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Astrocytes Potentiate Transmitter Release at Single Hippocampal Synapses

This paper gave support to the hypothesis that astrocyte calcium signaling in response to neuronal activation plays a role in potentiating the synapse. The authors of this paper performed recordings from CA1 and astrocytes. Calcium signaling was induced by uncaging calcium with UV pulses. These calcium increases were paired with Schaffer collateral stimulation. They found that the calcium elevations induced a short-term plasticity that was caused by a transient increase in release probability. They found that this plasticity was due to glutamate release from astrocytes that depended on group I mGluRs and SNARE proteins.

This figure is from an experiment in this paper that tested the effect of astrocyte calcium elevations on long-term potentiation (LTP). They paired the UV-flash uncaging of astrocyte calcium with postsynaptic neuronal depolarization (ND). A shows fifteen EPSCs on the left and the average EPSCs from 50 trials on the right, measured before and 60 minutes after the pairing procedure. B shows the effect of this procedure on synaptic efficacy, release probability, and synaptic potency. C is the same procedure but rather than uncaging calcium, calcium signaling was evoked by ATP. The white circles show the response in the presence of NMDARs antagonist AP5 and LY367385 and a control experiment of ND without astrocyte stimulation (white circles). D shows the summary of the experiment.

These results show that astrocytes calcium increases paired with postsynaptic activity do induce LTP through a presynaptic mechanism. The induction of LTP did depend on group I mGluRs but not NMDARs. This paper was important because it lent support to the hypothesis that astrocyte calcium increases and calcium-dependent gliotransmission had a role in inducing LTP, but it contradicts both Henneberger et al. found a post-synaptic mechanism of LTP induction that relied on NMDARs and Agulhon et al. found that astrocytic calcium had no effect on LTP induction.

See paper

Return to main project page See more about astrocyte calcium signaling Henneberger et al.–Summary Agulhon et al. – Summary