did not improve compared to the last training set in day 1; in addition, total sleep deprivation between day 1 and day 2 did not alter performance compared to sleeping controls (Donchin et al. 2002). This experiment studied arm reaching in interaction with external forces, which is known to require time-dependent consolidation (Shadmehr & Brashers-Krug 1997). Interestingly, performance was quantified with a learning index that measures quality, rather than speed, on task.

Similar observations can be made for finger skills. When motor skill accuracy (error rate) was measured as absolute number of wrong sequences per 30-second trial (Walker et al. 2002), it did not change significantly between 10:00 a.m., 2:00 p.m., 6:00 p.m., and 10:00 p.m.; nor did accuracy change between 10:00 a.m., 10:00 p.m., and post-sleep 10:00 a.m.; nor did it change between 10:00 p.m., post-sleep 10:00 a.m., and post-sleep 10:00 p.m. In contrast, when error rate was redefined as number of wrong sequences relative to number of correct sequences per 30-second trial (Walker et al. 2003b), significant differences could be observed between all pre- and post-sleep conditions above. Interestingly, Fischer et al. (2002) found that performance speed, but not accuracy, significantly improved during daytime awake retention without practice. Consistent with the proposed model is the hypothesis that an adaptive, compensatory response to increased sleep need could take place during extended wakefulness (Finelli et al. 2000).

In summary, these findings suggest that sleep may not have a uniform effect on all constituents of memory. Rather, specific aspects, or types, of internal representation may be selectively enhanced by mechanisms characterizing the sleep process. Understanding which features of behavioral performance are enhanced will help uncover the specific mechanisms influenced by sleep.

Hypotheses concerning the putative mechanisms that may underlie the consolidation of memory traces during sleep have focused on the role of either REM or non-REM sleep (Maquet 2001). The evidence in favor of one or the other hypothesis requires careful consideration of experimental design and method (Peigneux et al. 2001a). Walker's (2002) hypothesis makes no a priori assumptions about the sleep state that may be exerting an effect on memory consolidation. Thus, he and coworkers were able to infer post hoc a correlation between relative amount of stage 2 non-REM sleep and performance improvement after sleep. The independent study by Fischer et al. (2002) that used a similar task and design confirmed most results, except for dependence on stage 2, showing instead a correlation between amount of REM sleep and performance gain after sleep. For both studies the amount of time spent in one sleep stage across the night at best accounted for less than 44% of the variance in performance gain (the other stage accounted for less than 14%). Therefore, none of those factors can explain entirely the relation between sleep and performance improvement (see also Gais et al. 2000; Stickgold et al. 2000b). Factors other than time related to sleep staging (e.g., electrophysiological variables) should also be tested.

It has recently been proposed that sleep spindles, by virtue of a pattern of excitation-inhibition that provokes massive Ca entry that specifically activates Ca-dependent molecular gates in the spindling cells, could open the door to subsequent long-term changes in cortical networks (Sejnowski & Destexhe 2000). This hypothesis is consistent with the correlation observed by Walker (2002). Prominent in non-REM sleep stage 2, spindles >13 Hz have been shown to have their maximal expression in an area surrounding the head vertex, that is, in close correspondence with the motor cortex (Finelli et al. 2001). Sleep is a regulated process whose timing, duration, and intensity depend on the interaction of homeostatic (the prior sleep-wake history) and circadian factors (Borbély 1982). Would a non-dramatic extension (or reduction) of some stage of sleep cause a better (or worse) improvement in performance? Sleep deprivation prior to new acquisition and sleep-induced enhancement would increase slow wave sleep, and would probably not alter stage 2. However, EEG power density in the range of spindles would be significantly reduced (Finelli et al. 2001). The concept of memory enhancement through sleep could be tested in experiments of this kind.

Sleep and memory: Definitions, terminology, models, and predictions?

Jonathan K. Foster[a] and Andrew C. Wilson[b]

[a]*School of Exercise, Biomedical and Health Sciences, Edith Cowan University, Joondalup, and School of Paediatric & Child Health, University of Western Australia, Princess Margaret Hospital, Subiaco, Western Australia, Australia;* [b]*Department of Respiratory Medicine, Princess Margaret Hospital, Subiaco, Western Australia, Australia.* **j.foster@ecu.edu.au andreww@ichr.uwa.edu.au**

Abstract: In this target article, Walker seeks to clarify the current state of knowledge regarding sleep and memory. Walker's review represents an impressively heuristic attempt to synthesize the relevant literature. In this commentary, we question the focus on procedural memory and the use of the term "consolidation," and we consider the extent to which empirically testable predictions can be derived from Walker's model.

In recent decades, there has been increasing research focus on the topic of sleep and memory, transcending once and for all the "sleep and memory tapes" anecdotal speculation of yesteryear. Walker hails from one of the scientific hotbeds of contemporary sleep-memory research. His starting point is that neuroscientific evidence indicates that sleep plays a role in learning and memory, but that the mechanisms involved are currently unclear. In this heuristic review, Walker seeks to clarify the current state of knowledge, in the process formulating a model whereby, it is argued, sleep impacts favourably on specific elements of memory processing.

In his review, Walker focuses predominantly on procedural memory, proposing that learning in this domain comprises an acquisition phase which is followed by two specific stages of consolidation (the first involving a process of stabilization and the second involving a process of enhancement; the latter is proposed to underlie delayed learning). Walker argues that acquisition and stabilization of procedural information do not rely fundamentally on sleep. By contrast, he argues that procedural memory enhancement does appear to rely on sleep, presenting evidence for specific sleep-stage dependencies. Walker adduces relevant evidence from the sleep/memory literature and nominates potential candidate mechanisms at the molecular, cellular, and systems levels.

In the initial reading of Walker's article, there is some apparent confusion regarding the notion of consolidation. Presumably the sentence towards the end of the Abstract which begins "In contrast, the consolidation stage . . . does appear to rely on the process of sleep" should in fact read "In contrast, the enhancement phase," given that Walker has already stated that the stabilization phase of consolidation is not modulated by sleep per se.

Of interest is the manner in which Walker characterises the distinction between declarative and procedural memory; for example, in section 2.1 he refers to declarative memory in terms of "one or two readings of a text" or "one exposure to an event" and procedural memory in terms of being "passive." It is not entirely clear that these dichotomies are appropriate with respect to the extant literature. For example, a recent search of the Institute of Scientific Information Science and Social Science Citation Indexes found no association between the notions of procedural memory and passive processing. Although this point perhaps does not bear directly on the theoretical framework that is being articulated by Walker, it is germane to some of the neurocognitive mechanisms which he cites. Furthermore, we find the use of the term "consolidation" in this context to be potentially confusing, given its much more frequent use in the declarative memory literature (again evaluated via Institute of Scientific Information Science and Social Science Citation Indexes), and given the sleep-related distinction that is here being attempted by Walker between declarative and pro-

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cedural memory mechanisms. How does Walker define "consolidation" exactly in the procedural memory domain as distinct from its more common application in the declarative memory domain? Moreover, how are the fundamental mechanisms of (a) declarative memory "consolidation" and (b) procedural memory "consolidation" deemed to be different with respect to their putatively contrasting interactions with the mechanisms mediating sleep?

Any scientific model worth its salt should, of course, be able to predict novel findings as well as accounting for the current body of data. With respect to Walker's further thoughts concerning procedural memory mechanisms, articulated in section 2.2, it would be interesting if (based on the tenets of his model) Walker were able to offer some novel predictions regarding the types of memory which should or should not be affected by sleep. For example, Foster and colleagues (Gagnon et al. 2004) have recently demonstrated that a form of cognitively implicit learning which has previously been associated with temporal lobe memory mechanisms can in fact be acquired in SJ, a patient with pure hippocampal amnesia (Gagnon et al. 2004). This task involves the learning of supraspan sequences (i.e. Hebb's recurring digit task).

In the test used by Gagnon et al. (2004), the participant is exposed to a series of item sequences in which one embedded sequence is presented on several occasions (amongst other item sequences that are not repeated). Each item sequence has to be recalled immediately after presentation by the participant. With respect to the points articulated by Walker in section 2.3.2.2, hippocampal patient SJ also showed learning on an implicit serial-reaction-time motor-learning task. To what extent do these tasks (which are clearly non-declarative and not critically hippocampally dependent) "fit" with respect to the framework articulated by the author?

It would be helpful if Walker were to delineate more clearly the types of memory mechanisms which he believes to be sleep-dependent. This point is underscored by his reference towards the end of section 2.4.2. to "sleep-dependent neuronal replay" being "expressed throughout different memory domains, including medial temporal lobe structures." In contrast, earlier in the piece Walker has implied that the evidence suggesting that declarative memory is associated with sleep mechanisms is inconclusive. Yet, it is declarative memory with which the hippocampus and other medial temporal lobe structures have been most strongly linked in the literature.

Regarding the proposed role of sleep in different types of memory processing, it would be interesting to know why Walker believes that sleep may be less important in declarative memory than in procedural memory. For example, could this be related to selection pressures operating in our evolutionary past? If so, what are or were the operational characteristics of these proposed mechanisms? Furthermore, how does Walker's model dovetail with the literature indicating that sleep fragmentation may cause deficits in mental flexibility, attention, and executive functioning (as well as affecting mood). The cognitive deficits induced by sleep fragmentation do not appear to be limited to the domain of memory in general (and procedural memory in particular). What are the proposed neural mechanisms underlying these other types of sleep-related cognitive deficits? Moreover, we know that cognitive capacities such as attention and executive functioning can be improved by sleep, so is it possible that some of the sleep-memory enhancement findings reviewed by Walker have in fact been confounded by sleep-mediated improvements in attention and executive capacities?

How might one test Walker's model? For example, what are the critical predictions of the model regarding interventions which influence sleep (such as the types of arousing sound pulses that have been used by Douglas and colleagues; see Martin et al. 1996) and the implications of such interventions for memory enhancement? To what extent are the different neurocognitive mechanisms which Walker imputes, truly coupled to different sleep stages? From a clinical perspective, what are the implications of Walker's model for conditions such as obstructive sleep apnea (OSA) and sleep deprivation? (A recent study by Rouleau et al. 2002 has indicated that OSA patients do not show procedural skill learning deficits, but deficits were apparent in these patients in terms of frontal dysfunction and in psychomotor efficiency and vigilance.)

Finally, given that we know that there are distinctive changes in sleep and sleep patterns across the lifespan, would Walker wish to predict that these changes should have implications for our mnemonic capacity as we age?

Old wine (most of it) in new bottles: Where are dreams and what is the memory?

Ramon Greenberg

Department of Psychiatry, Harvard Medical School, Brookline, MA 02445.
rgreenberg@hms.harvard.edu

Abstract: I discuss how the work in Walker's article adds to the considerable body of research on dreaming, sleep, and memory that appeared in the early days of modern sleep research. I also consider the issue of REM-independent and REM-dependent kinds of learning. This requires including emotional issues in our discussion, and therefore emphasizes the importance of studying and understanding dreams.

Walker's article on sleep and memory is a real pleasure to see. He does a thorough job of pulling together recent work on the role of sleep in memory formation and he responds well to the many voices that still are skeptical of this relationship. This is a battle that has been going on since the early days of modern sleep research, when investigators such as Hawkins (1966), Breger (1967), Dewan (1970), Greenberg (1970), Hartmann (1970), Stern (1970), Fishbein (1971), Pearlman (1973), Glaubman et al. (1978), Hennevin and Leconte (1977), and Palombo (1978) began to publish studies related to the role of REM sleep in the integration of information or memory. Walker's summary of recent work builds on the earlier work in a most convincing manner. The criticisms of studies linking sleep, and especially REM sleep, with memory consolidation began immediately with a focus on the question of stress as the cause of the impairment of memory in REM deprivation (REMD) studies. Pearlman (1973) and Smith (1982), with their work on windows for the REM sleep relationship to memory, gave cogent reasons why stress was not a reasonable reason to discount the effects of REMD: They used the finding that there are periods when REM sleep is more related to memory processing and, studying REM deprived animals at different times, they found that only in the presumed sensitive windows of time was learning impaired. Hence, stress alone could not be blamed for the effects of REMD. Also, the fact that some studies of REMD failed to show any negative effect on learning became a reason to ignore all the positive results. By underlining the fact that there are different kinds of memory, which may or may not need the aid of sleep, Walker gets to one of the most important responses to this sort of criticism. As Walker notes:

> Evolutionarily, this trait for rapid improvement *during* the waking repetition of a new skill makes considerable sense, particularly if it were a beneficial procedure. It would not seem logical to have a system that requires hours or days, or periods of sleep, before the first signs of improvement emerge. (sect. 2.3.1.1, para. 2, emphasis in original)

This observation captures a distinction which has often been ignored in the debate. It is also very similar to one we made 30 years ago (Greenberg & Pearlman 1974): "Habitual reactions, which are closely linked to survival, are REM independent; but activities involving assimilation of unusual information require REM sleep for optimal consolidation." We compared this distinction to Seligman's (1970) ideas about prepared (hard-wired or instinctual) learning and unprepared learning. One-trial avoidance is considered prepared learning, while tasks which require both many trials and time for learning can be considered unprepared learning.

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