Tuning for Criticality: A New Hypothesis for Sleep

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We propose that the critical function of sleep [3] is to prevent uncontrolled neuronal feedback while allowing rapid responses and prolonged retention of short-term memories. The goal of learning is optimal behavior, and this sometimes requires the integration of sensory stimuli that are widely separated in time. At a neuronal level, this corresponds to persistent activity in local networks. Unfortunately, when a network exhibits persistent activity, small changes in the parameters or conditions can lead to runaway oscillations. Thus, the very changes that improve the processing performance of the network can put it at risk of runaway oscillation. To prevent this, stimulus-dependent plasticity should only be permitted when there is a margin of safety around the current network parameters. We propose that a critical role of sleep is to establish a margin of safety by exposing the network to a variety of conditions and inputs, observing for erratic behavior, and adjusting the parameters accordingly. During wakefulness this margin of safety is gradually consumed, ultimately requiring refreshment by another period of sleep. When sleep is not possible, an emergency mechanism comes into play to prevent runaway oscillations; this is done at the expense of processing efficiency, and constitutes tiredness. We contend that this theory matches the phenomenology of sleep and tiredness better than do alternative theories, such as the memory consolidation theory [1, 2, 5]. This theory also makes a number of novel testable predictions.

The most direct prediction is that different plasticity regimes should operate during wakefulness, tiredness and slow-wave sleep. Some experiments that relate to less direct predictions have already been conducted for other purposes. For instance, we would expect that the impulse response of cortex during wakefulness would be near the boundary of criticality, and that during slow wave sleep the impulse response would be moved closer to the boundary, which is consistent with the experimental data of Massimini et al. [4]. One success of this hypothesis is that it explains tiredness as an emergency mechanism. If this is true, then combating the symptoms of tiredness should increase the risk of suffering epilepsy. An increased risk of epilepsy would also be expected as a consequence of regular sleep deprivation, particularly among babies and children.

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