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Activity:

Learning-induced synaptic and intrinsic plasticity in hippocampus

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Synaptic (LTP, LTD) and intrinsic (membrane excitability) plasticity have been implicated in learning and memory. Understanding how these forms of plasticity interact as a function of associative learning may facilitate development of novel targets for enhancing or preserving memory function not only during normal aging but also in various neurodegenerative disorders. Thus, we have begun studying hippocampal plasticity and using trace fear conditioning as a model system for hippocampus-dependent learning. Adult rats were either naïve or received one 10-trial session of trace fear conditioning (15 s white noise CS, 30 s trace interval, 1 s footshock US; 5.2 min ITI). The next day, these rats were placed into a novel context where they received 2 CS-alone test trials. The rats were then removed from the chamber and brain slices (400 μm) were prepared. Dendritic field recordings were obtained from the stratum radiatum of CA1 using 2-6 MΩ pipettes filled with aCSF. Somatic intracellular recordings were obtained from nearby CA1 neurons using sharp microelectrodes filled with 3M potassium acetate (50-80 MΩ). Concentric bipolar stimulating electrodes were positioned in the stratum radiatum, on either side of the field electrode, to elicit field and intracellular EPSPs from both control and test pathways. After establishing a stable baseline for 10 min, LTP was induced in the test pathway by delivering a single 1 strain at 100 Hz. Both pathways were monitored for at least 30 min by stimulating every 30 s and recording the fEPSP and plotting the fEPSP slope as a percentage of the baseline slope. Recordings were made by an experimenter blind to the training condition. Analysis of the data from 41 experiments indicated that behavioral performance was positively correlated with amount of LTP in conditioned animals ($r = 0.61$, $p = .026$). Moreover, LTP was significantly greater ($F(4,36) = 4.84$, $p < .01$) in animals that were classified as “good learners” (150%; n= 6) compared with “poor learners” (127%; n = 7), naïve (127%; n = 19), or other control animals (chamber exposed [126%; n = 4], pseudoconditioned [120%; n = 5]). The enhanced LTP was also input specific (control pathway didn’t change). Intracellular recordings from CA1 pyramidal neurons revealed an increase in postsynaptic excitability in good learners, evidenced by significantly decreased slow AHP ($F(3,26) = 7.64$, $p < .01$) and less accommodation ($F(3,26) = 5.23$, $p < .01$). These data suggest that acquisition of trace fear conditioning enhances both synaptic and intrinsic plasticity in hippocampal CA1 neurons. Thus, an interaction between intrinsic and synaptic plasticity may underlie the acquisition of trace fear conditioning.

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