ASYMMETRY OF ASSOCIATIVE LONG-TERM POTENTIATION (LTP) IN THE HIPPOCAMPUS ACROSS THE CELL BODY OF CA1 PYRAMIDAL CELLS. J. Jester, P.K. Stanton, and T.J. Selnowski. Dept of Biophysics, Johns Hopkins University, Baltimore, MD 21218.

Long-term potentiation (LTP), a lasting increase in synaptic strength, occurs in hippocampus following high-frequency stimulation. Further, associative LTP has been shown in pathways receiving a weak attinuous concurrent with a strong stimulus to a converging input. We have investigated the association of inputs using patterned stimuli on the same or opposite sides of the cell bodies of CA1 hippocampal neurons.

Extra- and intracellular recordings were made from 400 µm thick slices of rat hippocampus in an interface chamber at 34°C. The stimulus sites were stratum oriens (SO), which synapses on the basal dendrites, and the Schaffer/commissural (SCH) pathway in stratum radiatum (SR) which synapses on the apical dendrites of CA1 pyramidal cells. The "strong" stimulus pattern (which alone induced LTP) consisted of trains of 10 bursts of 5 stimuli given at 100 Hz, with an interburst interval of 200 msec. The "weak" stimulus pattern (which alone had no effect) was a series of single shocks at 5 Hz which either coincided with or came between bursts to the strong site.

Pairing of the SO site as the strong stimulus and SCH as the weak resulted in no change of the epsp slope ($+5.4\pm4.8\%$, n=8) 15-60 minutes post-tetanus. However, when strong and weak sites were reversed, the SO site showed significant increases in both the epsp ($+62.7\pm19.9\%$, n = 7) and the amplitude of the population (pop) spike ($+58.5\pm15\%$, n = 7). When the stimulus sites were in SR on opposite sides of the recording site, pairing strong and weak led to an increase of $+37.0\pm11.1\%$ (n = 10) of the epsp and $+65.4\pm16.0\%$ (n=14) of the pop spike, whereas interleaving weak site shocks and strong site bursts resulted in long-term depression of the pop spike with no change in the epsp. Thus, associative LTP occurs across CA1 cell somata preferrentially when the weak input is in the basal dendrites. (Supported by Office of Naval Research Grant #N00014-88-K-0198)