Neuroscience and Biobehavioral Reviews 140 (2022) 104799

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review article

Is the role of sleep in memory consolidation overrated?

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ARTICLE INFO

Keywords:
Sleep
Memory consolidation
Electroencephalogram (EEG)
Reactivation
Replay
Wakefulness
Synaptic plasticity

ABSTRACT

Substantial empirical evidence suggests that sleep benefits the consolidation and reorganization of learned information. Consequently, the concept of "sleep-dependent memory consolidation" is now widely accepted by the scientific community, in addition to influencing public perceptions regarding the functions of sleep. There are, however, numerous studies that have presented findings inconsistent with the sleep-memory hypothesis. Here, we challenge the notion of "sleep-dependency" by summarizing evidence for effective memory consolidation independent of sleep. Plasticity mechanisms thought to mediate or facilitate consolidation during sleep (e.g., neuronal replay, reactivation, slow oscillations, neurochemical milieu) also operate during non-sleep states, particularly quiet wakefulness, thus allowing for the stabilization of new memories. We propose that it is not sleep per se, but the engagement of plasticity mechanisms, active during both sleep and (at least some) waking states, that constitutes the critical factor determining memory formation. Thus, rather than playing a "critical" role, sleep falls along a continuum of behavioral states that vary in their effectiveness to support memory consolidation at the neural and behavioral level.

1. Introduction

Over the last decades, a substantial body of empirical work has accumulated to suggest that sleep plays an important and, by some accounts, critical or essential role in the process of memory consolidation (Marquet, 2001; Walker and Stickgold, 2004; Stickgold, 2005; Diekelmann and Born, 2010; Rasch and Born, 2013; Chatburn and Lushington, 2014; Diekelmann, 2014; Schmid et al., 2020). The view that sleep is "pivotal" (Inostroza and Born, 2013, p. 79), "causal" Pocivavsek and Rowland (2018), p. 7), or "essential for memory formation" (Feld and Born, 2020, p. 31) is now widely accepted by the sleep research community, even though an active discussion continues regarding the mechanisms and sleep stages that mediate the so-called "sleep-dependent" consolidation of different classes (e.g., declarative, procedural) of memories. Further, while at least some in the sleep-memory field acknowledge that sleep may not be the only behavioral state that allows for consolidation to occur (e.g., Stickgold, 2005; Mednick et al., 2011; Wamsley, 2019), it is often assumed that sleep provides the ideal conditions and makes unique contributions to memory formation that are not shared by other (non-sleep) arousal states (e.g., Walker and Stickgold, 2004; Diekelmann and Born, 2010; Inostroza and Born, 2013; Feld and Born, 2020; Schmid et al., 2020).

Given the current state of the field, it might come as a surprise that there continues to be an ongoing debate regarding the "sleep-memory consolidation hypothesis" (Siegel, 2001; Benington and Frank, 2003; Vertes, 2004; Vertes and Siegel, 2005; Mednick et al., 2011; Dringenberg, 2019; Vertes and Linley, 2021). Important questions and controversial topics include: *Does the role of sleep in memory consolidation meet the criteria of* ***necessity*** *and/or* ***sufficiency****? Is sleep's contribution to consolidation* ***active*** or ***permissive****? Are there* ***states of wakefulness*** *that mimic the effects of sleep to allow for the effective consolidation of new information?*

In this paper, we will discuss these and related issues, with a particular focus on the questions of the necessity and sufficiency of sleep for memory consolidation processes. The empirical evidence reviewed below suggests that sleep is neither necessary nor sufficient, and that other (non-sleep) behavioral states also provide conditions for the effective consolidation of new information. Consequently, rather than ascribing a "special" or "critical" role, it seems prudent to view sleep along a continuum of behavioral states that vary in their effectiveness to support memory consolidation at the synaptic, systems, and behavioral level.

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https://doi.org/10.1016/j.neubiorev.2022.104799
Received 11 March 2022; Received in revised form 13 June 2022; Accepted 25 July 2022
Available online 26 July 2022

2. Consolidation mechanisms are active during sleep and wakefulness

In its most direct form, the sleep-memory consolidation hypothesis states that, after a learning experience, a period of sleep will benefit the consolidation and, hence, retrieval of information to a greater extent than an equivalent period of wakefulness (Stickgold, 2005). Importantly, rather than merely reducing interference by novel waking experiences, sleep is thought to *actively* promote memory consolidation (Marquet, 2001; Walker and Stickgold, 2004; Stickgold, 2005; Diekelmann and Born, 2010; Inostroza and Born, 2013; Rasch and Born, 2013; Diekelmann, 2014). This active process is assumed to be mediated by dedicated neural plasticity mechanisms that operate, either exclusively or predominantly, during sleep, explaining the apparent superiority of sleep over wakefulness to support consolidation. Important consolidation mechanisms include replay/reactivation of learning-related neuronal activity, slow wave and spindle activity, as well as changes in the hormonal and neurochemical milieu. In the following section, we will summarize evidence that these mechanisms are not specific to sleep; in fact, in many cases, sleep is not required for these mechanisms to actively promote memory consolidation.

2.1. Neuronal replay and reactivation

One of the most influential hypotheses regarding the mechanism of consolidation is the notion that replay/reactivation of learning-related neural activity during sleep acts to strengthen synaptic connectivity in neural engrams and, thus, memory storage (Peigneux et al., 2004; O'Neill et al., 2010; Margoliash and Brawn, 2012; Buzsáki, 2015). Neuronal replay was first observed in the rat hippocampus, where patterns of place-cell activity during spatial exploration re-emerged during subsequent slow-wave sleep (SWS; Wilson and McNaughton, 1994; Kudrimoti et al., 1999; Lee and Wilson, 2002; Diba and Buzsaki, 2007; Ji and Wilson, 2007). The strongest support for a role of replay in memory consolidation is provided by studies that directly manipulated replay events by electrical or optogenetic stimulation in rodents. This work has demonstrated facilitations or impairments of spatial memory recall following the enhancement or disruption, respectively, of replay during SWS (Girardeau et al., 2009; Ego-Stengel and Wilson, 2010; McNamara et al., 2014). These data suggest that, rather than being a consequence of learning, neuronal replay plays a causal role in memory formation.

While neuronal replay constitutes a prime candidate to support information storage, it is far from clear whether this function requires the state of sleep. In the rodent hippocampus, replay occurs during a specific state of local field potential activity termed sharp-wave ripples (SWRs; Buzsáki, 2015). It has been known for decades that, in rats, SWRs are prominent not only during SWS, but also wakefulness, specifically during behaviors such as waking immobility and various consummatory (also termed involuntary or Type 2) behaviors including chewing, licking, and grooming (Mednick et al., 2011; Vanderwolf, 1969, 1988). In non-human primates, hippocampal SWRs occur during quiet waking and active visual exploration (Leonard et al., 2015; Hussin et al., 2020). In the human hippocampus, SWRs have been recorded not only during napping, but also, and with a high frequency, wakefulness, particularly quiet rest (Axmacher et al., 2008).

Given the direct link between the occurrence of SWRs and neuronal replay, it is not surprising that replay events are also not confined to episodes of sleep, as was initially assumed. In fact, numerous reports of replay during wakefulness have accumulated over the last two decades. Kudrimoti et al. (1999) were perhaps the first to compare hippocampal place cell replay during SWS and waking. These authors observed similar levels of neuronal replay during SWS and quiet (largely immobile) waking, leading to the conclusion that "sleep per se is unnecessary for trace reactivation" and that "the presence of ripples may be a sufficient condition for pattern reactivation" to occur (Kudrimoti et al., 1999, p. 4098). Subsequent reports confirmed that, following spatial exploration, hippocampal place cells frequently exhibit episodes of replay during waking, particularly waking immobility (Foster and Wilson, 2006; Carr et al., 2011). In fact, detailed comparisons of replay events during wakefulness and sleep in rats showed that replay sequences during waking are stronger and more accurately match activity during a preceding spatial learning session than sequences during sleep (Karlsson and Frank, 2009; Carr et al., 2011; Tang et al., 2017). Thus, awake replay may be particularly well suited for the accurate consolidation and retention of memories in hippocampal and neocortical networks.

Awake replay events are frequent during the early phases of behavioral training (Tang et al., 2017) and, importantly, predict subsequent spatial memory strength (Dupret et al., 2010). Interestingly, awake replay is not restricted to the current spatial environment or recent experience, but can also encompass spatially remote, past experience (Karlsson and Frank, 2009; Carr et al., 2011). These observations are important since they suggest that replay during waking supports not only the initial storage of new spatial information, but also the continued consolidation of older experiences. Critically, a number of investigations have now demonstrated that disrupting SWR-associated replay during wakefulness (either during or following behavioral training) by means of electrical or optogenetic stimulation impairs learning and final task performance (Ego-Stengel and Wilson, 2010; Jadhav et al., 2012; Nokia et al., 2012). Recent experiments by Fernández-Ruiz et al. (2019) have extended this work by showing that, in addition to SWR-replay disruption impairing task acquisition, optogenetic prolongation of SWR-replay events during wakefulness actually facilitated task acquisition, an effect related to the recruitment of additional neurons in the CA1-area with place fields on the maze. These results, together with prior work, provide compelling support for the contention that SWRs and neuronal replay constitute plasticity mechanisms that serve to store memories during the waking state (Carr et al., 2011; Mednick et al., 2011; Dringenberg, 2019), even though this does not, of course, negate that they can exert similar effects during SWS, as initially postulated.

The detection of cellular replay requires intracranial recordings, which cannot be performed in healthy humans. However, a number of noninvasive recording techniques have been used to demonstrate that areas of the human brain that are active during an initial learning experience show similar patterns of activation during subsequent sleep. Ground-breaking work by Peigneux et al. (2004) detected activation of the hippocampal formation during spatial training in a virtual 3D environment, which re-emerged during SWS. Importantly, there was a positive correlation between reactivation during sleep and subsequent memory recall performance, consistent with an active role of reactivation in consolidation processes (Rasch and Born, 2013). However, similar to neuronal replay, reactivation is not restricted to periods of sleep. Electrocorticographic recordings in epileptic patients during and after visual stimulation showed reactivation of visually induced activity during quiet, alert wakefulness (Chelaru et al., 2016). Functional magnetic resonance imaging (fMRI) in neurologically healthy humans exposed to object-scene pairs revealed spontaneous reactivation during wakefulness in the hippocampus and anatomically connected cortical areas (e.g., entorhinal cortex; Staresina et al., 2013), as well as in visual cortex and the ventral visual stream (Deuker et al., 2013; Bang et al., 2018). Importantly, reactivation reflecting prior learning can be present even when participants engage in cognitively demanding distractor tasks to prevent rehearsal (Peigneux et al., 2006). Further, investigations have repeatedly shown that recall performance correlates with the number or accuracy of reactivation episodes during waking (Peigneux et al., 2006; Deuker et al., 2013; Staresina et al., 2013; Bang et al., 2018; Schapiro et al., 2018). It is also noteworthy that at least some evidence suggests that reactivation events in humans may be most frequent during wakefulness and become less common during successively deeper sleep stages (Deuker et al., 2013), observations that are not consistent with a critical role of sleep in memory consolidation.

Recent work has extended earlier correlational studies by directly manipulating reactivation events in humans. Tambini and D'Esposito (2020) applied transcranial magnetic stimulation (TMS) to disrupt post-learning reactivation activity in hippocampal-neocortical (lateral occipital cortex) networks during quiet wakefulness. Recall was impaired following TMS, indicative of a causal contribution of reactivation during waking to episodic memory consolidation.

The evidence summarized above demonstrates that SWR complexes, neuronal replay, and functional reactivation occur during both sleep and wakefulness. Among different waking states, quiet waking and a reduction of motor activity appear to constitute optimal (but not exclusively so) conditions for the activation of replay-related consolidation mechanisms. Most critically, similar results have been obtained in studies testing the contributions of replay/reactivation during sleep and wakefulness to memory: their spontaneous occurrence predicts memory strength, while their disruption impairs memory. These results are in line with suggestions that SWRs and the associated replay constitute general consolidation mechanisms that can operate during wakefulness and sleep (Foster and Wilson, 2006; Carr et al., 2011; Tang and Jadhav, 2019). Of course, this hypothesis does not preclude the possibility that wake- and sleep-related replay perform partially distinct or complimentary memory functions, as is currently being discussed in the literature (Tang and Jadhav, 2019; Findlay et al., 2020).

2.2. EEG slow oscillations

A number of early, groundbreaking studies suggest that SWS, rather than lighter sleep or REM sleep, supports the consolidation of declarative memories (Plihal and Born, 1997, 1999; Gais and Born, 2004b). Deeper sleep stages are characterized by the appearance of prominent slow activity patterns in the electroencephalogram (EEG), particularly in the delta (1–4 Hz) and slow oscillation (< 1 Hz) frequency bands (Achermann and Borbély, 1997; Borbély and Achermann, 1999; Vyazovskiy et al., 2009; Crunelli et al., 2018). Low-frequency activity can be highly effective in inducing synaptic plasticity, including long-term potentiation (LTP; Habib and Dringenberg, 2010; Miyamoto et al., 2017; Timofeev and Chauvette, 2017; Crunelli et al., 2018), offering a potential mechanism linking slow wave activity to memory consolidation. Indeed, increased slow wave activity during sleep following a training session correlates with subsequent recall performance (Huber et al., 2004). Further, electrical or TMS to enhance slow activity during sleep improves memory consolidation in rodents (Binder et al., 2014; Miyamoto et al., 2016) and humans (Marshall et al., 2006; Ladenbauer et al., 2016), indicative of a causal contribution of slow EEG oscillations to memory storage.

While there is no doubt that slow EEG activity is most pronounced during deeper sleep stages, brief episodes of anatomically restricted low-frequency events also occur during wakefulness. In the rodent neocortex, localized slow EEG oscillations emerge during waking immobility, similar to the appearance of hippocampal SWR described above (Petersen et al., 2003; Crochet and Petersen, 2006; Polack et al., 2013; Zagha et al., 2013). Surprisingly, slow waves in rodents may even occur during the performance of attention-demanding task Einstein et al. (2017)) or periods of active locomotion (wheel running) (Fisher et al., 2016). Anatomically restricted incidents of low frequency activity during waking have also been detected in non-human primates (Tan et al., 2014) and humans (Assenza et al., 2015; Sachdev et al., 2015). Localized low-frequency EEG activity, coupled to a profound suppression of firing of neocortical neurons, becomes more prominent after periods of prolonged waking, a phenomenon termed "local sleep" (Vyazovskiy et al., 2011; Rattenborg et al., 2012; Bernardi and Siclari, 2019). Local sleep occurs against a background of activated EEG- and neuronal activity, as well as clear behavioral wakefulness, even though performance on attention-demanding tasks is impaired when local sleep episodes become more frequent (Vyazovskiy et al., 2011). The characterization of local sleep serves to highlight the fact that sleep and waking states are more fluid and continuous than initially thought; low frequency EEG activity is compatible with behavioral wakefulness, in agreement with the argument that the EEG does not serve as an entirely unambiguous index of arousal states (Vanderwolf and Robinson, 1981; Vanderwolf, 1988).

As mentioned above, low-frequency activity (delta and the slow oscillation) can be a potent inductor of synaptic plasticity and has been suggested to gradually shift the dependency of memories from the hippocampus toward the neocortex (Klinzing et al., 2019; Oyanedel et al., 2020). Can slow waves during wakefulness exert similar effects on memory processes? It appears that this question has been tackled by only a small number of experiments. Kirov et al. (2009) employed a low-frequency transcranial stimulation protocol previously shown to enhance slow oscillations and memory consolidation when applied to sleeping humans (Marshall et al., 2006). When delivered during waking, stimulation improved immediate recall performance, but was ineffective over longer retention intervals, suggestive of a selective facilitation of encoding, but not memory consolidation (Kirov et al., 2009) However, procedural differences such as the time interval between training and subsequent stimulation (20 min during waking in Kirov et al., 2009 vs. > 30 min during sleep in Marshall et al., 2006) complicate a direct comparison between these studies.

Brokaw et al. (2016) used a correlational approach to examine potential EEG predictors of memory consolidation in humans over a period (15 min) of quiet, restful waking. Relative to active wakefulness (playing video games), quiet rest (awake with eyes closed) resulted in enhanced memory recall, an effect that correlated with slow activity in the 0.3–1 Hz range over the frontal cortex. The results obtained by Brokaw et al. (2016) support two intriguing conclusions: (a) quiet wakefulness, like sleep, can enhance memory consolidation in humans; and (b) slow EEG oscillations during wakefulness may contribute to consolidation, similar to their functions during sleep (note that a replication of this study by Humiston et al., 2019 failed to detect a statistically significant boost of recall by quiet waking, even though their effects fell within the 95% confidence interval of the results obtained by Brokaw et al.).

Of course, correlational evidence is not sufficient to establish a direct contribution of slow oscillations during waking (or any behavioral states) to consolidation processes. Thus, Miyamoto et al., (2016, 2017) used low-frequency optogenetic stimulation to elicit slow EEG activity in mice trained on a tactile discrimination task. Remarkably, stimulation enhanced long-term (4 days) memory recall regardless of whether it was delivered during sleep or wakefulness; note that mice were sleep-deprived, but kept awake during the stimulation period (Miyamoto et al., 2016). Based on these observations, Miyamoto et al. (2017) argue that slow oscillations, but not sleep itself, are the critical driver of memory consolidation.

Together, the evidence summarized above, mostly focused on declarative memories in humans, indicates that slow oscillations, along with other consolidation mechanisms (e.g., replay, SWRs), occur during both sleep and wakefulness brings into question the extent to which behavioral sleep is truly necessary for effective memory consolidation to proceed.

2.3. The hormonal and neurochemical milieu

The physiological mechanisms discussed in the previous sections (SWR, cellular replay, slow waves) are thought to play an *active* role in memory consolidation, that is, they directly initiate or contribute to the storage or transfer of information among memory networks of the forebrain. Active memory mechanisms likely work on concert with a number of secondary, permissive factors that aid information storage without making direct contributions to this process. Permissive factors known to facilitate memory consolidation during sleep include the hormonal and neurochemical milieu.

The first hours of nightly SWS in humans are characterized by the lowest levels of cortisol release (Weitzman et al., 1983; Plihal and Born,

1997). Interestingly, the same sleep period is also preferentially involved in promoting declarative memory consolidation (Plihal and Born, 1997, 1999), prompting the question whether reduced cortisol levels play a role in this effect. Indeed, infusions of cortisol or a glucocorticoid receptor agonist to prevent the trough of cortisol activity during the first half of the night abolished the effectiveness of SWS to enhance consolidation (Plihal and Born, 1999; Plihal et al., 1999). It is imperative to emphasize that these pharmacological manipulations did not affect the behavioral and EEG characteristics of sleep, that is, sleep architecture was equivalent in the drug and vehicle conditions, but consolidation was facilitated only in the vehicle group (Plihal and Born, 1999; Plihal et al., 1999). The most parsimonious interpretation of this dissociation between (behavioral, EEG) sleep and memory is that it is the hormonal milieu, but not sleep itself, that acts as the determining factor for successful consolidation of declarative information.

Experiments very similar to those summarized for cortisol were also conducted for the important sleep-wake related neuromodulator acetylcholine (ACh). Like cortisol, central ACh release is closely related to sleep-wake states, with the lowest levels of ACh in the forebrain seen during SWS (Kametani and Kawamura, 1990; Marrosu et al., 1995). In humans, preventing cholinergic hypofunction during SWS by administration of an acetylcholinesterase inhibitor reversed the effect of SWS to boost declarative (word pair) memory consolidation, an effect that can occur in the absence of alterations to polysomnographic sleep parameters (Gais and Born, 2004a). Remarkably, blocking cholinergic transmission with combined muscarinic and nicotinic receptor antagonist administration during wakefulness enhances declarative memory consolidation to the same extent as sleep (Rasch et al., 2006). In other words, low ACh activity in humans boosts memory consolidation completely independent of sleep.

The studies reviewed above point to an important role of the hormonal and neurochemical milieu in the consolidation of declarative information in humans. Unfortunately, relatively little information is currently available whether such effects extend to non-human species and other types of memories. In one of the few studies attempting to address these questions, acetylcholinesterase inhibition in mice or direct activation of muscarinic ACh receptors during sleep impaired motor memory consolidation (Inayat et al., 2020), but the interpretation of these results is complicated by significant alterations in sleep structure in drug-treated animals. As a control for sleep disturbances, the authors subjected mice to complete sleep deprivation after motor training. The fact that this manipulation did not affect motor performance led the authors to conclude that the prevention of low ACh activity, rather than sleep alterations, constitutes the most important factor hindering motor memory consolidation (Inayat et al., 2020). However, it is clear that more work is required to delineate the possible functions of ACh and other neurochemicals/hormones in the consolidation of non-declarative memories during different behavioral states.

The work summarized above is consistent with the view that it is not sleep itself, but the specific neurochemical/hormonal environment during the post-training period that acts as the *critical determinant* of the degree of memory formation and retention, at least with regard to declarative information in humans. Sleep after learning loses its effectiveness to sustain consolidation when some of its hormonal and/or neurochemical correlates are blunted. In this context, it is important to note that levels of many so-called "sleep-related" neuromodulators and other hormones also vary dynamically and significantly over the waking period. For example, the release of ACh is closely related to concurrent behavior, with significantly elevated ACh levels seen during active locomotion and exploration, sensory stimulation, high levels of attention and arousal, stress, and the performance of cognitively demanding tasks (Pepeu and Giovannini, 2004; Parikh et al., 2007; Zhang et al., 2021). Conversely, periods of reduced activity and cognitive engagement are characterized by a pronounced decline in ACh output, in parallel with the increase in SWRs (Zhang et al., 2021), replay events, and slow oscillations (see above). Many of the major neuromodulators (e.g., serotonin, adrenaline/noradrenaline, dopamine, cortisol) similarly exhibit dynamic fluctuations during different periods of wakefulness, in addition to circadian and sleep-related alterations in their release levels (for examples, see Kirschbaum et al., 1991; Jacobs and Fornal, 1997; Goto et al., 2007; Xiang et al., 2019). Given these fluctuations, and the well-established role of the neurochemical and hormonal milieu, it appears likely that there are numerous "windows of opportunity" for plasticity mechanisms to induce memory storage in the absence of sleep.

3. Quiet wakefulness and memory consolidation

As discussed, behavioral immobility (in rodents) and quiet/restful waking (in humans) constitute states characterized by the presence of SWR, neuronal replay/reactivation, as well as low levels of ACh and stress-related hormones. Thus, like sleep, quiet waking (QW) may also provide conditions for effective memory consolidation. A number of studies have investigated this hypothesis and generally confirm that, relative to active waking (AW), a period of QW after learning leads to better recall of learned material. In humans, recall for word-pairs (Dewar et al., 2007), stories (Dewar et al., 2012; Brokaw et al., 2016), spatial/navigational information (Craig et al., 2015, 2016), and motor skills (Humiston and Wamsley, 2018) is superior when learning is followed by a period of QW relative to AW. A recent meta-analysis confirmed this pattern of results, albeit with the caveat that the effect of QW on memory, while reliable and significant, is relatively small (Humiston et al., 2019). It is challenging to induce QW in rodents. However, recent evidence indicates that placing rats in a quiet, dark enclosure ("black box") to reduce sensory stimulation for 1 h after training in an object-location task leads to better memory recall than AW in the animals' home cage (note that rats in the "black box" were not allowed to sleep; Arkell et al., 2021). Together, these studies show that ongoing cognitive activity and/or sensory stimulation after learning disrupt recall, consistent with the notion that QW is a state that benefits memory consolidation (Wamsley, 2019).

Is QW as effective as sleep in promoting memory formation? Table 1 summarizes the results of the relatively small number of studies that have compared recall performance following sleep (napping) to both AW and QW. Gottselig et al. (2004), as one of the first to include a QW condition, found that a two-hour period of sleep or QW (lying in the dark) were equally effective in consolidating auditory learning, and both conditions were superior to AW (watching a movie). The authors conclude that "…conditions that accompany restful waking are sufficient to facilitate learning…" (Gottselig et al., 2004, p. 557). Similar results were obtained by Wang et al. (2021), who found that recall for

Table 1
Experimental studies in humans comparing memory consolidation over periods of sleep (S), active wakefulness (AW), and quiet wakefulness (QW).

Study	Memory task	Consolidation[a]
Gottselig et al. (2004)	auditory learning	S = QW > AW
Wang et al. (2021)	word pairs & motor learning	S = QW > AW
Piosczyk et al. (2013)	word pairs	S = QW = AW
	for S with high sigma activity	*S > QW = AW*
McDevitt et al. (2014)	visual learning	S = QW = AW
	for S with high REM sleep	*S > QW = AW*
Landry et al. (2016)	motor sequence	S = QW = AW
Maier et al. (2017)	motor sequence	S = QW = AW
Mednick et al. (2009)	visual search	S = QW = AW
Simor et al. (2019)	statistical & sequence learning	S = QW = AW
Tucker et al. (2020)	word pairs & concept learning	S = QW = AW
Schönauer et al. (2014)	declarative test battery	S > QW = AW
Schreiner and Rasch a, b) (2015)	cued memory reactivation	S > QW = AW

Note: [a] Memory consolidation is expressed as ranked recall performance following the experimental intervention (S, AW, or QW). See individual studies for methodological details.

declarative (word pairs) and non-declarative (motor sequence) memory showed similar benefits following a 30 min period of either sleep or QW (rest with closed eyes); again, participants in both conditions outperformed those in the AW group (distractor task). Wang et al. (2021) conclude that "…neurobiology specific to sleep might not be necessary to induce the consolidation of memory…" (p. 195). Another investigation comparing motor memory (motor pursuit) following napping and QW (listening to instrumental music for 90 min) also found equivalent recall performance in both conditions; unfortunately, AW was not examined in this work (Rieth et al., 2010).

Other work has confirmed that sleep and QW may similarly support memory, but also points to the role of some specific sleep characteristics that, when present, may further boost consolidation. Recall for word pairs was similar following a 60-min interval that contained a nap or QW (lying awake in the dark), as well as AW (physical activity, movies; Piosczyk et al., 2013). However, in nappers, higher EEG power in the sigma range (12–16 Hz) correlated with better recall, suggesting that the presence of specific EEG patterns may be critical for sleep to enhance memory storage. Similarly, visual perceptual learning shows equivalent improvements over a two-hour interval filled with napping or QW (sitting and listening to music), but not with AW (daily activities; McDevitt et al., 2014), again supporting the notion that QW may exert effects similar to those seen with sleep. In addition, a subset of participants in the nap condition who entered REM sleep showed the greatest performance gain (McDevitt et al., 2014). Thus, there may be specific processes operating during sleep that, when present, make this state particularly effective in supporting consolidation.

It is noteworthy that a majority of studies (7 out of 11) summarized in Table 1 have been unable to detect differences in memory recall following periods of sleep overall, AW, and one of several QW conditions (listening to music in Mednick et al., 2009 and McDevitt et al., 2014; lie awake in bed in a dark room in Piosczyk et al., 2013 and Maier et al., 2017; sit upright in bed without activity in Landry et al., 2016; sit quietly with closed eyes in Simor et al., 2019 and Tucker et al., 2020; see Table 1 for details regarding the role of specific sleep characteristics in Piosczyk et al., 2013 and McDevitt et al., 2014). These results that can be interpreted in several ways: it is possible that the specific experimental procedures and tests were insensitive to detect effects of QW and sleep. Alternatively, the fact that similar levels of recall were observed in all sleep- and wake conditions can also be taken as evidence that sleep's function to support consolidation is not nearly as pronounced and reliable as is often implied by most of the literature in the field. This argument is countered, however, by some evidence that, indeed, suggests a superior role of sleep in memory consolidation compared to both AW and QW. Schönauer et al. (2014) assessed several types of declarative memory following a 2-hour consolidation period filled with either sleep (nap), AW, or meditation as a specific type of QW. Recall performance was significantly better following sleep compared to AW and QW, which did not differ from one another. In addition, memory reactivation techniques (i.e., the explicit cueing of learned stimuli during post-learning time windows to trigger reactivation events; Klinzing and Diekelmann, 2019) appear to be effective in boosting recall when applied during sleep, but not during either AW or QW (Schreiner and Rasch, 2015a; b). Reactivation experiments rely on tightly controlled experimental techniques and, as such, do not necessarily provide insights into the natural, spontaneous memory functions of sleep and wakefulness. Nevertheless, this work does point toward some privileged role of sleep in allowing reactivation events and consolidation to proceed.

The results summarized in Table 1 provides some intriguing insights into the role of sleep and wakefulness in memory consolidation. First, and remarkably, the majority (9/11) of studies do not show an advantage of sleep in general over waking (either QW, AW, or both). Further, for studies (4/11) that demonstrate superior consolidation following sleep relative to AW, the sleep benefit disappears in 50% of the cases (i.e., 2/4) when QW is used as a comparison condition. Given that the large majority of published work has compared sleep to AW (Mednick et al., 2011; Brokaw et al., 2016; Wamsley, 2019), it is conceivable that the apparent memory benefits of sleep over waking (i.e., AW) are much smaller, or non-existent, if QW would have been included as an additional experimental condition. Thus, we strongly recommend the routine inclusion of a QW condition in studies on sleep and memory consolidation, particularly when shorter sleep periods (e.g., naps) are employed. It is also important to stress that the studies summarized in Table 1 use a variety of QW conditions (e.g., lying in the dark, listening to music, meditation) that may differ in their effectiveness to support consolidation processes; thus, a systematic analysis and comparison of different QW activities is also warranted.

Despite the arguments advanced above, it is important to acknowledge that some evidence (2/11 studies) suggest that there are specific sleep characteristics (sigma activity in Piosczyk et al., 2013; features associated with REM sleep in McDevitt et al., 2014) that may be particularly beneficial for memory consolidation, making sleep rich in those features superior to all forms of waking. On balance, however, the work summarized in Table 1 comparing sleep, AW, and QW clearly does not offer consistent support for a generally superior or "essential" function of sleep in consolidation processes.

3.1. *Meditation as one type of quiet wakefulness*

There has been a growing interest in the scientific study of meditation, given the multitude of cognitive and health benefits associated with meditative practices (Lomas et al., 2015; Tang et al., 2015; Lee et al., 2018; Tortella et al., 2021). For the purpose of the present review, meditation is of interest since it constitutes a particular type of QW that shows some neurophysiological parallels to sleep, such as increased power in the alpha and theta frequency bands, some of the EEG hallmarks of meditative states (Lomas et al., 2015; Lee et al., 2018). Behaviorally, meditation is characterized by reduced attention to, and awareness of the external sensory environment, together with reduced motor activity and signs of autonomic and hormonal relaxation, also mirroring changes seen during wake-sleep transitions. Thus, the question arises whether meditation benefits memory consolidation, and how such an effect may compare to those seen with sleep and various types of wakefulness. In addition, and from a practical standpoint, the use of meditation can ameliorate some of the methodological challenges associated with assigning participants to a QW condition, such as excessive boredom and fidgeting, falling asleep, and mental rehearsal of study items.

Does meditation facilitate memory consolidation? At present, the available evidence to answer this question is sparse and inconsistent. As discussed above, Schönauer et al. (2014) studied a group of experienced meditators to assess the effect of reduced interference during QW (a two-hour meditation session involving focused attention on a mantra or their breathing) or in the declarative memory consolidation. The results did not show significant differences between meditation and an AW condition, and participants in both groups experienced more memory decay than those in a nap condition. More recently, Collins and Wamsley (2020) found that breath-focused mindfulness meditation (for 10 min) was as effective as QW in supporting verbal memory consolidation. Unfortunately, AW and sleep were not examined in this study, even though this group has previously detected superior recall following QW relative to AW (Brokaw et al., 2016; Humiston and Wamsley, 2018; Humiston et al., 2019). Interestingly, participants who were more attentive to their breath during meditation showed increased forgetting compared to those less able to maintain breath focus (Collins and Wamsley, 2020). Thus, the style of meditation, the level of experience, and other factors related to the specifics of meditative practice likely play important mediating effects for the success of memory consolidation during this behavioral state.

Some evidence offers more direct support for a role of meditation in memory formation. Immink (2016) found that, in experienced

meditators, a 30-min yoga nidra meditation session (a cognitively engaged type of meditation involving attention to bodily sensations and visual imagery) resulted in enhanced motor memory consolidation relative to an AW condition (light physical work). In unpublished experiments (Dastgheib, 2020), we have assessed the effects of a 60-min post-learning period of self-guided mindfulness meditation, often conceptualized as focused attention on, and monitoring of, bodily sensations (Lomas et al., 2015). Declarative memory (word-pair) recall of participants in the meditation condition was superior to that of participants in an AW (watching a video) or a nap condition (Fig. 1A and B). A more detailed analysis of the meditation group revealed a small-to-moderate positive correlation between word recall and the time spent in a meditative state over the 60-min meditation opportunity (Fig. 1C; meditative state was defined as 30-s EEG epochs with a $\geq$ 50% increase over baseline of power in the alpha-theta frequency range, a hallmark EEG characteristic of meditation; Lomas et al., 2015; Lee et al., 2018).

The studies summarized above offer some preliminary support for a positive influence of meditation on the consolidation of specific types of memories. At the same time, more work is required to resolve discrepancies in the literature and develop a better understanding of the factors that account for the divergent results: the type, duration, and timing (relative to training and recall testing) of meditation, the level of experience with meditation, and the type of memory assessed are among the most obvious variables that likely influence the outcome of meditation on memory recall. Future work that systematically controls and manipulates these and other variables will provide a more comprehensive picture of how meditation, as one type of QW, influences consolidation over a wide range of time periods.

4. Implications and future directions

The concept of "sleep-dependent" memory consolidation has become deeply ingrained in the literature on the cognitive functions of sleep. A recent review by Feld and Born (2020) commences with the statement "Sleep is essential for memory formation." (p. 31). This view clearly implies that, in the absence of sleep, memory consolidation is impossible or, at the least, strongly compromised.

In contrast to this prevailing narrative, we summarize evidence to advance the argument that successful consolidation occurs during sleep and wakefulness (Mednick et al., 2011; Wamsley, 2019). Many of the plasticity mechanisms and conditions (neurochemical, hormonal) that support information storage at the neural level are active during waking, particularly QW and immobility. Behavioral studies demonstrate enhanced recall of information following QW relative to AW; at least in some instances, memory recall after QW is equivalent to that following sleep. Together, these findings clearly contradict the view that sleep is "essential" for the process of memory consolidation. Interestingly, some strong proponents appear to agree that the assumed "critical/essential" role of sleep does not hold up to the available evidence. Robert Stickgold (2005) rightfully points out that "…most sleep-related processes can occur during periods of wakefulness and vice versa…" (p. 1273), a view that closely matches the arguments advanced here: consolidation can take place during sleep and waking, making it neither sleep- nor wake-dependent.

It is not surprising that there are a number of open questions and considerations that remain. The majority of experimental evidence reviewed above has focused on the consolidation of declarative information in humans. As such, it will be critical for future work to include a much wider range of species and classes of memories (e.g., perceptual, motor, or various forms of classical conditioning) to assess the generality of the conclusions drawn from the existing literature. Even though a general consensus has not yet emerged, there are numerous suggestions in the sleep-memory literature that specific sleep stages preferentially aid the formation for specific classes of memory (e.g., Wagner et al., 2001; Gais and Born, 2004a; Cox et al., 2012; Piosczyk et al., 2013; Chatburn et al., 2014; McDevitt et al., 2014; Buzsáki, 2015; van den Berg et al., 2019). Similar hypotheses should also be formulated and tested for memory consolidation during wakefulness: are there some types of waking that act, selectively or preferentially, on specific classes of memory? What are the mechanisms that mediate such (potential) effects? While challenging to conduct, experiments to address these and related questions are clearly necessary to develop a comprehensive understanding of the interplay between memory formation, consolidation mechanisms, and the mediating role of different behavioral states.

A major limitation of work comparing sleep, AW, and QW is the fact that all studies published to date rely on naps as the sleep condition, rather than overnight sleep. Given the vast differences between nightly sleep and napping (total duration, sleep architecture, circadian timing, etc.), it is conceivable that overnight sleep provides memory benefits that differ from those seen with napping. The practical challenges of keeping participants in a QW condition for prolonged time intervals make it next to impossible to directly compare nightly sleep to QW; in effect, overnight QW would constitute a sleep deprivation condition, a highly problematic procedure for cognitive research (Vertes, 2004; Frank, 2006). The use of long-lasting meditation sessions, which can be of many hours for some experienced meditators, may be one strategy to assess the effects of extended periods of QW on memory processes.

It is also important to acknowledge that, despite the fact that sleep and QW share similarities, sleep contains at least some features not present in QW. Among such characteristics are sleep spindles, which have been repeatedly linked to successful memory consolidation (Cox et al., 2012; Kurdziel et al., 2013; Piosczyk et al., 2013; Rasch and Born, 2013; Antony et al., 2019; van den Berg et al., 2019). One of the hallmark characteristics of non-REM sleep, sleep spindles are bursts of highly synchronized 11–16 Hz EEG activity that are most prominent during intermediate (i.e., stage 2) sleep stages. Importantly, spindles have been proposed as a marker of replay/reactivation of memory traces, due to their precise temporal association with hippocampal SWR complexes, as well as neocortical slow oscillations (Rasch and Born, 2013; Antony et al., 2019; Oyanedel et al., 2020). Recent evidence suggests a particular role of sleep spindles in promoting weekly encoded declarative information (Denis et al., 2021). The fact that spindle activity is not a prominent feature of EEG activity of humans during wakefulness may suggest that they afford sleep a specialized role in memory processing (e.g., Antony et al., 2019; it is noteworthy that spindle activity is prominent during waking immobility in rodents; Vanderwolf, 1988). A promising avenue for future work will be to elicit or enhance activity in the spindle range in humans during wakefulness, (e.g., by TMS or electrical stimulation) and assess whether this activity patterns can modulate memory consolidation when induced independent of behavioral sleep.

In addition to spindle activity, a greater and more continuous degree of perceptual and cognitive disengagement during sleep relative to QW may also allow sleep to instill memory benefits that exceed those produced by QW. The neurophysiological and behavioral differences between sleep and QW (and other types of wakefulness) also raise the possibility that sleep and waking both provide important, and possibly specialized or unique, contributions to memory consolidation. For example, observations that neuronal replay characteristics differ between sleep and wakefulness has led to suggestions that the specific function of replay depend on the behavioral state of the animal (e.g., memory consolidation and action planning during wake vs. memory integration and gist extraction during sleep; for a detailed review see Findlay et al., 2020). At present, such views are controversial (e.g., Joo and Frank, 2018) and, as mentioned above, future work is required to critically assess whether or not consolidation mechanisms perform specialized functions during different stages of sleep and wakefulness.

Given these caveats, it seems prudent to conceive of memory consolidation along a continuum of behavioral states, ranging from deep sleep to highly active and engaged wakefulness (Fig. 2). There is a clear trend of increasing behavioral and cognitive quiescence to favor more

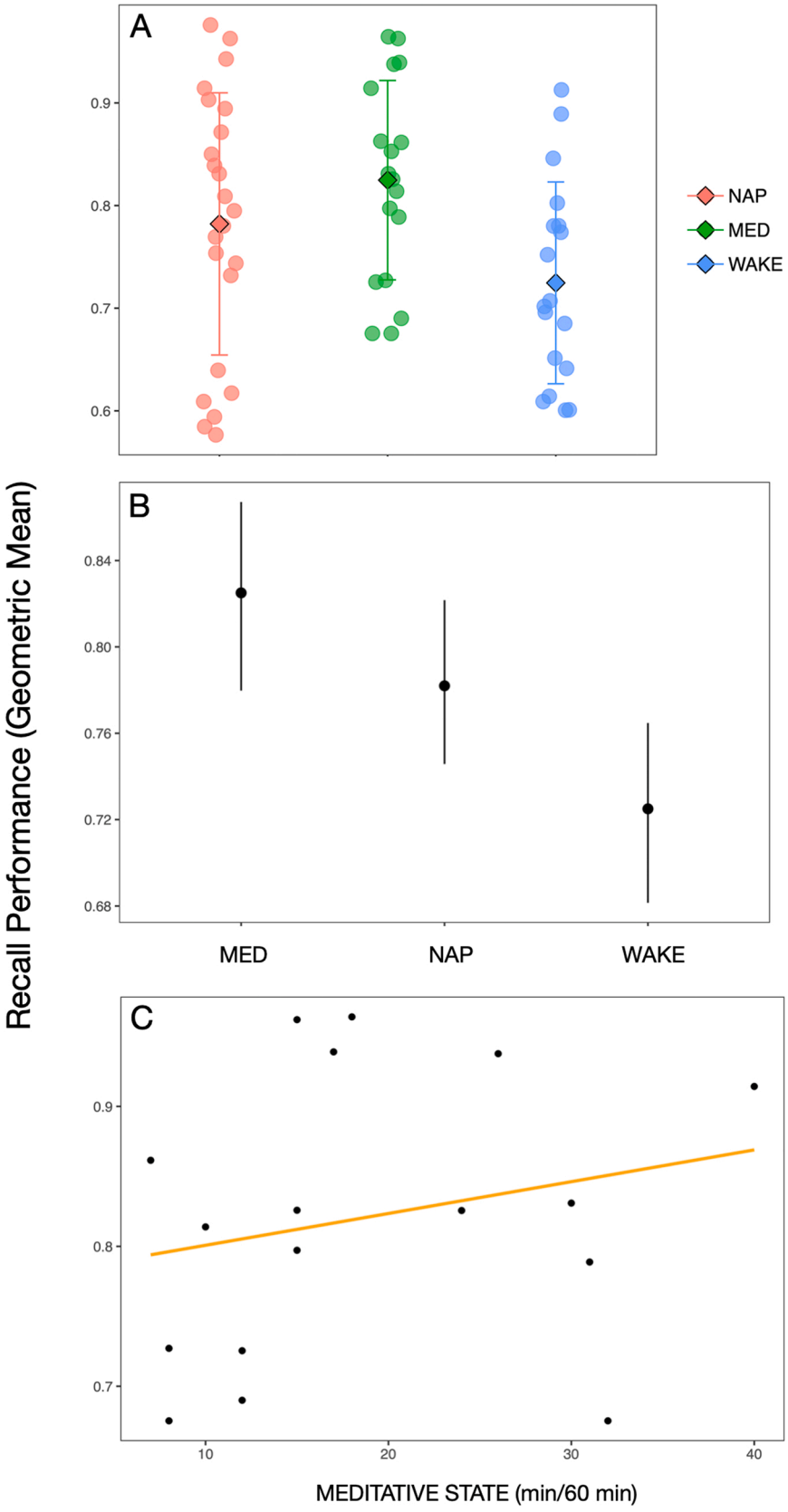

Fig. 1. A. Memory recall (word pair associates) in individual participants (circles) and as group means (± standard deviation) following a 1-hour period of either napping (NAP; n = 25), meditation (MED; n = 21), or wakefulness (WAKE; n = 18; watching a video). The Geometric Mean was calculated to provide an overall performance measure encompassing all possible responses on the paired-associated tasks (hits, false alarms, misses, and correct rejections). **B.** Participants in the meditation and nap condition outperformed those in the wakefulness condition. Importantly, a Bayesian general linear model revealed a 69.8% and 97.4% probability that meditation increased recall performance when compared to the nap and wakefulness condition, respectively. **C.** Memory recall in relation to the time (in minutes) spent in a meditative state over a 60-min meditation opportunity. Meditative state was defined as 30-s EEG epochs with increased (≥ 50%) power in the alpha-theta range, as assessed by Fast Fourier spectral analysis. Bayes analysis indicated a 1.47 greater probability over the null hypothesis (i.e., no association) that time in the meditative state was associated with recall performance (r = 0.16, this correlation can be considered as small-to-moderate with a probability of 56%; all data are from Dastgheib, unpublished M.Sc. thesis, 2020).[127].

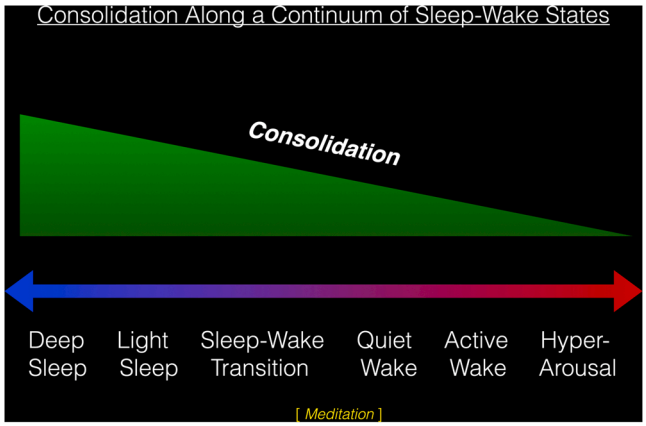

Fig. 2. Memory consolidation along a continuum of sleep- and waking states. Memory consolidation occurs during different sleep- and waking states. Plasticity mechanisms that mediate consolidation tend to be more active during sleep and quiet waking, making these states particularly effective for memory storage. However, there is no clear boundary between sleep and waking and both states are amendable to memory formation. Meditation, as a specific type of quiet waking, shares some characteristics with both sleep and wakefulness, making it a condition of particular interest for studies of memory consolidation.

effective memory consolidation, even though there are exceptions to this general pattern (Antony et al., 2017; Jentsch and Wolf, 2020). At the same time, there is no obvious demarcation to indicate a fundamental difference between sleep and wakefulness (Fig. 2): waking can be as effective as sleep, even if the presence of some sleep-specific characteristics may offer the most opportune conditions for successful consolidation. As such, sleep does not play an "essential" or "critical" function, and consolidation does not "depend" on sleep, even though it may benefit from it. Finally, given that sleep and AW constitute the opposite ends of this continuum of behavioral states (Fig. 2), the common practice of comparing sleep to AW serves to inflate the apparent benefits of sleep. The routine inclusion of QW conditions in future work would serve to provide a more conservative and accurate measure of the true contributions of sleep to consolidation above and beyond those provided by different states of wakefulness.

5. Summary

We opened this paper with the question: Is the role of sleep in memory consolidation overrated? The evidence reviewed here suggests that the answer to this question is: Yes. Effective memory consolidation occurs during both waking and sleep, mediated by many of the same plasticity mechanisms that perform information storage in neural circuits. Thus, sleep is not "critical" or "essential" in the sense that, as implied by this terminology, consolidation cannot occur in the absence of sleep. Further, minor alterations to the physiological or chemical milieu (e.g., increased cortisol levels) that leave the hallmark (behavioral, polysomnographic) characteristics of sleep intact render sleep ineffective in facilitating memory formation. Consequently, it is the presence of a set of specific chemical conditions and physiological mechanisms, rather than sleep per se, that constitute causal determinants of memory formation. Based on this evidence, we conclude that the notion of "sleep-dependency" and a "critical", "essential" or "causal" role of sleep does not provide an accurate reflection of the true contributions of sleep to the processes of consolidating and transforming memory traces in neural circuits.

Ethics clearance

Data collection for the unpublished experiments mentioned here was approved by the General Research Ethics Board, Queen's University. All participants provided written informed consent prior to the commencement of the study. We thank Ms. Elizabeth Legro for assistance with the data collection.

Conflict of interest

The authors report no conflicts of interest in this work.

Acknowledgements

This work was supported by a grant from the Natural Sciences and Engineering Research Council of Canada (NSERC; grant number RGPIN/04153-2019) to Dr. Hans C. Dringenberg. The authors report no conflicts of interest in this work.

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