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Perspective

Treatment of Sleep Disorders with Adenosine Receptors

Ivandro Menke*

Department of Psychology, University of Vienna, Vienna, Austria

DESCRIPTION

Adenosine receptors have long been recognized as potential therapeutic targets for the pharmacological treatment of sleep disorders. The next section describes how pharmacological and physiological interventions affect wakefulness and sleep. It probably acts on potential targets of the adenosine activator and adenosine neuro modulatory systems to improve sleep–wake and associated disorders.

Hypersomnia and excessive daytime sleepiness

Acute caffeine intake (about 150-500 mg/day) commonly consumed by individuals from coffee or tea is aroused by blocking A1 and A2A receptors, especially in the case of sleep deprivation and increased drowsiness and can promote arousal and mood. Coffee and caffeine were one of the earliest recommended treatments for excessive daytime sleepiness in narcolepsy. More effective medicines are available, but because the diagnosis is pending, many patients with narcolepsy are "self-treated" with high levels of caffeine to improve their alertness. Adenosine A2A and dopamine D2 receptors are co-localized in the indirect pathway of medium-sized spiny neurons in the striatum. Activation of excitatory A2A binding sites in this pathway strongly induces sleep, and blockade of A2A receptors on these cells enhances dopaminergic neurotransmission through functional A2A-D2 receptor antagonism. A2A receptors should be a promising target as an additional treatment to relieve motor and non-motor symptoms such as fatigue in Parkinson's disease. Caffeine (up to 400 mg/day) was unable to reduce daytime sleepiness in Randomized Controlled Trials (RCTs) in patients with Parkinson's disease. Importantly, the effect of caffeine depends on chronic caffeine intake and sleep pressure. Antagonists with a more selective affinity for the A2A receptor (eg, istradefylline) have a more favorable safety profile for promoting wakefulness compared to high doses of caffeine and psychostimulants that can disturb sleep and cause anxiety and dependence.

Depressiveness

60%-90% of people face depression due to disturbed sleep. Acute sleep deprivation (or "wake therapy") improves depressive symptoms in about 60% of people. The antidepressant response to sleep deprivation is quantitatively similar to standard therapies such as medication, psychotherapy, and electroconvulsive therapy. Unfortunately, arousal-induced mood improvements are usually short-lived and relapse into depression after a restful sleep.

Changes in A1 receptors due to sleep loss may contribute to the neurochemical mechanisms underlying the rapid mood-enhancing effects of sleep deprivation therapy. Interestingly, a recent meta-analysis of observational studies of nearly 370,000 adults in the general population found that coffee and caffeine intake was associated with a dose-dependent reduction in the risk of depression. In this view, neither the existence of a causal relationship between coffee/caffeine consumption and depression can be concluded, nor the underlying mechanism of the observed relationship. Future studies are needed to investigate the role of adenosine-regulated neurotransmission and its effect on homeostasis and circadian sleep-wake regulation processes on the antidepressant effects of therapeutic sleep deprivation.

CONCLUSION

The available evidence concludes that, adenosine is likely involved in the adaptive homeostatic response to previous sleep and wake periods, as levels of adenosine increase during sleep deprivation and decrease during recovery sleep. In prolonged wakefulness, adenosine may be involved in inducing subsequent increases in non-rem sleep time and non-rem sleep EEG. For clearer insights, experiments are needed to examine adenosine levels in different brain regions in parallel with simultaneous sleep recordings. Such experiments can shed light on the role of adenosine in tracking sleep needs on a shorter time scale than the entire sleep and wake cycle.

Correspondence to: Ivandro Menke, Department of Psychology, University of Vienna, Vienna, Austria, E-mail: ivandro.menke@031.at

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