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Opinion Article

Effects of Adenosine and Caffeine on Sleep

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DESCRIPTION

Adenosine is widely recognized as an important sleep regulator. It is widely believed that adenosine contributes to sleep-wake regulation by binding to high-affinity A1 and A2A receptors. These receptors are differentially expressed in different brain areas and can play different roles in sleep regulation and control of sleep. Surprisingly and apparently challenging the notion that A1 binding sites contribute to sleep homeostasis, mice exhibiting constitutive and central nervous system specific, conditional ablation of A1 receptors showed virtually unaltered sleep-wake behaviour and cognitive functions when studied under baseline sleep-wake conditions. However, when conditional knockout animals were exposed to prolonged arousal, their working memory was impaired, they did not prolong sleep and recovery after sleep deprivation did not show normal recovery with Electroencephalographic Slow-Wave Activity (EEGSWA) during sleep. Adenosine is a degradation product of the breakdown of Adenosine Triphosphate (ATP) in the brain. ATP levels in the wake-active brain region of the rat increase during spontaneous sleep and decrease during sleep deprivation. After prolonged arousal neuronal activity, ATP accumulates in the extracellular space and is broken down by 5'-EN into adenosine. Adenosine is thought to increase in extracellular space during prolonged arousal and has been proposed as a homeostatic accumulator for sleep needs. Some authors have suggested that levels of adenosine, especially in the basal forebrain, are important for sleep-wake regulation. In some areas of the brain, stimulation of the adenosine A1 receptor suppresses glutamate release and reduces the amplitude of postsynaptic currents. Therefore, not only the accumulation of adenosine, but also other sleep-promoting substances can reduce the activity of the arousal area and thus unblock the sleep-promoting area. Among the four different subclasses of adenosine receptors (A1, A2A, A2B, and A3 receptors), not only the A1 receptor, but also the A2A receptor appears to be important for sleep-wake regulation. Pharmacological stimulation of A1 and A2A receptors by specific agonists increases slow-wave sleep and EEG Slow-Wave Activity (SWA; spectral power in the range 1-4 Hz) in Non-Rapid Eye Movement (NREM) sleep. However, the observed kinetics indicate that extracellular adenosine is unlikely to be responsible for the diurnal changes in sleepiness and sleep pressure observed under undisturbed baseline conditions. Nonetheless, adenosine can play a role even if arousal is prolonged beyond the normal daily duration, contributing to increased sleep pressure and slow EEG in these conditions.

Caffeine is a potent adenosine receptor antagonist and has almost the same affinity for A1 and A2A receptors. Studies in mice provided strong evidence that caffeine promotes alertness primarily by blocking the A2A subtype of the adenosine receptor. Caffeine (15 mg/kg dose) did not interfere with sleep in mice with genetically disrupted A2A receptor function, whereas stimulants were transgenic lacking wild-type mice and functional A1 receptors. Subsequent experiments have shown that gene deletions of the A2A receptor, especially within the nucleus accumbens envelope, block caffeine-induced arousal.

Human studies have typically relied on caffeine studies to investigate the role of adenosine in sleep homeostasis. Administration of caffeine in the evening just before bedtime consistently prolonged polysomnography during sleep onset latency and reduced sleep efficiency. The situation changes when you take caffeine daily. The continued presence of caffeine and its major metabolite, paraxanthine, probably causes changes in the adenosine system, which can affect sleep.

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