Agency (DARPA), in part, say aides, because lawmakers felt some programs were growing too fast while others duplicated work being done by other agencies. Although the exact impact of the cuts won't be known for months, "it's ironic that IT got cut just when everyone is saying that new computers and software will be so important to the future military," says Robert Parker, head of the University of Southern California's Information Sciences Institute in Arlington, Virginia. "We need to get much more proactive in making the case" for Pentagon spending on IT.

The cuts come at a time when some question DOD's stomach for the type of risky research that led to the Internet and radar-avoiding stealth aircraft. Jim Richardson, a former DARPA program manager who is now vice president of the Arlington-based Potomac Institute, believes that DOD's basic and applied research today "are focused on the nearer term than was the case 5 years ago," although he admits that the shift is hard to quantify.

Reversing that trend, he and other observers hope, will be high on the agenda of the next president. But although both Al Gore and George W. Bush have said they would significantly increase Pentagon R&D—with Bush calling for a $20 billion boost by 2006—neither has spelled out where the money would go. The details could emerge in January, when the new president submits his first budget to Congress.

–DAVID MALAKOFF

CANNIBALISM

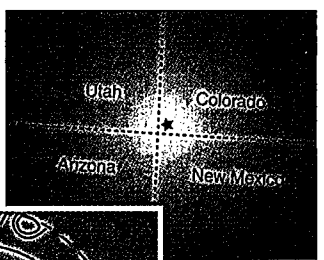
Molecule Shows Anasazi Ate Their Enemies

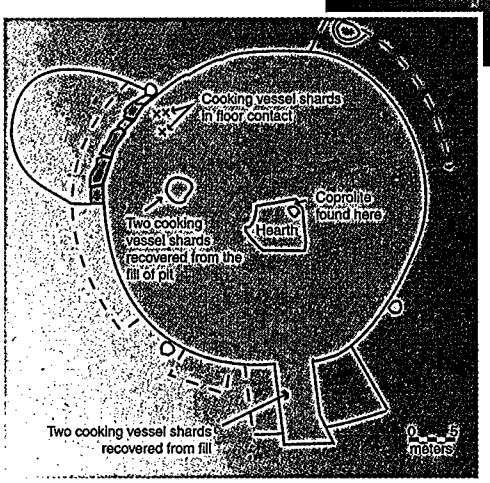
It's official: Scientists say they have definite proof that prehistoric Indians in the Southwest not only killed, butchered, and cooked, but actually ate other human beings. The evidence takes the form of a dried chunk of human excrement, or coprolite, containing a telltale human protein that could have gotten there only by being ingested.

"The bottom line," says biochemist Richard Marlar of the University of Colorado Health Sciences Center in Denver, is "yes, [cannibalism] did occur in the Anasazi population. Now it's up to the anthropologists and archaeologists to say how and why it happened."

In this week's issue of *Nature*, Marlar and colleagues claim their data refute those who would "take the archeological and osteological evidence and say there was a potential for cannibalism" but no proof that butchering and cooking led to actual consumption of human flesh. The coprolite, he believes, has forged the final link in this gruesome food chain. A handful of critics are unswayed, however, insisting that alternative interpretations of the evidence have not been fully explored.

The 850-year-old coprolite was found near Cowboy Wash in southwestern Colorado. The Four Corners area (above) contains a number of sites offering strong evidence of cannibalism: human bones disarticulated, cut, burned, and cast about in exactly the same fashion as the bones of animals known to have been used for food. Investigating this small settlement of three half-buried "pit houses," scientists found two that contained mutilated remains of seven men, women, and adolescents—apparently massacre victims whose bodies were butchered. The coprolite was found in a cold fire pit in the third pit house.





Hard evidence. Analysis of human coprolite found in the hearth of this pit house in southwestern Colorado solidifies the argument for cannibalism.

The scientists looked for traces of myoglobin, an oxygen-transporting molecule that occurs in skeletal and heart muscles but not in the gut. They developed an assay that distinguishes between human myoglobin and that of nine food animals. The assay found human myoglobin in the coprolite, but no traces of it in any of 25 control human fecal samples. It also identified beef myoglobin in feces from people who had recently eaten cooked beef. The authors report finding more human myoglobin on shards of a cooking pot, as well as human blood on two cutting tools.

"It appears that in two of the pit structures, people cooked and processed human remains and then left the bones on the floors," says the lead archaeologist at the site, Brian Billman of the University of North Carolina, Chapel Hill. "In the third pit structure, they appear to have cooked human remains in a pot and then, after they were done [eating], they defecated into the hearth and smashed the cooking pot."

Not everyone agrees, however. For example, Debra Martin of Hampshire College in Amherst, Massachusetts, says the fecal chunk could have been contaminated with human proteins during handling by scientists. And Peter Bullock of the Museum of New Mexico in Santa Fe questions whether the coprolite is even human. "It's most likely from a coyote," a common scavenger in these parts, he says. But Billman says the coprolite lacks the bone chunks, hair from grooming, and fur that are almost always found in canine coprolites, and there were no canine tooth marks on the human remains.

Those who worked on the site acknowledge that their findings pack a cultural wallop. "Unfortunately, it's a very emotional debate," Billman says. But he doesn't agree that cannibalism "dehumanizes" early native Americans: "This is, unfortunately, what human beings do."

–CONSTANCE HOLDEN

SPONGIFORM DISEASE

Experts Downplay New vCJD Fears

The thought of coming down with *Salmonella* after eating a tainted chicken sandwich may be alarming—but "mad cow disease"? That kind of chilling scenario was splashed across the mainstream British press last week based on a report from a leading U.K. lab that prions—abnormal proteins linked to bovine spongiform encephalopathy (BSE) and its human form, variant Creutzfeldt-Jakob disease (vCJD)—can jump from one species to another more easily than previously believed.

CREDIT: (DIG SITE) SOIL SYSTEMS INC./MARLAR *ET AL.*, *NATURE* 407, 6800 (7 SEPTEMBER 2000)

Although some experts say the findings raise concern about a threat to human health, others decry the media frenzy and contend that the new research adds little to what is known about the mysterious, fatal diseases.

What's beyond dispute is that scientists have only a vague idea about how vCJD victims become infected and when the brain begins to deteriorate. That is itself alarming given that the incidence of vCJD is rising 23% per year in the United Kingdom and may yet accelerate into a devastating epidemic (*Science*, 1 September, p. 1452). So neurologist John Collinge and his colleagues at St. Mary's Hospital in London decided to take a new look at the so-called "species barrier" that appears to block or slow transmission of prions between most species. By convention, the species barrier is determined by the time between experimental inoculation of an animal with prion-infected brain extracts (from an infected individual from another species) and the onset of symptoms, such as loss of balance.


Scientists find new BSE links

James Meikle

Scientists are to re-examine controls against variant CJD, the deadly human form of BSE, in the wake of evidence that both diseases may be highly infectious even when their unwitting carriers do not display outward signs of suffering from the condition.

They will also consider whether a new system of checks on sheep, pigs and chickens is needed to ensure that existing safeguard methods have not created other potentially fatal routes for BSE to enter humans.

New research has raised fears that animals thought to be healthy and incapable of acquiring BSE can in theory pass the disease on. The results have also renewed concern that humans not displaying vCJD symptoms but incubating the condition may infect others undergoing medical or dental procedures.

Experiments on mice and hamsters have suggested that the barrier to infection between species most feared than had been supposed and spread within species even when obvious symptoms are not yet evident.

Although scientists in the field of BSE research have suspected this, Professor John Collinge, who headed the research funded by the medical research council, said its results could have important public health implications.

The latest developments come amid signs that the incidence of vCJD is growing at between 20% and 30% a year in Britain. 70 people have died of the disease, all of them suffering the always fatal condition. There have been two deaths in France and one in Ireland. All are thought to have been infected years ago through exposure to cheap meat from parts of cattle containing high concentrations of the agent responsible.

Since 1989, the government has introduced a series of controls, including a ban on certain offal entering food, a ban on all cattle over 30 months old being used for food, and measures to stop the use of mechanically recovered meat in products for human consumption.

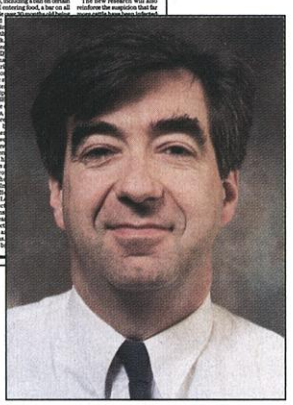
WHO THE HARVEST FROM COWS ARE DRAWING CLOSE AT ALL

injected into other mice and hamsters, it eventually killed them. The suspicion is that the process created a new strain, or strains, of more virulent disease.

The findings, published last night in the Proceedings of the National Academy of Sciences, will cause concern that contaminated material from people not displaying vCJD symptoms but incubating the condition may infect others undergoing medical or dental procedures, or the recycling of contaminated instruments.

Present sterilisation and cleaning techniques cannot remove all traces of the vCJD agent, although experts say they can substantially reduce levels of infectiousness.

The new research will also reinforce the suspicion that many cattle have been infected



Sounding the alarm. British newspapers pounced on a report from a team led by John Collinge (*inset*) suggesting that infectious prions may be lurking in asymptomatic livestock or even in people.

The best studied species barrier is that between hamsters and mice. Previous research has found that mice show no signs of disease over their lifetimes, about 2 years, after being infected with a hamster prion strain known as Sc237. Thus mice are considered highly resistant to this strain. Collinge's group replicated these experiments and confirmed that mice infected with Sc237 prions do not get sick. But the researchers went a step further, examining the brains of infected mice as they age.

They discovered that good health was only skin deep. Brain slices revealed that some mice had neurological damage typical of that seen in some human and animal prion diseases, including clumps of prion protein. Moreover, using antibodies to distinguish similar proteins from one another, the researchers found that the brain tissue was riddled with mouse prions—not the hamster Sc237. Writing in the 29 August issue of the *Proceedings of the National Academy of Sciences*, the authors conclude that the hamster prions had converted healthy mouse prion protein into the aberrant form. Indeed, the researchers were able to make other healthy mice ill by injecting them with brain extracts from the asymptomatic mice, demonstrating that the newly created mouse prions are infectious.

The study "undermines what we have thought about species barriers," Collinge told *Science*. His group speculates that chickens, pigs, or other livestock fed BSE-infected animal feed may be silent carriers of the disease. That grim prospect set off a frenzy in the British press. Newspapers suggested not only that animals deemed healthy and incapable of acquiring BSE could transmit the disease, but also that people not displaying vCJD symptoms might infect others.

Some researchers have taken Collinge's group to task for extrapolating from rodents to livestock. The findings "do not say anything directly about whether pigs, poultry, or sheep have become silently infected," says biologist Moira Bruce of the Institute for Animal Health in Edinburgh, Scotland. She argues that the findings speak only to mice and add little to the prion debate. "All that has happened is to change the story from mice are resistant to mice are susceptible but with very long incubation periods."

Similar reservations are expressed by Oxford University mathematical biologist Angela McLean. Her investigations of the potential danger of BSE infection in sheep have come up reassuringly empty so far. Scientists, McLean says, "were already worried about preclinical infections" in livestock long before the Collinge study.

Others, however, say that the findings might have public health implications. The paper "does suggest that species in which we've never seen disease might be carriers," says epidemiologist Peter Smith of the London School of Hygiene and Tropical Medicine. Smith, who is acting director of the U.K.'s Spongiform Encephalopathy Advisory Committee, says the panel will discuss the study at its next meeting later this month.

But whereas Collinge's group argues that their data should prompt a program of screening apparently healthy cattle for "subclinical" levels of BSE, Smith doubts the findings will persuade his panel to call for changes in anti-BSE control measures now in place. These controls, which ban the use

ScienceScope

Biological Riches Conservationists are shaking the tropical copaiba tree, hoping it will rain cash. Their goal, set last month at a California conference, is $5 billion to save the richest—and most threatened—areas of biodiversity.

The weeklong summit, held at the California Institute of Technology in Pasadena and organized by Conservation International (CI) of Washington, D.C., produced a draft "practical agenda" for saving 25 "hotspots" that cover 1.4% of Earth's surface. They range from the deserts of Morocco to the cloud forests of the Andes and are believed to hold 44% of the world's vascular plants (such as South America's copaiba, above) and 35% of all mammals, amphibians, birds, and reptiles.

CI, the World Bank, and the Global Environment Facility have already pledged $75 million over the next 5 years to create, expand, or improve protected areas, beginning in three of the "hottest" spots: Madagascar, West Africa's Ghanaian forest, and the tropical Andes. They hope to double that seed fund with private and foundation gifts. Says CI chief Russ Mittermeier: "If we don't start investing in living biodiversity with the same enthusiasm and commitment as we did in the space race of the 1960s, we're going to fail."

Adding SALT South Africa has broken ground on what will be the Southern Hemisphere's single largest optical telescope. Thousands of onlookers gathered on a windy hill near the town of Sutherland last week to celebrate the start of the Southern African Large Telescope (SALT), a 10-meter-class observatory that will take advantage of the Karoo Plain's clear skies to track nebulae and other celestial objects.

South African officials are banking on the telescope to produce more than imagery. The multinational project, which includes partners from the United States, Poland, Germany, New Zealand, and the United Kingdom, "will provide a focus for the development of basic sciences on the African continent," predicts Ben Ngubane, South Africa's science minister. He also sees it drawing tourists to the rural region, long targeted by astronomers as prime observing grounds.

Visitors, however, have a while to wait before there will be anything to see: Construction isn't due to be completed until 2004.

CREDITS: (LEFT TO RIGHT) COURTESY *THE GUARDIAN*; IMPERIAL COLLEGE SCHOOL OF MEDICINE; MISSOURI BOTANICAL GARDEN

of animal feed derived from animal carcasses and require cattle slaughtered for food to be no older than 30 months, "already take into account the possibility of subclinical infection," says Smith. He notes that previous studies have turned up no evidence that pigs and chickens are infected with BSE.

Although Smith thinks the findings might raise a warning flag, he too believes the media went overboard. But he's not surprised. "Anything that has BSE attached to it is going to produce a big media response," he says.

–MICHAEL BALTER

DEVELOPMENTAL BIOLOGY

Brain Cells Turning Over a New Leaf

Many of us may pine for our lost youth, but we know we can't turn back the clock. Biologists had long assumed that the same is true in development—that once a cell becomes committed to a particular fate, it can't reverse its tracks and become something else. A spate of recent work seems to have turned this biological dogma on its head, however. New findings described on page 1754 offer the strongest evidence yet that certain cells can be steered onto new career paths after all.

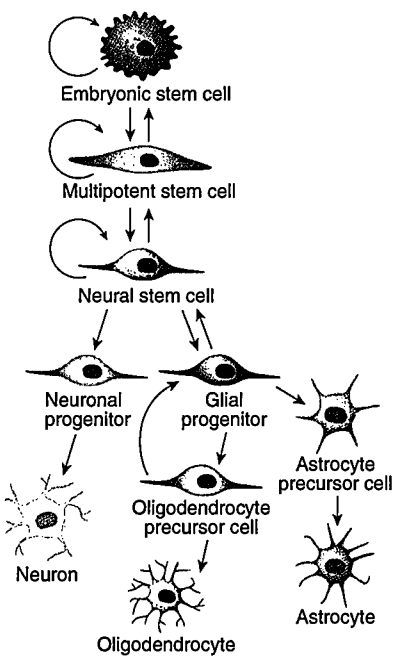
Previous reports that adult cells can be reprogrammed have been dogged by the question of whether scientists had really tapped a cellular fountain of youth or whether immature cells hiding in their culture dishes gave rise to unexpected cell types. In the current work, Toru Kondo and Martin Raff of University College London, managed to coax rat oligodendrocyte precursor cells (OPCs), which scientists thought were irreversibly committed to becoming neuronal handmaidens called oligodendrocytes or astrocytes, into becoming neurons. Because Kondo and Raff ran several experiments to test the purity of their well-characterized OPCs, they say it is unlikely that the effect was due to undetected immature cells.

"Until recently in most people's minds, there was no reverse arrow" for OPC development, says stem cell researcher Ron McKay of the National Institute for Neurological Disorders and Stroke in Bethesda, Maryland. With this work, he says, "it's pretty clear there is a reverse arrow." If the feat can be duplicated with human OPCs, it raises the tantalizing prospect of a new approach to treating Alzheimer's or other diseases marked by the loss of functional neurons: OPCs drawn from fetal tissue or even an adult patient might be reprogrammed to serve as replacement neurons.

Kondo and Raff began by isolating OPCs from the optic nerves of newborn rats and used previously described techniques—culturing the cells in fetal calf serum or exposing them to bone morphogenetic proteins—to turn them into astrocyte-like cells. They then treated the cells with basic fibroblast growth factor, a protein known to stimulate proliferation of neural stem cells. Nearly half started dividing, producing cells resembling nervous system stem cells, which the team induced to develop into neurons and astrocytes as well as oligodendrocytes.

Fetal calf serum and fibroblast growth factor, it appears, had somehow induced the OPCs to revert to a more primitive state. "There had been all sorts of evidence that these were committed precursor cells," says stem cell biologist Ben Barres of Stanford University. But the evidence that the OPCs' developmental clock can indeed be wound back, he argues, is "totally convincing."

Not to everyone, however. To test for renegade stem cells, the researchers cultured the OPCs in a series of factors that turn neural stem cells into neurons. After 2 weeks, they found no sign of neuronal markers in their cells. But neuroscientist Fred Gage of the Salk Institute for Biological Studies in La Jolla, California, says that "it still remains possible that there are undetected multipotent stem cells that exist within the culture." Last fall, his team reported that cells from the optic nerve of adult rats could, when treated with similar proteins, also become neurons. But adult cells are harder to purify than those from fetal nerves, and Gage says he wasn't convinced that his team's preparation did not contain immature stem cells.



Reversing course. Extracellular signals can prompt oligodendrocyte precursor cells to "dedifferentiate" and become neural stem cells.

The evidence that OPCs can rejuvenate in the lab is good news for eventual disease treatments. But it is not yet clear what it means for researchers who study how normal brains develop and repair themselves, notes Sean Morrison of the University of Michigan, Ann Arbor. "We have to be very careful to distinguish what happens to a cell in vivo and the potential it can have after being reprogrammed in vitro," he says. Still, he adds, attempts to pin down the molecular signals that drive reprogramming should help scientists better understand the signals that govern normal development.

Together with previous reports of shape-shifting cells, Barres says, the new work "raises the question, 'What else can be reprogrammed?' " If more elixirs can be discovered for various cell types, then more degenerative diseases might fall victim to newfound fountains of youth.

–GRETCHEN VOGEL

PALEONTOLOGY

Biggest Extinction Hit Land and Sea

There was no hiding from the greatest mass extinction of all time. Two hundred and fifty million years ago, at the end of the Permian period and the opening of the Triassic, 85% of the species in the sea and 70% of the vertebrate genera living on land vanished. Whatever pummeled life in the sea did its dirty work in a geologic moment of less than half a million years (*Science*, 15 May 1998, p. 1007). Now from South Africa comes evidence that the Permian-Triassic extinction of land plants was equally brutal and swift.

The new signs of ecological catastrophe come from rocks that started as sediments laid down in South Africa's Karoo Basin 250 million years ago. In a paper on page 1740 of this issue of *Science*, paleontologist-geologist Peter Ward and geomorphologist David Montgomery of the University of Washington, Seattle, and sedimentologist Roger Smith of the South African Museum in Cape Town report that the rocks tell of an abrupt switch in style of sedimentation, as if the land had been permanently stripped of the rooted plants that held it in place. "It looks a lot like we just lost the forests," says paleontologist Gregory Retallack of the University of Oregon, Eugene. Something with the power of an asteroid impact seems to have shattered life on Earth. But in the absence of any trace of an impact, researchers are groping for an equally far-reaching explanation.

The Karoo ecological disaster left its mark in the mud and sand laid down as water drained from the landscape. At seven spots scattered across 400 kilometers of the basin, Ward and colleagues found the same pattern of changing sedimentation. Their benchmark

ILLUSTRATION: C. CAIN