

Project fruit fly: What accounts for insect taste?

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fruit fly

A Johns Hopkins team has identified a protein in sensory cells on the "tongues" of fruit flies that allows them to detect a noxious chemical and, ultimately, influences their decision about what to eat and what to avoid.

A report on the work, appearing April 19 in the online Early Edition of the <u>Proceedings of the National Academy of Sciences</u> (PNAS), raises the possibility that the protein — TRPA1 — is a new molecular target for controlling insect pests.

"We're interested in how TRPA1 and a whole family of so-called TRP channels affect not just the senses, like taste, but also behavior," says Craig Montell, Ph.D., a professor of <u>biological chemistry</u> and member of



the Center for Sensory Biology in Johns Hopkins' Institute for Basic Biomedical Sciences.

Montell notes that when his team knocked out the TRPA1 sensor, the behavior change — an alteration in food preference — was stark. "This is the first TRP channel in insects that responds to a naturally occurring plant chemical known as an antifeedant, so now we have a target for finding more effective chemicals to protect plants from destruction by insect pests."

Montell discovered TRP (pronounced "trip") channels in 1989 in flies and, a handful of years later, in humans, noting their abundance on sensory cells that communicate with the outside world. The job of these pore-like proteins — activated by a bright light, a chilly breeze or a hot chili pepper — is to excite cells to signal each other and ultimately alert the brain by controlling the flux of atoms of calcium and sodium that carry electrical charges. Montell's lab and others have tallied 28 TRP channels in mammals and 13 in flies, improving understanding about how animals detect a broad range of <u>sensory stimuli</u>, including the most subtle changes in temperature.

"We already knew that TRP channels have these broad sensory roles, having previously discovered that the insect TRPA1 had a role in helping flies to detect small differences in sub-optimal temperatures within their comfort range," Montell says. "We wondered if it had any other sensory roles, so we went looking."

First, the team genetically altered a normal TRPA1 gene. This experiment let them show that the protein was made in the fly's major taste organ (called the labellum) and trace its manufacture to a subset of sensory cells that respond to noxious chemicals. Separate taste cells in mammals are also known to respond to either noxious or appealing chemicals in foods.



The researchers then conducted a series of behavioral tests comparing the feeding of wild type flies to those of mutants in which the TRPA1 gene was knocked out — unable to manufacture the protein.

The team placed 50 to 100 flies that had been purposely starved for a day in a covered plate with 72 wells full of two concentrations of sugar water. The wells containing the high concentration of sugar water were laced with different bitter compounds, including quinine, caffeine, strychnine and aristolochic acid. This bitter/sugar water was distinguished with blue food coloring as opposed to the pure sugar water, colored red. A wild type fly normally would consume the more sugary water because, like humans, it has a "sweet tooth." However, if the more sugary water is laced with an aversive flavor, they choose the less sugary water.

After allowing the hungry wild type and mutant flies to feed from the wells, the team froze and then counted the insects, separating them based on belly color: red, blue or purple. Surprisingly, most of the mutants avoided all but one of the bitter compounds — aristolochic acid, a naturally occurring chemical produced by plants to prevent themselves from being eaten by insects. The majority of the wild type were red, the appropriate color for having chosen the less sugary water; and the mutants mostly were blue, the color associated with the high concentration of sugar laced with aristolochic acid, because they couldn't taste the noxious chemical.

"To our surprise, it was looking at first like TRPA1 didn't have a role in responding to anything," Montell said. "The aristolochic acid was literally the last compound we tried. I certainly wasn't expecting that the TRPA1 would be so specific in its response."

The team followed up with electrophysiology tests on both wild type flies and those lacking the TRPA1 gene. By attaching electrodes to the



tiny taste hairs on the labellum, the scientists were able to measure the taste-induced spikes of electrical activity resulting from neurons responding to the noxious chemicals. <u>TRPA1</u> was required for aristolochic acid-induced activity by neurons, meaning it's essential for aristolochic acid avoidance.

TRP channels also play important roles in taste in mammals, but the requirement is very different, Montell says. While one mammalian TRP channel is required for tasting all sugars and bitter chemicals, no single insect TRP has such a broad role.

"It's important to make this discovery in insects, not only because it's interesting to trace the similarities and differences through millions of years of evolution, but also because of the possible practical applications" Montell says. "By targeting this TRP channel, we might be able to prevent insects from causing crop damage."

More information: Craig Montell lab: <u>neuroscience.jhu.edu/CraigMontell.php</u> PNAS: <u>www.pnas.org/</u>

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