in that layer. But because the thickness of the layer has not increased during that time, the hole's depth hasn't, either. And its breadth—the width of the hole—has increased only slightly since the mid-1990s. "That's positive news," says atmospheric physicist and assessment chapter co-author Paul Newman of NASA's Goddard Space Flight Center in Greenbelt, Maryland. With ozone-destroying halocarbons expected to be on the decline, Newman says, "by 2010, we could see 5 to 6 years when the hole looks consistently smaller than during the past 5 years."

Encouraging news is coming from the Arctic, as well. That scary string of low-ozone years in the mid-1990s (researchers never rated them "holes") ended with 1997. Four of the 5 years since have seen minimal springtime ozone losses. The Arctic, it turns out, was not plunging into a full-blown, Antarctic-like ozone hole. New modeling reported in the assessment suggests that it might never do so. An early model study had suggested that the greenhouse gases that cool the stratosphere would encourage PSC formation and cause a massive ozone loss (*Science*, 10 April 1998, p. 202). "It's really looking like the more detailed models don't give that [low-ozone] result," says atmospheric chemist Susan Solomon of the National Oceanic and Atmospheric Administration in Boulder, Colorado.

Outside the polar regions, ozone has fared better than feared too. In the 1990s, rather than worsening over the northern midlatitudes, ozone depletion all but ground to a halt. Researchers aren't sure what caused the slowdown. Plateauing halocarbons certainly played a major role, but some researchers have suggested that changes in atmospheric circulation have been a key factor as well (*Science*, 22 June 2001, p. 2241). If natural variations, global warming, or even ozone depletion itself increased the amount of air moving into midlatitudes from the ozone-rich tropics, for example, midlatitude ozone would be bolstered.

There is increased evidence that atmospheric dynamics has in fact contributed to the leveling off of midlatitude ozone depletion, says dynamical meteorologist and chapter co-author William Randel of the National Center for Atmospheric Research in Boulder: "Some fraction of ozone changes—probably less than 50%—may be associated with changes in the dynamics of the stratosphere." No one can say what proportion of dynamically induced ozone change might be natural and how much is human induced.

A certain amount of optimism runs through the assessment, but so does a note of caution. The effect of climate change remains uncertain, not just on the Arctic but the whole stratosphere. The assessment also notes that although damaging ultraviolet radiation has increased on the order of 10% in some regions, ozone depletion has not been the only cause. Difficult-to-predict changes in cloud cover and pollutant hazes have altered and will continue to alter the amount of ultraviolet reaching the ground, it says. And then there's the human element. Further reductions in the production of ozone-destroying halocarbons are required in the next few years under the Montreal Protocol, especially by developing countries. Without continued reductions, the assessment concludes, ozone recovery could be delayed decades or even indefinitely. **–RICHARD A. KERR**

CREDITS (TOP TO BOTTOM): ELAINE THOMPSON/AP; THIERRY CENNEN/CGIAR

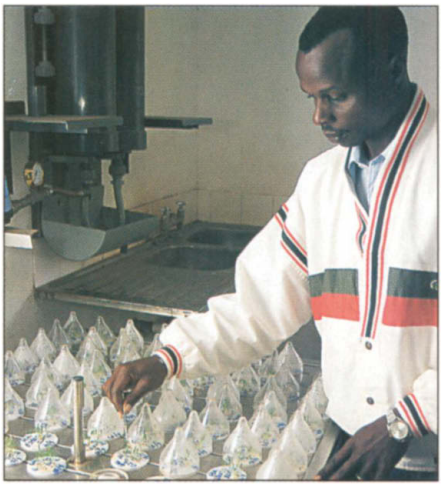
PLANT SCIENCES

Rescue Planned for Seed Banks

Plant germ plasm is a political hot potato. The issue of access to—and payments for—samples stored in gene banks was a sticking point for a treaty signed last fall by 116 nations (*Science*, 26 October 2001, p. 772). Now it could haunt a new proposal, announced last week, aimed at preserving a deteriorating global network of gene banks.

On 29 August, a new organization called the Global Conservation Trust used the United Nations (U.N.) World Summit on Sustainable Development in Johannesburg, South Africa, to announce a drive to raise a $260 million endowment to rejuvenate these seed banks. A report also released at the summit shows that shrinking budgets and smaller staffs are hindering repositories' ability to keep seeds available to breeders for improving strains or fighting diseases.

Crop gene banks around the world hold perhaps 2 million varieties of plants. Some of these, such as wheat, can be stored for years as seed. Others must be maintained in tissue culture. But even seeds need occasional replanting to ensure a viable supply, a laborious



Undernourished. Many gene banks lack resources to care for rare crop varieties.

ScienceScope

Anthropologists Win on Kennewick A federal judge has ruled that the U.S. government must allow scientists to study the bones of Kennewick Man, an ancient skeleton unearthed on the banks of the Columbia River near Kennewick, Washington. The 30 August decision marks a clear victory for a team of eight anthropologists who have fought to gain access to the 9300-year-old skeleton, arguing that it could offer new clues to how people first arrived in America. But the ruling might not end the 6-year legal tussle, as the Justice Department can still appeal the decision.

Kennewick Man, known as "the ancient One" to Native Americans, was discovered in 1996. The 380 bones and bone fragments compose one of the most nearly complete sets of ancient remains ever found in North America. Government researchers completed an initial analysis of the skeleton in 1998. But it was placed out of scientific bounds 2 years ago, when then–Secretary of the Interior Bruce Babbitt ruled that a 1990 law called the Native American Graves Protection and Repatriation Act required the skeleton to be given to the five modern Native American tribes that claimed him as an ancestor and sought to have him reburied (*Science*, 29 September 2000, p. 2257).

In his 73-page ruling, U.S. Magistrate John Jelderks of Portland, Oregon, called Babbitt's decision "arbitrary and capricious." After reviewing some 22,000 pages of documents, Jelderks ruled that there was insufficient evidence to link the skeleton to any modern tribe. "Allowing study is fully consistent with applicable statutes and regulations, which are clearly intended to make archaeological information available to the public through scientific research," Jelderks wrote. Plaintiff attorney Alan Schneider calls the decision a "landmark" because it sets an important precedent that should give researchers access to future discoveries of ancient remains.

"We are delighted with the decision," says Robson Bonnichsen, who heads the Center for the Study of the First Americans at Texas A&M University in College Station and was a plaintiff in the case. He says researchers hope to carry out a wide variety of tests on the skeleton, including skull measurements and possibly DNA tests, to pinpoint the origin of the bones. The ruling gives the researchers 45 days to submit a study proposal to the Department of the Interior and another 45 days for the government to respond.

arthritis," says Lee. Although mast cells riddle arthritic tissue, no one knew how they contribute to the disease, in part because the cells are difficult to study in humans. So the team took advantage of two strains of mice, one prone to inflammatory arthritis and another lacking mast cells. The mice are "a very nice model to elegantly show the involvement of mast cells. Previously, it was guilt by association," says rheumatologist Maripat Corr of the University of California, San Diego.

In rheumatoid arthritis, the two prongs of the immune system cooperate. One, known as innate immunity, immediately pounces on pathogens with cells that devour germs and inflame tissues. The other, called adaptive immunity, forges antibodies to fight invaders it has encountered. Going astray in arthritis, they destroy the synovium, a cushion wedged between bones in joints. Researchers think the disease begins when antibodies are somehow generated against a protein in the synovium. These so-called autoantibodies orchestrate the collapse of the joint lining by drawing in inflammatory immune processes. The inflamed cushion swells and eventually hardens to make joints distinctively gnarled. Until now, no one had determined how the autoantibodies muster up inflammation.

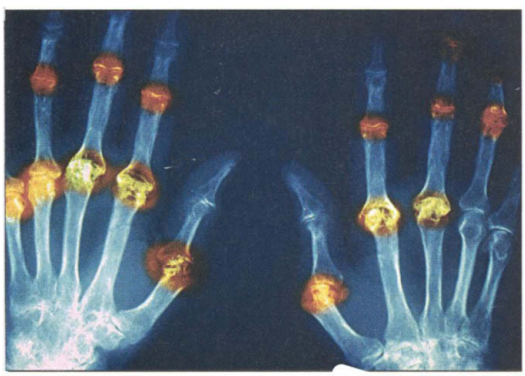
Lee's team examined whether mast cells spur the interaction between antibody-based and innate immunity. The researchers suspected the cells in part because they have receptors for both autoantibodies and inflammation-inducing proteins known as complement. What's more, mast cells can release inflammatory molecules called cytokines.

To test this idea, the team turned to so-called K/BxN mice, which have a genetic mutation that causes them to spontaneously develop inflammatory arthritis. Serum taken from these animals and injected into mice of almost any other strain will cause the receiving mouse's paws to swell. The researchers injected K/BxN serum into mice that lack mast cells as well as littermates with normal immunity. As expected, the normal mice acquired full-blown arthritis within 10 days of injection. However, mice without mast cells never manifested the disease. The team also transplanted mast cells into the mastless mice; if then injected with K/BxN serum, their paws flared with inflammation. When the researchers examined arthritic tissue, they saw that the mast cells had spewed their cytokines and other inflammatory chemicals within 2 hours of serum injection.

The researchers suggest that mast cells residing in synovial tissue are a cellular link between the free-floating autoantibodies and inflammation. Autoantibodies and complement bind to mast cells, the team proposes, which prompts them to dump their cytokines and other inflammatory chemicals, thus calling in the inflammation brigade. Rheumatologist Cornelia Weyand of the Mayo Clinic in Rochester, Minnesota, says the "beautiful study" clearly shows that "mast cells are the key effector cells in translating adaptive immunity to inflammatory disease. When you read the paper, it leaves you very satisfied."

How the study translates to human disease isn't as clear, however. Rheumatologist Joseph Craft of Yale University says that mast cell involvement will be hard to verify, because such experiments can't be done in humans. However, the mouse result might explain data showing that a cytokine named TNF-α that is released from mast cells "serves as such a dominant force" in human disease. The most recent therapy developed for rheumatoid arthritis targets this cytokine. The rheumatologists agree that the paper will cause a surge of interest in mast cells—as if the ornery rabble-rousers don't get enough attention from the allergists. –MARY BECKMAN

Mary Beckman is a writer in southeast Idaho.



CREDIT: SCOTT CAMAZINE/PHOTO RESEARCHERS

Inflammatory ruckus. Mast cells might be the missing link necessary for arthritis.

SCIENCE BUDGETS

Japan's Ministries No Longer Call the Shots

TOKYO—When Japan's ministries last week unveiled their budget requests for the fiscal year beginning next April, they revealed eye-popping increases in science-related spending. The Ministry of Education, Culture, Sports, Science, and Technology wants to boost its budget for research in four economically strategic fields by 36%; the Ministry of Economy, Trade, and Industry wants 44% more money for the same areas.

In past years, the ministries could be confident that they would end up with close to what they asked for. Not this year. The prime minister's cabinet office will now cut and shape the ministries' requests, putting its own stamp firmly on the priorities by deciding which projects actually get increases while holding overall science spending flat. Researchers fear this will further tilt the scales toward economically strategic areas. "I am strongly protesting the fact that funding will be available only when a

ScienceScope

Banking on Stem Cells The United Kingdom's plans for a stem cell bank are expected to take concrete shape next week. The Medical Research Council (MRC), which will oversee the cell repository, will announce details of the bank's location and operation at an 11 September symposium in London.

According to a plan strongly endorsed last February by a House of Lords special committee on stem cell research, the bank will hold both embryonic and adult stem cell lines and distribute them to academic scientists in the United Kingdom and abroad. Any new human embryonic stem cell lines derived in Britain must be deposited in the bank.

The planned announcement made headlines in the United Kingdom last week as several newspapers questioned the meeting's timing on the anniversary of last year's terrorist attacks, charging that MRC hoped any potential controversy stirred by the meeting would go unnoticed. But an MRC spokesperson says the date was chosen for logistical reasons—and noted that both the press and opponents of stem cell research have been invited.

Splitting Cells Australia's quest for national legislation regulating human embryonic stem cell research has hit another speed bump. The House of Representatives last week voted to split the proposed legislation (*Science*, 30 August, p. 1461) into two bills—passing one that bans human cloning for reproduction but delaying a vote on the other, which allows researchers to use and derive certain human stem cell lines for research. Lawmakers are expected to revisit the issue later this year, but researchers worry that opponents of stem cell research will use the time to organize.

Prime Minister John Howard, meanwhile, has ordered a review of the government's $25 million commitment to a new stem cell research center in the wake of a controversy sparked by researcher Alan Trounson of the Monash Institute of Reproduction in Melbourne. Trounson, head of the new Center for Stem Cells and Tissue Repair, admitted to misrepresenting a video of a crippled rat he showed to Parliamentarians. He claimed that the rat had regained partial muscle function after being treated using rat stem cells; in fact, researchers had used other kinds of human fetal tissue. Howard said he was "disturbed" by the incident, but he supports stem cell research.

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