Activation of mGlu receptors induces LTD without affecting postsynaptic sensitivity of CA1 neurons in rat hippocampal slices.

Two forms of long-term depression (LTD) of excitatory synaptic transmission have been identified in the mammalian CNS, which are induced by the synaptic activation of N-methyl-D-aspartate (NMDA) and metabotropic glutamate (mGlu) receptors, respectively. The mGlu receptor-dependent form of LTD can be activated by application of 3,5-dihydroxyphenylglycine (DHPG), a group I selective mGlu receptor agonist. DHPG-induced LTD is increasingly being used to investigate the mechanisms of mGlu receptor-dependent LTD. However, recent experiments have argued for both a pre- and postsynaptic locus of expression of DHPG-induced LTD. In the present study we report that DHPG-induced LTD is not associated with changes in the sensitivity of CA1 neurons to bath applied AMPA. Furthermore, in contrast to homosynaptic LTD, DHPG-induced LTD is also not associated with changes in sensitivity to focally uncaged L-glutamate. These data do not support the notion that DHPG-induced LTD requires a modification of AMPA receptors, such as their internalisation, but are compatible with a presynaptic mechanism of expression.