## Abstract

Biological rhythms are common feature of living organisms, which are present in physiological processes and behaviour at every level of the organization. Such rhythms are endogenously generated by suprachiasmatic nuclei (SCN) of the hypothalamus, which are our internal biological clock. Genetically coded circadian rhythm are synchronized to cyclic changed external conditions by two kinds of information: photic (light) and nonphotic (i.e. arousal, social interactions or food availability). A second neuronal structure that is functionally connected with the SCN is the intergeniculate leaflet of the thalamus (IGL). This small flat nucleus receives photic information directly from the retina of the eye, and also receive nonphotic signals from nonspecific brain systems, such as the serotoninergic or orexinergic systems. Both received signals are integrated on the level of IGL network, and then transmit to the master biological clock - SCN.

The aim of my PhD dissertation was to examine the influence of different substances on single IGL neurons. These substances were split into endogenous neurotransmitters such as  $\gamma$ -aminobutyric acid (GABA), enkephalin (ENK) and neuropeptide Y (NPY), and exogenous neurotransmitters of the nonspecific systems, such as orexin (OX) and serotonin (5HT). In addition to verifying how these substances impact on the investigated structure, there was also an attempt to reveal the biochemical nature of the recorded neurons.

All experiments were conducted with a patch clamp *in vitro* technique. In addition to electrophysiological experiments, some immunostaining was performed in order to verify the biochemical nature of the recorded neurons.

The data obtained indicated that GABA and ENK are two main inhibitory neurotransmitters of the IGL. The presence of both GABA<sub>A</sub> and GABA<sub>B</sub> receptors, and the presence of inhibitory synaptic transmission in every recorded IGL neuron were confirmed. In the case of ENK, my research indicated an inhibitory postsynaptic effect that was mainly mediated by type μ opioid receptors. My study concerned a third endogenous neurotransmitter of the IGL – NPY, confirmed its main projecting character, because only 30% of recorded neurons were affected by this neuropeptide. The second stage of my research focused on the influence of orexinergic and serotonergic systems on the neuronal activity of the IGL. The performed experiments indicated that both subpopulations of IGL neurons, NPY-positive and NPY-negative, are affected by orexin A (OXA). The observed effect of OXA in the IGL was

depolarization. Moreover, my experiments have shown an involvement of both orexin receptors, orexin receptor type 1 and type 2, in the response of IGL neurons. In the case of serotonin application, the observed effect was both depolarization on one neuron, and hyperpolarization of another. The influence of serotonin was presynaptic as well as postsynaptic. These data supplement the current state of knowledge about the cellular mechanisms involved in activation of the IGL network. Additionally, this research enables us to identify, with a high probability, the subpopulation of NPY-positive neurons as being responsible for photic and nonphotic signal integration.

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