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Editorial

Kainate Receptors: Molecular Physiology

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Introduction

In the mammalian nervous system, glutamate is primarily involved in fast excitatory neurotransmission. Despite the fact that the importance of this amino acid for brain function has been constantly emphasized, there is no way to exaggerate it. On the one hand, glutamate functions as transmitter at most excitatory synapses and is implicated in the production of long-term changes in neurotransmission efficacy, which are assumed to be neuronal correlates of memory formation. Glutamate, on the other hand, plays an important role in the nervous system's development, assisting in the extension of processes, the production and deletion of synapses, and the activity-dependent fine tuning of finely precise patterns of connection in various brain locations.

Finally, changes in glutamatergic neurotransmission have been linked to neuronal injury following ischemia and hypoglycemia, as well as the etiology of a number of neurological disorders such as epilepsy, Alzheimer's disease, Huntington's chorea, and amyotrophic lateral sclerosis. The number of studies devoted to understanding glutamate-mediated transmission has progressively increased over the years as a result of this diversity of roles. It's no surprise, then, that inotropic glutamate receptors, cationic channels that translate synaptic amino acid release into an immediate neuronal response, are among the most studied and best-understood molecules in the nervous system.

Molecular Physiology

The existence of three kinds of inotropic glutamate receptors, named after their preferred ligands, is now well established: Amino-3-Hydroxy-5-Methyl-4-Isoxazole Propionic Acid (AMPA)-preferring, N-Methyl-D-Aspartate (NMDA)-preferring, and kainate-preferring receptors. The validity of this pharmacological subdivision has been proven by the cloning of a large number of glutamate receptor proteins and the finding of their structural links. At the same time, it has paved the way for the majority of our present understanding of each subtype's biophysical features and physiological role in the mammalian brain.

The research of kainate receptors is one of the best examples of this feature. Although early glutamate receptor classification schemes based primarily on pharmacological and radio ligand binding experiments suggested the existence of a different class of kainate-selective ligands, the evidence for this was confusing. The fact that a given neuron might show a fast desensitizing response to AMPA (or quisqualate, another agonist widely utilized in early experiments) but not any desensitizing response to kainate was regarded as evidence that each ligand was working on a different molecular entity.

However, the discovery that homomeric channels constructed of this protein could be triggered by AMPA and kainate led to the cloning of GluR1, the first AMPA receptor subunit. Furthermore, the responses evoked by each ligand on GluR1 were strikingly similar to those previously seen in nerve cells. Both agonists were demonstrated to act on the same receptor protein in this fashion, calling into question the existence of kainate receptors.

The subsequent cloning of additional glutamate receptor subunits led to the unequivocal identification of a group of true kainate receptors, channels with a strong preference for this agonist over AMPA and rapid desensitizing responses similar to those seen in preparations like the Dorsal Root Ganglia (DRG). Thus, the cloning of a number of kainate receptor subunits was a true milestone in the study of these molecules, laying the groundwork for the phenomenal progress made in their knowledge over the last decade.

In this review, we summaries our present understanding of kainate receptors by first describing their molecular, biophysical, and pharmacological features, areas where the insights gained from studying AMPA and NMDA receptors have shown to be quite useful. The potential activities of these receptor molecules during synaptic transmission are then discussed, as well as their importance for brain function, using several recent examples of kainate receptor participation at the systems level. Finally, we believe that this receptor subtype may be involved in some clinical disorders, most notably epilepsy, emphasizing the need for more selective pharmacological agents to investigate the potential of kainate receptors as antiepileptic therapeutic targets.

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