technology for the future," says Kinney. Agency managers are pondering a 2004 initiative to come up with better and lighter mirrors, detectors, coatings, and other technologies. So far, such long-term vision has suffered from near-term budget troubles. A NASA proposal to spend some $5 million in 2003 on solar sails and lightweight optics was shot down by White House budget officials to cover cost overruns in other programs. But NASA officials say they are confident that O'Keefe will prove amenable to spending money on long-term technologies for space telescopes.

Telescopes of every wavelength need bigger and lighter optics. But the details differ. Ultraviolet instruments, for example, require greater precision in their optics because of the shorter wavelengths; mirror contamination, which absorbs those shorter wavelengths, also poses a major threat. Infrared astronomy demands low temperatures, to reduce the amount of contaminating heat and light. And x-ray astronomy requires higher precision and multiple mirrors; the current use of cone-shaped mirrors is limited because increasing their size does not rapidly increase light collection. Harley Thronson, NASA's chief astronomy technologist, says that better detectors are essential for all wavelengths, pegging total research and development costs at $30 million to $40 million a year.

Meanwhile, NASA managers say they want to keep close tabs on advances in ground-based astronomy. The 2004 initiative, adds Thronson, might include collaboration with the National Science Foundation, which oversees ground-based telescopes.

Such cooperation would no doubt please the White House, which pushed unsuccessfully last year to combine the two agencies' largely separate efforts under one roof. Coordinating technologies could be a first step toward determining which missions are best met by space- or ground-based instruments. "Hubble has been king of the hill for so long, it's hard to think of a different direction," says Angel. With Hubble's demise in sight, however, the time may be ripe for a new approach.

–ANDREW LAWLER

NEUROSCIENCE

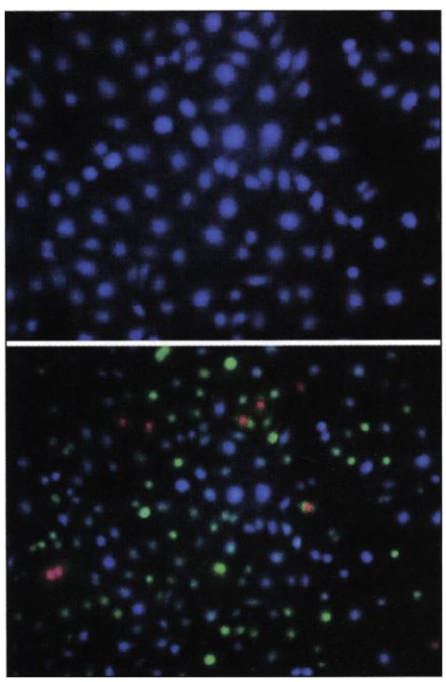
How Neurons Know That It's C-c-c-c-cold Outside

A newfound mint receptor and a cold-sensitive balance of ion movements can each send the chilling message

Mouthwash and chewing gum makers advertise that their minty products feel and taste cool. It's not just a metaphor. To a cold-sensitive neuron, menthol, the active ingredient in mint, might just as well be ice. Until recently, however, researchers didn't have a good grasp of how nerve cells transmit cold sensations. Now a spate of papers published this month offers two answers. Sometimes specialized menthol sensors do the job; other times neurons choreograph the movement of ions in response to cold in the absence of a specific receptor. Researchers suspect that the two mechanisms operate in concert, either in separate populations of cells or together in the same neurons.

Fifty years ago, researchers discovered that some nerve endings react both to cold temperatures and menthol. That meant that menthol could be used to identify cold-sensitive neurons, says neuropharmacologist David Julius of the University of California, San Francisco. These neurons should be mirror images of neurons that are sensitive both to hot temperatures and capsaicin, the chemical that gives chili peppers their sting. In 1997 Julius identified a receptor that registers both types of heat sensations, and since then he's turned his attention from the oven to the freezer. "Is there a bona fide menthol receptor?" he wondered. If so, "what does it look like, and is it [also] a cold receptor?"

Last year work by Gordon Reid's group at the University of Bucharest, Romania, pointed to the existence of such a receptor. The team observed a flow of ions into certain neurons when stimulated by cold and found that exposure to menthol increased that flow—exactly the behavior one would expect if a cold- and menthol-sensitive receptor were at work. That receptor would likely be a channel in the cell membrane that allows ions to flow into the cell and activate the neuron when it gets cold. (Because they looked at only the electrical characteristics of the current, the researchers did not identify any specific neural receptors.) They were a bit perplexed, however, as the new findings seemed to contradict the team's earlier work suggesting that menthol stimulated neurons not by allowing ions to flow into the cell but by preventing ions from leaving the cell.



Cool customers. Neurons expressing the newfound receptor relax at room temperature (top) but fill with calcium ions at 15°C (bottom). Color changes from blue to green to red as the ion concentration inside the cell increases.

CREDIT: PEIER *ET AL.*, *CELL*, PUBLISHED ONLINE 11 FEBRUARY 2002

The new observations, by three independent research groups, support both mechanisms. Two teams cloned proteins that appear to be the long-sought cold receptor. Under cold temperatures, the proteins shuttle positive ions into the cells. The third team reports that cooling blocks a protein channel through which positive ions leak back out of the cell, trapping extra charge inside the neuron. "They're both going on," Reid says of the two mechanisms, but "the relative importance of the two is still open."

In search of a channel responsive to cold, Julius and his colleagues isolated menthol-sensitive neurons from the faces of rats. Even in the face, which is particularly sensitive to cold, only about 10% to 15% of neurons responded to the cooling compound. After verifying that cold temperatures also stimulated the cells, the researchers inserted various genes expressed by those cells into other cells that don't normally register cold or menthol. One gene opened up a new world of sensations for the cells, making them respond to low temperatures as well as the minty chemical, the group reports online 11 February in *Nature*. The gene encodes a receptor that they named, straightforwardly enough, CMR1, for cold and menthol receptor. The team found that the receptor is present primarily in small-diameter neurons, which typically play a role in sensing pain.

A second group, led by Ardem Patapoutian of the Scripps Research Institute in La Jolla, California, and Stuart Bevan of

the Novartis Institute for Medical Sciences in London, approached the problem from the opposite direction. They searched DNA databases for sequences similar to the recently identified heat and capsaicin receptor and pulled out a mouse gene they called *TRPM8*. When transferred into cultured cells, the gene conferred cold and menthol sensitivity, the group reports in the 11 February online edition of *Cell*.

Although the two groups didn't realize it until a few days before their papers were published, TRPM8 and CMR1 are the same receptor. Neither group yet knows exactly how cold affects the receptor, but the "most straightforward" explanation, Patapoutian says, is that "colder temperatures change the conformation of the channel," allowing positive ions to flood into the cell. A similar adjustment could occur when minty-cool menthol binds to the receptor.

But "by no means can this [receptor] by itself explain perception of cold," Patapoutian says. "Other mechanisms and channels will be involved, given that humans can detect differences as little as 1°C."

Indeed, neurons with designated cold receptors aren't the only ones to feel a chill. Félix Viana and colleagues at Miguel Hernández University in San Juan de Alicante, Spain, propose that cold sensitivity results from an interaction among channels that hold potassium ions inside the cell and those that let them out—a process that has no need of a specific cold receptor. The team isolated a population of cold-sensitive neurons from the faces of mice. These neurons, when exposed to cold or menthol, close a so-called "leak channel" that normally allows positive potassium ions to trickle out of the cell, the researchers found. Closing the channel holds positive ions inside the cell, exciting the neuron, they report online on 11 February in *Nature Neuroscience*. These observations confirm similar findings in nonfacial neurons reported by Reid's group in January 2001.

Viana's group found another difference between cold-sensitive and cold-insensitive sensory cells that are otherwise quite similar. The latter seem to have a "braking" mechanism that is absent from cold-sensitive cells. This brake, called a voltage-gated channel, slows neural excitation by shuttling potassium ions out of the neuron, despite the closure of the leak channel. Cells' sensitivity to cold, therefore, rests not on the presence or absence of one particular channel but rather on "the unique combination of channels [the cells] express," Viana says.

Neurons aren't necessarily committed to a life of sensitivity or obliviousness to cold. Viana's team found that blocking the voltage-gated potassium channel in neurons normally impervious to cold made about 60% of those cells cold-sensitive. This mechanism could explain how nerve injury sometimes causes painful sensitivity to cold, the researchers suggest. Previous reports indicate that nerve injury disrupts certain voltage-gated potassium channels. If a neuron contains too few of these channels, the researchers suggest, it could become hypersensitive to cold.

"It looks like you have a population [of cells] that can become responsive [to cold] in the absence of CMR1," says neuroscientist Michael Gold of the University of Maryland, Baltimore. "My gut feeling is that in the end, it's going to turn out to be probably a combination of both," with specialized receptors working in concert with a balance of ion channels to let people know that, baby, it's cold outside.

—CAROLINE SEYDEL

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ASTRONOMY

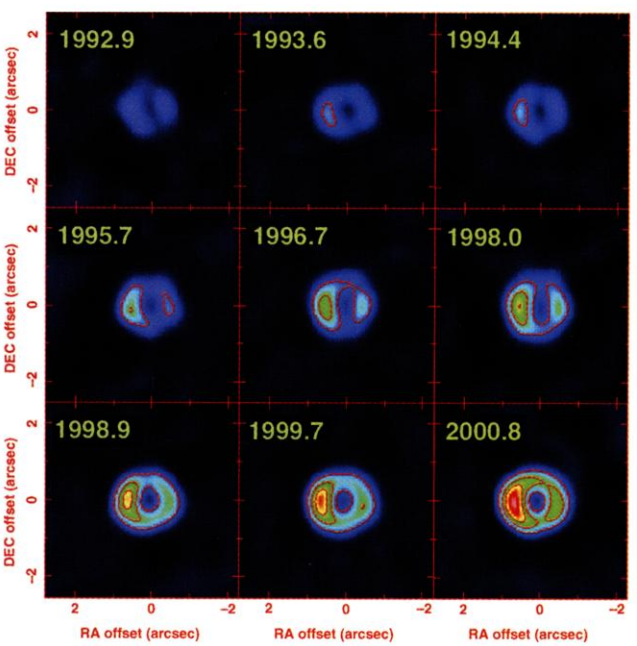
The Tumultuous Teens of Supernova 1987A

With the explosion long faded, the most spectacular star in centuries is treating astronomers to a rip-roaring new light show

When Supernova 1987A burst onto the cover of *Time* 15 years ago this week, astrophysicist Stan Woosley of the University of California, Santa Cruz, told the magazine: "It's like Christmas. We've been waiting for this for 383 years." Today, the supernova is the gift that keeps on giving. Although the explosion has dimmed, space around it now rages with energy. This maelstrom promises the closest look yet at how nature churns fresh elements into the cosmos.

The rebirth started a few years ago, when the supernova's blast wave hit part of a ragged ring of gas shed by the bloated star before it collapsed and self-destructed. Recently, that fierce impact has spread from a single "hot spot" to a dozen, and more are coming. "It's all going to become one solid hot spot," says astrophysicist Richard McCray of the University of Colorado, Boulder. "It will become 100 to 1000 times brighter in the next decade."

That's not bright enough to make 1987A visible to the unaided eye. However, the fireworks should reach beyond the inner ring to light up a puzzling cloud of gas and dust, also cast off by the star long ago. The shape and motions of this cocoon may reveal whether the doomed star had a companion. The shock wave itself will start to echo throughout the supernova's debris, reflecting like a sonic boom in a canyon. That chaos will disperse the star's pristine elements far into space: carbon, oxygen, iron, and heavier substances flash-forged by the explosion.



Tuning up. Radio waves from Supernova 1987A keep getting brighter as a shock wave boosts electrons to near the speed of light.

CREDIT: RICHARD MANCHESTER *ET AL*./CSIRO AUSTRALIA TELESCOPE NATIONAL FACILITY

It's all part of 1987A's transformation into a supernova remnant like the famed Crab Nebula, whose explosive birth Chinese astronomers noted 948 years ago. This time, astronomers have a ringside seat with modern telescopes at their disposal. "We've never had a good record of how a supernova feeds heavy elements into the interstellar medium to make future stars and planets," says astronomer Arlin Crotts of Columbia University in New York