Behavioral forgetting of olfactory learning is mediated by interneuron-regulated network plasticity and multiple signaling in Caenorhabditis elegans

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論 文 名　：Behavioral forgetting of olfactory learning is mediated by interneuron-regulated network plasticity and multiple signaling in *Caenorhabditis elegans*
（線虫において、嗅覚学習の忘却行動は、介在ニューロンの神経回路可塑性と複数のシグナル経路に担われている）

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論 文 内 容 の 要 旨

Forgetting is important for animals to manage acquired memories to enable adaptation to changing environments; however, the neural network in mechanisms of forgetting is not fully understood. To understand the mechanisms underlying forgetting, I examined olfactory adaptation, a form of associative learning, in *Caenorhabditis elegans (C. elegans). C. elegans* displays weak chemotaxis behavior toward diacetyl, one of the attractants, after they are exposed to it for a while but able to recover the chemotaxis behavior later. Such behavioral change is considered as forgetting. The forgetting of diacetyl olfactory adaptation in *C. elegans* is regulated by secreted signals from AWC sensory neurons via the TIR-1/JNK-1 pathway. These signals cause a decline of the sensory memory trace in AWA neurons where diacetyl is mainly sensed. Yet, the neuron network of this forgetting mechanism is not fully revealed. To further understand the neural network and mechanism that regulate this forgetting, I investigated the function of interneurons downstream of AWA and AWC neurons. I found that a pair of interneurons, AIA, is indispensable for the proper regulation of behavioral forgetting of diacetyl olfactory adaptation. Loss of or inactivation of AIA not only caused the impairment of the chemotaxis recovery after adaptation without causing severe chemotaxis defects in naïve animal, but also cause defect in forgetting behavior of two distinctive olfactory adaptation regulated by AWAs and AWCs respectively. Furthermore, based on the AWA and AIA Ca^{2+} imaging result in reported studies, even though AIAs Ca^{2+} response can be seen in naïve animal and after recovery, loss of AIAs cause behavioral defect without causing declination of sensory memory trace in AWAs. In addition, lack of both neuropeptide and glutamate cause prolonged retention of the olfactory adaptation, suggest that multiple chemicals signaling involved in regulating forgetting of olfactory adaptation. Here, I propose that 1) forgetting mechanism might regulated via multiple signaling and 2) the functional neuronal circuit for attractive chemotaxis to diacetyl is changed temporally at the recovery phase so that AIA interneurons are required for chemotaxis, although AIAs are dispensable for attractive chemotaxis to diacetyl in naïve animals.