Neuroscience research may help patients recover from brain injury

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New neuroscience research by life scientists from UCLA and Australia may potentially help people who have lost their ability to remember due to brain injury or disease.

By examining how we learn and store memories, these scientists have shown that the way the brain first captures and encodes a situation or event is quite different from how it processes subsequent similar events.

The study is published in the Sept. 29 edition of the online journal PLoS ONE, a publication of the Public Library of Science.

Memories are formed in the part of the brain known as the hippocampus, a seahorse-shaped structure that plays critical roles in processing, storing and recalling information. The hippocampus is very susceptible to damage through stroke or lack of oxygen and is critically involved in Alzheimer's disease, said study co-author Michael Fanselow, a UCLA professor of psychology and a member of the UCLA Brain Research Institute.

When a memory is first formed, a small protein involved in synaptic transmission — the NMDA receptor — is indispensable to the process, said study co-author Bryce Vissel, a group leader of the neuroscience research program at Sydney's Garvan Institute of Medical Research. Activation of the NMDA receptor allows calcium to enter a neuron, and calcium permeability enables a chain of molecular reactions that help encode experience and consolidate memory, Fanselow and Vissel said.

Learning theorists have assumed that learning cannot occur without NMDA receptors. But the new findings show that NMDA receptors are not essential in "second-learning," when the rules of "first-learning" are applied to new yet similar scenarios. Instead, another class of receptors known as AMPA receptors, also calcium permeable, appears to take up the task.

Although the findings are still preliminary, Fanselow is optimistic about what it could mean for people whose memory formation has been impaired.

"The system we are working with is one that we know is critically involved in Alzheimer's disease and other kinds of brain deficit memory impairment," he said. "This is just the start. We have uncovered a mechanism that contributes to learning and memory, and we now have to figure out what to do with it. When is it important normally? When can we harness it to take over function when the normal mechanisms aren't working? Can we use it to have some protective effect in conditions like Alzheimer's disease, where neurons are dying? Can we stimulate these pathways and keep them participating in memories?

"We can see that we might now have a target for drugs that are different from the standard class of cognitive enhancers," he added. "We can see the possibilities for different styles of training that better activate this newly discovered mechanism."

If the processes involved in second-stage learning can be mimicked therapeutically, he said, the health benefits potentially could be substantial.

Fanselow and Vissel have worked closely over the last six years, along with Thomas O'Dell, a UCLA professor of physiology at the David Geffen School of Medicine at UCLA, to unravel the two different

synaptic mechanisms and their meanings.

"When we started this research, we knew that the NMDA receptor was implicated in learning and memory, and we decided to see if we could mimic its process through another receptor system," said Vissel, a molecular neuroscientist. "Instead of having to create a new receptor system, we discovered one already in existence — one that was NMDA-independent. This amounted to uncovering a whole new mechanism of learning."

Provided by University of California Los Angeles