Together again. Wads of clay show where both femurs were broken.

Jelderks in Portland, Oregon, will toss the Kennewick find back into their domain.

Kennewick Man's bones are well traveled. Shortly after they were found, they were seized by the U.S. Army Corps of Engineers in response to concerns from Native Americans. Stashed in the county coroner's office, then sent to Battelle Pacific Northwest National Laboratory in Richland, Washington, the bones are now stowed at Burke Museum in Seattle.

An inventory taken at Battelle in 1997 revealed that most of Kennewick's thighbones—two pieces from each femur—were absent. Now they have apparently reappeared as mysteriously as they vanished. Richland anthropologist James Chatters, who studied the bones before the government took them, says workers demolishing an old storage building used by the sheriff found them in the coroner's evidence locker—in a box labeled as containing some other Columbia River bones that had been returned to Indians for burial in 1998.

"I'm utterly baffled," says Chatters, who notes that the FBI ransacked the sheriff's locker in a search for the bones in 1998. So is Michael Trimble, chief curator for the corps, who says "I haven't a clue" how they turned up again.

FBI spokesperson Roberta Burroughs says the bones have been tentatively identified through comparison with photos. The FBI is awaiting approval from the U.S. attorney's office before returning them to the corps.

Chatters says that the femurs should yield information about racial origins, because the femoral head in American Indians is more highly rotated in relation to the shaft than it is in Europeans. But the U.S. legal system will ultimately decide whether scientists will have another go at them. **–CONSTANCE HOLDEN**

CREDITS: (TOP TO BOTTOM) JAMES CHATTERS; HANS SUPÈR

NEUROSCIENCE

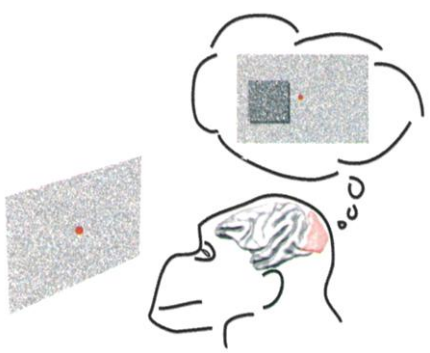
Neurons Fix Memories In the Mind's Eye

When a monkey has to remember something, it holds that thought in its mind's eye, a new study suggests. Earlier memory research showed that higher order brain regions such as the frontal lobes buzz madly when monkeys (and people) remember something briefly. But this study, which appears on page 120, is the first to show that even the lowest level, workaday region of the visual cortex also hums with anticipation while maintaining a memory. The researchers suggest that this part of the brain holds on to a simple sensation that helps guide more sophisticated parts of the memory system.

The work comes from Hans Supèr and colleagues at the University of Amsterdam in the Netherlands, who looked at the responses of neurons in the primary visual cortex (V1) while animals were engaged in a test of working memory. This type of memory holds information at the ready, temporarily, while an animal prepares to act. The prototypical example is reciting a telephone number while walking from the phone book to the phone to dial.

Neurobiologists used to think that V1 simply sorts incoming visual information before passing it on to higher brain centers for interpretation. As vision scientist Jeffrey Schall of Vanderbilt University in Nashville, Tennessee, points out, the brain area is "just one step removed from the retina." But the Supèr team showed that neurons in V1 actively store memories of an image, briefly holding them until the animal makes the appropriate response.

The result follows other studies in the past several years showing that V1 neurons aren't simple receptacles of light and shadow. For example, neurons in V1 fire more enthusiastically in response to a stimulus that tells a monkey how to get food than to an irrelevant stimulus. Now this study shows that V1 neurons don't even need a stimulus—they con-



You must remember this. Neurons in V1 (red) fire to a remembered stimulus even after it vanishes.

ScienceScope

Torn Loyalties A nasty fight is brewing between the Bush Administration and Congress over who should administer proposed math and science education partnerships involving universities, schools, and industry (*Science*, 25 May, p. 1463). Hundreds of millions of dollars are at stake, and the National Science Foundation (NSF) is caught in the crossfire.

The House Science Committee last month passed a bill that would put universities in the driver's seat by funneling federal funds to academics and nonprofits working with the schools. That time-tested approach is fine with NSF officials, sources say. But some Administration officials object, and in a 19 June letter to congressional leaders, NSF director Rita Colwell followed her bosses' wishes and argued that the program should give awards directly to state and local school districts. They are "closer to the needs of students" and more accountable for their performance, Colwell wrote. She also complained that a larger education reform bill moving through Congress goes against the Administration's plans by putting the Department of Education—and not NSF—in charge of the partnerships.

The disagreement won't be resolved until Congress finishes the education package later this year.

Money Talk A proposal to charge researchers up to $500 to post their papers on a free-access Web site is drawing mixed reviews from scientists. BioMed Central—a free online publisher—last week said that it is mulling a sliding scale for author charges. Publisher Jan Velterop says the charges will help maximize the distribution of papers and eventually reduce the amount of money that the scientific community overall spends on publishing fees and journal subscriptions.

The fee idea is backed by the Public Library of Science (PLoS), an advocacy group that has challenged journal publishers (including AAAS, publisher of *Science*) to provide free access to back issues (*Science*, 23 March, p. 2318). But in an online debate on the proposal (www.biomedcentral.com/editorial/charges.asp), some researchers argue that a fee will drive researchers to submit their best work to commercial journals that have no charges and will possibly drive up costs in the short run, as institutions pay both to publish and maintain subscriptions. If BioMed Central does impose the fees, officials say they would come no earlier than 2002.

Contributors: Jeffrey Mervis, David Malakoff

tinue to fire to an image they have to keep in working memory even after it has disappeared. As Schall says, "V1 is a lot smarter than it used to be."

To test V1's role in memory, Supèr and Amsterdam colleagues Henk Spekreijse and Victor Lamme taught monkeys to watch a computer screen filled with flickering black and white pixels and wait for directions. While the animals kept their eyes focused on a central red dot, a small, rectangular patch of pixels somewhere in the monkey's peripheral vision would occasionally jerk to one side and then quickly vanish into the background flicker. Then, when the central dot disappeared—which could happen up to 2 seconds after the patch came and went—the monkey had to move its eyes to where the patch had been. If successful, the animal earned a treat.

The team monitored V1 neurons, which are location specific, that were tuned to respond to a spot where the patch sometimes appeared. When the animals had learned that the patch (and not some other object on the screen) was tied to a reward, these neurons fired more robustly, as earlier studies have shown. The heightened firing then continued while the animal was waiting to move its eyes—presumably keeping the location in mind.

What's more, the continued firing appeared to help the animals remember correctly. The monkeys sometimes failed to move their eyes to the right spot. When the researchers compared neuronal firing patterns in the incorrect trials to patterns in the correct ones, they found that the V1 neurons' firing had dwindled to baseline levels shortly before the monkey made a mistake.

The team suggests that V1 neurons are communicating with other, higher level areas of the brain that are responsible for understanding the task and formulating a plan to respond. The V1 neurons contribute by keeping in mind the exact location that has to be remembered. So even if V1 isn't the ringleader of the memory gang, the new work shows that it plays an important role as a lookout.

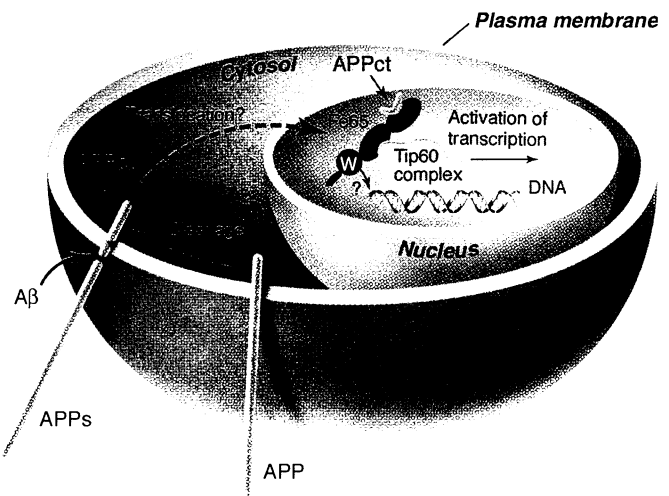
–LAURA HELMUTH

NEUROSCIENCE

Elusive Protein Auditions For Several New Roles

Few proteins are as hot as the amyloid precursor protein (APP), at least among neurobiologists. Highly expressed in brain neurons, APP is the source of amyloid-β (Aβ), the small protein whose abnormal deposition in the brain is thought to cause Alzheimer's disease. But APP's normal role has remained elusive, despite years of study. Now, on page 115, neurobiologists Xinwei Cao and Thomas Südhof of the University of Texas Southwestern Medical Center in Dallas provide an intriguing clue about one possible function.

APP appears to be located in the cell membrane, with part extending outside the cell and part—the so-called cytoplasmic tail—projecting inside. Cao and Südhof now have evidence that the cytoplasmic tail, when released from APP, can activate gene expression in the nucleus, possibly acting directly as a transcription factor, a protein that binds to regulatory regions and turns genes on and off. This is the first time anyone has linked APP to control of gene expression, and Alzheimer's researcher Sam Sisodia of the University of Chicago School of Medicine predicts that the finding "will capture the attention of many folk in the cell biology and Alzheimer's worlds." He cautions, however, that more work will be needed to confirm this "tantalizing" result.



Gene regulator? When released from APP, the protein's cytoplasmic tail (APPct) may activate gene expression with the help of Fe65 and the Tip60 complex.

This is new territory for Südhof, whose research has dealt mainly with understanding the synapse, the specialized structure through which neurons communicate with one another. But he's been thinking about the protein for a long time. "It's hard not to be interested in APP, given the importance of Alzheimer's," he notes.

A couple of findings prompted him to focus on a possible physiological function for the cytoplasmic tail. It associates with a variety of cell proteins. In addition, one of the two enzymes that cuts Aβ from the APP molecule also releases the cytoplasmic tail. Together these findings suggested to Südhof that APP might behave like another membrane protein called Notch, which plays an important role in embryonic development. When appropriately stimulated, Notch releases its cytoplasmic segment, which migrates to the nucleus, where it interacts with other proteins to turn on various genes.

To find out whether APP might play a similar role in gene transcription, Cao and Südhof introduced the gene for luciferase, an easily detectable enzyme, into various mammalian cells. Then, the researchers introduced the *APP* gene, modified so that at least theoretically its product could bind to and activate the luciferase gene. But the APP constructs alone had little effect on luciferase production, leading the researchers to conclude that APP might need help from another protein or proteins to stimulate gene transcription. They searched for such partners by looking for proteins that bind to the cytoplasmic tail of APP in yeast. They came up with a protein of unknown function called Fe65, which was already known to bind APP.

When Cao and Südhof then added the *Fe65* gene along with the *APP* genes to the luciferase gene–bearing cells, luciferase production shot up—more than 2000-fold in some cells. But when the APP tail is mutated so that it can't interact with Fe65, transcription remains low, confirming that the two are partners. The researchers also showed that the APP tail and Fe65 bind a protein, called Tip60, which is part of a large complex of proteins involved in gene transcription.

Südhof proposes that when the tail is cleaved from APP, it moves to the nucleus, where it binds Fe65 and the Tip60 complex, thus activating gene transcription. If APP is involved in such a signaling pathway, he says, "the results imply there's some degree a regulation of [APP] cleavage." That could be important for understanding Alzheimer's if, for example, that regulation goes awry and fosters Aβ production or other neuronal abnormalities. But the case is not yet airtight, says Südhof, because the results were obtained in altered cells; the researchers still need to show that the cell's own proteins act the same way.

And the APP-Fe65 partners may have other functions to boot. In the 25 June issue of the *Journal of Cell Biology*, a team led by Paul Greengard of Rockefeller University and Joseph Buxbaum of Mount Sinai School of Medicine, both in New York City, report that the two proteins foster the cell movements needed in wound healing. Fur-

ILLUSTRATION: C. CAIN