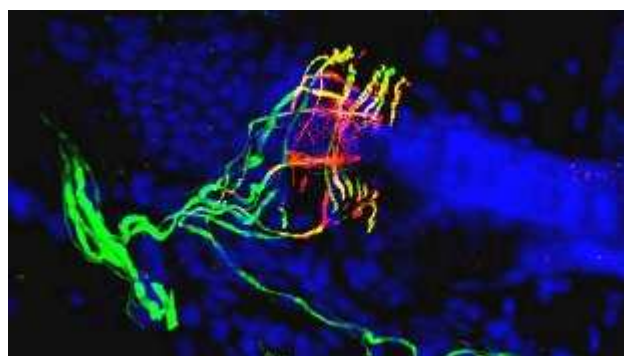


[News & Opinion](#)

Deafness Gene Heightens Touch

People with a defect in an ion channel that causes deafness are more sensitive to certain types of touch.

By Tia Ghose | November 20, 2011



KCNQ4 channel (red) at skin mechanosensory nerve endings (green) Matthias Heidenreich

A super sense of touch may be the unexpected byproduct of a gene mutation that causes deafness, according to a paper published today (November 21) in *Nature Neuroscience*. The new study reveals that a potassium ion channel on sound-detecting hair cells also modulates touch sensitivity, and that those with defective channels are better able to sense low frequency touch vibrations.

“Our sense of touch is something that is really poorly understood,” said Stanford University School of Medicine neuroscientist [Miriam Goodman](#), who was not involved in the study. The study “opens the door

to really knowing how it is that this ion channel regulates the vibration sensitivity of different touch-sensitive neurons,” she said. In addition, the study shows that “defects in this gene that may cause you to lose your sense of hearing may heighten your sense of touch, which I think is just cool!”

In 1997, [Thomas Jentsch](#), a neuroscientist at the Leibniz-Institut für Molekulare Pharmakologie in Berlin, and his colleagues [discovered](#) that mutations in one type of potassium channel called KCNQ1 caused heart rhythm abnormalities. Mutations in other subtypes led to epilepsy and mutations in one, called KCNQ4, caused a form of progressive deafness in people. The team also found that the that KCQN4 channel was found in just a few specific locations: inner ear hair cells which translate vibration into sound, a part of the brain stem involved in hearing, and curiously, neurons in the skin that help us sense touch.

In the current study, they further investigated the KCNQ4 channel in the skin by electrically stimulating the skin of mice. They found that the channel was only located in neurons that quickly adapt to low-frequency touch vibrations, where it appeared to quickly dampen the signals being fired. Cells with the deafness-causing mutant form of KCNQ4, on the other hand, maintained frequent firing in response to low-frequency vibration.

“What these genes do in other neurons is they act as a brake for excitability,” said neurobiologist [Ellen Lumpkin](#) of Columbia University, who was not involved in the study. “So if you inactivate that gene you should get more excitability for these touch receptors.” The same excitability is likely going on in the inner ear to cause deafness, she added—the exquisitely delicate hair cells “are so excitable that they become overloaded and they die.”

The researchers also tested members of large Dutch and Spanish families that carried the mutation in KCNQ4 gene, whose hearing had slowly degenerated from childhood. Sure enough, family members were more sensitive to low frequency vibrations—a difference that may translate to being better able to distinguish between textures with your fingertips, Goodman said, such as corduroy and silk.

But while being “super feelers,” sounds great on paper, Jentsch is not convinced it’s a benefit . “I would rather suspect this is a disadvantage, otherwise nature wouldn’t have put KCNQ4 in these nerve endings.” It’s also unlikely to be an adaptation to deafness, as the people were likely born with higher touch sensitivity but lose their hearing only gradually.

Furthermore, the KCNQ4 mutation represents only one of more than 60 different gene mutations that can cause deafness, so it’s unlikely that those with other forms of deafness experience this boost in touch sensitivity, Jentsch added.

M. Heidenreich, et. al, “KCNQ4 K⁺ channels tune mechanoreceptors for normal touch sensation in mouse and man,” *Nature Neuroscience*, doi:10.1038/nn.298, 2011.

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