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The central objective of this proposal is to understand the molecular mechanisms of long-term synaptic plasticity at both excitatory and inhibitory synapses in the vertebrate retina. Previous work has shown that in the retina, three forms of synaptic plasticity can be induced in parallel by subretinal injections of N-methyl-D-aspartate (NMDA), cholinergic and GABAergic agonists, and the metabotropic glutamate receptor (mGluR) agonist, mGluR1 activating protein (mGluR1-aP2). I will use these three types of stimuli and their combinations to address the following Aims. 1) To test the hypothesis that mGluR1-aP2-induced LTP is mediated by CaMKII and activation of p38 MAPK signaling. 2) To test the hypothesis that LTP induced by mGluR1-aP2 and NMDA converge at a functional level. 3) To test the hypothesis that NMDA-induced and cholinergic-induced LTP each are mediated by CaMKII and activation of p38 MAPK, with either one of the forms of LTP normally dominating the others. 4) To test the hypothesis that mGluR1-aP2-induced LTP is blocked by application of a GABA receptor blocker or by activation of G-protein-gated inwardly rectifying potassium (GIRK) channels. All of these pathways are known to converge in specific pathways in the brain, and are thought to play a role in synaptic plasticity in the brain. A multidisciplinary approach will be used to reveal the possible role of these pathways in the retina. I will test for possible involvement of these pathways by pharmacological block of the signals in the various pathways, and by means

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