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Commentary

Pharmacological Treatment of Gamma-Aminobutyric Acid System for Sleep Disorders

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DESCRIPTION

Gamma-Aminobutyric Acid (GABA) is the Central Nervous System (CNS) primary inhibitory neurotransmitter. It is generally known that GABAA receptor activation promotes sleep. GABAA receptor-mediated inhibitory processes are the basis of three generations of hypnotics. The first and second generations of hypnotics (barbiturates and benzodiazepines, respectively) reduce waking, increase slow-wave sleep, and improve the intermediate stage between Slow-Wave Sleep (SWS) and paradoxical sleep at the price of the last sleep stage. The third generation of hypnotics (imidazopyridines and cyclopyrrolones) has similar effects on waking and slow-wave sleep, but the minor reduction in paradoxical sleep in the early hours is not due to an increase in the intermediate stage. GABAB receptor antagonists have been demonstrated to promote brain-activated behavioral states (waking and paradoxical sleep: Dreaming stage). A particular GABAC receptor antagonist was recently developed and found to increase waking at the expense of Slow-Wave Sleep and paradoxical sleep when given intravenously. Because GABAC receptors are more sensitive to GABA than GABAA and GABAB receptors, GABAC receptor agonists and antagonists, once ready for therapeutic use, could usher in a new era of treatment for insomnia, epilepsy, and narcolepsy. They may be able to work at lower doses and with fewer adverse effects than currently available medications. Insomnia treatment, on the other hand, encompasses both pharmaceutical and non-pharmacological approaches. Understanding of the basic neurobiology of sleep-wake control has recently encouraged the development of pharmaceutical treatments for insomnia. Benzodiazepines that target the GABAergic system are the most often utilized hypnotics for pharmacological therapy. Gamma-Aminobutyric Acid (GABA) is an inhibitory neurotransmitter that regulates alertness levels.

GABA is a primary inhibitory neurotransmitter that aids in the general balance of neuronal excitation. It also plays a key role in brain growth and function. GABAergic neurons account for around 20% of all neurons in the brain. GABAA, GABAB, and GABAC are three distinct GABA receptors involved in sleep and arousal regulation. The most commonly used hypnotics work by allosterically modulating the benzodiazepine site, which is how they affect GABA systems. Benzodiazepines are a class of CNS depressants that produce feelings of relaxation (anxiolysis), tiredness, and sleep. These function by assisting the inhibitory neurotransmitter GABA in binding to specific GABA receptors throughout the CNS. In humans, activation of the benzodiazepine binding site improves slow-wave sleep, especially stage II (with spindle augmentation) over stages III and IV, and inhibits paradoxical sleep. GABAA chloride conductance can depolarize postsynaptic neurons and may have stimulating effects, in addition to the fast inhibitory transmission. GABAA receptor activation may aid action potential formation when paired with other depolarizing inputs within a specified time window. Thus, depending on intracellular chloride concentrations at the site of GABAergic input as well as the spatiotemporal summation with other inputs, GABAergic inputs at the same synapse may result in various effects. Moreover, GABAA receptors functions not only on postsynaptic neurons but also on presynaptic neurons.

GABAA receptors functional properties and pharmacological profiles are influenced by their structural complexity, multiple receptor subtypes with distinct electrophysiological properties (channel kinetics, affinity for GABA, rate of desensitization, and phasic *vs.* tonic conductance), and specific regional, cellular, and subcellular distribution. The GABAA receptors are targeted by the majority of tranquil hypnotics used to treat insomnia. The anxiolytic, anticonvulsant, and myorelaxant effects of these medicines, as well as their amnestic, sedative, and hypnotic effects, are thought to be attributable to their general inhibitory influence on neuronal activity. It is crucial to find out how these various sedative-hypnotic medications boost phasic and tonic GABAA-mediated transmission.

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