

Researchers uncover previously unknown mechanism of memory formation

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(Medical Xpress)—It takes a lot to make a memory. New proteins have to be synthesized, neuron structures altered. While some of these memory-building mechanisms are known, many are not. Some recent studies have indicated that a unique group of molecules called microRNAs, known to control production of proteins in cells, may play a far more important role in memory formation than previously thought.

Now, a new study by scientists on the Florida campus of The Scripps Research Institute has for the first time confirmed a critical role for microRNAs in the development of memory in the part of the brain called the amygdala, which is involved in [emotional memory](#). The new study found that a specific [microRNA](#)—miR-182—was deeply involved in memory formation within this [brain structure](#).

"No one had looked at the role of microRNAs in amygdala memory," said Courtney Miller, a TSRI assistant professor who led the study. "And it looks as though miR-182 may be promoting local protein synthesis, helping to support the synapse-specificity of memories."

In the new study, published in the *Journal of Neuroscience*, the scientists measured the levels of all known microRNAs following an [animal model](#) of learning. A [microarray analysis](#), which enables rapid genetic testing on a large scale, showed that more than half of all known microRNAs are expressed in the [amygdala](#). Seven of those microRNAs increased and 32 decreased when learning occurred.

The study found that, of the microRNAs expressed in the brain, miR-182 had one of the lowest levels and these decreased further with learning. Despite these very low levels, its overexpression prevented the formation of memory and led to a decrease in proteins that regulate neuronal plasticity (neurons' ability to adapt) through changes in structure.

These findings suggest that learning-induced suppression of miR-182 is a main supporting factor in the formation of long-term memory in the amygdala, as well as an underappreciated mechanism for regulating [protein synthesis](#) during memory consolidation, Miller said.

Further analysis identified miR-182 as a repressor of proteins that control actin—a major component of the cytoskeleton, the scaffolding that holds cells together.

"We know that [memory formation](#) requires changes in dendritic spines on the neurons through regulation of the actin cytoskeleton," Miller said. "When miR-182 is suppressed through learning it halts, at least in part, repression of actin-regulating proteins, so there's a good chance that miR-182 exerts important control over the actin cytoskeleton."

Miller is now interested in whether or not high levels of miR-182 accumulate in the aging brain, something that would help to explain a tendency toward memory loss in the elderly. She also notes that other research has shown that animal models lacking miR-182 had no significant physical or cellular abnormalities, suggesting that miR-182 could be a viable target for drug discovery.

More information: "MicroRNA-182 Regulates Amygdala-Dependent Memory Formation," January 23, 2013, *The Journal of Neuroscience* 33(4):1734-1740; [doi:10.1523/JNEUROSCI.2873-12.2013](https://doi.org/10.1523/JNEUROSCI.2873-12.2013)

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