

Researchers identify changes that may occur in neural circuits due to addiction

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A research team from the Friedman Brain Institute of the Icahn School of Medicine at Mount Sinai has published evidence that shows that subtle changes of inhibitory signaling in the reward pathway can change how animals respond to drugs such as cocaine. This is the first study to demonstrate the critical links between the levels of the trafficking protein, the potassium channels' effect on neuronal activity and a mouse's response to cocaine. Results from the study are published in the peer-reviewed journal *Neuron* on May 7, 2014.

The authors investigated the role of sorting nexin 27 (SNX27), a PDZ-containing protein known to bind GIRK2c/GIRK3 channels, in regulating GIRK currents in dopamine (DA) neurons on the [ventral tegmental area](#) (VTA) in mice.

"Our results identified a pathway for regulating the excitability of the VTA DA neurons, highlighting SNX27 as a promising target for treating addiction," said Paul A. Slesinger, PhD, Professor, Department of Neuroscience, Friedman Brain Institute, Icahn School of Medicine at Mount Sinai.

"Future research will focus on the role that [potassium channels](#) and trafficking proteins have in models of addiction," said Dr. Slesinger.

Dr. Slesinger was the lead author of the study and joined by Michaelanne B. Munoz from the Graduate Program in Biology, University of California, San Diego and the Peptide Biology Laboratories, The Salk Institute for Biological Studies, La Jolla, California.

Provided by The Mount Sinai Hospital

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