

Gene mutation enhances cognitive flexibility in mice, study suggests

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Researchers at the National Institutes of Health have discovered in mice what they believe is the first known genetic mutation to improve cognitive flexibility—the ability to adapt to changing situations.

The gene, *KCND2*, codes for a protein that regulates potassium channels, which control [electrical signals](#) that travel along neurons. The electrical signals stimulate chemical messengers that jump from neuron to neuron. The researchers were led by Dax Hoffman, Ph.D., chief of the Section on Neurophysiology at NIH's Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD). It appears in *Nature Communications*.

The *KCND2* protein, when modified by an enzyme, slows the generation of electrical impulses in neurons. The researchers found that altering a single base pair in the *KCND2* gene enhanced the ability of the protein to dampen nerve impulses. Mice with this mutation performed better than [mice](#) without the mutation in a cognitive task. The task involved finding and swimming to a slightly

submerged platform that had been moved to a new location. Mice with the mutation found the relocated platform much faster than their counterparts without the mutation.

The researchers plan to investigate whether the mutation will affect neural networks in the animals' brains. They added that studying the gene and its protein may ultimately lead to insights on the nature of cognitive flexibility in people. It also may help improve understanding of epilepsy, schizophrenia, Fragile X syndrome, and [autism spectrum disorder](#), which all have been associated with other mutations in *KCND2*.

More information: Jia–Hua Hu et al, Activity-dependent isomerization of Kv4.2 by Pin1 regulates cognitive flexibility, *Nature Communications* (2020). [DOI: 10.1038/s41467-020-15390-x](https://doi.org/10.1038/s41467-020-15390-x)

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