Cold feeling traced to source

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For the first time, neuroscientists have visualized cold fibers – strands reaching from sensory neurons near the spinal cord to nerve endings in the skin tuned to sense different types of cold. The study and pictures appear in the Dec. 19 issue of the *Journal of Neuroscience*.

Surprisingly, given the highly diversified sensory system and the range of sensations studied – harmless cool, stinging cold, soothing coolness – the fibers lead back to one place in the neuron: a protein known as TRPM8 that relays a cold signal up the spinal cord to the brain.

The idea of a cold fiber is simple. When the dentist chills a tooth with compressed air, the fiber carries a signal from nerve ending to sensory neuron. The neuron relays the signal to the brain, and the patient shivers.

In practice, said USC study leader David McKemy, “no one’s actually seen a specific cold fiber.”

McKemy’s study solved that problem by genetically engineering mice in which neurons that express TRPM8 molecules also included a fluorescent tracer that lights up the fibers.

McKemy’s study provides the first visualization of cold-sensing, TRPM8-expressing neurons. Previous studies had shown that mice lacking TRPM8 lose much of their cold sensitivity (video available at www.nature.com/nature/journal/ … nfo/nature05910.html).

Humans and other mammals appear to share the same mechanism, McKemy said.

By following the fluorescent cold fibers, the researchers added to the evidence that TRPM8 is involved in several types of cold sensing. In teeth, the distinct nerve endings involved in the initial shooting pain and the subsequent dull ache both lead back to TRPM8, McKemy said.

Sensations such as the pleasant coolness of menthol, the sting of ice on the skin, the heightened cold sensitivity after an injury and the soothing cool of some pain relief lotions also involve TRPM8, he added.

Removing TRPM8 does not eliminate all sensitivity to all types of cold. Extreme cold not only activates TRPM8 but also burns the skin, turning on many other warning circuits.

“Cold is going to be activating these cool and cold cells that likely are the ones we’re studying in this paper as well as activating these neurons that are probably responding to tissue damage,” McKemy said.

“So your higher cognitive centers are processing a cool signal and a pain signal, and so we get cold pain.

“As with anything with biology, it’s not as simple as you would think.”

McKemy was the lead author of a landmark 2002 study, published in Nature, that first identified the cold-sensing role of TRPM8.

One larger goal of such research is to understand the molecular mechanisms of sensation, in the hope of developing better drugs for relief of chronic pain states, such as arthritis and inflammation.

“If we understand the basic nuts and bolts of the molecules and neurons

and how they detect pain normally," McKemy said, "then perhaps we can figure out why we detect pain when we shouldn't."

Source: University of Southern California

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