Altered synaptic physiology and reduced susceptibility to kainate-induced seizures in GluR6-deficient mice

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L-glutamate, the neurotransmitter of the majority of excitatory synapses in the brain, acts on three classes of ionotropic receptors: NMDA (N-methyl-D-aspartate), AMPA (alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) and kainate receptors. Little is known about the physiological role of kainate receptors because in many experimental situations it is not possible to distinguish them from AMPA receptors. Mice with disrupted kainate receptor genes enable the study of the specific role of kainate receptors in synaptic transmission as well as in the neurotoxic effects of kainate. We have now generated mutant mice lacking the kainate-receptor subunit GluR6. The hippocampal neurons in the CA3 region of these mutant mice are much less sensitive to kainate. In addition, a postsynaptic kainate current evoked in CA3 neurons by a train of stimulation of the mossy fibre system is absent in the mutant. We find that GluR6-deficient mice are less susceptible to systemic administration of kainate, as judged by onset of seizures and by the activation of immediate early genes in the hippocampus. Our results indicate that kainate receptors containing the GluR6 subunit are important in synaptic transmission as well as in the epileptogenic effects of kainate.

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