surface and a mirrorlike flat bottom surface. The combination helped newly created photons bounce around inside the device until they could find an escape route, raising the efficiency to more than 1%.

The next step for the team is to convert the silicon LED's steady stream of light into a series of pulses that can encode information, by connecting the LED to a device called a modulator. Then both devices will be placed directly onto a silicon computer chip. **–ROBERT F. SERVICE**

ECOLOGY

African Elephant Species Splits in Two

As the largest land mammal, elephants should be hard to miss. But scientists have apparently overlooked an entire species. On page 1473, a team of geneticists and elephant experts describe new molecular evidence showing that forest- and savanna-dwelling elephants, currently lumped together in a single species called *Loxodonta africana*, each merits its own species name.

For more than 100 years, scientists have argued about the distinctiveness of forest elephants. The shy creatures are difficult to spot in their thick forest habitat, and only one is in captivity in the Paris Zoo. But those fortunate enough to have seen both them and their better known savanna-dwelling cousins note that the difference is striking. Forest elephants are not only smaller, but they also have straighter, longer tusks and round, as opposed to pointed, ears. "If you see a forest elephant for the first time, you think, 'Wow, what is that?,' " says team member Nicholas Georgiadis, a biologist at the Mpala Research Center in Nanyuki, Kenya.

Despite the differences between savanna and forest elephants, most biologists had assumed that the two populations readily mix on the edges of forests. At best, forest elephants were designated as a subspecies, *Loxodonta africana cyclotis*. But when Georgiadis and his collaborators analyzed the DNA of the elephants, the results indicated that the two populations are as genetically distinct as lions are from tigers. Indeed, the researchers propose two separate species names: *Loxodonta africana* for the savanna elephants and *Loxodonta cyclotis* for the forest dwellers. "The morphological evidence has been very, very strong," says conservation biologist Samuel Wasser of the University of Washington, Seattle. "When you see the genetic data, it seems almost a no-brainer."

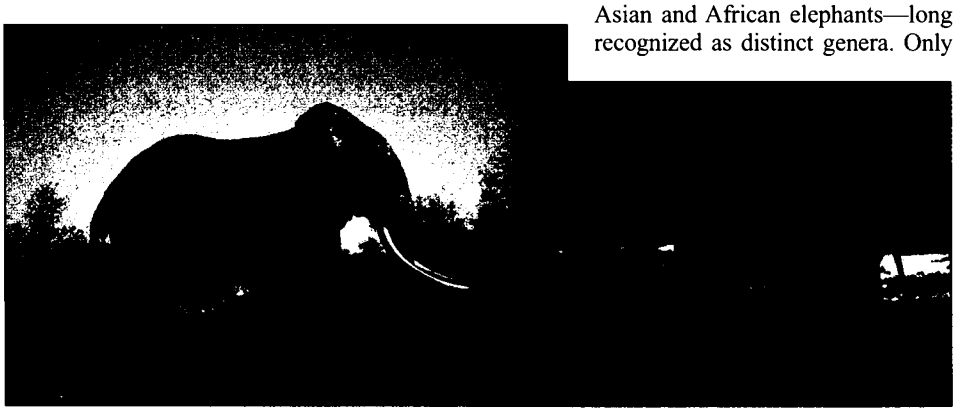
The team bases its claim on data from an extensive collection of tissue samples from 195 animals in 21 different elephant populations. Georgiadis spent 8 years collecting the samples, shooting needlelike darts into free-ranging elephants. The darts collected a plug of skin and then fell to the ground, enabling Georgiadis to retrieve them after the startled elephant ran away. The project was originally designed to collect genetic signatures so that ivory samples could be traced to their elephant populations—a goal other geneticists are pursuing. A preliminary analysis of the mitochondrial genes suggested significant differences between forest and savanna dwellers (*Science*, 7 March 1997, p. 1418), a finding that piqued Georgiadis's interest, but a more robust test with nuclear genes was needed to cinch the case, he says.

To pursue the question, Georgiadis teamed up with researchers at the National Cancer Institute (NCI) in Frederick, Maryland, to measure the genetic variation between the populations. Alfred Roca, a postdoc at NCI, with geneticists Stephen O'Brien and Jill Pecon-Slattery, sequenced portions of four nuclear genes, a total of 1732 nucleotides, from each of the samples. The researchers focused on the noncoding intron regions of the genes, which are not subject to natural selection; this makes them more reliable indicators of the random genetic changes that occur over time.

The team pegged the genetic distance between forest and savanna samples as more than half as large as the distance between Asian and African elephants—long recognized as distinct genera. Only one of the populations showed the type of genetic mixing that could come from interbreeding, and that apparently happened several generations ago. To O'Brien, that means that crossbreeding between the two populations "does occur once in a while, but not very often."

The new genetic evidence has implications for conservation, says Georgiadis. Instead of assuming that 500,000 elephants exist in Africa, "there are many fewer than that of each kind, and they're both much more endangered than we presumed," he says. Researchers estimate that up to one-third of African elephants are forest dwellers.

Ivory from forest elephants is especially prized for its hardness and sometimes pinkish hue. Wasser cautions that conservation organizations must be alert: The current international regulations list only *Loxodonta africana* as protected. If the law is not changed quickly to reflect a new species name, an inadvertent loophole might leave the vulnerable forest elephant even more at risk. **–GRETCHEN VOGEL**



Distinctly different. African forest elephants *(right)* have rounder ears and straighter tusks than savanna-dwelling elephants *(left)*.

CREDITS: (LEFT TO RIGHT) MICHAEL BISCEGLIE/ANIMALS, ANIMALS; PETER WEIMANN/ANIMALS, ANIMALS

VIROLOGY

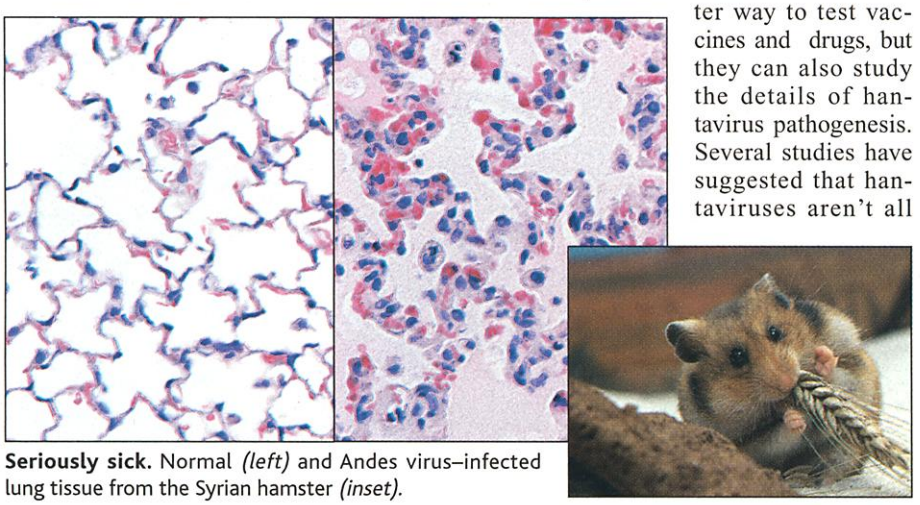
Finally, a Handle on The Hantaviruses

A group of U.S. Army virologists has found by accident what researchers had been seeking for decades: an animal model to study hantaviruses, a fearsome group of rodent-borne pathogens that cause disease and death across the globe. In a paper accepted by the journal *Virology*, they report that Syrian hamsters get sick and die when injected with a hantavirus from South America—and that the animals' disease looks strikingly like hantavirus pulmonary syndrome (HPS), one lethal manifestation of the infection in humans.

The report, from a group at the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID) in Fort Detrick, Maryland, means that researchers at last have a way to study how one member of the family sickens and kills—and how the disease can be stopped. "I'm impressed," says Heinz Feldmann, a virologist at the Canadian Science Centre for Human and Animal Health in Winnipeg. "This will definitely speed up vaccine and drug development." Virologist Stuart Nichol of the U.S. Centers for Disease Control and Prevention in Atlanta hails the study as "a major breakthrough for the field."

The hantavirus family grabbed world headlines in 1993 when an outbreak occurred in the Four Corners area of the southwestern United States. The culprit, now called Sin Nombre virus, is one of several hantaviruses that cause HPS throughout North and South America. No specific

antiviral treatments exist, and usually between one-third and one-half of the victims die. In Europe and Asia, a quartet of different hantaviruses causes tens of thousands of cases yearly of a disease called hemorrhagic fever with renal syndrome (HFRS); mortality rates range from 0.1% to 15%. Hantaan virus, the first of that bunch to be discovered, caused more than 3000 cases of HFRS among United Nations troops during the Korean War between 1951 and 1954; the U.S. Army has had a keen interest in developing hantavirus vaccines ever since.



Seriously sick. Normal *(left)* and Andes virus–infected lung tissue from the Syrian hamster *(inset)*.

But Army researchers and others have been handicapped by the lack of animal models. No matter how many animals they injected with various hantaviruses, they could not produce anything resembling the ravages of either severe HFRS or HPS. For example, two teams have injected monkeys with a European hantavirus called Puumala, but it caused symptoms that would likely be too subtle for a vaccine trial, says USAMRIID team leader Jay Hooper.

Confronted with these obstacles, the team had to make do with another strategy. Instead of testing whether a vaccine protects against disease, they tested whether it could prevent infection altogether—a much stricter test because it flunks vaccines that let the virus enter the body and replicate but prevent illness.

The team recently started looking into vaccines for Sin Nombre and the Andes virus from South America, which together cause the great majority of HPS cases in the Americas. A key initial step was determining how much virus is needed to infect an unvaccinated animal. When Hooper injected hamsters with Sin Nombre, they became infected but stayed healthy. But after he injected adult Syrian hamsters with Andes virus, something unusual happened. One by one, the animals developed difficulty breathing, and most died within days—just as fast as human victims. Further studies of the hamsters by USAMRIID pathologist Tom Larsen revealed that after an incubation period of about 11 days, the microscopic blood vessels in the animals' lungs became permeable and their lungs and chest cavity rapidly filled with fluid, essentially causing them to drown. All these symptoms closely resemble HPS in humans, says Hooper.

Not only do researchers now have a better way to test vaccines and drugs, but they can also study the details of hantavirus pathogenesis. Several studies have suggested that hantaviruses aren't all that lethal to individual cells, says virologist Clarence Peters of the University of Texas Medical Branch in Galveston. Instead, the human immune system's response may wreak the real havoc. Researchers can now test that theory by blocking or stimulating the suspect immune messengers—studies that could provide new drug leads.

The model does have drawbacks, however. Because it works only with Andes virus (the team is still not sure why), it may not tell researchers much about the HFRS-causing hantaviruses. And the hamster, unlike the mouse, isn't a common lab animal, so researchers lack both an intimate knowledge of its biology and a wealth of reagents to study it. Even so, the dearth of models has been so frustrating that Peters predicts others will jump on the findings.

–MARTIN ENSERINK

CREDITS: (TOP TO BOTTOM) THOMAS LARSEN/USAMRIID; BOB SCHATZ/ANIMALS, ANIMALS; AP PHOTO/UNSCOM

ANIMAL CARE

Coulston Loses NIH Tie, Faces Hard Times

A major U.S. primate facility has lost its permit to house and experiment on federally owned chimpanzees, raising questions about its viability.

In June the National Institutes of Health (NIH) ended its funding of the Coulston Foundation of Alamogordo, New Mexico, after finding a new caretaker, Charles River Laboratories, for 300 chimps housed there (*Science*, 10 September 1999, p. 1649). Later that month NIH officials let lapse a document, called an Animal Welfare Assurance,

ScienceScope

Red Flight NASA engineers are celebrating the success of a prototype plane that one day could swoop over martian dunes and canyons, looking for water and providing a detailed view of the planet's complex surface. The small glider was dropped 9 August from a helium-filled balloon that carried it to an altitude of more than 30,000 meters above the Oregon coast. Designed at the Ames Research Center in Mountain View, California, the glider has a long, straight wing nearly 3 meters long to help it stay aloft high in Earth's atmosphere, an analog to the thin martian atmosphere at low altitudes. Both the recent flight and a low-altitude mission last month by another model met engineering expectations, agency officials say. But don't expect scheduled flights soon; NASA still must develop a craft with wings that could be folded up to fit inside a spacecraft as well as a suitable propeller propulsion system.

Going Nowhere Negotiations over measures to ensure compliance with the 30-year-old Biological and Toxin Weapons Convention have come to an ignominious end. Last month the U.S. delegation at the talks announced its staunch opposition to the measures, set out in a draft treaty protocol (*Science*, 20 July, p. 414). So the representatives from 55 nations instead tried to craft a consensus statement to preserve the current draft protocol as a basis for future discussions.

But even that document landed in the wastebasket in the final minutes of a monthlong negotiating session that ended 18 August. The U.S. delegation objected to language that hinted at its opposition to the protocol. The disagreement doesn't bode well for a November review conference at which treaty states are meant to take stock of potential bioweapons threats that have emerged during the past 5 years. Bioweapons expert Graham Pearson of the United Kingdom's University of Bradford predicts "a lot of recrimination" at that meeting. Others share the pessimism. "I hate to think you can't get countries to act unless a disaster strikes," says Barbara Hatch Rosenberg, chair of the Federation of American Scientists' biological weapons working group.

Contributors: Martin Enserink, Constance Holden, Andrew Lawler, Richard Stone