

ACTION POTENTIAL-EVOKED CALCIUM TRANSIENTS IN SINGLE PRESYNAPTIC TERMINALS OF CULTURED HIPPOCAMPAL NEURONS. V. N. Murthy*, T.J. Sejnowski and C.F. Stevens. Computational Neurobiology, and Molecular Neurobiology Laboratories, The Salk Institute, 10010 N. Torrey Pines Rd., La Jolla, CA 92037.

Exocytosis of synaptic vesicles is triggered by entry of calcium into presynaptic terminals. Presynaptic calcium also plays a role in short-term synaptic plasticity, an example of which is paired-pulse facilitation. In order to assess directly the role of presynaptic calcium in synaptic transmission, we have combined fluorescence imaging and electrophysiological recordings in cultured hippocampal neurons. Whole-cell recordings were obtained using electrodes containing the calcium indicator fluo-3 (100 - 250 μ M). Calcium levels in small axonal varicosities that were in apposition to dendrites were measured with confocal laser scanning microscopy at a temporal resolution of 4 ms. Transient rises in calcium were observed in putative presynaptic boutons in response to single action potentials elicited at a rate of 0.05 - 0.1 Hz in the soma (under voltage clamp). In some recordings, transients with fast rise times < 10 ms, and decay time-constants < 150 ms could be resolved in single trials. In other recordings, calcium transients were smaller and were evident only after averaging across 5 - 10 trials. The peak amplitude of the calcium transients in varicosities appeared to be more variable than predicted from the background noise. Further experiments are aimed at comparing transients evoked by single action potentials, and by pairs of action potentials separated by 50 - 500 ms. Supported by NIH grants to T.J. Sejnowski and C.F. Stevens.