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CB1 cannabinoid receptor-dependent long-term depression at excitatory synapses onto hippocampal fast spiking interneurons.

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Group I metabotropic glutamate receptor (mGluR) activation induces long term depression (LTD) at excitatory synapses on pyramidal cells in the CA1 region of the hippocampus in a CB1 cannabinoid receptor (CB1R) dependent manner. The goal of this study was to uncover whether the mGluR-induced LTD at excitatory synapses onto fast spiking interneurons also involve activation of CB1Rs. To address this question, we performed whole-cell patch clamp recordings in CA1 hippocampal neurons in slices prepared from mice followed by a post hoc anatomically identification of the recorded cells. Our results indicate that an mGluR agonist DHPG could induce LTD both in pyramidal cells and fast spiking interneurons in wild-type mice, but not in CB1R knockouts. Pretreatment of the slices with an mGluR5 antagonist MPEP or a DAG lipase inhibitor THL blocked DHPG-induced LTD in both pyramidal cells and fast spiking interneuron. While DHPG in 10 μM or 50 μM induced LTD in CA1 pyramidal cells, DHPG could trigger LTD in fast spiking interneurons only in a concentration of 50 μM. Our results demonstrate that DHPG-induced LTD is dependent on mGluR5, DAG lipase, and CB1R function both in CA1 pyramidal cells and fast spiking interneurons. Thus, fast spiking interneurons can also modulate their excitatory inputs via endocannabinoid signaling, but this control may be achieved at higher activity states compared to that seen in CA1 pyramidal cells.