and costly process called regeneration. A 1996 survey of 151 countries by the U.N. Food and Agriculture Organization (FAO) found that many facilities were rapidly deteriorating and had a large backlog of samples needing regeneration. A follow-up survey in 2000, analyzed by Chris Higgins and colleagues at Imperial College London, U.K., concludes that "the situation has gotten worse," says Geoffrey Hawtin, director of the International Plant Genetic Resources Institute in Rome, Italy.

For Enrique Suárez, director of the National University of Agricultural Technology's main gene bank, in Castelar, Argentina, that means insufficient staff to regenerate samples. And the recent devaluation of the peso has left him too poor to buy specialized sample bags to refrigerate some 30,000 samples awaiting curation. "The work at the gene bank is stopped," he says. "It's very frustrating." Plant samples are stacking up in two-thirds of the countries surveyed, and the budgets of a quarter of gene banks have been trimmed.

The Global Conservation Trust teams FAO with the Consultative Group on International Agricultural Research, which runs 11 major gene banks. At the summit, the Swiss government ponied up $10 million, but trust officials say they need 10 times that figure before they will solicit proposals.

Uncertainty about the governance of the trust could spell trouble, warns Pat Mooney of the ETC Group in Winnepeg, Canada, which defends the rights of farmers in developing countries. Some governments might misconstrue the endowment as a way for industrialized countries to gain control over germ plasm resources, he cautions. "What worries me a lot is that it will open a wide vista for agribusinesses to serve their own purposes," says Melaku Worede, an agricultural consultant based in Addis Ababa, Ethiopia. But the trust's Ruth Raymond says that developing countries will have a voice in the trust's governance structure, and that several countries are interested in contributing. **–ERIK STOKSTAD**

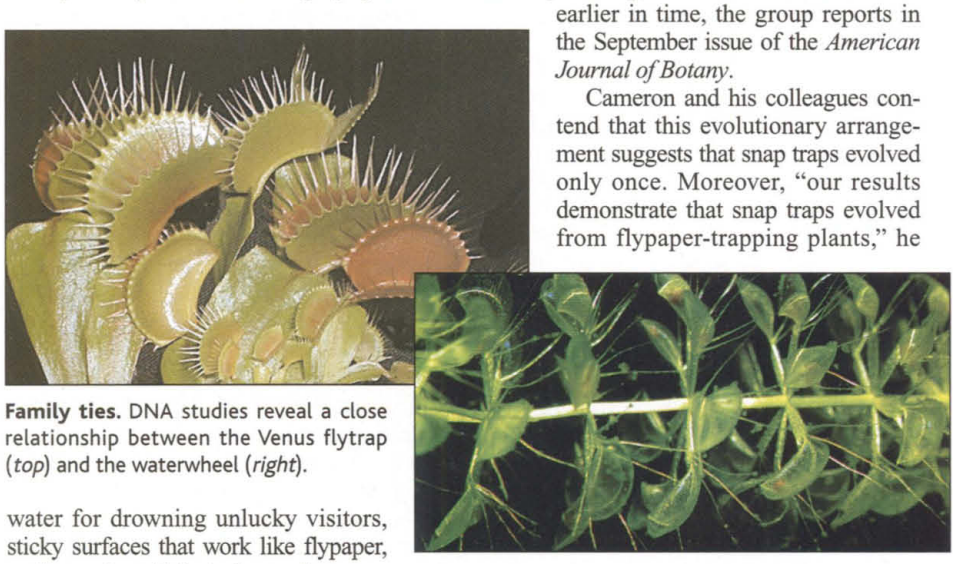
PLANT EVOLUTION

Elaborate Carnivorous Plants Prove to Be Kin

The Venus flytrap has a muddled family history. Charles Darwin thought this elegant bug eater from the southern United States had close ties to a European aquatic weed called the waterwheel. A century later, researchers decided that the waterwheel's closest kin was not the Venus flytrap but the terrestrial sundew, which also dines on insects. Now a DNA analysis of these botanical carnivores suggests that Darwin's hunch was right after all.

In many ways, this revised family history makes sense, comments Mark Chase, a plant systematist at the Kew Royal Botanical Gardens in Surrey, U.K.—even though he once suggested otherwise. Of all the plants that feast on animals, waterwheels and Venus flytraps "have taken carnivory to the extreme," he notes: Each has leaves reshaped into traps that snap shut. Now that their close relationship is "nailed down, it sets the stage for people to ask more intelligent questions about how these mechanisms evolved," Chase points out.

Carnivorous plants have come up with a variety of ways to snare their prey: pools of water for drowning unlucky visitors, sticky surfaces that work like flypaper, or "snap traps" that clamp down on morsels in milliseconds. Sundews are flypaper predators; waterwheels and Venus flytraps depend on snap traps. All use their prey not as a food source but to provide minerals.



Family ties. DNA studies reveal a close relationship between the Venus flytrap (*top*) and the waterwheel (*right*).

Evolutionary biologists have long speculated about how these features evolved. In the late 1800s, Darwin picked up on similarities in the stamens and pistils—a flower's reproductive parts—of waterwheels and the Venus flytrap and suggested that these two plants were closest kin. However, in the early 1990s, Chase and his colleagues threw a fly in the ointment, so to speak, when they compared the DNA of about a dozen carnivorous plants and took a closer look at their morphology. They had no DNA from waterwheels and so relied solely on morphology (*Science*, 11 September 1992, p. 1491).

The 20th century study led researchers to conclude that they should lump the sundew in with the waterwheel and push the Venus flytrap out of the tight-knit group. This family tree had evolutionary implications, says Richard Jobson, a plant systematist at Cornell University in Ithaca, New York. Snap traps might have evolved twice, once in the waterwheel and once in the Venus flytrap. Alternatively, the alignment could mean that a snap-trapping ancestor gave rise to sundews, in which case the less elaborate flypaper traps represented simple, modified snap traps.

Now a 21st century DNA analysis tells a different evolutionary story. Jobson, Kenneth Wurdack, a plant systematist at the Laboratories of Analytical Biology of the Smithsonian Institution in Suitland, Maryland, and Kenneth Cameron of the New York Botanical Garden have compared four genes instead of the one studied in the 1990s. They conclude that even though the Venus flytrap is terrestrial and the waterwheel aquatic, the world's only two snap-trapping plants are nonetheless siblings. The sundew is no closer than a cousin, sharing a common ancestor much earlier in time, the group reports in the September issue of the *American Journal of Botany*.

Cameron and his colleagues contend that this evolutionary arrangement suggests that snap traps evolved only once. Moreover, "our results demonstrate that snap traps evolved from flypaper-trapping plants," he says. They also think that among snap-trappers, the Venus flytrap came first.

Chase thinks the snap-trap story might be more complicated than it now looks. The two species "don't live in the same parts of the world," he explains, and although fossils show that the waterwheel was once common throughout Eurasia, the Venus flytrap is known to grow only in North and South Carolina. That leaves open the question of where the snap-trap plants got started and how they spread. **–ELIZABETH PENNISI**

INFLAMMATORY ARTHRITIS

How Immune System Gangs Up on Joints

Mast cells are best known for releasing the dastardly allergy compound histamine, which induces sniffles and swollen eyes. Now rheumatologists have found the troublemaker cells embroiled in another dysfunctional immune system activity: inflammatory arthritis. On page 1689, David Lee of Harvard Medical School in Boston and colleagues report that mast cells in mice act as a bridge linking arthritis's self-attacking antibodies and the inflammation that swells joints.

It took time to accumulate evidence on the suspects. "There are 20 years of literature documenting mast cells in human inflammatory

CREDITS: (TOP TO BOTTOM) K. CAMERON/THE NEW YORK BOTANICAL GARDEN; BARRY RICE/WWW.SARRACENIA.COM

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.

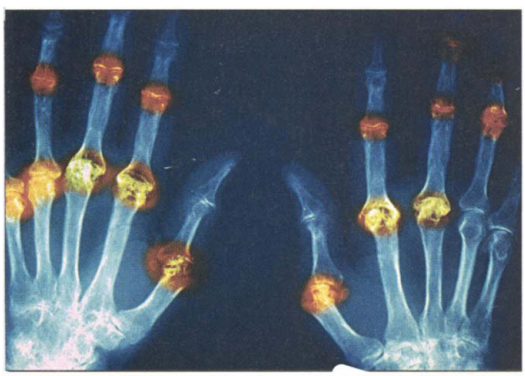
Lee's team examined whether mast cells spur the interaction between antibody-based and innate immunity. The researchers suspected the cells in part because they have receptors for both autoantibodies and inflammation-inducing proteins known as complement. What's more, mast cells can release inflammatory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

Contributors: Robert F. Service, Gretchen Vogel, Leigh Dayton