow wave sleep; SWA, slow wave activity; REM sleep, rapid eye movement sleep; NonREM sleep, non-rapid eye movement sleep; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; IMM, intermediate and medial mesopallium; RA, robust nucleus of arcopallium; NCL, nidopallium caudolaterale; NCM, caudomedial nidopallium; HVC, a letter based name for a premotor association region in the songbird brain.

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1. Introduction

Learning is tiring! While everyone would agree from his own experience, research confirms a strong link between learning, memory and sleep (Diekelmann and Born, 2010; Rasch and Born, 2013; Stickgold, 2005). Over the last two decades the search for the function of sleep has expanded from the almost exclusive study of mammals to birds and invertebrates, most extensively in respect to work with *Drosophila* (Bushey and Cirelli, 2011). Still the field of sleep research is divided based on the model organisms used: (i) mammals (humans and rodents), (2) birds (chicks, zebra finches and starlings), and (3) fish and invertebrates (Zebrafish, honey bees, *Drosophila* and *Caenorhabditis elegans*). While all of these models have their own strong advantages, little work has been presented extracting the shared features and possible common functions of sleep among these models. Here we review findings in respect to the memory function of sleep in the named models. We will start with a short introduction into the concepts and knowledge about the function of sleep that has been collected from the work with humans and rodents, and then ask whether in birds and invertebrates sleep might play a comparable role for memory.

2. Sleep and memory in mammals

Memory is typically divided into three fundamentally different sub-processes: encoding, consolidation and retrieval. (I) Encoding refers to the up-take of the information to be stored into a neural representation. (II) Consolidation refers to some kind of stabilization of the memory that follows encoding and enables the retention of a memory over time. In the absence of such consolidation the information would be rapidly forgotten. Forgetting can result from a decay of the memory trace or from retroactive interference as the encoding of new information leads to an overwriting of the information encoded before. (III) Retrieval of the stored information refers to the reactivation of a stored memory in the context of more or less goal-directed behavior. Above all, sleep appears to support the consolidation of memory. However, sleep is also known to benefit the subsequent encoding of new information (Tononi and Cirelli, 2014). This second function of sleep will not be covered here.

That sleep supports memory consolidation is known for more than a century. Experimental demonstrations of this effect go back to Heine (1914), a student of Ebbinghaus, and Jenkins and Dallenbach (1924). The latter researchers basically showed that when subjects slept after learning a list of nonsense syllables (encoding), they were able to recall more of the nonsense syllables at a later retrieval test than when they had stayed awake during the retention interval following learning. Since then, numerous other studies, mostly performed in humans, confirmed the benefitting effect of sleep on the retention of different kinds of memory materials and tasks. This research has been in depth reviewed in several recent publications (Abel et al., 2013; Conte and Ficca, 2013; Diekelmann and Born, 2010; Fogel and Smith, 2011; Huber and Born, 2014; Inostroza and Born, 2013; Lewis and Durrant, 2011; Rasch and Born, 2013; Ribeiro, 2012; Stickgold, 2013; Stickgold and Walker, 2013; Wilhelm et al., 2012b). Rather than reiterating these previous reviews, here we want to accentuate several features that appear to hallmark the consolidation process during sleep.

2.1. Sleep-dependent memory consolidation is selective

Hundreds of studies demonstrate a beneficial effect of post-encoding sleep on the consolidation of different types of memory whereas less than a handful of studies claim an opposite effect. However, sleep does not equally benefit all newly encoded representations. Sleep appears to preferentially enhance memories involving the prefrontal–hippocampal memory system during encoding. In rats, sleep affects context conditioning, a hippocampal dependent task, while it does not affect cued conditioning, which is not hippocampus dependent. Five hours of sleep deprivation after context fear conditioning (i.e., learning that a certain surrounding is dangerous) impaired the fear response at a later retrieval test, whereas cued fear conditioning (i.e., learning that a certain tone is followed by a shock independent of the surrounding) was not affected by sleep deprivation (Graves et al., 2003). In two other studies in rats, an object-place recognition task, a temporal order memory task and an episodic-like memory task benefited from post-encoding sleep whereas a novel-object recognition task did not (Inostroza et al., 2013; Oyanedel et al., 2014). Indeed, novel-object recognition in these studies was the only task that does not critically depend on intact hippocampal function (Bussey et al., 2000; Mumby et al., 2002).

Also in humans, sleep seems to preferentially help consolidate hippocampus-dependent memory (e.g., contextual types of memory) rather than hippocampus-independent memory (e.g., item memory) (Aly and Moscovitch, 2010; Rauchs et al., 2004; van der Helm et al., 2011; Weber et al., 2014). Memory for the spatio-temporal context of an episode critically depends on hippocampal function, whereas item memory, like object recognition memory, does not (Davachi, 2006; Eichenbaum et al., 2007). For example, when participants learned lists of words (item memory) while facing two different posters (contexts), napping following learning led to better memory for the posters but not for the list words compared with a no-nap control condition. Recognition of the list words does not critically depend on hippocampal function (van der Helm et al., 2011). Other studies likewise revealed selectively improving effects of sleep on spatio-temporal context memory (Griessenberger et al., 2012; Rauchs et al., 2004; Wilhelm et al., 2011b), and also showed that such enhancing effects on context can be blocked by the administration of glucocorticoids during retention sleep, which affect in particular hippocampal circuits (Griessenberger et al., 2012; Kelemen et al., 2014; Wilhelm et al., 2011b).

In humans hippocampus-dependent memory is traditionally equated with declarative memory which refers to memory for episodes and facts, and is explicitly (i.e., consciously) encoded and retrieved (Squire and Zola, 1996). Procedural memory for perceptual and motor skills, in contrast, is thought of not essentially relying on hippocampal function. However, more recent imaging studies revealed that learning of such procedural tasks entailing a sequential feature, at least in the initial stages of training, typically also involves hippocampal function (Henke, 2010; Schendan et al., 2003). Regarding sleep-dependent consolidation, the hippocampal involvement at training, specifically the functional connectivity between hippocampus and prefrontal areas, appears to even predict the overnight gain in skill (Albouy et al., 2008, 2013a,b). Also, the involvement of the prefrontal–hippocampal system in procedural learning is enhanced when the training takes place under

explicit conditions (e.g., Destrebecqz et al., 2005; Schendan et al., 2003; Strange et al., 2001). In a motor sequence learning task participants improved their skills after sleep when they were aware of the sequence during training, whereas sleep-dependent gains after implicit task training were negligible in this study (Robertson et al., 2004). Overall these studies show that not only for episodic memory but also for procedural memory the involvement of the prefrontal–hippocampal system critically determines the extent to which a memory is enhanced by sleep.

Ultimately, the importance of prefrontal–hippocampal circuitry for sleep-dependent memory consolidation is underlined by recent human studies indicating strong benefits for memories for the future, i.e., memories with a prospective component (Diekelmann et al., 2013a,b; Scullin and McDaniel, 2010). In these studies, subjects were asked to perform specific behaviors at a test session taking place one or two days later. When they had slept during a 12-h interval after the instruction session they remembered to execute these plans much better, than when staying awake during this interval (for related results see Fischer and Born, 2009). Also, the mere expectancy that recall of the learned materials will be tested after the retention interval appears to strengthen the enhancing effect of sleep on memory (Wilhelm et al., 2011a). Planning and the allocation of expectancies are central executive prefrontal cortical functions (Burgess et al., 2001, 2007; Miller and Cohen, 2001). Indeed, prefrontal planning functions appear to tag newly acquired hippocampal memories during wakefulness, during encoding or shortly afterwards, such that these memories preferentially enter subsequent sleep-dependent consolidation (Rasch and Born, 2013). In light of the obvious importance of such prospective prefrontal tagging of memories for sleep-dependent consolidation it has been speculated that, rather than merely enhancing memory traces, consolidation during sleep might primarily act to enhance the accessibility of memories in the context of planned actions and behavior (Inostroza and Born, 2013).

2.2. Memory representations are transformed during sleep

Beyond merely strengthening or weakening of certain memories (quantitative effect), there is evidence that sleep can also induce changes to a memory representation and thus transform the memory (qualitative effect). Although rodent studies have so far largely neglected this aspect of sleep-associated memory processing, a recent study in rats provided first cues that sleep might promote generalization of inhibitory behavioral control (Borquez et al., 2013). This study used a go/nogo conditional discrimination learning task to examine the effects of 80-min retention periods filled with sleep (vs wakefulness). Re-learning performance at the delayed retest indicated that sleep benefitted the discrimination behavior in particular by enhancing correct nogo responses. Thus animals who were allowed to sleep improved in withholding and, therefore, actively controlling their response, a task typically associated with prefrontal cortex function. Interestingly, the effect of sleep was independent of whether the animals were retested in the same or in a different context as during learning. Such generalization across contexts reflects a de-contextualization of memory. De-contextualization might be favored by slow wave sleep (SWS) rather than rapid eye movement (REM) sleep, as sleep-induced enhancement of nogo responses did not occur after selective deprivation of REM sleep (Fu et al., 2007). That sleep promotes the de-contextualization of a memory has been likewise suggested by human studies, where such effect appeared to develop gradually over several succeeding nights (Cairney et al., 2011; Cox et al., 2014; Deliens et al., 2013; Deliens and Peigneux, 2013). In conjunction with findings of a sleep-induced enhancement of episodic memory (discussed in Section 2.1), findings of sleep-induced context generalization

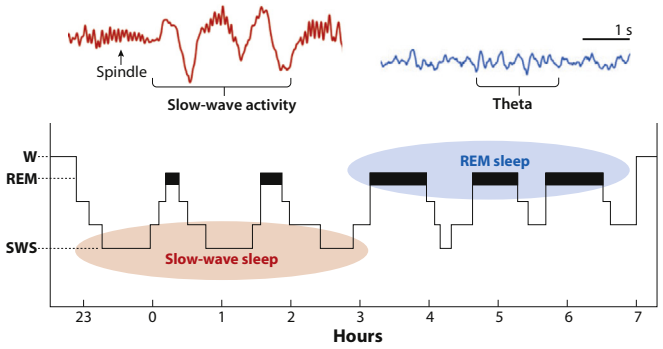

Fig. 1. *Sleep stages in humans.* Sleep in humans and other mammals is divided into different sleep stages, mainly into slow wave sleep (SWS) which represents the deepest form of non-rapid eye movement (NonREM) sleep and REM sleep. Periods of NonREM and REM sleep alternate in cycles of ~90 min. SWS is hallmarked by high amplitude slow oscillatory EEG activity in the 0.5–4.0 Hz frequency band and spindle activity in the 12–15 Hz band. REM sleep is characterized by low amplitude mixed frequency activity and theta activity. The first half of nocturnal sleep is dominated by SWS with little REM sleep, whereas the second half of the night is dominated by REM sleep. Modified from Inostroza and Born (2013).

points to a twofold function of sleep: On the one hand, to an immediate enhancing effect on the memory for episodes and its binding into spatiotemporal context, which entails a sleep-induced improvement for the context itself. On the other hand, sleep favors the de-contextualization of the experienced event and the generation of a more schema-like context-independent representation, i.e., a process that appears to develop more gradually over time (Inostroza and Born, 2013; Kumaran and McClelland, 2012).

In humans, sleep facilitates the insight into, and abstraction of hidden rules and structures in learned materials. Thus, implicit memory becomes explicit which in fact reflects a qualitative transformation of memory (Lewis and Durrant, 2011; Wagner et al., 2004). For example, Fischer et al. (2006) asked subjects about their explicit knowledge of the sequence underlying a serial reaction time task on which they had been trained implicitly before retention periods of sleep or wakefulness. After the sleep interval they exhibited a distinctly greater explicit sequence knowledge than after the wake interval. Sleep seems to favor abstraction processes also in the procedural memory system, for instance, by supporting the formation of an effector-independent representation in a finger sequence tapping task (Cohen et al., 2005; Witt et al., 2010). Sleep after training a sequence with the left hand improved also tapping the same sequence when performed with the (untrained) right hand. At the functional anatomic level, the sleep-induced transformation of memory has been characterized via functional magnetic resonance imaging (fMRI). Typically, post-encoding sleep produced two effects at the delayed retest: (i) an increased activation in hippocampal networks and (ii) an increased functional connectivity of the hippocampus with extra-hippocampal regions and/or an increased activity in these extra-hippocampal regions (Fischer et al., 2005; Gais et al., 2007; Orban et al., 2006; Payne and Kensinger, 2011; Takashima et al., 2009). Enhanced activation and connectivity to extra-hippocampal regions at retrieval after sleep was often observed in neocortical areas, mainly prefrontal cortical areas, and the striatum, and might be considered a manifestation of the redistribution of the memory representation promoted by sleep.

2.3. Memory consolidation is caused by slow wave sleep and associated reactivations of neuron assemblies

Sleep in humans, and similarly in rodents, consists of the alternating occurrence of non-rapid eye movement (NonREM) sleep periods and intermittent REM sleep periods (Fig. 1). The deepest kind of NonREM sleep is termed slow wave sleep (SWS). For a

long time, sleep-associated memory processing was suspected to take place mainly during REM sleep, probably because in human subjects awakenings from this sleep stage are usually associated with the report of vivid dreams. However, approaches using selective REM sleep deprivation to test possible memory consolidating effects of this sleep stage revealed overall rather mixed results (Gais and Born, 2004a). In fact, more recent research accumulated compelling evidence that, rather than REM sleep, NonREM sleep, and specifically SWS plays a more important role for consolidation, in particular of hippocampus-dependent memory. For example, a number of human studies compared the effects of 3 to 4-h retention intervals, filled with either early (SWS-rich) or late (REM-rich) nocturnal sleep. The studies consistently showed that, hippocampus-dependent declarative and prospective types of memories (words, texts, plans etc.) encoded before the respective sleep intervals profited from early SWS-rich sleep, but not from late REM-rich sleep (Diekelmann et al., 2013b; Drosopoulos et al., 2005; Plihal and Born, 1997, 1999; Yaroush et al., 1971). The REM-rich late sleep in these studies appeared to convey an additional benefit on the retention of emotional stimuli, as well as for procedural tasks (but see Rasch et al., 2009).

The notion of a leading role of SWS for hippocampus-dependent memory consolidation is corroborated by studies more closely examining the EEG phenomena that hallmark this sleep stage, i.e., EEG slow wave activity (SWA) and the slow oscillation as an underlying neuronal substrate of SWA, as well as spindle activity. In humans, SWA is typically defined by the EEG power during SWS in the 0.5–4.0 Hz frequency band, with a spectral peak around 0.8 Hz. The slow oscillation represents an alternation of neuronal network activity between down-states, in which the great majority of neurons hyperpolarize and is silent, and subsequent depolarizing up-states in which firing activity distinctly increases (Steriade, 2006). The slow oscillation is generated primarily within neocortical networks, partly as a function of the use of these networks for the uptake of information during prior wake periods (Huber et al., 2004a; Molle et al., 2004). In adult humans, slow oscillations most often originate from prefrontal cortical areas and travel toward posterior sites, however they also reach the hippocampus and subcortical sites (Murphy et al., 2009; Riedner et al., 2011; Wolansky et al., 2006). The depolarizing up-state of the slow oscillation drives the generation of spindle activity, which originates from thalamic networks and reaches the neocortex via widespread thalamo-cortical fibers. In the human sleep EEG, spindle activity refers to waxing and waning oscillatory activity between 12 and 15 Hz that forms discrete spindles during lighter NonREM sleep stage 2 but is likewise present during SWS in particular during the initial periods of SWS (De Gennaro and Ferrara, 2003). Both SWA and spindle activity occur preferentially in synaptic networks that were potentiated, indicating that prior information encoding favors their generation (Behrens et al., 2005; Bergmann et al., 2008; Tononi and Cirelli, 2006). Conversely, both oscillations can support plastic synaptic processes such as long-term potentiation (Chauvette et al., 2012; Rosanova and Ulrich, 2005). Moreover, both slow oscillations and spindle activity seem to be closely linked to enhanced sleep-associated consolidation processes in rats and humans (Binder et al., 2012; Fogel and Smith, 2011; Gais et al., 2011; Oyanedel et al., 2014; van der Helm et al., 2011; Wilhelm et al., 2011a). For example, children show more SWA than adults. Corresponding with this increased SWA, the sleep-dependent gain of explicit sequence knowledge from a serial reaction task that was trained under implicit conditions before sleep was distinctly greater in children than adults (Wilhelm et al., 2013). Moreover, in each age group the gain of explicit sequence knowledge showed a robust correlation with SWA. Directly suppressing or enhancing slow oscillations through electrical or acoustic stimulation effects the consolidation of hippocampus-dependent memories (Marshall et al., 2006, 2011; Ngo et al., 2013).

Consolidation is probably caused by "replay" of neuronal memory representations during sleep. During sleep following performance on a spatial task specific firing patterns in hippocampal place cell assemblies are reactivated in the same temporal order as during actual task performance (Ji and Wilson, 2007; O'Neill et al., 2010; Skaggs and McNaughton, 1996; Wilson and McNaughton, 1994). Reactivations of place cell assemblies occur during SWS, and also during quite wakefulness, but are normally not observed during REM sleep (Kudrimoti et al., 1999). Place cell assemblies are also reactivated following exploration of novel environments whereby assemblies associated with place fields that were more intensely explored displayed stronger reactivation during succeeding SWS (O'Neill et al., 2008; Ribeiro and Nicolelis, 2004). Assembly reactivations in hippocampal circuitry are typically accompanied by so-called sharp wave–ripple events (O'Neill et al., 2010). Ripples represent bouts of fast oscillatory activity with frequencies above 180 Hz. Sharp wave–ripples and associated assembly reactivations, like thalamic spindles, occur phase-locked to the up-state of the neocortical slow oscillation (Clemens et al., 2009; Ji and Wilson, 2007). Assembly reactivations occur also in neocortical and striatal areas, where they emerge some milliseconds later than in the hippocampus (Euston et al., 2007; Ji and Wilson, 2007; Lansink et al., 2009; Pennartz et al., 2004). This temporal pattern suggests a spreading of reactivations that originate in hippocampal circuitry and travel to extrahippocampal sites. Human fMRI studies basically confirm these findings: After learning hippocampus-dependent declarative materials, blood oxygenation level dependent (BOLD) patterns are reactivated during NonREM and SWS in hippocampal and specific neocortical regions (Bergmann et al., 2012; Peigneux et al., 2004).

Notably in humans and rats, hippocampal reactivations and ripples seem to play a casual role for memory consolidation. In humans, when auditory and olfactory stimuli (cues) were presented together with items at specific spatial locations during the learning phase, the memory for the spatial locations was enhanced when these stimuli were presented again during subsequent SWS. (Rasch et al., 2007; Rudoy et al., 2009). Re-exposure of the cues during REM sleep remained ineffective. Comparable results were obtained in rats (Bendor and Wilson, 2012; Girardeau et al., 2009). Altogether, these studies identify assembly reactivations during post-encoding SWS as a key mechanism for memory consolidation during sleep.

2.4. Active system consolidation during sleep

The concept of an "active system consolidation" during sleep has been proposed to integrate findings on memory formation during sleep in humans and rodents (Diekelmann and Born, 2010; Inostroza and Born, 2013; Rasch and Born, 2013). The concept originates from the standard consolidation theory, considering also more recent conceptual developments such as the trace transformation theory (McClelland et al., 1995; Squire and Alvarez, 1995; Winocur et al., 2010). It assumes a two-stage memory system entailing a rapidly encoding initial storage system, essentially represented by the hippocampus, and a long-term storage system which encodes at a much slower pace, essentially represented by neocortical and striatal networks (Fig. 2A). During wakefulness new information is encoded under control of the prefrontal–hippocampal episodic memory system both in hippocampal and neocortical networks, whereby the hippocampus specifically encodes the episodic features of this information, binding experienced events into their unique spatio-temporal context. During subsequent sleep and specifically during periods of SWS, slow oscillations predominantly originate from prefrontal

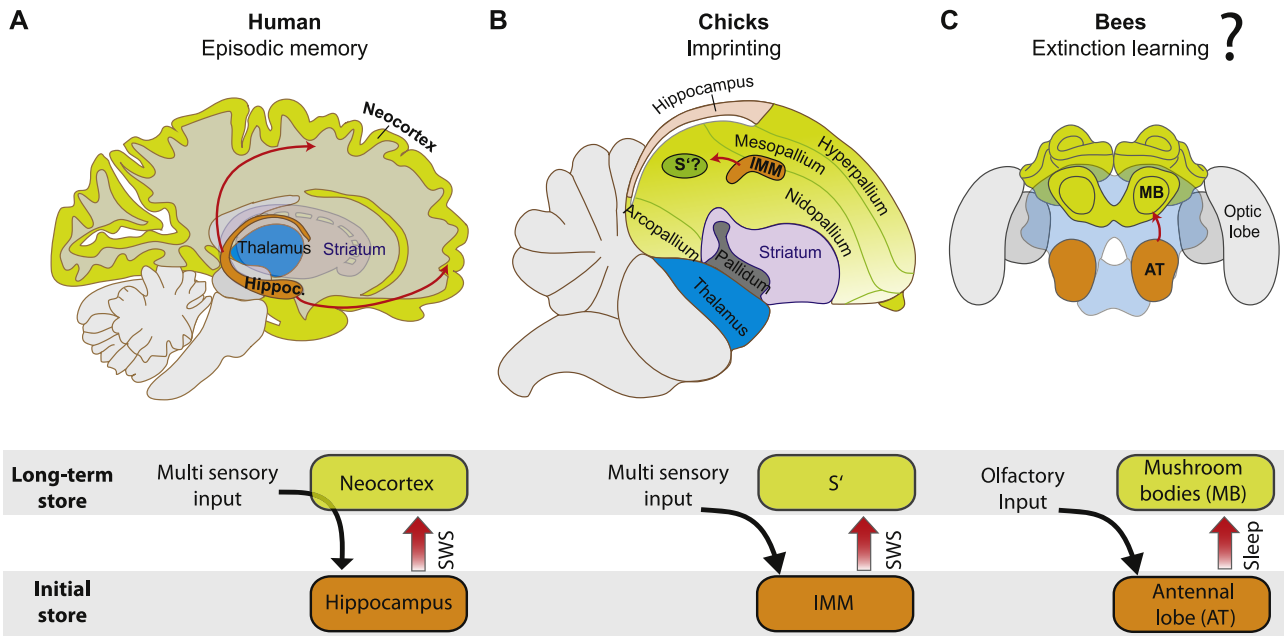

Fig. 2. *Two-stage long-term memory formation during sleep in different species.* (A) In humans newly encoded episodic memories are stored for initial usage in the hippocampus (orange). During SWS they are reactivated and presumably redistributed towards long-term storage sites, mainly the neocortex (green) where they reside in more abstract and de-contextualized form. (B) Sleep is essential for the formation of imprinting memory in chicks. Imprinting memory is initially encoded in the left IMM (intermediate and medial mesopallium). Presumably during SWS occurring within 9 h after imprinting training, the imprinting memory becomes redistributed to an unknown locus termed S′, where it can be more flexibly accessed in different contexts. (As the location of S′ is presently unknown the actual placement of S′ in the figure is meaningless) (C) Similar two-stage processes of memory formation supported by sleep might be established in song learning birds (not shown) and in bees during extinction learning. In bees, classical olfactory reward conditioning of the proboscis extension response occurs at the level of the antennal lobe and is not affected by sleep. However, extinction learning of this response requires sleep. We speculate (indicated by "?") that sleep-dependent consolidation of extinction originates from the redistribution of representations (related to the conditioned response and its inhibition) from the antennal lobe to the mushroom bodies, which is a higher order processing area containing more flexibly controlled memory representations. Representations in the mushroom bodies are known to be sensitive to sleep effects. Respective upper panels provide anatomical locations for the relevant structures. (A) and (B) modified from Jarvis et al. (2005). (C) modified from Menzel et al. (2006). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

neocortical circuitry, used during prior wake for encoding. Through their depolarizing up-states the slow oscillations drive the repeated reactivation of newly encoded neuronal representations in the hippocampus. Simultaneously, the slow oscillations serve to globally down-scale and renormalize synaptic potentiation to prevent excess connectivity (Tononi and Cirelli, 2014). The repeated reactivations in the hippocampus produce, on the one hand, a transient strengthening of select representations. On the other hand, these reactivations spread to extra-hippocampal networks, alongside with the passage of the reactivated memory information from hippocampal to extra-hippocampal networks. The spreading of reactivations and passage of reactivated memory information promotes a more gradual redistribution of the original episodic representation such that essential parts of the representation that are accessed during retrieval are stored outside of the hippocampus. However, this redistribution does not implicate a complete transfer of the memories, as some specific representations remain in the hippocampus. Psychologically, the immediate strengthening effect of reactivations on hippocampal representations expresses itself in a sleep-induced enhancement of episodic memory including its spatio-temporal context. The more gradual effect of hippocampal reactivations redistributing representations toward preferential extra-hippocampal networks is accompanied by a qualitative transformation of the representation, in which the memory becomes unbound from its specific context in which it was originally experienced. Such de-contextualized schema-like memories are mainly stored in neocortical association areas in the case of semantic memories (for facts) and in striatal areas in the case of procedural skills.

Memory reactivations occur also during wakefulness. However, two factors favor that sleep is better suited for the reactivation-induced redistribution of memory representations toward extra-hippocampal networks. First, mainly because of the minimal acetylcholinergic activity during SWS, the output from the hippocampal CA1 region to extra-hippocampal sites is disinhibited (Gais and Born, 2004b; Hasselmo and McGaughy, 2004). Second, the top-down synchronizing influence of the slow oscillation up-states allows for the formation of so called spindle–ripple events whereby ripples and the associated reactivated hippocampal memory information is nested into the excitable troughs of a spindle (Molle and Born, 2011; Siapas and Wilson, 1998). These spindle–ripple events might be a mechanism that does not only ease the passage of reactivated memory information to mainly neocortical and striatal sites but concurrently enables the storage of information via longer-lasting plastic changes in the respective target networks (Bergmann et al., 2012; Chauvette et al., 2012; Rosanova and Ulrich, 2005).

Although this concept of an active system consolidation provides a rather integrative view on memory processing during sleep, there are several issues that need to be clarified. Above all, the specific features of the processes that determine the putative abstraction of more generalized, schema-like memory representations during sleep needs to be elaborated (Kumaran and McClelland, 2012). Computation of such generalized representation might be specifically promoted by recurrent information flow between hippocampus and neocortex. The neocortical slow oscillation, beyond timing of hippocampal reactivations, might also convey specific recurrent information that biases reactivations in hippocampal networks and thereby helps the shaping of generalized representations in extra-hippocampal circuits. Another unsolved question refers to the function of REM sleep. It has been proposed that REM sleep serves synaptic consolidation and, thereby, the stabilization of representations that underwent

transformation during preceding SWS (Diekelmann and Born, 2010). However, experimental evidence for this sequential hypothesis on the function of REM sleep is currently scarce.

3. Sleep and memory in birds

3.1. Characterization of sleep in birds

Birds are the only non-mammalian taxonomic group to exhibit high amplitude slow oscillatory EEG activity during SWS and low amplitude mixed frequency EEG activity during REM sleep. Compared with human sleep, sleep in birds is quite fragmented, occurring in short bouts of 1–4 min duration. Periods of SWS last around 50 s in the beginning of the sleep period and decrease to ~25 s at the end of the night. The average increase in SWA from wake to SWS is smaller than in mammals (Tobler and Borbély, 1988). While birds spend most time in SWS, REM sleep accounts typically for less than 10% of total sleep time and is distributed across sleep in relatively short epochs (Roth et al., 2006). Unlike mammals with the exception of whales and dolphins, birds regularly display unihemispheric sleep with one eye open, especially in threatening environmental conditions. Under safe conditions they prefer SWS with both eyes closed, suggesting that SWS expanding over the whole brain represents the more effective state (Rattenborg et al., 1999). Consistent with this view, chickens spend more time sleeping with both eyes closed when they were deprived of sleep during the prior day (Bobbo et al., 2008; Boerema et al., 2003; Rattenborg et al., 2009). Whereas SWS can occur unihemispherically REM sleep does not.

In birds, like in humans, SWA primarily reflects the homeostatic regulation of sleep. SWA is higher after longer periods of wakefulness and highest at the beginning of the night declining exponentially across the sleep period (Martinez-Gonzalez et al., 2008; Szymczak et al., 1996). Sleep deprivation in pigeons and white-crowned sparrows produced compensatory increases in SWA during subsequent sleep (Jones et al., 2008; Rattenborg et al., 2008). Comparable with conditions in mammals, SWA in birds seems to be additionally locally regulated, increasing specifically in brain regions that were used more extensively during prior wakefulness. Pigeons after watching David Attenborough's 'The Life of Birds' with only one eye, showed increased SWA (i.e., power in the 0.5–4.5 Hz frequency band) in the primary visual processing area (the hyperpallium) of the corresponding eye during subsequent sleep (Lesku et al., 2011). The homeostatic regulation of SWA in birds, like that of humans, has been linked to an underlying process of synaptic down-scaling and renormalization that serves to globally balance synaptic connectivity, and thereby prepares the neuronal network for encoding of new information during the upcoming wake phase (Tononi and Cirelli, 2014). In contrast to SWS, REM sleep in birds, like in mammals, appears to be primarily driven by the circadian rhythm and typically increases across nocturnal sleep (Jones et al., 2008; Low et al., 2008; Tobler and Borbély, 1988), thus pointing to a differential function of both core sleep stages also in birds. However, there are observations suggesting that sleep deprivation in birds can be followed by increases in REM sleep as well (Tobler and Borbély, 1988; Martinez-Gonzalez et al., 2008; Newman et al., 2008) which would implicate the existence of additional homeostatic mechanisms also for REM sleep regulation.

Because reptiles and amphibians lack distinct forms of SWS and REM sleep stages, it can be assumed that SWS in birds coevolved independently from mammals (Fig. 3a; Rattenborg, 2006). Indeed, the convergent evolution of this prominent feature of sleep might bear the answer as to the function of this sleep stage, which is possibly related to the comparably large and strongly interconnected brain in both mammals and birds (Rattenborg, 2006, 2007).

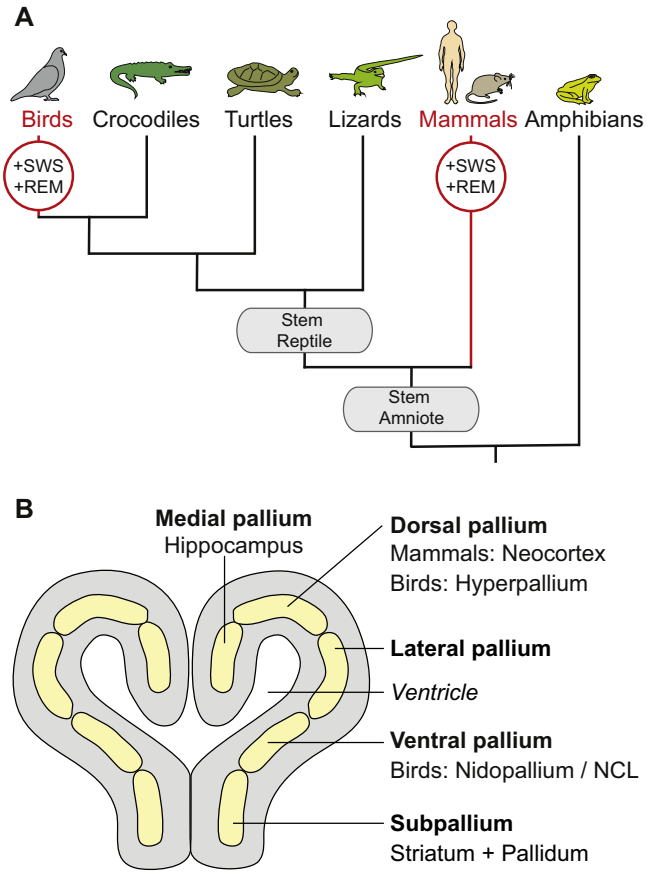

Fig. 3. *Convergent evolution of relevant brain characteristics in mammals and birds.* (A) Birds are the only taxonomic group besides mammals to show SWS and REM sleep. Closely related reptiles, within the shared clade of amniotes, lack these features of sleep, suggesting the co-evolution of SWS and REM sleep in birds to serve similar functions as in humans. Tree depicts the inferred evolutionary relationship between the descendant species. Nodes represent evolutionary separation with shared common ancestor. Modified from Rattenborg (2006, 2009). (B) The avian and mammalian brains show analogous functions, though based on different expansions of the derived pallial structures. Whereas in mammals the dorsal pallium strongly expanded giving rise to the neocortex including the prefrontal cortex to serve higher cognitive function, in birds the ventral pallium expanded to the nidopallium including the nidopallium caudolaterale (NCL), and this region appears to represent the functional analog of the mammalian prefrontal cortex. The hippocampus in both species is derived from the medial pallium. Thus whereas in mammals hippocampus and neocortex including prefrontal cortex originated from neighboring pallial regions, in birds the analogous structures originated from quite distant pallial structures. This explains the lack of close connectivity between the hippocampus and the NCL in birds. The difference in connectivity suggests that the avian hippocampus serves different functions then the mammalian hippocampus. While in mammals the hippocampus in conjunction with the prefrontal cortex, serves the quick encoding of episodic memories into an initial storage system this does not seem to be the case in birds. Yellow fields depict gray matter, gray fields depict white matter. Modified from Looie496 (2011).

3.2. Same function—Different brain structure

Birds, like mammals, do not only exhibit signs of SWS and REM sleep. Furthermore, sleep in birds has been linked to a putative memory function. Yet, despite the functional similarities, there are obvious morphological differences in brain structure and allocated function between these taxonomic groups that should caution against premature generalizations.

As to sleep, SWA appears to originate, preferentially from areas with high interconnectivity, like the neocortex in mammals and the hyperpallium in birds. This high interconnectivity bears a possible explanation of the high capabilities of learning and information encoding in the wake state in both taxonomic groups (Rattenborg

and Amlaner, 2009; Rattenborg et al., 2011). However, the avian pallium – the avian homologue of the mammalian neocortex – is not layered like the mammalian cortex, but rather divided into regions or fields often separated by fiber tracks (nuclear structure). For instance, the hyperpallium consists of stretched nuclei stacked on top of each other across the dorsal–medial–anterior surface of the brain (Beckers et al., 2014). This nuclear structure results in distinct pathways that appear to mirror the function of the layered mammalian cortex. Each of these pathways comprises a granule cell layer receiving ascending input from a different dorsal thalamic nucleus. Information from the granule cell layer is projected to secondary neurons, probably via interneurons, as found for example in the meso- and nidopallium, resembling the mammalian neocortical layers 2/3 (Medina and Reiner, 2000). Secondary neurons in turn propagate the information to neurons that serve as output neurons, as found in the arcopallium, which are thus comparable with the mammalian neocortical layer 5 neurons (Margoliash and Brawn, 2012). In reptiles the lack of SWA appears to coincide with an absence of similarly highly interconnected neuronal regions like the avian cortex. The more simply structured, three-layered reptilian cortex shows homology only with the mammalian layers I, V and VI lacking homology with layer II and III, which show the most extensive cortico-cortical projections in mammals (Jarvis, 2009; Martinez-Cerdeno et al., 2006; Medina and Reiner, 2000; Molnar et al., 2006). Taken together, these comparisons between species suggest the presence of SWA to be associated with high regional interconnectivity rather than to the presence of a specifically multi-layered cortex. This characteristic of SWA is consistent with the notion that the slow oscillations underlying SWA support the communication between widely distributed brain regions and the integrative processing of information in these areas during SWS.

This view is supported by a recent study analyzing the propagation of slow oscillations in zebra finches (Beckers et al., 2014). While in mammals slow oscillations propagate two-dimensional across the neocortex due to its lamination, in birds slow oscillations appear to travel as local plumes of local field and action potential activity in three dimensions through different corresponding structures such as the hyperpallium, the nidopallium caudolaterale (NCL) and caudomedial nidopallium (NCM). Slow oscillations are thought to process spatially distributed information by means of spike timing-dependent plasticity. The coexistence of traveling slow wave activity hints at a shared function possibly for the transfer and integration of information, irrespective of differences in the cytoarchitectonic organization (Beckers et al., 2014). However, the difference in cortical layer structure between birds and mammals does not exclude that SWA in each of these groups serves additional specific functions.

Regarding the memory function of sleep, research in mammals has focused on system consolidation processes evolving during sleep from a dialogue between hippocampus, serving as initial storage system, and extra-hippocampal, mainly neocortical structures, serving as long-term storage system. Interactions between prefrontal cortex and hippocampus are considered of particular importance, as they might select the memories that are consolidated during sleep. In the avian brain the homologous structures do not appear to serve equivalent functions (Fig. 3B). During phylogeny, the largest part of the avian as well as the mammalian brain originates from the dorsal telencephalon, i.e. the pallium, which is composed of four distinct embryonic fields (medial, dorsal, lateral and ventral pallium). In both mammals and birds the medial pallium develops into the hippocampus. However, whereas in mammals the adjacent dorsal pallium develops into the prefrontal cortex, which is thus in rather close connection to the hippocampus, the avian structure analogous to the prefrontal cortex, i.e., the nidopallium caudolaterale (NCL) develops from the ventral pallium and is most distant to the hippocampus (Güntürkün, 2005; Mogensen and Divac, 1982). Thus, in mammals the developmental proximity of the hippocampus and prefrontal cortex indeed favors interactions between these brain structures that might serve the consolidation of select memories initially encoded in the hippocampal memory system.

It may not come as a surprise that in birds the hippocampus obviously serves different functions, due to a lack of immediate connections to the nidopallium caudolaterale. Instead, the avian hippocampus receives only olfactory and visual input mostly from the hyperpallium, although a role of the avian hippocampus for long-term spatial memory has been discussed (Rattenborg et al., 2011). One idea links the difference in evolution between birds and mammals to the use of olfactory inputs as primary sense in early mammals, whereas avian ancestors relied on visual inputs. Avian ancestors presumably first enlarged the dorsal ventricular ride – possibly already a structure for high-level associations then – while the hippocampus might have continued to receive visual input from the dorsal pallium, aside from olfactory input. The hyperpallium, in contrast to the mammalian neocortex, might have been less involved in forming multimodal associations. By contrast, in early mammals relying to a greater extent on olfactory stimuli, olfaction might have boosted connections to the association circuitries and thus strengthened the olfactory–hippocampal–dorsal cortex circuitry (Aboitiz et al., 2003; Rattenborg and Martinez-Gonzalez, 2011). Whatever the reasons for the divergent brain development, so far, it is not clear whether in birds a unitary network exists analogous to the mammalian hippocampus that integrates multimodal sensory and motor inputs to form and initially store episodic-like memory representations. If at all, such function might be supported by a subregion of the nidopallium caudolaterale or by a variety of higher association regions in the nidopallium and mesopallium (Rattenborg et al., 2011; Salwiczek et al., 2010). Given the disparate development of the hippocampus and prefrontal cortex, it may also not surprise that the avian brain lacks an EEG theta rhythm, which in mammals spans the prefrontal–hippocampal system during wake encoding and, especially in rodents, dominates hippocampal activity during REM sleep. Also, sharp wave–ripples are not observed in the bird's hippocampus, during quite wakefulness or SWS, and there also seems to be no equivalent of thalamocortical spindles during SWS (Rattenborg and Amlaner, 2009; Rattenborg et al., 2011). Collectively, these observations argue against a prominent function of the hippocampus and hippocampo-neocortical interactions in the initial storage and system consolidation of memory in the avian brain comparable with that described in mammals. Yet, this does not exclude that such function is taken over, perhaps in a less generalized manner, by other structures.

3.3. Sleep and filial imprinting: The legacy of Gabriel Horn

The first study to imply a role of SWS for memory consolidation in birds came from Gabriel Horn's lab, close to the end of his life (Jackson et al., 2008). Horn dedicated part of his career to the search for the engram, i.e., the memory trace induced by learning. He used filial imprinting as a most robust memory formed in birds during a critical period at an early stage of life, approximately during the first 3 to 4 days in the life of chicks. Filial imprinting refers to a strong social recognition and bonding response learnt after short exposure to an object, which enables the chick to selectively follow this object. Naturally the object of attachment is a member of its own species, mostly the mother. However, in experimental conditions, this bonding can be done to humans or even objects like a moving red box, as Horn used in his experiments (Fig. 4A, Horn, 2004).

Imprinting does not manifest in the hippocampus in terms of plastic synaptic changes, even though the hippocampus, as an area

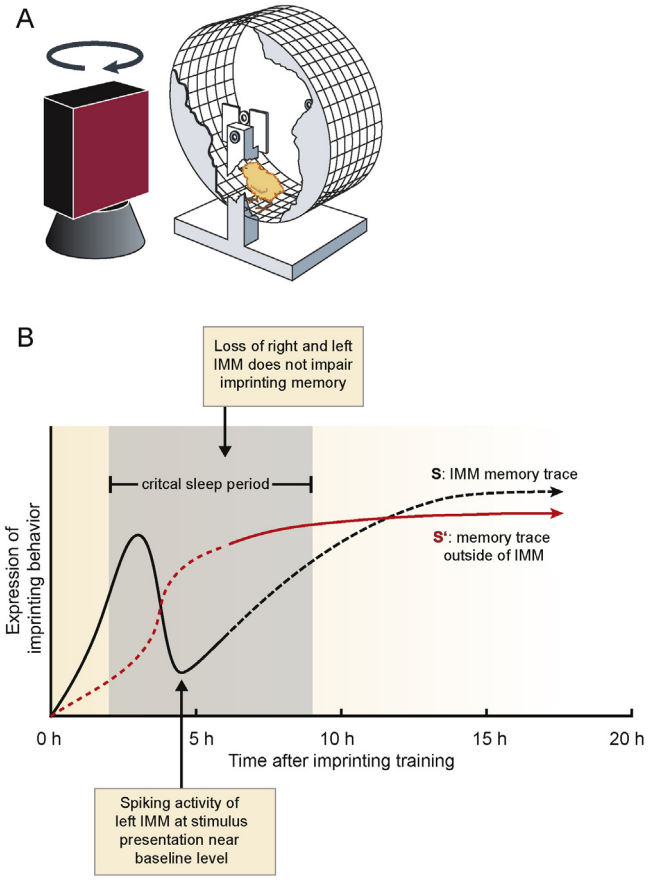

Fig. 4. *Formation of imprinting memory in chicks.* Imprinting memory is first stored in an initial store (IMM) and then probably redistributed to a long-term store (S′). Long-term storage depends on sleep. (A) Experimentally, imprinting memory is induced by exposing chicks during the first 48 h after hatching to a moving stimulus (red box). During training the chick is in a running wheel. (B) After training, neurons in the left IMM (also termed S) selectively respond to the imprinting stimulus. Thus behavioral imprinting response and IMM firing activity are correlated. About 4.5 h after imprinting training IMM neurons cease to fire in response to the imprinting stimulus, yet the animal continues to respond behaviorally to the imprinting stimulus. Bilateral lesion to the IMM does not impair the behavioral imprinting response if performed 6 h or more after training. Also, suppression of slow oscillatory (0.5–2.5 Hz) EEG activity during sleep impairs the formation of imprinting memory, when applied within the first 9 h after training. During this time interval the redistribution of the memory trace from the IMM toward the unknown locus S′ is assumed to occur. Black and red lines indicate the strength of the putative memory traces formed respectively in the IMM and S′, as derived from the experimental findings. Solid line indicates that behavioral expression of the imprinting response during the first ~5 h critically relies on the initial storage system of the left IMM. Although later on responsiveness of left IMM neurons to the imprinting stimulus increases again and remains elevated, activation of the IMM trace is not anymore critical for behavioral expression of imprinting (dashed line), as during this time behavior essentially relies on the long-term memory trace formed in S′ (solid line). Modified from Horn (2004). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

with sensory input, responds to the imprinting stimulus (Brown and Horn, 1994; Nicol et al., 1995, 1998). Instead, studies tracking protein synthesis and neuron structure in chicks identified the intermediate and medial mesopallium (IMM) as the area where the strongest neuronal changes take place upon imprinting. The IMM plays a more general role in memory formation as it seems to be involved in passive avoidance learning as well as visual discrimination learning, and is likely analogous to the mammalian association cortex (Daisley et al., 1998; Horn, 2004). Behavioral imprinting was correlated with an increase in the size of postsynaptic densities and a delayed up-regulation of NMDA receptors selectively in the left IMM, and not in the right IMM (Bradley et al., 1981; McCabe and Horn, 1988; Solomonia and McCabe, 2015). Neurons in the left IMM do not only selectively respond to the imprinting stimulus, but imprinting also increases the number of neurons that selectively respond to the imprinting stimulus (Jackson et al., 2008). Interestingly, 4.5 h after exposure to the imprinting stimulus the number of neurons in the IMM that responded to the imprinting stimulus declines to pre-exposure levels, although the chick continues to respond to the imprinting stimulus. Later on, the number of responding neurons increases again (Fig. 4B; Horn, 2004). This temporal dynamics in neuronal firing in the IMM strongly speaks for the notion that there is an additional storage site for the imprinting stimulus, i.e., a so far unidentified network called S′, which mediates the behavioral response at least during the time of decreased IMM response (Cipolla-Neto et al., 1982; Honey et al., 1995). Lesion studies showed that the representation of the imprinting stimulus in S′ persists for at least 26 h, and that its formation depends on the right IMM (Cipolla-Neto et al., 1982). Bilateral lesions to the IMM 6 h or more after the training does not affect the persistence of the imprinting memory, whereas lesions to these structures at earlier time points impair imprinting memory (Cipolla-Neto et al., 1982; Davey et al., 1987; McCabe et al., 1982). In combination, the findings converge to the view that the imprinting memory is encoded in the left IMM especially for initial use as a fast encoding store, from where it is redistributed to S′ for longer-term storage. The right IMM might play a mediating role in the redistribution of the representation to S′. Thus, the formation of imprinting memory appears to represent a two-stage process: First an initial memory is rapidly formed in the IMM during training and second, a longer-term representation is established in a different network termed S′. This second step takes place off-line after imprinting training and takes several hours to manifest.

Horn and colleagues (Jackson et al., 2008) demonstrated that sleep during the first 9 h after imprinting training is critical for the consolidation of a lasting imprinting memory, with this period likely covering the transfer of memory information from the IMM to S′. This study compared two groups of animals: One group was allowed to sleep for 6 h after imprinting training followed by 6 h of rest deprivation. This group developed a stable memory for the imprinting stimulus and the number of neurons in the IMM responding to the imprinting stimulus doubled toward the end of the experiment. In the other group, chicks underwent first a 6-h period of disturbed sleep after imprinting followed by a 6-h period of undisturbed rest. Those chicks did not form any memory of the imprinting stimulus in terms of attachment behavior, and the number of neurons in the IMM responding to the imprinting stimulus was significantly reduced at the end of the rest period. Thus, in the chicks allowed to sleep during the first 6 h after imprinting, sleep strengthened the response of the initial storage site in the IMM and possibly simultaneously facilitated the redistribution of the memory to S′. Overall, the observed pattern is remarkably consistent with reports from studies in mammals where sleep, specifically the repeated reactivation of neuronal representations during SWS, is thought to exert a twofold effect: An immediate strengthening of the initial memory representation (in the IMM in chicks), and a simultaneous more gradual redistribution and formation of a second representation in networks (S′) designated to serve as long-term store (Inostroza and Born, 2013), expressed by molecular changes in different areas of the brain (Solomonia and McCabe, 2015). Furthermore, the two memories formed are distinct in their function. While both memory traces are sufficient to elicit a response toward the imprinting stimulus, only memories in S′ influenced the acquisition of a heat-reinforced discrimination task in which the imprinted objects served as discriminanda (Honey et al., 1995). This suggests that a putative sleep-induced redistribution of the imprinting memory toward S′ goes along with a transformation of the memory such that the representation in S′ can be more flexibly applied in a different context.

There is an increase in EEG SWA (1–4 Hz) during sleep in birds. Chicks allowed to sleep after imprinting training showed an increase in this and the neighboring 5–6 Hz band, suggestive for a link between sleep slow oscillations and the formation of imprinting memory (Jackson et al., 2008). To test this possibility in a subsequent study the same researchers selectively manipulated slow EEG activity recorded in the left IMM during sleep after imprinting training (Nicol and McCabe, abstract presented at the SfN, 2013). After imprinting, chicks were allowed to sleep for 6 h either (i) undisturbed, or (ii) with sleep disturbed when EEG activity in the 5–6 Hz frequency band exceeded a threshold criterion, or (iii) with sleep disturbed when EEG slow oscillations in the 0.5–2.5 Hz frequency range exceeded a threshold criterion. For sleep disturbance chicks were placed on a running wheel, which turned for 15 s as soon as the threshold criterion was exceeded. While the first two groups of chicks, i.e., undisturbed sleep, and sleep disturbances in the 5–6 Hz range, exhibited behavioral signs of imprinting memory tested after 5, 21 and 29 h, the groups with sleep disturbances in the 0.5–2.5 Hz slow oscillation range displayed a significantly weaker long-term imprinting memory after 21 and 29 h. These observations provide evidence for a causal role of EEG SWA in the formation of a long-term memory representation of the imprinting stimulus. The findings, of course, do not exclude additional contributions of REM sleep to the formation of an imprinting memory. Such contribution is suggested, for example, by the fact that REM sleep seems to be a prevalent sleep stage during early development not only in mammals but also in birds (Scriba et al., 2013). However, presently clear evidence for an involvement of REM sleep in filial imprinting is missing (Solodkin et al., 1985).

To summarize, sleep is of critical importance for the formation of long-term imprinting memory in chicks. Specifically, sleep leads to the recruitment of additional neurons responding to the imprinting stimulus in the left IMM which suggests an immediate strengthening effect of sleep on the initial representation formed in this network. Moreover, the studies revealed hints for a causal role of EEG SWA after imprinting training for the formation of a long-term imprinting memory. In particular, SWA suppression effectively impairing long-term memory formation when applied during a 6-h post-training interval. This speaks for the view that SWA specifically supports the redistribution of the representation from the IMM to the unknown locus S′.

3.4. Sleep and song learning

Song learning in juvenile birds is another developmental model of memory formation that has been extensively used to examine the role of sleep for memory. Beyond benefitting effects of sleep, song consolidation appears to be linked to reactivation of song representations during sleep. In fact, besides mammals, birds are the only taxonomic group, of the ones studied so far, where neuronal reactivations of newly encoded representations have been identified during sleep.

The study of song learning in birds has received strong interest because of its unique resemblance to human speech acquisition. Birds develop their song in two discrete stages between 30 and 90 days after hatching. First a template of a tutored song is formed and only in a second step the bird learns the song by imitation and auditory feedback. Song learning, like speech learning in humans, is a demanding task which might be accompanied by additional sleep need, as birds being first exposed to the singing of an adult tutor tend to fall asleep quickly after exposure (Margoliash and Brawn, 2012). The dependency of song learning on sleep shows a particular dynamic which has been first described by Deregnaucourt et al. (2005) in young zebra finches. While juvenile birds improved their song quality during the day due to intense practice, song structure and quality declined across nocturnal sleep. This dynamics did not merely reflect a circadian rhythm, since juvenile birds induced to sleep by administration of melatonin during daytime likewise sang a less precise song after awakening. Yet, this decline was again followed by practice-induced improvements in song structure during the subsequent wake time (Deregnaucourt et al., 2005). Importantly, the birds that showed the highest degree of song deterioration after sleep turned out to be the best learners in the long run.

The main brain structures participating in song processing during waking and sleep are the caudomedial nidopallium (NCM), the so-called HVC, and the robust nucleus of the arcopallium (RA). The HVC is a premotor-association region thought of as an analog to the human Broca area (Moorman et al., 2012). It is crucial for song motor output and song modification and projects to the motor pathway via the RA. RA neurons show firing patterns during sleep that match in sequential structure those seen during singing in the preceding wake phase, and these assembly reactivations are probably driven by HVC neurons (Dave and Margoliash, 2000; Hahnloser and Fee, 2007; Hahnloser et al., 2006, 2008; Margoliash, 2005). Offline reactivations of firing patterns associated with song performance might also occur in the HVC itself. In RA neurons of juvenile zebra finches bursting spike activity, possibly related to song reactivation, is increased during sleep especially after the animals had listened to the tutor song during the day (Shank and Margoliash, 2009). Interestingly, bursting activity only increased if the birds were allowed to listen to their own singing after listening to the tutor song. When such auditory feedback was prevented by presenting continuous 100 dB white noise, the birds showed no increase in RA bursting spike activity even with the possibility to sing. Hence, bursting of RA neurons during sleep reflects the interacting effects of sensory information from the tutored song and sensory feedback from the bird's own singing, possibly for integration in the premotor network.

The caudomedial nidopallium (NCM) has been considered the avian equivalent to the auditory association cortex and specifically of the Wernicke area. The left NCM contains the neural template for the tutored song (Bolhuis and Gahr, 2006); however, the learning process leads to the formation of an independent sensorimotor representation of the song possibly in areas down-stream from the NCM, such as HVC and RA, since lesions to the NCM in adults impair recognition of the tutor song, but have no effect on the bird's own song production (Gobes and Bolhuis, 2007). In awake adult birds the left NCM is activated in response to the tutor song and also by the bird's own song imitation, with the response depending on the accuracy of the imitation. In juveniles, the NCM spontaneously activates during sleep and this activation is related to the amount of tutor song stimulation and accuracy of song performance during prior wakefulness (Phan et al., 2006).

Interestingly, sleep appears to modulate the lateralization of activity in the song network (reviewed by Moorman and Nicol, 2015). During wakefulness activity in the HVC (the premotor association region) of juvenile zebra finches shows a left hemispheric dominance and a shift toward the right HVC during sleep (Moorman et al., 2013). The left NCM (the auditory association region) shows as well a left sided dominance during wakefulness, but an activity shift toward the right NCM during sleep is only seen in birds that showed poor imitation of the tutored song during the previous day. A strong sleep-associated involvement of right NCM might thus characterize initial stages of learning when the level of imitation is low and the formation of a separate representation of the bird's own song is still in progress. We speculate that the right NCM plays a role for song consolidation similar to that played by the right IMM for the redistribution of imprinting memories during sleep toward the area S′ (discussed in Section 3.3.). Sleep-associated activity of the right NCM might crucially support the redistribution and transformation of auditory song representations into

sensorimotor representations of the bird's own song that are eventually formed in the premotor and motor areas of the HVC and RA.

Interestingly, the study of Deregnaucourt et al. (2005) in juvenile zebra finches showed that in the initial phase of the song learning period sleep had an acute deteriorating effect on performance of the tutored song. This phenomenon is puzzling since in adult birds and mammals sleep normally produces an immediate enhancement in memory performance. However, it is remarkably reminiscent of findings in humans which indicated that children unlike adults, after one night of post-training sleep also do not show consistent performance gains in skills like finger sequence tapping, and in some cases even a significant deterioration in skill performance (Fischer et al., 2007; Wilhelm et al., 2012b). One factor contributing to the sleep-induced decrease in skill is a low pre-sleep level in skill performance, because performance levels in children are normally distinctly lower than in adults, and children did benefit from sleep in a finger sequence tapping task after they had received intense pre-training on the task (Wilhelm et al., 2012a). The pre-training raised tapping skill in the children to levels close to untrained adults. A dependency on pre-sleep performance levels might likewise explain that in juvenile zebra finches the deteriorating effect of sleep was strongest in the initial phase of learning declining with practice time and age.

Why does sleep produce an immediate deteriorating effect on singing skill when the bird's performance is still rather low? This is presently an unsolved question. Experimental deafening can induce a deterioration in song performance in adult song birds similar to that seen after sleep in juveniles (Nordeen and Nordeen, 1992), and it has been speculated that neuronal reactivations of newly acquired song representations during sleep produce such deterioration, because they likewise take place in the absence of auditory feedback (Nick and Konishi, 2005a,b). Thus, in the initial learning phase sleep-associated reactivations induce an unsupervised learning process that might also enhance inaccurate aspects of song performance, due to the lack of auditory feedback. After a more generalized representation of the song has been formed, during sleep such pre-existing representations might replace the missing acute auditory feedback. Those pre-existing representations might then serve as a reference template for the reactivation of newly encoded sensorimotor information to produce a further shaping and improvement in song quality. Brain maturation might play an additional role. Crandall et al. (2007) found that the degree of song deterioration across sleep was inversely correlated with the amount of neuronal spiking in the HVC. Juveniles with the greatest deterioration showed the lowest HVC firing rate during sleep. Importantly, there is also a general increase in HVC firing rate during development. Juveniles during song learning show generally a distinctly lower firing rate of the HVC during sleep than adults. Thus, it was proposed that the increasing spiking activity of HVC neurons stabilizes the maturing song (Crandall et al., 2007). Whatever the underlying mechanisms, the similar dynamics in the effects of sleep on skill memory in song birds and humans during development further adds to the notion of a general role for sleep in long-term memory formation that is independent of the species.

3.5. Sleep and song discrimination learning

The majority of research on the role of sleep for memory in birds has employed developmental models of learning (filial imprinting and song learning). There is some evidence that sleep in birds, like in mammals, also benefits memory formation in adult brains. Brawn et al. (2010, 2013) trained European starlings on an auditory discrimination go/nogo task. Starlings were trained to go for a food reward after 5 s of a specific song segment played to them (go). For another song segment they were trained to withhold the response (nogo). If the bird went for the food reward at the second song segment, it was punished with a 20-s period of lights off. With reference to discrimination performance in the learning phase, performance was significantly increased at a retest when this took place after a retention period filled with nocturnal sleep. Retest performance after retention sleep was also distinctly better than after an interval of daytime wakefulness during which discrimination performance even tended to decline (Brawn et al., 2010). In a more recent extension of this study the same research group tested how interference learning on another pair of go/nogo song segments affects sleep-dependent consolidation (Brawn et al., 2013). As expected from research in humans (e.g., Diekelmann et al., 2011; Drosopoulos et al., 2007; Ellenbogen et al., 2009), interference learning during the wake retention condition, impaired memory for the go/nogo discrimination on both the original and the newly learned interference task. Yet, a night of retention sleep improved discrimination performance on both tasks. In this condition, the performance improvement for the original discrimination task was even greater than that of a control group of starlings which were not subjected to interference learning before sleep. These data indicate that sleep does not only nullify the impairing effects of interference on memory retention. On the contrary, the sleep-dependent consolidation process appears to be even stimulated by interference occurring during prior waking. Such changes toward a better separation of interfering representations speak for an active transformation in the memory representations that is induced by sleep. The tempting question whether such transformation is linked to SWS and SWA needs to be answered in future studies including electrophysiological sleep recordings.

4. Sleep and memory in invertebrates

The existence of a spinal cord does not seem to matter for the occurrence of sleep. Still invertebrates were greatly neglected as an attractive model of sleep research for a long time. Sleep research in invertebrates started with bees, cockroaches and scorpions (Kaiser and Steiner-Kaiser, 1983; Tobler, 1983; Tobler and Stalder, 1988). More recently, *Drosophila* (Hendricks et al., 2000; Shaw et al., 2000), and the roundworm *C. elegans* were discovered as promising models for sleep research (Raizen et al., 2008). All of these models were mainly exploited for the genetic dissection of sleep, as the identification of genes regulating normal and aberrant sleep requires massed screening. For this purpose, these models are advantageous because they are easy to maintain, have a short life cycle and are equipped with a genetic tool box (Crocker and Sehgal, 2010; Sehgal and Mignot, 2011).

In the beginning, the field was rather preoccupied with proving the existence of sleep in invertebrate species. The research strongly contributed to the implementation of general criteria for the definition of sleep (Table 1). As no typical mammalian EEG signal is present in these species, the identification of sleep-like states in invertebrates relies primarily on behavioral signs like inactivity and the presence of a specific body posture, an increased threshold to arousing stimulation, as well as the demonstration of a rebound in the sleep-like state that occurs as a consequence of experimental sleep deprivation. In fact, invertebrates studied so far like honey bees and flies, with the exception of crayfish, seem to lack synchronous neuronal activity during sleep (Mendoza-Angeles et al., 2007, 2010). A change in brain state from wakefulness to sleep has been shown for honey bees and flies where sleep expresses itself in reduced spontaneous neural firing rates (Kaiser and Steiner-Kaiser, 1983; Nitz et al., 2002; van Swinderen et al., 2004). Moreover, sleep intensity seems to vary as seen in cockroaches, bees and flies suggesting the existence of different sleep stages possibly serving different functions (van Alphen et al., 2013;

Table 1
Criteria for sleep.

(1) Behavior
- Increased threshold for arousal and latency for reactivity (Piéron, 1913)
- Rapid state reversibility to distinguish sleep from hibernation, torpor or coma (Piéron, 1913)
- Physical quiescence (Piéron, 1913)
- Specific body posture (Flanigan, 1973) or behavior during sleep
- Preferred sleeping site (Bruce Durie, 1981)
- Specific pre-sleep behavior (e.g. grooming, yawning, circling)
- Persistence throughout life, possibly specific change of sleep quotas during the life cycle

(2) Homeostatic regulation
- Rebound sleep after sleep deprivation (Tobler, 1983)
- Intensification of sleep after sleep deprivation or a cognitive demanding task (e.g., less fragmented and longer sleep bouts (Huber et al., 2004b), increased SWS in mammals (Borbely, 1982))

(3) Physiological
- Change in heart rate, breathing, body temperature
- Change in muscle tone

(4) Electrophysiological
- Change in specific electrophysiological properties (Kaiser and Steiner-Kaiser, 1983; Nitz et al., 2002; van Swinderen et al., 2004)
- In mammals and birds SWS and REM sleep (Aserinsky and Kleitman, 1953; Dement and Kleitman, 1957; Szymczak et al., 1996)

(5) Pharmacological/endocrinological
- Specific effect of stimulants and hypnotics on sleep and wake (caffeine, amphetamines, antihistamines, benzodiazepines)/(Hendricks et al., 2000; Shaw et al., 2000)
- Change in hormonal signaling

Gray shaded: essential criterion. Adapted from McNamara et al. (2009), Campbell and Tobler (1984), Moorcroft (2003) reviewed in Hartse (2011), Cirelli and Tononi (2008) and Zimmerman et al. (2008).

Eban-Rothschild and Bloch, 2008; Sauer et al., 2003; Tobler and Neuner-Jehle, 1992). Meanwhile, there is compelling evidence that most if not all invertebrates exhibit sleep. Even organisms with nervous systems as simple as that of *C. elegans* appear to display signs of sleep. Sleep in *C. elegans* is apparently associated with periods of developmental transformation, and does not persist throughout life (Iwanir et al., 2013; Raizen et al., 2008). Even though invertebrates can learn and form memories, studies on the role of sleep for memory formation are overall scarce.

4.1. Sleep, extinction learning and spatial navigation in bees

Bees are an outsider among the invertebrate models currently most intensely studied regarding sleep (*Drosophila*, *C. elegans*). There is neither a genetic toolbox nor any knockout strain for bees. Yet, bees possess a highly developed brain with a 10-fold increased number of neurons compared to *Drosophila*. With this brain bees are able to fulfill quite complex navigation and communication tasks (Menzel, 2013). In their natural environment bees need to associate spatial relationships of the landscape to their hive and the sun compass. Additionally they interpret information from the waggle dance (distance and direction), and associate this information with olfactory cues sensed during the dance. At the feeding site, they associate odor, color, shape and spatial location of the feeding ground in relation to nearby landmarks and to the time of the day, quality of reward and quantity of nectar and pollen. Thus, bees form truly episodic-like memory representations about the what, where, and when of experienced events (Menzel, 2012, 2013).

A first study testing effects of sleep on memory in bees (Hussaini et al., 2009) used olfactory reward conditioning of the proboscis extension response (Fig. 5). During training harnessed bees are presented with a neutral odor (conditioned stimulus) followed by a sucrose reward (unconditioned stimulus) and, thus, learn to extend their proboscis in response to the odor. After successful conditioning, presenting the bees repeatedly with the odor without the sucrose reward, leads to the formation of an extinction memory (extinction learning) that mediates the inhibition of the originally conditioned proboscis extension response. Sleep or sleep deprivation after the learning period, did not affect the retention of the conditioned response at a later retest. However, sleep promoted the formation of extinction memory. When the bees were prevented from sleep after extinction learning, they failed to inhibit the originally trained proboscis extension response at a later retest. By contrast, bees that could sleep after extinction training successfully inhibited proboscis extension to the odor on the next day.

Notably, the outcome pattern in bees with sleep selectively benefitting extinction memory, and leaving unaffected original conditioning, corresponds well with findings in mammals where effects of sleep have been mainly examined using fear conditioning paradigms. These studies do not only consistently demonstrate a supporting effect of sleep on the retention of fear extinction learning in rats and humans (e.g., Datta and O'Malley, 2013; Pace-Schott et al., 2012; Spoormaker et al., 2012), but also provide evidence in rats for a selective sensitivity of extinction memory to the effects of sleep, in comparison with the original classical fear conditioning (e.g., Fu et al., 2007; Silvestri, 2005). Extinguishing the original conditioned memory is considered new learning of an inhibitory response, rather than a process erasing the original representation, a view which is also supported by studies in invertebrates (Bouton, 1993; Eisenhardt and Menzel, 2007; Maren, 2011). In mammals, inhibitory learning during extinction involves a complex network of brain structures including, the prefrontal–hippocampal system as a main component. Inhibitory learning in this regard fundamentally differs from classical fear conditioning, which does not critically rely on this system. Specifically the involvement of the prefrontal–hippocampal system during learning is thought to favor access of memories to sleep-dependent consolidation in mammals, and thus explains also that sleep selectively benefits extinction memory over classical conditioned responses (Inostroza and Born, 2013). Which neural circuitries mediate classical conditioning and extinction of the proboscis extension response in bees is currently unknown. Acquisition of a classical conditioned response is thought to occur at the level of the antennal lobe (Giurfa, 2003). Other networks like the mushroom bodies might specifically support extinction memory. The mushroom bodies do not only play a major role in the formation of more complex memory in insects, but have also proved sensitive to the effects of sleep vs sleep deprivation in *Drosophila* (Joiner et al., 2006). Yet, the contribution of the mushroom bodies to the formation of extinction memories in bees has still to be scrutinized.

Employing a navigation task, another study (Beyaert et al., 2012) showed that sleep in honeybees supports the formation of spatial memory, very much in accord with the effects of sleep on spatial memory in mammals (Nguyen et al., 2013; Peigneux et al., 2004). Bees were collected and transferred to an unknown release site from which they had to find a new way back to their hive. On the subsequent night, the bees were either allowed to sleep or were sleep deprived. After being collected again and set free at the same newly learned release site, a significantly greater number of bees of the sleep group found their way back to the hive. Whereas only 58% of the bees were able to find their way home during the first release, 83% of the sleep group and less than 50% of the sleep deprived group mastered the task on the retest run. Additionally, bees after the first exposure to the new release site, slept longer than usual, and this was not an effect of added exhaustion, because bees showing different length of foraging flights did not show differences in sleep duration. Also, sleep deprivation before the forced navigation task did not affect learning on the task. The persistent mapping of a new

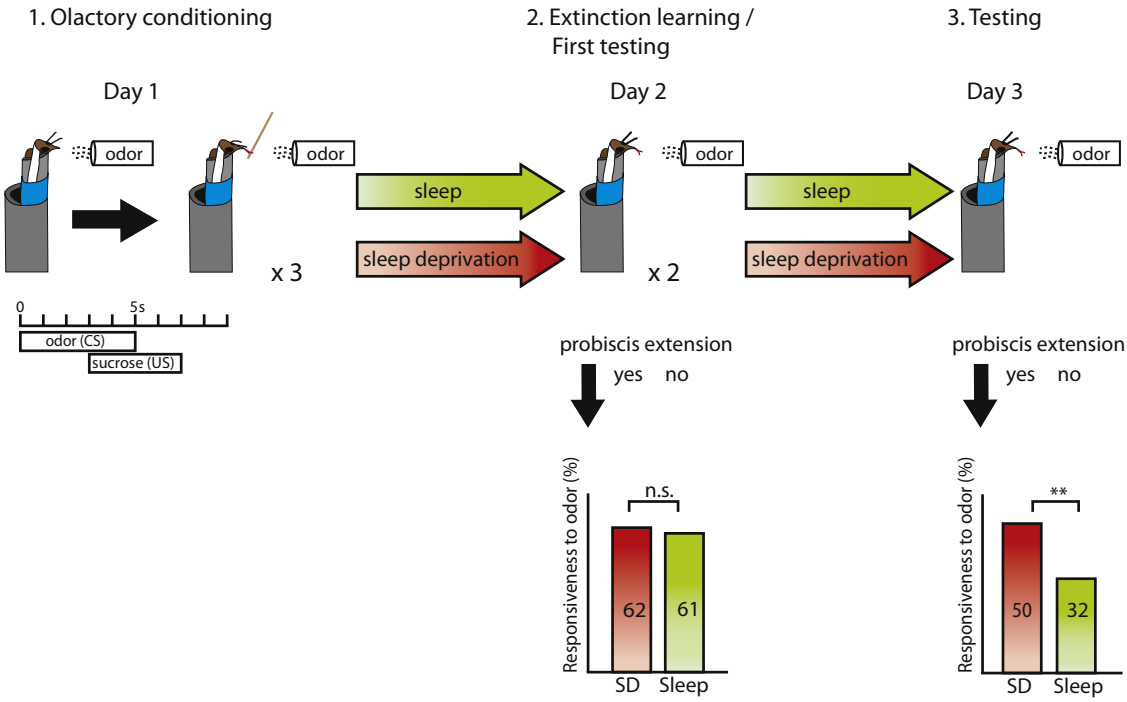

Fig. 5. Sleep in bees benefits extinction of the classical conditioned proboscis extension response, but not classical conditioning of the response itself. On day 1 bees were conditioned on three trials (×3) in which an odor (conditioned stimulus, CS) was shortly followed by the presentation of a toothpick with sugar solution (unconditioned stimulus, US). During the subsequent night, one group of bees was sleep deprived (SD) for 16 h by gentle vibration on a vortex machine every 5 min whereas the other group had undisturbed rest. On the following day animals were tested on two trials (×2) only including the presentation of odor. There was no difference in the proboscis extension response between the two groups. The two test trials simultaneously served as extinction trials, as odor presentation was not rewarded by sucrose. Animals of the original sleep group were then divided into a sleep deprivation group which was deprived from sleep the following night and a sleep group which had undisturbed rest on this night. When re-tested on the subsequent day, the bees of the sleep deprivation condition persisted to respond to the odor, whereas the sleep group successfully suppressed the proboscis extension response. ** $p < 0.01$, n.s., not significant, for comparisons between groups. Data from Hussaini et al. (2009).

flight route into memory is a highly demanding spatial task, which requires the integration of different types of sensorimotor information about the novel flight route as landmarks into the existing spatial navigation memory, and sleep might enhance this type of spatial integration.

4.2. Sleep and memory formation in Drosophila

Considering the rapid increase in studies of sleep in *Drosophila* since the first characterization of sleep-like states in flies (Hendricks et al., 2000; Shaw et al., 2000), surprisingly little work has been devoted to the link between sleep and memory in this species. First indirect evidence for a role of sleep in memory formation in *Drosophila* derived from experiments using enriched environments. *Drosophila* respond with a distinct increase in sleep after exposure to an enriched environment (Bushey et al., 2011; Donlea et al., 2009; Ganguly-Fitzgerald et al., 2006). Such increase in sleep is also observed in the flies after learning of a specific behavior, like the suppression of courtship behavior (Donlea et al., 2009; Ganguly-Fitzgerald et al., 2006). Exposures to enriched environments trigger an increase in the number of synaptic terminals (Bushey et al., 2011; Donlea et al., 2009). In the optic lobe, the increase in the number of synapses and dendritic length returned to baseline values only if the flies were allowed to sleep, in line with the assumption that sleep is involved in synaptic renormalization and homeostatic regulation of synaptic connections (Tononi and Cirelli, 2006, 2014). Also in line with the concept of synaptic renormalization supported by sleep, these studies revealed that the successful acquisition of certain tasks can be hampered if the flies were deprived of sleep for 6–24 h before learning (Li et al., 2009; Seugnet et al., 2008).

Indirect evidence for a link between sleep and memory derived from studies of mutant *Hyperkinetic* flies, which sleep distinctly less than wild type flies, as these mutants also show diminished capabilities to form memory. In a heat box paradigm, in which the two sides of a box are heated to different levels, flies typically prefer the less heated side and maintain the memory for the temperature difference for up to 2 h after the temperature difference is eliminated (Bushey et al., 2007). *Hyperkinetic* mutants rapidly lost their preference for the previously non-heated side, showing little memory. However, rather than on the formation of long-term memory, this study focused on short term memory.

Direct evidence for an effect of sleep on the consolidation of long-term memory in *Drosophila* comes from work on classical aversive olfactory conditioning (Le Glou et al., 2012). Around 50 flies were first exposed to an odor paired with electric shocks, and were subsequently transferred to a second odor in the absence of shocks. Training was repeated 5 times and the flies were then placed in single glass tubes to monitor their activity. For retrieval testing, the flies had to choose (for 1 min) between two arms of a T-maze, each arm equipped with one of the odors. Depriving flies of sleep before training did not produce any memory impairment, whereas 4 h of sleep deprivation after conditioning strongly impaired memory consolidation. At a first glance, this outcome diverges from observations in bees where sleep did not affect classical conditioning of the proboscis extension response (see Section 4.1.). However, unlike in the bees study, in the flies study retrieval was tested in a new context (T-maze) that differed from the context during conditioning training. It might be this transfer testing of the memory in a different context, which unmasks aspects of the representation that are sensitive to the effects of sleep. Also, results differed depending on the time of training and retrieval testing, suggesting an interaction with the circadian rhythm. For example, detrimental effects of

post-training sleep deprivation were abolished when retrieval was placed at the time of circadian peak activity.

Like in bees, more robust effects of sleep in *Drosophila* were revealed for more complex learning tasks, like inhibitory conditioning of courtship behavior (Ganguly-Fitzgerald et al., 2006). In this paradigm, sexually naïve males are situated together with female-pheromone producing males. Due to the pheromones, males chase the female-scented males to start courtship, but without success. After three 1-h training sessions, flies develop long-term memory if left undisturbed with the possibility to sleep for at least 8 h after conditioning, and do not chase other female-scenting males the next two days. However, when flies were sleep deprived for 4 h during an 8-h period after training no such memory was formed. Delayed sleep deprivation 24 h after training did not affect memory formation. Basically, the same effect was observed when flies were 'remotely' put to sleep for 4 h after learning through the temperature-gated expression of a non-specific cation channel in the fan-shaped body, which presumably regulates the rest state in the animals (Donlea et al., 2011). By raising the temperature to 31 °C, the cation channel was expressed and flies entered a sleep-like state that produced long-term memory for the conditioned suppression of courtship behavior. With remote induction of sleep, only one learning trial was sufficient to induce long-term memory, whereas flies that were not 'remotely' put to sleep did not develop significant memory after one learning trial.

5. Further vertebrate and invertebrate models

Some further animal models should be shortly mentioned here, as they might offer promising approaches to the study of sleep's role in memory from a comparative biological perspective, although so far these species have not been examined regarding these issues. Zebrafish is one of these models. For zebrafish quite a number of learning and memory tasks have been established; it sleeps, and it offers the remarkable opportunity for long-term optical live imaging of brain processes at the cellular level, although only during early development until the age of ~25 days (Appelbaum et al., 2010; Norton and Bally-Cuif, 2010). To the best of our knowledge, there is so far just one study in zebrafish that indirectly addressed the link between sleep and memory formation. It was shown that melatonin in these diurnal animals impairs learning of an active avoidance task, in which the animals learned to associate a lighted compartment in the tank as a safe environment (Rawashdeh et al., 2007). However, the study remained inconclusive regarding the issue of sleep-dependent memory consolidation, because it included neither an assessment of sleep manipulations nor an assessment of performance effects with melatonin administered after the task training.

Aplysia represents an invertebrate model that pioneered the research of the electrophysiological and molecular basis of learning and memory (Kandel, 2001; Lee et al., 2008), and it exhibits sleep (Vorster et al., 2014). The features that fostered the success of this model still stand out today: A relatively simple nervous system with approximately 20,000 neurons, which are huge (up to 1 mm), easy to visualize and identifiable. *Aplysia* bears the unique possibility to study the effects of sleep on memory in well-characterized neuronal circuits at the single cell level. Recent experiments provided first hints that operant learning is inhibited by sleep deprivation before and after training in *Aplysia* (Krishnan et al., 2013), and these findings might stimulate research on the mediating molecular mechanism.

Finally, *C. elegans* appears to be a highly attractive model in this context as well, since it offers optimal conditions for the study of essential, conserved genes (Singh et al., 2014). Of particular interest is that sleep in these worms seems to occur only transiently during specific developmental periods (Iwanir et al., 2013; Raizen et al., 2008). These temporal dynamics could provide rather specific insights into the dependence of memory on the presence of sleep. Additionally, the model may offer an approach to study the interaction between sleep-dependent memory formation and maturational processes.

6. Conclusion and perspective

Studies in humans and rodents have consistently shown that sleep supports memory consolidation. This research provided the basis for the concept of active system consolidation during sleep. Simplified, the concept assumes a two-stage process of long-term memory formation, where episodic information is first encoded primarily into an initial store represented by the hippocampus. At a second stage, some of the newly encoded representations are reactivated and redistributed such that representations become predominantly stored in an extra-hippocampal circuitry which serves as long-term store. The redistribution of representations from initial to longer-term-storage sites goes along with a qualitative transformation of the representations, specifically the formation of more generalized and abstract representations, which can be flexibly used independent from the context in which they were originally acquired. Sleep is thought to support this second stage of memory formation in particular through the slow oscillations of SWS.

Here, we have discussed research on the memory function of sleep from a comparative biological perspective in different taxonomic groups, in order to clarify whether this function represents an evolutionarily conserved core function of sleep. Indeed, we found that sleep produced an enhancing effect on memory for all species that were examined.

Although there is still a lack of research on sleep and memory formation in non-mammalian groups, it appears that in birds SWS coevolved to serve the same function. Strikingly, slow oscillatory activity is required during sleep after filial imprinting in chicks for the stable formation of imprinting memory, linking long-term memory with SWS. Furthermore, studies of filial imprinting in chicks and of song learning in song birds suggest the view that sleep supports the redistribution of memories from networks serving as the initial storage of information to different networks serving as a long-term store (summarized in Fig. 2). Additionally, electrophysiological recordings in song birds have suggested that the putative redistribution originates from neuronal reactivations of memory representations during sleep. Although there is no evidence for a separate SWS stage in invertebrates, spatial learning in bees revealed hints that sleep might favor qualitative transformations of newly acquired memory representation. Bees after sleep had a higher probability of returning to their hive from a newly learned place suggesting that sleep enhances integrative spatial mapping. Furthermore, studies in bees demonstrated the selectivity of sleep-dependent memory formation, with sleep strengthening extinction memory but leaving unaffected the original classical conditioned proboscis extension response.

Collectively, these observations indicate that cornerstones of the active system consolidation process during sleep – i.e., neuronal reactivations, redistribution of representations, qualitative transformations, and the selectivity of consolidation – can be identified not only in mammals but also in birds and invertebrates. Thus, the concept might indeed either describe an evolutionarily conserved process of memory formation associated with sleep, or be an example of evolutionary convergence pointing at a fundamental need. If so, then the central question arises: What is the adaptive advantage of using sleep for consolidating memory? Presently, this question cannot be clearly answered. If we assume that the storage capacities

of the nervous system are limited and thus do not suffice for representing the complexity of environmental conditions as a whole, then there is a basic need to reduce information. The transformation of memory during active system consolidation might solve this problem by producing more general, abstract, and schema-like representations that lack contextual detail, and by favoring the formation of memories for the gist of an experience that is relevant to advance future adaptive behavior (Inostroza and Born, 2013; Kumaran and McClelland, 2012; Wilhelm et al., 2011a). Shifting system consolidation to the offline period of sleep basically enables the abstraction of gist memory in conditions unbiased by external stimulus inputs. Because the same networks that are used for system consolidation in a two stage memory system are also used for acute processing of environmental stimuli during wakefulness, ongoing processing of environmental inputs would bear the risk that external stimuli interfere and disturb the effective abstraction of gist from previous experience.

Although the concept of an evolutionarily conserved process of active system consolidation taking advantage of the offline conditions during sleep might appear conclusive, we have to caution that currently the experimental evidence for such a species-independent process is more than meager. A patchwork of findings indicates that each of the essential building blocks of the concept can be identified in one or the other species. However, with the exception of rats and humans, there is no single species for which a larger coherent set of data exists in support of the theory. For example, in song birds neuronal reactivations of song representations occur during sleep, but it is unknown whether they play a causal role in the consolidation process. In bees, first hints for memory transformations occurring during sleep have been revealed, but it is unknown whether this is a consequence of neuronal reactivations during sleep, etc. These and many further questions of this kind disclose the obvious gaps that are to be filled by future research.

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