nature
neuroscience

Why do we sleep?

Although there is still no satisfying answer to the question of why we sleep, rapid progress in the last several years suggests that this may soon change. Perhaps a better understanding of the function of sleep will also help to change attitudes about sleep at a societal level. The average person requires about eight hours of sleep per night, but many otherwise healthy people continually deprive themselves of adequate sleep with consequences that include fatigue, poor decision-making and increased risk of accidents. New research, including two papers in this issue demonstrating that sleep is required for memory consolidation (p. 1237 and 1335; see also p. 1235), may convince people to take sleep more seriously.

A recent poll taken by the US National Sleep Foundation illustrates the extent of the problem. Twenty percent of American adults reported being so sleepy during the day that it interferes with their daily activities at least a few days per week, and a frightening 17% reported falling asleep while driving within the last year. The risk of sleep-related accidents is compounded by the fact that people are unable to judge the likelihood that they will fall asleep, and by the related misconception that falling asleep is a slow process. In fact, sleep-deprived people commonly enter so-called 'microsleep' states, where they fall asleep for brief episodes lasting several seconds, during which time they are perceptually 'blind', often unaware that they have fallen asleep.

Accidents aside, one likely consequence of sleep deprivation is memory impairment. It was shown several years ago that a particular type of memory consolidation—improvement after practicing a visual discrimination task—does not occur until many hours after practice has ended. Using cleverly designed sleep deprivation experiments, the two papers in this issue extend this result by demonstrating an absolute requirement for sleep within 30 hours of training. Importantly, it was the occurrence of sleep and not the simple passage of time that was critical. This conclusion was strengthened by the fact that sleep-deprived subjects were tested after two full nights of recovery sleep, thus excluding the possibility that a nonspecific effect of sleep deprivation (such as a poor attention) was interfering with memory.

The results also shed light on a long-standing controversy as to the relative importance of the two major types of sleep. REM (rapid eye movement) sleep and is characterized by low amplitude, high-frequency EEG rhythms, whereas non-REM sleep involves high amplitude, low frequency rhythms. Based on physiological measures, REM and non-REM sleep seem as different from each other as either is from wakefulness—but do they play different roles in memory consolidation? Whereas a full night of sleep was needed for maximum improvement, the new findings demonstrated a critical role for slow-wave sleep (a type of non-REM sleep), which occurs more often early in the night; late-night sleep, dominated by REM, was not sufficient for memory improvement. This observation may eventually help to understand how the particular physiology of the different sleep states is related to the process of memory consolidation.

If sleep involves processing of information acquired while awake, then it should be possible to detect some representation of this information in neuronal activity patterns during sleep. A recent report[1] provides evidence for this idea, based on recordings from single neurons in songbirds. By recording from a forebrain region involved in the production of learned vocalizations, the authors were able to show that the firing patterns during sleep closely resembled those recorded during singing, as though the song-producing circuits were rehearsing these patterns 'off-line'. Similar observations have previously been made in the rat hippocampus, and also in humans (using functional imaging), but the songbird work is notable because of the tight correlation between the neuronal firing and behavior (when the bird is awake). An important next step will be to determine whether sleep deprivation in birds interferes with learning as it does in humans.

A major challenge in sleep research will be to link behavioral and electrophysiological findings with discoveries at the molecular level. The discovery of hypothalamic neuropeptides (hypocretins/orexins) that are linked to narcolepsy, as well as recent reports demonstrating that *Drosophila* have periods of rest that are remarkably similar to mammalian sleep, offer hope that genetic tools will help to forge such links. For example, in addition to sharing many other features of mammalian sleep, such as an increased arousal threshold, *Drosophila* increase their rest after prolonged waking, indicating that their rest, like ours, is under homeostatic control. A key issue for the field is to determine what parameter is being maintained by this homeostatic process, and by studying mutants with altered responses to rest deprivation (several have already been identified), it may be possible to address this question. It will also be of great interest to understand the link between homeostatic mechanisms, which track sleep debt and determine the probability of falling asleep, with circadian mechanisms, which help to organize sleep into characteristic bouts[2].

A comprehensive theory will also have to take into account large species differences in sleep patterns; for example, horses sleep about 3 hours per day compared with 20 hours for bats, and the ratio of REM to non-REM sleep also differs greatly between species. Moreover, there are large developmental differences that remain to be explained: for human infants, about 50% of sleep is REM sleep, whereas this drops to about 25% in adults. Perhaps as a more comprehensive theory of sleep emerges, common attitudes about sleep will also change. For instance, napping is considered normal in children, but in adults it carries a stigma of laziness and inefficiency, despite the fact that it can be extremely effective in improving alertness for many hours afterward.

1. Dave, A. S. & Margoliash, D. *Science* **290**, 812–815 (2000).
2. Naylor, E. *et al. J. Neurosci.* **20**, 8138–8143 (2000).