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Mutations found in individuals with autism interfere with endocannabinoid signaling in the brain

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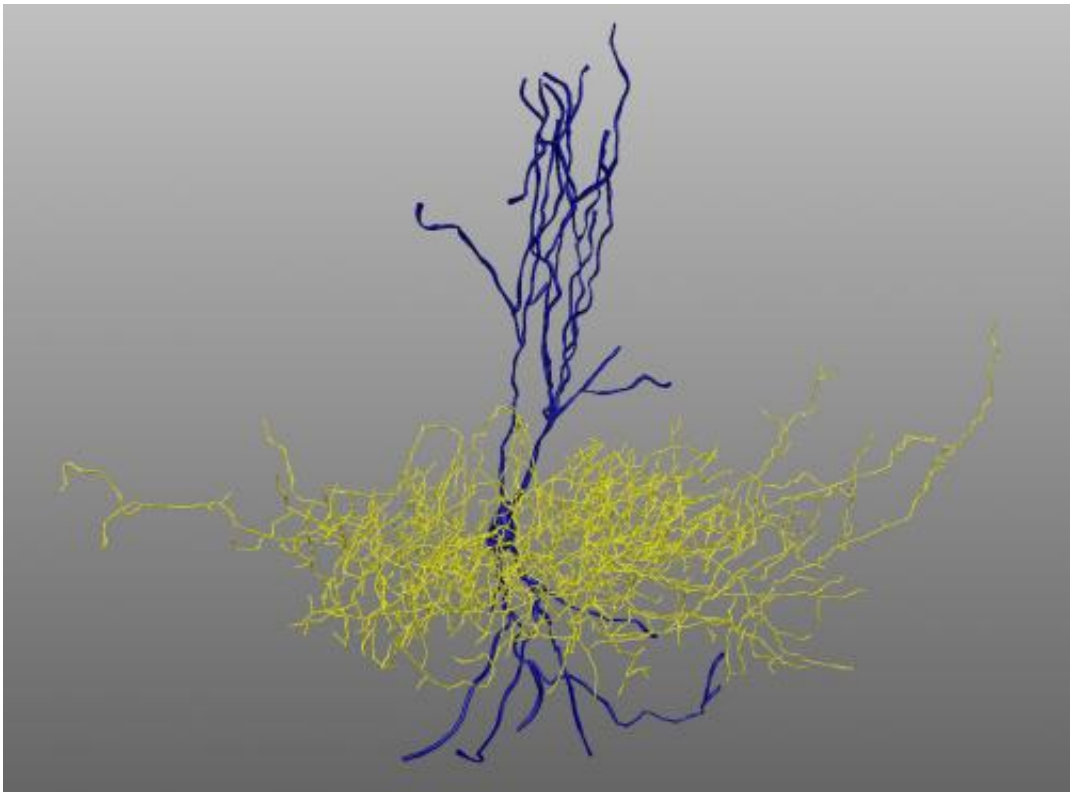

This image shows an inhibitory neuron whose function is affected by neuroligin mutation. Credit: *Neuron*, Foldy et al.

Mutations found in individuals with autism block the action of molecules made by the brain that act on the same receptors that marijuana's active

chemical acts on, according to new research reported online April 11 in the Cell Press journal *Neuron*. The findings implicate specific molecules, called endocannabinoids, in the development of some autism cases and point to potential treatment strategies.

"Endocannabinoids are molecules that are critical regulators of normal neuronal activity and are important for many brain functions," says first author Dr. Csaba Földy, of Stanford University Medical School. "By conducting studies in mice, we found that neuroligin-3, a protein that is mutated in some individuals with autism, is important for relaying endocannabinoid signals that tone down communication between neurons."

When the researchers introduced different autism-associated mutations in neuroligin-3 into mice, this signaling was blocked and the overall excitability of the brain was changed.

"These findings point out an unexpected link between a protein implicated in autism and a signaling system that previously had not been considered to be particularly important for autism," says senior author Dr. Thomas Südhof, also of Stanford. "Thus, the findings open up a new area of research and may suggest novel strategies for understanding the underlying causes of complex brain disorders."

The results also indicate that targeting components of the endocannabinoid signaling system may help reverse autism symptoms.

The study's findings resulted from a research collaboration between the Stanford laboratories of Dr. Südhof and Dr. Robert Malenka, who is also an author on the paper.

More information: Foldy et al.: "Autism-Associated Neuroligin-3 Mutations Commonly Disrupt Tonic Endocannabinoid Signaling."

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