



SLEEP PHYSIOLOGY AND THE EFFECTS OF INSOMNIA ON THE NERVOUS SYSTEM.

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Annotation

This scientific article analyzes the main principles of sleep physiology, the neurobiological mechanisms of sleep phases, and the effects of insomnia on the central and peripheral nervous systems. The article discusses physiological, psychological, and pharmacological factors of sleep disorders, diagnostic methods, and therapeutic approaches (both pharmacological and non-pharmacological). It provides theoretical foundations and practical recommendations for identifying and managing sleep-related problems in pharmaceutical practice.

Keywords sleep physiology, insomnia, nervous system, melatonin, GABA, irritability.

Introduction

Sleep is an essential biological process vital for maintaining physical and mental health, supporting cognitive functions, regulating the immune system, and modulating metabolism. In recent years, sleep disorders—especially insomnia—have become a major global health issue. Understanding sleep physiology and developing effective diagnostic and therapeutic approaches are crucial in medical and pharmaceutical sciences.

CHARACTERISTICS AND PHASES OF SLEEP

Sleep is divided into two main states: Rapid Eye Movement (REM) and Non-Rapid Eye Movement (NREM). NREM is subdivided into three stages (from light to deep sleep), each characterized by distinct brain electrical activity and physiological parameters. The REM phase is associated with memory consolidation and emotional processing, while NREM plays a major role in physical restoration and metabolic regulation.

NEUROTRANSMITTERS AND ANATOMICAL STRUCTURES REGULATING SLEEP

The main neurotransmitters involved in regulating sleep and wakefulness include GABA, serotonin, noradrenaline, dopamine, acetylcholine, and melatonin. The hypothalamus (particularly the ventrolateral preoptic nucleus), reticular formation, locus coeruleus, dorsal raphe, and occipital brain regions regulate sleep-wake cycles. Melatonin, secreted by the pineal gland, defines circadian rhythm and promotes sleep onset.

INSOMNIA: DEFINITION AND CLASSIFICATION

Insomnia is characterized by difficulty falling asleep, insufficient sleep duration, or poor sleep quality. It can be acute, chronic, or recurrent. Pathogenetically, insomnia may result from psychophysiological, emotional, or pharmacological factors.

EFFECTS OF INSOMNIA ON THE NERVOUS SYSTEM

Chronic insomnia may induce neuroplastic changes in the central nervous system. Persistent sleep deprivation leads to cognitive decline, attention and memory impairments, mood disturbances, and increased stress reactivity. Insomnia alters neuroendocrine activity,

elevating cortisol levels, enhancing sympathetic activity, and increasing inflammatory markers, potentially contributing to neurodegeneration.

RISK FACTORS AND ETIOLOGY

Risk factors for insomnia include stress, depression, anxiety, chronic pain, smoking or alcohol consumption before bedtime, late-night eating, and limited sun exposure (circadian rhythm disruption). Some medications (stimulants, certain antidepressants) also negatively affect sleep quality.

DIAGNOSTIC METHODS

Diagnosis is based on clinical interviews, sleep diaries, and questionnaires such as the Pittsburgh Sleep Quality Index (PSQI). Biological tests and polysomnography (PSG) are used in complex cases or when other sleep disorders are suspected. Actigraphy helps monitor sleep-wake cycles in home settings.

NON-PHARMACOLOGICAL APPROACHES

Primary interventions include sleep hygiene, cognitive-behavioral therapy for insomnia (CBT-I), behavioral monitoring, and relaxation techniques. CBT-I improves sleep by correcting maladaptive thoughts and habits, showing long-term effectiveness over medications.

PHARMACOTHERAPY AND MEDICATIONS

Pharmacological treatment of insomnia includes benzodiazepines and related sedative-hypnotics, non-benzodiazepine GABA-A agonists, melatonin receptor agonists, orexin receptor antagonists, and certain antidepressants. Medication selection depends on metabolism, age, adverse effects, drug interactions, and addiction risks. Pharmacotherapy should be short-term and minimal, especially for benzodiazepines due to dependency risks.

NEUROLOGICAL CONSEQUENCES AND LONG-TERM OUTCOMES

Chronic insomnia may cause neurotoxic stress; studies show a potential link to Alzheimer's disease. Sleep deprivation increases oxidative stress, mitochondrial dysfunction, and impairs synaptic integrity. It also aggravates mental health conditions such as depression and anxiety.

PHARMACEUTICAL APPROACHES AND RECOMMENDATIONS

Pharmacists play a key role in advising patients on sleep hygiene, selecting medications, and adjusting doses. They must consider drug interactions, metabolic conditions, and age-appropriate strategies. Short-term melatonin use, evening dosing of orexin antagonists, or CBT-I combination therapy can be effective.

RESEARCH DIRECTIONS AND FUTURE PERSPECTIVES

Emerging areas include genetic factors, microbiome-sleep interactions, and neuroimaging advancements. New pharmacological targets, such as receptor subtype-specific ligands and therapies restoring synaptic plasticity, are promising for future treatment.

CONCLUSION

The physiology of sleep and the effects of insomnia on the nervous system are complex and interrelated. Accurate diagnosis, a combination of non-pharmacological and pharmacological approaches, and the pharmacist's role are essential for effective management. Future research may contribute to sleep restoration and reduce risks of neurodegenerative diseases.

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