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Title: Pre- and postsynaptic roles of PKA, Ca²⁺, and CamKII in sensitization of the *Aplysia* siphon withdrawal reflex
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Sensitization of the *Aplysia* siphon-withdrawal reflex in a simplified preparation is reduced by perfusing the abdominal ganglion with inhibitors of PKA or CamKII but not PKC (Antonov et al., 2005). We have now explored the pre- and postsynaptic roles of PKA and CamKII in experiments in which we recorded from an LE siphon sensory neuron and an LFS siphon motor neuron during behavioral sensitization by 1 or 4 tail shocks. A single shock to the tail produced sensitization of siphon withdrawal and facilitation of the sensory-motor neuron EPSP that both lasted about 30 min, and also broadening of the sensory neuron action potential that lasted less than 15 min. Injection of a peptide inhibitor of PKA (PKAi) into the sensory neuron almost completely blocked the facilitation and spike broadening, whereas injection of BAPTA into the motor neuron had no effect. A train of 4 shocks to the tail produced sensitization and facilitation that both lasted about 60 min, and also broadening of the sensory neuron action potential and increases in its evoked firing and membrane resistance that lasted less than 15 min. Injection of PKAi into the sensory neuron reduced the entire time course of facilitation as well as the transient changes in sensory neuron membrane properties. Injection of BAPTA into the motor neuron reduced facilitation after 15 min, and had no significant effect on the sensory neuron membrane properties. Injection of a peptide inhibitor of CamKII (CamKIIi) into the sensory neuron had effects similar to PKAi, and injection of CamKIIi into the motor neuron had effects similar to BAPTA. These results suggest that sensitization by 4 but not 1 shock involves postsynaptic Ca²⁺ and CamKII. Furthermore, sensitization by both 4 and 1 shock involves presynaptic PKA and CamKII, which cause transient spike broadening as well as some longer lasting mechanism of facilitation.

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