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Synaptic Facilitation of Naive and Depressed Synapses in Aplysia

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To evaluate the contribution of cAMP/PKA signal pathway in short-term facilitation, we overexpressed Ap oa1 receptor in sensory neurons that do not normally express this receptor. We have previously shown that activation of this receptor in sensory cells, by a brief treatment with octopamine (OA), produced short-term facilitation such as membrane depolarization, increase in membrane excitability, spike broadening, and enhanced neurotransmitter release in non-depressed synapse. To assess contribution of PKA pathway on the different degrees of synaptic depression, we examined the ability of octopamine to facilitate depressed synapse in sensory cells expressing Ap oa1. When synaptic connections were moderately depressed to 30-40% of initial EPSP amplitude (ISI=1min) in the pleural-pedal ganglia, the application of OA to the sensory cells expressing Ap oa1 showed a moderate synaptic facilitation compared with that achieved by treatment with 10 uM 5-HT. In the case of highly depressed synapse (up to 10-30% of initial EPSP amplitude, ISI= 1min or ISI= 20sec), PKA pathway activated by Ap oa1 had less effect on synaptic facilitation than that of moderately depressed synapse in the pleural-pedal ganglia. These data are consistent with the previous observations that the role of PKA in synaptic facilitation becomes less effective as the sensori-motor synapse becomes depressed with repeated activity.