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INTRACELLULAR CORRELATES OF EPSP-TO-SPIKE POTENTIATION. L.W. Campbell* and T.J. Sejnowski. Howard Hughes Medical Institute, The Salk Institute for Biological Studies, La Jolla, CA 92037

EPSP-to-Spike (E-S) potentiation has been observed in the rat hippocampus following stimulation trains which lead to LTP in the CA1 cell layer. Most commonly, E-S potentiation is noted as a potentiation in the population spike amplitude in excess of that predicted by the potentiation of the field EPSP slope. Jester, *et al.* (J Physiol, 1995) reported an associative stimulation paradigm which directly produces an E-S potentiation with no change to the EPSP slope. Further work with field potentials recordings and $GABA_B$ antagonists suggest that $GABA_B$ receptors are necessary for the expression of this associative E-S potentiation. What are the intracellular correlates of associative E-S potentiation and in what way are $GABA_B$ receptors involved in its expression?

Intracellular recordings were made from 75 g male Sprague-Dawley rats using sharp electrodes filled with 2M potassium acetate. In order to measure Na spike excitability and accommodation, a series of intracellular depolarizing 150 ms D-C current injections were given at approximately 5 minute intervals. The population spike amplitude and EPSP slope were monitored every 20 seconds with a single shock to the Schaffer-Collateral pathway . The associative tetanization consisted of 50 bursts of 5 antidromic pulses at 100 Hz with an interburst interval of 200 ms, paired with one shock to the Schaffer-Collaterals per antidromic burst.

Tetanization resulted in a slowly developing depolarization of the RMP and an increase in spikes generated in the intracellular current injection series. Counteracting the depolarization with holding current did not fully counteract the increase in spikes generated in the series. Changes in the $GABA_B$-mediated IPSP do not appear to contribute to the expression of E-S potentiation.