Epilepsy is closely related to an altered transmission of GABA, the major inhibitory transmitter in the brain. GABA acts through two classes of receptors, ionotropic GABA(A) receptors and metabotropic GABA(B) receptors. Using in situ hybridization, receptor autoradiography and immunocytochemistry, we now investigated temporal changes in the expression the GABA(B)-1 and GABA(B)-2 subunits (GABA(B)-1R and GABA(B)-1R, respectively) in the hippocampus following kainic-acid-induced seizures. Significant decreases (by about 40%) in mRNA levels of both splice variants (a and b) of GABA(B)-1R and of GABA(B)-2R were observed in the principal cell layer of the hippocampus 6-12 h after kainic acid injection in the rat. Whereas mRNA levels in the granule cell layer returned to basal after 24 h, the decreases persisted in sectors CA1 and CA3, presumably due to progressing neurodegeneration. In the sector CA3, GABA(B)-R mRNA levels and GABA(B)-R1 immunoreactivity partially recovered 30 days after the initial kainic acid seizures indicating receptor upregulation in surviving neurons.