physicochemical alterations, increased intimal proteoglycan binding, and incorporation by macrophage scavenger receptors and specific PLA2R. Lp-PLA2 requires oxidized LDL as a substrate, and it has no effect on native lipoproteins. The hydrolysis of phospholipids on lipoproteins and cell membranes results in bioactive lipids (NEFAs, lysophospholipids, and eicosanoids) that activate pro-inflammatory redox-sensitive transcription factors and enhance proapoptotic effects.

BRAIN ADENOSINE RECEPTORS AS TARGETS FOR MEDICATIONS

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Introduction. Adenosine as a neuromodulator of the central nervous system has specific receptors, which have been the target of medical therapy for many years. Such compounds that have high affinity to adenosine receptors (ARs) can act both like antagonists and agonists. Despite this, there are still only a limited number of adenosinergic medications on the market.

The aim of our work was to review publications devoted to study medications, which mechanism of action is mediated by interaction with ARs in brain.

Materials and methods. Search for scientific publications was conducted in PubMed and ScienceDirect textual databases until December 2022. The following search terms were used: adenosine receptors, modulators of adenosine receptor activity, adenosine receptors antagonists, pharmacology of adenosine receptors.

Research results. Currently, four ARs are found in different tissues to be expressed, they named A1, A2A, A2B, and A3. All these receptors are metabotropic, particularly, G protein-coupled receptors. AR density is very high in the brain. Thus, A1AR and A2AAR are mainly localized at excitatory synapses, although they are also present in glial cells.

Both activation and blockade of ARs are important mechanisms in the correction of such brain conditions as ischemia and epilepsy. For example, in ischemic stroke, the dual and opposite control of A1 activation and A2A blockade provide reliable neuroprotection with abnormally increased load, which is also characteristic of epileptic conditions. This suggests that combined A1AR activation and A2A blockade may be more effective in limiting acute brain injury. The prospect of treating neurodegenerative diseases such as Parkinson's disease (PD) is also important. The particularly high density of A2A in the basal ganglia and their close antagonistic interaction with dopamine D2 receptors has prompted the targeting of A2A to alleviate the dopaminergic depletion characteristic of PD. Selective A2A antagonists also attenuate other motor conditions such as catalepsy and tremor, and others. In experiments, A2A blockade prevents memory deficits in models of Alzheimer's disease. Antagonism of A2A also prevents memory dysfunction associated with other states of cognitive dysfunction such as seizures, demyelination states.

Conclusions. Over the past several decades were generated a number of highly specific agonists and antagonists of ARs, which are important modulators of physiological and pathological processes.