

Psychiatric disorders linked to a protein involved in the formation of long-term memories

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Researchers have discovered a pathway by which the brain controls a molecule critical to forming long-term memories and connected with bipolar disorder and schizophrenia.

The discovery was made by a team of scientists led by Alexei Morozov, an assistant professor at the Virginia Tech Carilion Research Institute.

The mechanism – a protein called Rap1 – controls L-type [calcium channels](#), which participate in the formation of long-term memories. Previous studies have also linked alterations in these [ion channels](#) to certain psychiatric disorders. The discovery of the channels' regulation by Rap1 could help scientists understand the physiological genesis of bipolar disorder and [schizophrenia](#).

"People with [genetic mutations](#) affecting L-type calcium channels have higher rates of bipolar disorder and schizophrenia," said Morozov. "This suggests that there might be a relationship between the activation of L-type calcium channels and these psychiatric disorders. Understanding how these ion channels are controlled is the first step to determining how their functioning or malfunctioning affects mental health."

A single neuron in the brain can have thousands of synapses, each of which can grow, strengthen, weaken, and change structurally in response to learning new information. [Electric signals](#) traveling from neuron to

neuron jump across these synapses through chemical neurotransmitters. The release of these chemicals is caused by the flow of electrically charged atoms through a particular subset of ion channels known as voltage-gated calcium channels.

Previous studies have shown that blocking these ion channels inhibits the formation of long-term memories. Although it was known that L-type calcium channels are activated in response to learning, how they are controlled was a mystery.

In the experiment, Morozov and colleagues knocked out the gene responsible for coding the enzyme Rap1, which he suspected played a role in activating L-type calcium channels. The researchers then used live imaging techniques to monitor the release of neurotransmitters and electron microscopy to visualize L-type channels at synapses. They discovered that, without Rap1, the L-type calcium channels were more active and more abundant at [synapses](#) all the time, increasing the release of neurotransmitters. The results showed that Rap1 is responsible for suppressing L-type calcium channels, allowing them to activate only at the proper moments, possibly during long-term memory formation.

"Our next step is to determine whether this new signaling pathway is altered in cases of mental disease," said Morozov. "If so, it could help us gain a better understanding of the molecular underpinnings of channel-related [psychiatric disorders](#), such as bipolar disorder and schizophrenia. Such knowledge would go a long way toward developing new therapeutic methods."

More information: The discovery appeared in *The Journal of Neuroscience* in the study "Rap1 Signaling Prevents L-Type Calcium Channel-Dependent Neurotransmitter Release," by Jaichandar Subramanian: www.jneurosci.org/content/33/17/7245.abstract

Provided by Virginia Tech

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