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Molecular and Neural Mechanisms of Salt Chemotaxis Learning

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Summary

Learning ability is essential for animals to better adapt and survive in variable environment, and such ability is documented even in animals with simple neural circuit. Salt chemotaxis of the soil nematode *Caenorhabditis elegans* is a memory-dependent navigation behavior: animals are attracted to the salt concentration at which they have been fed, whereas they avoid it if they have been starved (salt chemotaxis learning).

Input from a single taste neuron, ASER, is required and sufficient for salt chemotaxis under well-fed conditions, and this neuron is activated by salt concentration decrease irrespective of cultivation salt concentrations. Such responsivity of ASER raise a fundamental question as to how animals generate bidirectional migration behavior on salt gradient according to salt experiences. It is suggested that salt experience modulate synapse strength between ASER and a pair of primary interneurons AIB, and this synaptic plasticity is responsible for behavioral change. However, the molecular and cellular mechanisms of ASER-AIB synaptic plasticity remains largely elusive.

To elucidate the relationship of neuronal responses of ASER and AIB as well as behavioral responses of animals to salt stimuli, we observed salt responses of the neurons in free-moving animals. ASER was depolarized by salt down-steps irrespective of cultivation conditions, whereas AIB was activated either by salt decrease after high salt cultivation or by salt increase after low salt cultivation. Increase in turning frequency correlated with AIB activation. These results demonstrate that the sign of synaptic transmission between ASER and AIB is reversed according to salt experience: it is excitatory after high salt cultivation, whereas inhibitory after low salt cultivation. Mutant analyses revealed that both excitatory and inhibitory transmissions are glutamatergic in which EAT-4 (VGLUT) acts in ASER and GLR-1 (AMPA receptor) acts in AIB.