Light-sensing receptor plays role in temperature sensation: study

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A light-sensing receptor that's packed inside the eye's photoreceptor cells has an altogether surprising role in cells elsewhere in the body, Johns Hopkins scientists have discovered. Using fruit flies, they showed that this protein, called rhodopsin, also is critical for sensing temperature.

A report on the work appears March 11 in *Science*.

“For decades, this well-known molecule — one of the most-studied sensory receptors — was thought to function exclusively in the eye as a light receptor, but now we have found that fly larvae and possibly other organisms use it to distinguish between slight temperature differences,” says Craig Montell, Ph.D., a professor of biological chemistry and member of the Center for Sensory Biology in the Institute for Basic Biomedical Sciences. “And it makes you wonder about what was the more ancient role for rhodopsin — was it used originally for light or temperature detection?”

The Hopkins team identified rhodopsin while investigating the process that results in the activation of a temperature-sensor protein known as a TRPA1, one of many so-called “trip” channels abundant on sensory cells that receive communication from the outside world. Montell discovered earlier that TRPA1 enables fly larvae to detect tiny changes in the range of temperature that’s optimal for their survival. However, unlike TRP channels that function in avoiding hot and cold temperatures, TRPA1 was not directly turned on by changes in temperature in the comfortable temperature range, which extends from 18 to 24 degrees centigrade

(equivalent to about 64 to 75 degrees Fahrenheit).

The team set out to determine what receptor responds to the temperature in order to set off the signaling cascade that results in TRPA1 activation.

A reasonable place to start looking for likely suspects, Montell said, was the large family of G-protein coupled receptors, because they are cell-surface molecules known to activate TRP channels. Still, the researchers were faced with more than a hundred possible gene candidates, each coding for a different G-protein coupled receptor in flies: If it was a GPCR, then which one?

“There were no precedents for a GPCR functioning in thermosensation, leaving us wonder where to start,” Montell says. “We considered rhodopsin, even though it was thought to be required exclusively for light reception, because some of the other proteins that we showed previously to function in thermosensation were required in photoreceptor cells.”

Using larvae missing the gene that codes for rhodopsin, the team conducted a series of tests to compare their behaviors with normal (wild-type) animals. The researchers released about 75 larvae on a plate with two temperature zones; half of the plate was kept at their favorite temperature of 18 degrees C, and the other at an alternative temperature, ranging from 14 to 32 degrees C. After 10 minutes, the researchers counted the number of larvae that had crawled to the 18-degree side, and the number on the side with the alternative temperature. They discovered that in contrast to the wild-type larvae, which preferred 18 degrees over any other temperature, the larvae lacking rhodopsin couldn’t discriminate temperatures in comfortable range, just like the larvae lacking TRPA1. However, the rhodopsin mutant larvae were able to choose 18 degrees over temperatures that were too hot or cold.

“The genetics and the behavior show that rhodopsin is required for thermosensation,” Montell says. “Larvae that contain mutations disrupting rhodopsin are profoundly defective in their ability to sense temperatures, but only in the comfortable range. The simplest interpretation of these results is that rhodopsin is activated by temperature and this in turn, activates TRPA1. However, we cannot exclude that there is an additional accessory protein required for rhodopsin to act as a thermosensor.”

This rhodopsin which functioned in “feeling” temperature was required in a new type of thermosensory neuron in the body wall of fruit fly larvae as well as in neurons in the head region of the animals.

Montell says this new thermosensing role for rhodopsin has absolutely nothing to do with light. Wild-type fly larvae kept in a dark box were able to choose the preferred 18 degrees centigrade over 24 degrees C.

The indirect activation of the TRPA1 channel via a signaling cascade that requires rhodopsin most likely represents “a quality of life issue” for the larvae, Montell muses. It allows to them to give up avoiding temperatures that are slightly less preferred than 18 degrees and to adapt if they can’t find their favorite temperature in their thermal landscape. Direct activation of TRP channels by noxious temperatures is more about survival.

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