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Opinion

AN OVERVIEW ON METABOTROPIC GLUTAMATE RECEPTORS

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INTRODUCTION

Most excitatory synapses in the mammalian central nervous system use L-glutamate as their neurotransmitter (CNS). Using second messenger signaling pathways, neuromodulatory glutamate receptors known as metabotropic glutamate receptors (mGluRs) can alter synaptic transmission and cell excitability. The fact that mGluR proteins are widely distributed suggests that these neuromodulatory receptors have the capacity to participate in several CNS processes and may make excellent therapeutic targets for a wide range of CNS illnesses. The metabotropic glutamate receptors (mGluRs), which belong to the family C of G-protein-coupled receptors, participate in the central nervous system-wide control of synaptic transmission and neuronal excitability.

The mGluRs bind glutamate within a sizable extracellular region and send signals to intracellular signaling partners via the receptor protein. The methods by which mGluRs are activated, the proteins with which they interact, and the orthostatic and allosteric ligands that can alter receptor

function have all made considerable progress. Recent studies have confirmed the therapeutic value of mGluR ligands in neurological and psychiatric illnesses such Alzheimer's disease, Parkinson's disease, anxiety, depression, and schizophrenia. The broad expression of mGluRs makes these receptors particularly appealing pharmacological targets.

DESCRIPTION

The G-protein-coupled receptor (GPCR) superfamily, which contains the most receptor gene families in the human genome, includes mGluRs. GPCRs are membrane-bound proteins that transduce intracellular signals through interactions with G proteins. Extracellular ligands like light, peptides, and neurotransmitters can activate GPCRs and cause them to become active. The G protein, which is made up of a heterotrimeric complex comprising, and subunits, is activated because of the change in conformation of the GPCR brought on by ligand interaction. When G proteins are inactive, they are attached to guanosine 5/-diphosphate (GDP); nevertheless, when G proteins are activated, GDP is exchanged for GTP inside the subunit.

The action of several effector molecules, including enzymes, ion channels, and transcription factors, is then modified by activated G protein subunits. When the bound GTP is hydrolyzed to GDP and the heterotrimer is reassembled, the G protein becomes inactive. The majority of classical neurotransmitter GPCRs are members of family A, one of numerous subgroups within the GPCR family. The structural similarities between these receptors-often referred to as rhodopsin-like GPCRs—include the presence of an extracellular N-terminal domain, seven transmembrane-spanning domains, and an intracellular C-terminus [1-3].

mGluRs are class C GPCRs, as opposed to family A receptors. The inclusion of a sizable extracellular N-terminal domain, which houses the endogenous ligand-binding site and is described in more detail below, sets these receptors apart from their family A cousins. GABAB receptors, calcium-sensing receptors, pheromone receptors, and taste receptors are other members of the family C GPCRs (1). Eight mGluR subtype-specific genes have been discovered, several of which have numerous splice variants and are differently expressed throughout the CNS in various cell types.

Based on sequence homology, G-protein coupling, and ligand selectivity, mGluRs are divided into three classes. The mGluRs 1 and 5 are in Group I, the mGluRs 2 and 3, and the mGluRs 4, 6, 7, and 8 are in Group III. For many illnesses, combination therapy techniques are the most appealing therapeutic approaches, and multiple recent studies have highlighted the potential of multifunctional pharmacological approaches. Multiple therapy modalities will be more effective than those aimed at a single target because trauma and neurodegeneration in the CNS are influenced by several factors. In response to acute and subacute injury as well as chronic neurodegeneration, neurons, astrocytes, microglia, oligodendrocytes, endothelial cells, and other circulating immune cell's function. Targeting mGluRs, which are expressed in a variety of cell

types and are widely distributed throughout the CNS, is one multifunctional therapeutic approach. The reduction of neurotransmitter release is one of the main roles of presynaptic mGluR2/mGluR3. It is well recognized that both receptor types contribute to the control of synaptic plasticity, especially through promoting LTD of excitatory synaptic transmission. In the mouse olfactory bulb, a particular configuration of synaptic plasticity has been noticed where activation of mGluR2 lessens GABA inhibition of mitral cells [4,5].

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