cell can do better than a hexagon if appropriately penalized for having more than six sides or outward curves," says John Sullivan of the University of Illinois, Urbana-Champaign. Although other mathematicians, including Weaire, had discovered a penalty for the number of sides, Hales is the first to find the right penalty for the curvature of the sides and to combine both penalty terms.

Other geometers seem quite pleased with the proof. Unlike Hales's proof of the Kepler conjecture, which involved thousands of elaborate computer calculations, the proof of the honeycomb conjecture does not require a computer at all. "The overall idea just seems right," Sullivan says. "There should be an easy reason for a pattern this simple, and I think Hales has found it." **–DANA MACKENZIE**

Dana Mackenzie is a writer in Santa Cruz, California.

DEVELOPMENTAL BIOLOGY

Selenium's Role in Infertility Explained

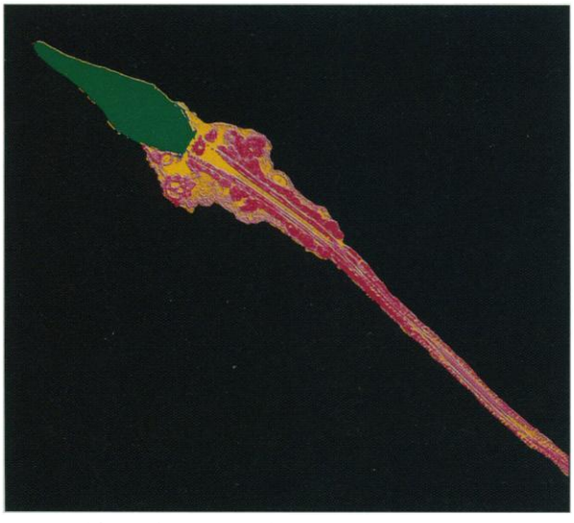
Many proteins lead multiple lives, depending on environmental conditions or on the presence of particular partners. It's rare, however, for a protein to change its stripes completely, acting as a soluble enzyme under some circumstances and an insoluble structural component under others. But that's what new research suggests for a particular selenium-containing protein of sperm—a discovery that may help explain the long-standing mystery of why selenium deficiency in lab and domestic animals leads to male sterility.

On page 1393, biochemists Leopold Flohé of the Technical University of Braunschweig in Germany, Fulvio Ursini of the University of Padova in Italy, and their colleagues report that the protein, previously identified as an enzyme that helps rid developing sperm of dangerous reactive oxygen molecules, moonlights as part of the glue that holds together mature sperm. "This is a new function for a selenoprotein—to form a structure, not just to carry out a reaction," says Raymond Burk, a selenium expert at Vanderbilt University in Nashville, Tennessee.

Although selenium deficiency is rarely a problem for humans, who get the element from common foods such as seafood, liver, lean red meat, and grains grown in soil that is rich in selenium, scientists showed decades ago that animals fed selenium-deficient diets produce sperm that break in the middle and can therefore no longer fertilize eggs. Beyond demonstrating that selenium is concentrated in the midpiece, the region between the head and tail, of normal sperm, scientists made little headway in explaining this effect. Several years ago, they thought they had an answer: The selenium deficiency might be interfering with another protein they had identified in the mitochondrial capsule, a structure that holds the energy-producing mitochondria in the sperm midpiece.

But that idea dropped out when sequence analysis of the corresponding gene revealed that in some perfectly normal animals it doesn't encode the amino acid that carries selenium—evidence that the element isn't required for the protein's function. "There's been a question of whether there is such a thing as a real structural selenoprotein in sperm," says Thressa Stadtman, a selenium biochemist at the National Heart, Lung, and Blood Institute in Bethesda, Maryland.

Flohé and his colleagues have now shown that there likely is, by studying a known selenoprotein called phospholipid hydroperoxide glutathione peroxidase (PHGPx). The enzyme, which likely protects the developing sperm cell against damage by converting toxic peroxides to harmless alcohols, climbs to extremely high levels in testes. Because the levels are much higher than would be expected for protection against the amounts of peroxides probably present in that tissue, Flohé describes the situation as "kind of strange." About 2 years ago, however, his team's work began pointing to a structural role for the protein. Their analysis of the mitochondrial capsule showed that PHGPx is its most abundant component, accounting for about 50% of the capsule material.



CREDIT: (LEFT TO RIGHT) TONY BRAIN/PHOTO RESEARCHERS; CHUN *ET AL.*, *NATURE NEUROSCIENCE* 2, 844 (1999)

Sperm aid. A selenoprotein known as PHGPx may help keep normal sperm, such as this one, from breaking apart.

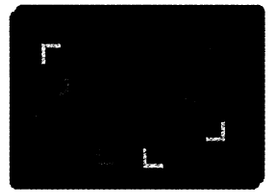
But even though it constitutes such a large proportion of the capsule, tests revealed that the protein from mature sperm had lost its enzymatic activity, apparently because the protein molecules had become linked together in an inactive form. Based on these findings, the researchers propose that PHGPx acts as a soluble enzyme early in sperm development and later polymerizes into a protein mesh that contributes to the structural integrity of the midpiece. If so, says Burk, "this [work] may explain the head-to-tail separation seen in sperm of selenium-deficient animals."

In addition, the result opens the door to a better understanding of the mechanisms underlying normal sperm development, presumably in humans as well as in animals, say experts. Currently, Stadtman notes, the triggers for the switch from active enzyme to inactive structural protein are not known. "The next step is to work out the signals that tell the sperm to undergo this developmental change," she says. Indeed, as anyone who has juggled identities knows, timing is one key to success. **–EVELYN STRAUSS**

NEUROBIOLOGY

New Role Found for The Hippocampus

When you remember a friend, your first day of work, or your address, you're fully aware of what you're remembering. But memory has another guise: nonconscious skills like riding a bicycle or knowing how to tie your shoes. Subjectively, the two kinds of memory seem very different, and the brain structures responsible for them are different as well. Results reported in this month's issue of *Nature Neuroscience* suggest, however, that the hippocampus, a twist of tissue deep in the brain long believed to help form only conscious memories, also serves certain memories that don't rise to the level of awareness.



T is for target. Without a healthy hippocampus, subjects couldn't learn from seeing the same complex pattern twice.

The report comes from psychologists Marvin Chun of Vanderbilt University in Nashville, Tennessee, and Elizabeth Phelps of New York University. Chun and Phelps compared how normal people and those with anterograde amnesia, a memory defect caused by damage to the hippocampus, respond to certain complex patterns. They found that the normal subjects, but not the amnesiacs, could learn to remember repeated patterns they weren't consciously aware of. "What they've con-