LETTER TO THE EDITOR

Sleep Homeostasis and the Function of Sleep—Comments

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BENINGTON’S INTERESTING ARTICLE ON SLEEP HOMEOSTASIS AND THE FUNCTION OF SLEEP, PUBLISHED RECENTLY IN SLEEP,1 can be related to our recently published review2 of the evolution of sleep. We would like to add some comments.

Firstly, Benington rejects the hypothesis by Webb3 and Meddis4 that the primary function of sleep is to eliminate behavioral responsiveness during the body’s spare time, as the organism is not well adapted during part of the light/dark cycle. Benington considers that the existence of homeostatic regulation would be counterproductive after a loss of sleep. This might seem initially to be true, but there are also additional factors to take into account. Let us consider the case of reptiles. They show clear circadian activity and rest periods, but they also show some signs of homeostatic regulation: activity and rest rebounds can be seen when the normal light/dark photoperiod is restored after a period of continuous dark and light, respectively.5 For animals whose activity is completely dependent on light and heat, what possible objective could there be in the homeostatic control of activity and rest? One explanation for this control system is redundancy, which would guarantee a certain amount of activity and rest, even after several days of low light and heat. Sleep homeostasis could have developed as a low-level, redundant need for behavioral adjustment to better environmental conditions.

Continuing on the theme of reptiles, we proposed that reptilian activity and rest were transformed into NREM and REM after the development of mammalian cortical waking. Assuming that our hypothesis was true, this would have resulted in mammals experiencing round-the-clock continuous activity: with cortical waking during dark hours and activity controlled by the brainstem during daylight hours. This untenable situation would have been avoided by increasing the degree of what, in the reptile’s case, had originally been a low level of homeostatic regulation. The main burden of this regulatory mechanism would have been focused on ensuring behavioral inhibition during the new NREM phase, after having turned it from reptilian activity into sleep. On the other hand, there was little need to develop a homeostatic control of REM and this has remained the case up to now, as reptilian inactivity and mammalian REM were and continue to be very similar, from both a structural and functional point of view.

In addition, Benington proposes that sleep only serves one primary function, while accepting that other benefits are the result of secondary forms of behavioral adaptation, which originally evolved for other reasons. If we follow his line of thought, disregarding for the moment the previously described NREM and REM system of evolution, two stages in the evolutionary development of sleep regulation might occur to us. In the first one, sleep would be dependent on circadian clocks, without the need for homeostatic regulation. However, newly acquired secondary functions would have required the development of more powerful regulatory mechanisms.

Whether it was the result of the transformation of reptilian waking into NREM or due to secondary adaptive processes (or indeed both), sleep could have acquired the well-known circadian and homeostatic regulation as proposed in the two process model of sleep regulation6 and Webb and Meddis’ hypothesis could still be maintained. This hypothesis is simple, elegant, easily understandable and, above all, fitting. This is because the consequence of being asleep (i.e., inactivity) corresponds exactly with the requirements of its presumed function. On the contrary, other hypotheses require an additional explanatory step over and above their empirical support: if sleep is necessary for the maturation of behavior, learning, “unlearning,” or for the dynamic stabilization of synapses, we should continue to ask why these functions can only be achieved while the body is asleep and not during its waking hours.

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