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COMPUTER SIMULATIONS OF E-S POTENTIATION IN HIPPOCAMPAL CA1 PYRAMIDAL CELLS. J.C. Wathey, S. Chattarji, W.W. Lytton, J.M. Iester and T.J. Sejnowski. The Salk Institute, La Jolla, CA 92037.

Long-term potentiation (LTP) of hippocampal excitatory synapses is often accompanied by E-S potentiation (i.e., an increase in the probability of spiking to an EPSP of fixed strength). We used computer simulations of a CA1 pyramidal cell to test the plausibility of the hypothesis that changes in dendritic excitability contribute to E-S potentiation. These changes were simulated by adding "hot spots" of voltage-sensitive Ca^{++} conductance to various dendritic compartments. This typically caused spiking in response to previously subthreshold synaptic inputs. The magnitude of the simulated E-S potentiation depended on the passive electrical properties of the cell, the excitability of the soma, and the relative locations on the dendrites of the synaptic inputs and hot spots. The specificity of the simulated E-S potentiation was quantified by co-localizing the hot spots with a subset (40/80) of the synaptic contacts, denoted "tetanized", and then comparing the effects of the hot spots on these and the remaining (untetanized) synaptic contacts. The simulated E-S potentiation tended to be specific to the tetanized input if the untetanized contacts were, on average, electrically closer to the soma than the tetanized contacts. Specificity was also high if the two inputs were segregated to different primary dendrites. The results also predict, however, that E-S potentiation by this mechanism will appear to be nonspecific (i.e., heterosynaptic) if the synapses of the untetanized input are sufficiently far from the soma relative to the tetanized synapses.

To test this hypothesis we recorded field EPSP and population spikes from area CA1 in rat hippocampal slices. Tetanization of a proximal Schaffer collateral input induced LTP at that input and potentiated population spikes evoked via a more distal untetanized perforant path input. This suggests that postsynaptic excitability changes can contribute to E-S potentiation.