Hold that thought? Scientists find sensor that may explain working memory

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(Medical Xpress) -- In many cases, a delay occurs between the time you are presented information and the time you respond with an action or decision. Most of us call it a thought, while some scientists call it working memory.

University of Wisconsin-Madison researchers believe they now understand on the cellular level how working memory holds a piece of information - or thoughts linger. They found the molecular sensor that controls a little-understood phase of nerve cell communication that keeps a message alive well after it has been delivered.

"The sensor could play a role in keeping a thought 'on line' until it is either lost or burned into longer-lasting forms of memory," says lead author Dr. Edwin Chapman, a Howard Hughes Medical Institute professor at the UW School of Medicine and Public Health.

The study explaining sustained slow nerve cell communication appeared recently in the journal *Cell*.

When one nerve cell sends a signal to another at a synapse, most of the communication takes place instantaneously, with an electrical impulse causing calcium in the sending cell to release a shot of neurotransmitter into the receiving cell. The much-studied action, which makes up the bulk of activity between communicating nerve cells, ends in milliseconds.

But a second, slower phase often follows, in which the signal hangs on such that residual levels of calcium continue to drive the release of neurotransmitters over a much longer period-seconds. Overlooked for a long time, this slow phase of nerve cell communication (also called asynchronous) is now clearly in the spotlight following the Wisconsin studies.

"We knew that different calcium sensors controlled these two distinct phases of synaptic transmission," says Chapman, based in the Department of Neuroscience.

He is an expert on the sensor that gets the fast synchronous phase going, called synaptotagmin 1 (Syt 1), but nobody knew what started the process in the slow phase. So he and his team searched Syt 1-like proteins with different properties that might govern asynchronous transmission.

Jun Yao, Jon D. Gaffaney and Sung E. Kwon were also involved in the research.

The scientists thought the studies could answer important questions about a phenomenon called persistent reverberation, a form of neuronal activity that lasts for seconds after even a single millisecond firing of a neuron. Slow transmission might produce nerve network activity like persistent reverberation, and therefore working memory, they reasoned.

In addition to retaining information that is just experienced but quickly disappears, working memory can also retrieve items from long-term memory for re-evaluation.

The Wisconsin team discovered that Doc2, a protein structurally similar to Syt 1, is the calcium sensor that operates on a timescale consistent with asynchronous release.

"Doc2 took its time responding to calcium, unlike synaptotagmin, which responded immediately," Chapman says.

The researchers could change the speed of slow release with higher and lower levels of Doc2 in brain cells without affecting fast release. And they blocked persistent reverberation in networks of nerve cells when they inhibited Doc2.

Going forward, the UW team hopes to test how different speeds of release affect lab animals' ability to remember or forget.

"Defects in release mechanisms are seen in many nerve diseases, including autism and schizophrenia," says Chapman. "If we could learn more about these mechanisms, we might get insights into the origins of these diseases."

Provided by University of Wisconsin-Madison

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