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MEMORABLE SNAIL

E.A. Kuznetsova, A.A. Artemjewa
dunfrogg@gmail.com, artemjewa.nas@yandex.ru

Studies of the marine snail Aplysia californica have contributed significantly to our modern understanding of the biology of learning and memory. The behavioural modification of Aplysia's siphon withdrawal reflex has been a particularly useful focus of research.

In the 1960s, James Schwartz and Eric Kandel embarked on a research program seeking to establish the biochemical and neuroanatomical basis of learning and memory. Their initial breakthrough came in the 1970s when they established that cAMP and later serotonin were synthesized in Aplysia ganglia during the process of short-term memory formation. Subsequent research showed that cAMP-dependent protein kinase (PKA) and its regulatory effects on potassium channels were relevant to learned behaviour and memory.

The synthesis of C reactive element-binding (CREB) and its influence upon the formation of synaptic connections helped to show that short-term memory was associated with functional changes in existing synapses, whereas long-term memory was associated with a change in the density of synaptic connections. This research earned Kandel the Nobel Prize in 2000.

In 2017 a collaborative study by research groups from Columbia University Medical Center and the Montreal Neurological Institute and Hospital of McGill University (The Neuro) has improved our understanding of memory retention. The researchers stimulated two sensory neurons of Aplysia, both having synaptic connections with the same motor neuron, one to induce associative memory, and another to induce non-associative memory. Their findings could one day lead to therapies that alleviate anxiety disorders and posttraumatic stress disorder (PTSD) by selectively erasing pathological memories. They found that by targeting specific variants of protein kinase M (PKM) in the motor neuron, they could erase the associative and non-associative forms separately because the variants responsible for strengthening the synapses of each of the two sensory neurons are different. In addition, they found that specific memories are also erased by targeting distinct variants of other molecules that either protect specific PKMs from degradation or participate in the generation of specific PKMs.

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