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Research sheds light on memory by erasing it

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For years, scientists have studied the molecular basis of memory storage, trying to find the molecules that store memory, just as DNA stores genetic memory. In an important study published this week in the Journal of Neuroscience, Brandeis University researchers report for the first time that memory storage can be induced and then biochemically erased in slices of rat hippocampus by manipulating a so-called "memory molecule," a protein kinase known as CaMKII.

"The core problem in memory research has been understanding what the storage molecule actually is. Identifying this molecule is essential to understanding memory itself as well as any disease of memory, " explained lead author John Lisman. "With this study, we have confirmed CaMKII as a memory molecule."

The research involved electrically stimulating neuronal synapses to strengthen them, a process known as long-term potentiation (LTP). This process has served as a model system for studying memory. CaMKII has been a leading candidate as a memory molecule because it is persistently activated after LTP induction and can enhance synaptic transmission, properties that are necessary for a memory molecule.

Like a computer whose electronics change with the addition of new information, molecular activity in the hippocampus, where memory is stored in the brain, changes as memory is being stored. In this study, Lisman and his colleagues showed that they could saturate the memory stores. However, when CaMKII was chemically attacked and previous memory erased, it then became possible to insert new memories in the

synapses.

Alzheimer's and other diseases in which memory loss plays a major role will benefit from this new understanding. Of particular importance may be conditions like epilepsy, which involves synapses that have become overly strengthened. The new research shows how synapses can be weakened by attacking memory molecules.

Lisman's lab plans further research to better understand what happens to the CaMKII after it is attacked. By using fluorescent forms of CaMKII, it will be possible to determine whether the kinase leaves the synapse after inhibitor is applied. This provides a way to directly visualize the forgetting process and complements previous work done in Lisman's laboratory showing that when LTP is induced (as during learning), CaMKII moves into the synapse.

Source: Brandeis University

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