loss through a crystal lattice.

Yet the high-temperature superconductors, made of copper oxides, behave nothing like conventional ones. Although the temperature has to be lowered to turn them into superconductors, their properties seem to depend strongly on impurities in the copper oxide crystal. "How did it occur to Bednorz and Müller [discoverers of HTSC] to look for a superconductor in the most terrible conductor, a ceramic insulator?" marvels Shou-Cheng Zhang, a theorist at Stanford University. "By doping atoms into copper oxide, they found that somehow this insulator becomes a perfect conductor." The fact that the change depends on something besides temperature, namely the doping of other elements into the crystal lattice, suggests a quantum phase transition.

Last year, Zhang came up with a possible scenario for this phase transition by applying mathematical tools borrowed from high-energy physics. The insulating state of the high-temperature superconductors is an antiferromagnet, a delicate balance of alternating spins, like an array of tiny bar magnets pointing alternately north and south. Zhang and his colleagues found that they could mathematically "unify" antiferromagnetism and superconductivity, much as particle physicists have learned to unify the electromagnetic force with the nuclear weak force. This unification allowed Zhang to describe the transition to superconductivity in a high-temperature material as a process radically different from the one in a conventional superconductor, where the resistance drops to zero precisely when the electrons form pairs.

"The pairing can actually occur at high temperatures, well above those at which the material is superconducting, but the fate of these pairs is governed by lower temperature effects," Zhang says. As the temperature is lowered, the pairs can form an antiferromagnetic quantum solid or melt into an electron-pair superconductor. At zero temperature, Zhang's model suggests, these two ground states might emerge as two different quantum phases, with composition alone governing the transition between them.

So far, it's only a theoretical picture, but experimenters are beginning to find hints of quantum phase transitions in high-temperature superconductors. Mason and his co-workers at Oak Ridge, for example, probed single crystals of $LaSrCuO_4$ with neutrons to measure magnetic fluctuations in the material when it had been doped almost enough to make it into a superconductor (*Science*, 21 November 1997, p. 1432). The neutrons are like a beam of tiny bar magnets bouncing off the spins in the sample; sorting out the scattered neutrons tells how the spins are fluctuating. Although the experimenters could not reach absolute zero—the place where quantum phase transitions come into their own—they found that the spin fluctuations varied with temperature in a way that would be expected if a quantum phase transition were lurking somewhere nearby.

Not only could quantum phase transitions lift the lid on HTSC, but Mason and others think that studying these transitions may equip physicists to solve equally tough puzzles in the future. The research, says Mason, is part of physics's quest for universality—for ways to explain the behavior of complex, many-body systems in terms not of microscopic details but of large-scale properties. "Because of universality, the things we are exploring now can be applied to cases we don't know about yet," he says. "It's a remarkable thing that you can experiment on a simple system, then discover there's a whole other set of transitions that follow the same behavior." **–DAVID VOSS**

David Voss, a former editor of *Science*, is a physics writer in Silver Spring, Maryland.

ADDITIONAL READING

S. L. Sondhi, S. M. Girvin, J. P. Carini, D. Shahar, "Continuous quantum phase transitions," *Reviews of Modern Physics* **69**, 315 (1997).

MEETING SYMPOSIUM ON THE BRAIN

New Clues to Movement Control and Vision

Last month, neuroscientists gathered at Boston University School of Medicine for a conference on the brain, held to commemorate the late computational neurobiologist David Marr, who pioneered theories about brain regions as varied as the cerebellum, the visual system, and the cerebral cortex. The meeting's topics were similarly diverse, ranging from the workings of the retina to computer models of the cerebellum.

Guiding a Robot's Movements

When a tennis ball flies over the net, U.S. Open champion Lindsay Davenport has to predict where it will go so she can race to hit it. If she fails to guess the right spot, she'll lose the point. It's crucial to be a split-second ahead of the game, and computational neurobiologist Terrence Sejnowski of the Salk Institute in La Jolla, California, aided by a robot his team developed, has new evidence to support the idea that this skill is learned with the help of a brain area called the cerebellum.

As the robot turns. Adding a cerebellar circuit helped this robot follow a moving light.

CREDIT: ALAIN HERZOG

At the meeting, Sejnowski showed that a software model of the human cerebellum gave the 2-centimeter-wide cylindrical robot an ability it didn't have before: It could predict, a second in advance, the position of a moving light based on the light's pattern of movement. The result suggests that the cerebellum learns how to anticipate where a fast-moving object will go. Other work had already suggested that the cerebellum—known for its role in stabilizing the body, moving the eyes, and performing multijoint movements—might have a role in learning to make very short-term predictions. The details of Sejnowski's model have not yet been published, but if the software really is a good model of the cerebellum, says computational neuroscientist Tomaso Poggio of the Massachusetts Institute of Technology, the new work could be "an important and necessary step" toward nailing down this function for the cerebellum.

Sejnowski's group originally set out to use the robot as a real-world test of a model they had made of a brain circuit that governs motivation and reward-seeking. This circuit, shared by both humans and bees, is thought to assess the chances of rewards that may appear in the next few seconds and direct behavior to maximize a reward. It had accurately simulated, on a computer screen, the behavior of bees given a

choice of stationary blue or yellow flowers that offer different amounts of nectar (the reward). The computer bees learned to move to the colored flowers that had the highest probability of containing nectar.

For the robot, the reward was not nectar but the light emitted from a horizontal array of diodes that flashed in sequence from left to right and then back again. The reward circuit caused the tiny robot to roll toward the array of diodes, because this makes the light appear brighter. But because the light moved from left to right, changing position every second, the robot would lose track of it. The robot seemed unable to predict where the light would go next.

Sejnowski and his graduate student, Olivier Coenen, wondered whether the robot might do better if it had a software version of the cerebellum. This brain structure was already known to have a role in predicting movement. Some of this work involved the so-called vestibular-ocular reflex, in which the eyes move to compensate for a head movement so that the image on the eyes remains still. Studies in monkeys suggested that the cerebellum delivers signals that predict how much the eyes must move in response to a future head movement. That way the brain can plan the eye movement before the head turns.

Sejnowski suspected that this predictive role might extend to tuning other types of motion to sensory cues as well. So, he enlisted the aid of engineers Marwan Jabri and Jerry Huang at the University of Sydney in Australia to help him and Coenen program the robot with a model of cerebellar learning based on recent physiological data. In this model, the cerebellum cooperates with a brain structure called the inferior olive, which compares sensory data indicating what happened with input from the cerebellum, which predicted what should happen. If there is a difference between the two, the olive transmits that to the cerebellum, changing connections between simulated neurons in a way that is thought to mimic learning. In this case, the learning is supposed to reduce the difference between prediction and reality to improve future predictions and thus performance.

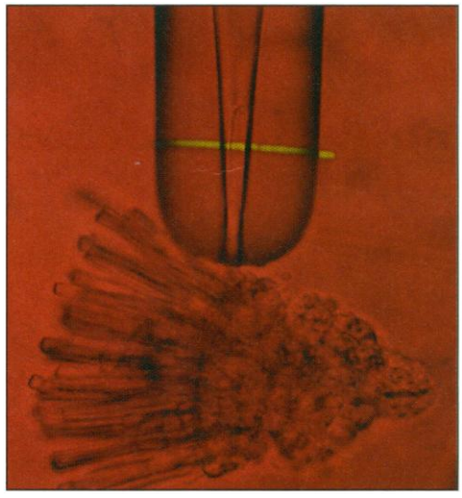
Equipped with software based on this model, the robot's performance did improve: It learned to predict the light's movement well enough to follow, and sometimes lead, the light by turning its body. Sejnowski believes an animal's ability to track a moving target is similarly "improved" by the cerebellum. "The model has taught us that each piece of brain can be greatly helped by other parts of the brain," Sejnowski says. "These parts fit together in a temporal progression," with the cerebellum acting within a second and the reward circuit acting within several seconds, followed by longer term predictors like the cerebral cortex.

Sejnowski says his group now plans to test whether the model will enable the robot to perform other animal-like behaviors thought to depend on the cerebellum. He also hopes the robot will "evolve" to perform even more complicated tasks as additional brain structures are added to it. "We might be able to reproduce simple creatures, then more complex creatures, and ultimately, ourselves," he says.

CREDIT: DENIS BAYLOR/STANFORD UNIVERSITY SCHOOL OF MEDICINE

Seeing in the Dark

Although humans are not nocturnal creatures, we can see well enough at night to find our way to the bathroom or walk along a beach guided only by starlight. We owe this ability to our rods, specialized cells in the retina of the eye that can respond to as little as a single photon of light, triggering neural impulses that will eventually relay an image to our brains. Scientists have largely worked out the chemical reactions that enable rods to make these responses. They know much less, however, about an equally important step: how the rods turn off that response when the light that elicited it is gone, thereby keeping the eyes ever-ready to perceive changes in light patterns at night.



Probing vision. The micrograph shows an electrode recording the activity of a single rod in a cluster.

Now, neurobiologist Denis Baylor of Stanford University School of Medicine and his colleagues have pinned down a key reaction in resetting the rods. At the meeting, Baylor described new results with knockout mice showing that an enzyme called rhodopsin kinase, which adds phosphate groups to the rods' light-sensing protein rhodopsin, initiates the chain of events that returns rhodopsin to its off state. The work, which neuroscientist Edward Pugh of the University of Pennsylvania, Philadelphia, calls "really elegant," could also help researchers understand—and perhaps design better treatments for—diseases in which rods become dysfunctional. In such ailments, which include retinitis pigmentosa, people's night vision may be so impaired they cannot go out at night without someone to guide them.

The first inklings of how rhodopsin is shut down after it absorbs a photon and triggers a neural signal came in the late 1980s and early 1990s. Working with suspensions of rod proteins, biochemists found that rhodopsin inactivation requires two types of proteins: a kinase, which attaches a phosphate group to one of several amino acids clustered at one end of the molecule, and arrestin, a protein which then sticks to phosphorylated rhodopsin. As a result, rhodopsin can no longer bind to a so-called G protein, which transmits the light-initiated signal through the cell.

Roughly the same thing happens in intact rods, as Baylor's group, in collaboration with Melvin Simon, Jeannie Chen, and their colleagues at the California Institute of Technology (Caltech) in Pasadena, showed in 1995. When the researchers genetically altered mice so that their rhodopsin lacked the postulated phosphorylation sites, their rods recovered very slowly from light, taking about 20 times as long to return to the resting state as did rods in normal mice. And last year, the team also confirmed a role for arrestin by showing that rods can't reset all the way in mice that lack a working gene for that protein. It still wasn't clear, however, which enzyme performs the crucial phosphorylation. In test tube studies, the reaction could be catalyzed equally well by protein kinase C, a kinase that is commonly involved in cells' signaling paths, and by an enzyme specific to rods named rhodopsin kinase.

To find an answer, Jason Chen in the Caltech group genetically engineered mice to inactivate the rhodopsin kinase gene. Rods from these animals, Stanford postdoc Marie Burns found, recovered as slowly after light exposure as did those of mice without the rhodopsin phosphorylation sites. This shows, Baylor says, that "rhodopsin kinase is the enzyme that normally turns off rhodopsin," and that protein kinase C plays a minor role, if any.

Now Baylor's team is probing the mystery of how the activity of a single rhodopsin molecule generates a cellular response that is exactly the same every time a photon is absorbed, a property necessary for us to make sense of what we see. His group is also studying how other proteins that mediate a rod's response to light are reset. Solving such mysteries will shed more light on our still-hazy view of how we see in the dark.

–INGRID WICKELGREN