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A theoretical computer model of cellular modification associated with olfactory learning in the rat piriform cortex

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Abstract

Learning-associated cellular modifications were previously studied experimentally in the rat piriform cortex after operand conditioning. The results showed a 19% reduction in the level of the action potential after-hyperpolarization (AHP) in trained rats, while the spike trains indicated decreased adaptation during long depolarization. Paradoxically, this reduced AHP amplitude was associated with a level of depression in the EPSP amplitude, which was significantly higher in trained rats than in the control groups, the pseudo-trained and naive rats.

Our goal in the present study is to analyze and explain through computational techniques the effect of increased EPSP depression after learning. We apply three different models to simulate the exact reduction in the AHP amplitude: (1) "*Conductance change*:" Controlled by decreasing g_{kCa} by 40 %. (2) "*Moving*:" Shifting the location of the dendritic segment that exhibits active conductances, including AHP conductance, distally from the soma, while decreasing g_{kCa} by only 15%. (3) "*Shrinkage*:" Decreasing the length of the AHP dendritic segment, while increasing g_{kCa} by 9%. Moving the synaptic input distally from the soma enhances EPSP depression by the AHP conductance. Hence, the learning process could be simulated by a "jump" from the control curve to any other curve, representing decreased AHP

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amplitude. At the same time, the enhanced EPSP depression requires an additional shift of the EPSP input to more distal locations.
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Keywords: Computer simulations; Pyramidal piriform neurons; Learning; Enhanced inhibition; Decreased AHP amplitude

1. Introduction

We have previously conducted laboratory experiments of cellular modifications, associated with olfactory learning in the rat piriform cortex following operand conditioning [2,8,9,10,12,]. Trained rats were trained in a 4-arm maze to discriminate positive cues in pairs of odors. Training continued until rats acquired the rule of learning, typically after learning the first or second pair of odors. Pseudo-trained and naive rats served as controls. Pseudo-trained rats were randomly rewarded, thus preventing them from acquiring any learning skills, while the naive rats were never exposed to the experimental protocol. The training led to a reduction of the action potential after-hyperpolarization (AHP) in the pyramidal cells in the trained rats, while also decreasing the cells' firing adaptation during depolarizing voltage step. A later study showed that the learning process mainly reduced the slow I_{AHP}, a Ca^{2+}-dependent potassium current that dominates AHP amplitude (Bosh and Barkai, unpublished). EPSPs evoked by neighboring pyramidal cells were increased after training. Intriguingly, inhibition of these EPSPs during maximal AHP conductance, following a short train of action potentials, was more effective after learning than before learning [14].

The goal of the present study is twofold: first to computationally simulate the results, and second to explain why the trained rats' inhibition of EPSP, during maximal AHP conductance is more effective, despite the reduction in the AHP amplitude. We explore alternative models, defined by changes in size, location, or maximal potassium conductance of the AHP. We suggest that a change in the distance between the EPSP's input site and the proximally AHP conductance location [13] could potentially be the cause for the paradoxical phenomena.

2. Methods

The modeling of the piriform pyramidal neuron is executed by a NEURON simulator [4]. The cell is composed of a soma and a single unbranched dendrite [6]. The length and width of the dendrite are set at 1300 and 2 μm, while the length and width of the soma are at 10 and $10/\pi$ μm. The dendrite is reconstructed by 26 isopotential compartments of 50 μm length each. The passive parameters are the uniformly distributed membrane resistance (R_{m}) of 30,000 $\Omega\,\mathrm{cm}^2$ and the capacitance (C_{m}) of 1 $\mu\mathrm{F/cm}^2$. Cytoplasmic resistance (R_{i}) is defined at 100 Ωcm. At the soma, the maximal voltage-dependent channels are sodium conductance (g_{Na}) of 8571 $\mathrm{pS}/\mu\mathrm{m}^2$

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