are lacking in modern humans and other primates. (Two later hominids—*A. africanus* and *Paranthropus robustus*—lacked these specializations.) "These features in the early hominid bones can't be explained except that they are uniquely related to knuckle walking," says John Fleagle, a paleoanthropologist at the State University of New York, Stony Brook, the institution from which Richmond and Strait received their Ph.D.s. And these common traits imply that the common ancestor of australopithecines, chimps, and gorillas was a knuckle walker. The knuckle-walking traits were lost in hominids—by about 2.5 million to 3.0 million years ago, according to specimens of *A. africanus*, Richmond says.

But the finding raises other questions, such as why a climbing creature already adapted for traveling on the ground would evolve the ability to stand on two feet as well. "In some ways, for me, it makes it more difficult to understand the evolution of bipedalism," Potts says. One idea is that walking upright freed the hands for other uses, such as carrying food, tools, or weapons, says Carol Ward, a paleoanthropologist at the University of Missouri, Columbia. "The big problem is that we don't have a fossil record of the chimp-human-gorilla ancestor," she notes. "So what you have to do is build an argument based on parsimony and hope for the best."

–ERIK STOKSTAD

LOOKING AHEAD

Medalists Gaze Out on A Familiar Future

Who says good scientists need data to voice an opinion? Last week the newest winners of the national medals of science and of technology (*Science*, 4 February, p. 785) spent an hour speculating on what the world might look like in 2025 and whether "innovation will surpass science fiction." The spirited discussion among the 15 medalists—part of a daylong series of events that culminated in presentation of the awards by President Clinton—flowed freely around such knotty questions as the impact of technology on the quality of life and what drives human behavior. Not surprisingly, there was no consensus. But although a few scientists declined to venture outside their own discipline, most were happy to extrapolate from today the shape of tomorrow.

Computer scientist Raymond Kurzweil spoke glowingly of nanobots communicating directly with our neurons to repair damaged tissue, part of a panoply of technological advances that would bring good health and prosperity to all. His sunny view, however, clashed with conservation biologist Jared Diamond's warning about a collection of 25-year "time bombs"—in particular the loss of biodiversity—that must be defused before humanity can prosper. Cellular biologist Lynn Margulis was even gloomier, fretting about how the desire to procreate could lead to unsustainable population levels that would overwhelm the capacity of any technology. In rapid succession, the three scientists gave thrust and parry, conceding nothing.

The issue of how to monitor where the world was headed proved equally hard to pin down. Economics Nobelist Robert Solow objected to overly optimistic predictions of ever-expanding productivity from computers and electronic communications, saying that the slight gains in recent years have yet to survive a recession. Kurzweil disagreed, saying that traditional economic measures were no match for the new economy, but Solow stuck to his guns. Taking another tack, medicine Nobelist David Baltimore opined that productivity itself was a poor measure of progress and that, for most people, an improved quality of life from modern pharmaceuticals was a more meaningful indicator.

Moderator Ira Flatow, a science journalist, seemed happy to let participants state their views and take their shots, leaving the audience to draw its own conclusions. But at least one panelist expressed displeasure at how the issues were being framed. "I don't know what life will be like in 2025, and I don't think scientists have much that's useful to say about the topic," commented physics Nobelist James Cronin after the roundtable ended. "But I can promise you that in 25 years we will know a lot more about the composition of the universe. That's what science can give the world. And I think that's pretty important."

–JEFFREY MERVIS

On stage. This year's medalists include (top row, from left): Stewart Rice, Robert Solow; (second row): Leo Kadanoff, Jerome Swartz, Susan Solomon, Kenneth Stevens, Felix Browder; (third row): Judy Swanson (for Robert), John Ross, Lynn Margulis, James Cronin; (bottom): Glenn Culler, Ray Kurzweil, Jared Diamond, David Baltimore, Ronald Coifman. Not shown: Robert Taylor.

NEUROSCIENCE

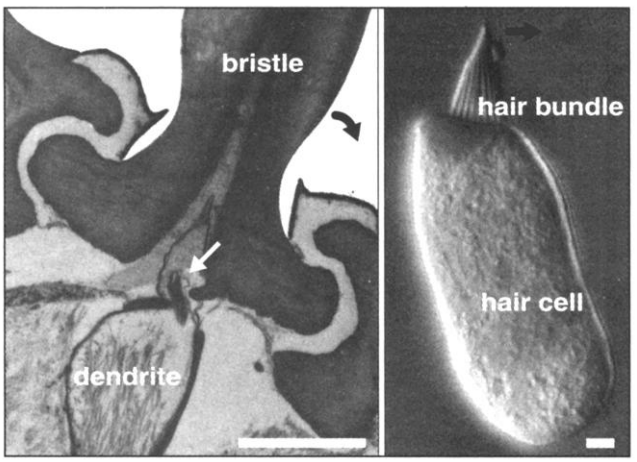
New Ion Channel May Yield Clues to Hearing

As every biology student learns, the sense of hearing depends on the operation of the hair cells in the inner ear. These cells bear microscopically fine projections, the hairs or cilia, that bend in response to passing sound waves, setting off nerve impulses that the brain recognizes as sounds—a clap of thunder, say, or a hushed whisper. But even though neuroscientists have learned a great deal about hair cells, they have been unable to track down a key element needed for the cells' operation—the ion channel that opens when the hairs bend to produce the electrical signal. Now, working with a seemingly different system, they've made a discovery that may help them get their hands on the elusive channel.

On page 2229, a team led by Charles Zuker of the University of California (UC), San Diego, reports that it has cloned an intriguing ion channel from the neurons that underlie the sensory bristles of the fruit fly. It is a mechanically sensitive channel—in other words, it responds to mechanical force instead of voltage changes or biochemical modifications. At first blush, the fruit fly bristles, visible with a magnifying glass, appear to be quite different from the microscopic bundles of hair cells within the human ear. But the Zuker team has shown that the neurons beneath the bristles operate much like the hair cells as they convert movement into electrical impulses. That has some researchers thinking that the functioning of the two types of cells may depend on structurally similar ion channels. If so, the new gene could provide a useful probe for fishing out the channel in human hair cells—an accomplishment that could lead to new insights into the causes of hereditary deafness and perhaps ways to correct it.

"I am really excited about [this] channel," says Cornelia Bargmann, who studies sensory systems at UC San Francisco. Although there are other candidates for such mechanically sensitive channels, including one discovered by Bargmann's team, she calls Zuker's the "most intriguing candidate right now" because of its possible connection to hair-cell channels, with their "clear medical relevance and interesting biophysics."

Hair-cell physiologists have long wanted to see what the hair-cell channel looks like, because their experiments had shown that it has fascinating biophysical properties. By studying the electrical currents passing through the membranes of hair cells as they are stimulated, they learned that hair-cell channels are stunningly fast, opening up within microseconds, compared to the milliseconds needed by biochemically activated channels. They are also exquisitely sensitive to the slightest movement and to direction; they open when the tip of the cell's cilia bundle is deflected by a mere atom's width—akin to bending the tip of the Eiffel Tower by the width of your thumb. If the cilia bundle moves one way, the channel opens; the other way and it shuts. The channels are also able to register tiny cilia movements on top of a larger constant deflection—a trait that lets us discern meaningful sounds from background noise.



Nonidentical twins. Both this insect sensory bristle *(left)* and this hair cell from the inner ear of a frog *(right)* have ion channels that respond to the deflections shown by the black arrows.

CREDITS: (TOP TO BOTTOM) PHOTOGRAPHS COURTESY OF THOMAS KEIL [*MICROSC. RES. TECH.* **39**, 506 (1997)]; R. A. JACOBS, AND A. J. HUDSPETH; ZUKER LAB/HOWARD HUGHES MEDICAL INSTITUTE

Efforts to isolate the channels have been stymied, however, primarily because hair cells are so sparse and contain relatively few channel molecules. So Zuker decided to apply the power of fruit fly genetics to the problem, on the hunch that the flies' bristles might contain channels similar to those in hair cells. In the first phase of the work, begun about 7 years ago, Zuker and then-postdoc Maurice Kernan, now at the State University of New York, Stony Brook, created mutant flies and screened them for those that were defective in their sense of touch. Some of those flies, they reasoned, would have mutations in genes specific to the touch response—including the gene for the touch-sensitive channel itself.

In a separate phase of the work, postdoc Richard Walker, who arrived in Zuker's lab in 1996, examined whether the bristle system would be a good model for hair cells. It was. Using electrophysiological methods similar to those employed to study hair cells, Walker found that when the fly neurons respond to touch, they share key characteristics of hair cells: fast responses to even the tiniest movements, directional sensitivity, and adaptability to new bristle positions. Hair-cell researcher David Corey of Massachusetts General Hospital in Boston calls the comparison "beautiful." Walker "repeated the last 20 years of human hair-cell physiology on this bristle system," he says, "and everything looks the same."

Walker then applied the same methods to the bristle neurons of the mutant flies to search for those in which the mutations caused defects in the channel's function—a good indication that the affected gene encodes the channel. He found that a gene called *nompC* (for no mechanoreceptor potential C) seemed to fit the bill. Mutations in *nompC* either blocked the opening of the channel in response to bristle movement or in one case altered the channel so it opened but let through less current than normal. To Bargmann, this is the "most convincing" evidence that the NOMPC protein is the mechanically sensitive bristle channel.

The sequence of the *nompC* gene supports that view, as it encodes a protein with the general structural features of proteins that form ion channels. The gene sequence also contains a clue to how mechanically sensitive ion channels open. To be tugged open, a channel must be anchored so that pulling on it changes its shape. NOMPC appears to have "a great way of anchoring the channel" to the cell's skeleton, says Corey. This is a set of 29 so-called ankyrin repeats—short amino acid sequences that link up to other proteins.

Although all these data constitute strong evidence that NOMPC is a mechanically sensitive channel, definitive proof would require putting it into cultured cells and showing that it renders them responsive to touch. That is a tough experiment, because other specialized proteins are likely required for NOMPC function. And even if NOMPC does turn out to be a mechanically sensitive channel in flies, that doesn't necessarily mean that it will be related to the elusive hair-cell channel in vertebrates.

So far, opinion on that issue is mixed. Neuroscientist Denis Baylor of Stanford Medical School is cautious. "The anatomy [of bristles and hair cells] is so different that I wouldn't be surprised if [the hair-cell channel] is a completely different molecule, not even a relative," he says. But Corey and fellow hair-cell researcher James Hudspeth of The Rockefeller University in New York City come down on the other side. Given the similarities that Walker found between the hair-cell responses and those of the bristle neurons, "chances are very good" that the two are related, Hudspeth says.

To find out, his team is now using the Zuker group's cloned gene to look for expression of a similar gene in hair cells from chickens. Researchers will also want to determine, Hudspeth suggests, whether a human version of *nompC* might turn out to be mutated in any of the many forms of hereditary deafness for which genes have not yet been identified. If either of these searches is successful, then the similarity of bristles to hair cells will indeed have paid off.

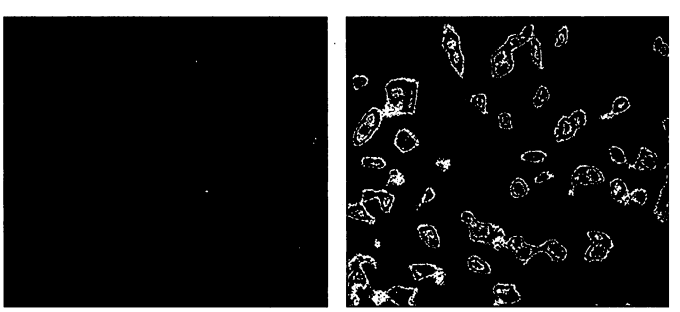
–MARCIA BARINAGA

NEUROSCIENCE

Family of Bitter Taste Receptors Found

Our ability to savor the sweetness of a fig or the sour tang of a lemon may seem more like a pleasure than a necessity, but the sense of taste is actually honed for survival. Sweetness, for example, means that a food has high caloric value, while bitterness tells us that it may be poison. For neuroscientists, however, bitter has been a perplexing flavor, because a wide range of unrelated chemicals all taste similarly bitter even though their diverse structures suggest that they must trigger different receptor molecules. The solution to that puzzle may now be at hand—along with other insights into the phenomenon of taste.

A team led by Nicholas Ryba of the National Institute of Dental and Craniofacial Research and Charles Zuker of the University of California, San Diego, reports in the current issue of *Cell* that it has identified a



Bitter match. Cultured cells containing a bitter receptor fluoresce in response to cycloheximide *(right)*, but not to three other bitter-tasting chemicals *(left)*.