Muscarinic activation regulates developmental and experience-dependent structural plasticity of honey bee Kenyon cells

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The honey bee (*Apis mellifera*) brain is a model for the study of experience-dependent neuronal plasticity. Young worker bees perform in-hive tasks while older bees exit the hive to forage. The Kenyon cell dendritic fields of 1 day old bees are more complex than those of their 7 day old sisters, and bees with more foraging experience have larger arbors than age-matched bees with less foraging experience [1]. We hypothesize that foraging-induced mushroom body growth is mediated via the same synaptic activity-dependent signaling mechanisms used during development. In the present study, we manipulated cholinergic signaling in the brains of young pre-foragers and experienced foragers. On either the first day of adult life or after accumulating 1 week of foraging experience, bees were caged in the laboratory and treated with pilocarpine (muscarinic agonist) or scopolamine (muscarinic antagonist) for one week. The Golgi method was used for detailed studies of collar Kenyon cell arborizations. Pilocarpine treatment prevented, but scopolamine treatment enhanced, the normal developmental pruning that occurs in young bees. Foragers responded to pilocarpine treatment with increased dendritic complexity. These data suggest that the cytoarchitecture of honey bee Kenyon cells is regulated by the level of muscarinic activation.

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[1] Farris et al. J. Neuroscience 2001 21, 6396-6404