

RANDOM SAMPLES

edited by CONSTANCE HOLDEN

NASA's Fling With Anti-Gravity

Anti-gravity—this year's "cold fusion"? Gravity is one force that has always seemed immutable. But if dreams come true at NASA, a device that uses a spinning disk of superconductor to defy gravity could one day revolutionize spacecraft propulsion.

Anti-gravity research jumped into the news last month when the London *Sunday Telegraph* came out with a story touting a soon-to-be-published paper by engineer Eugene Podkletnov, a sometime researcher at Finland's University of Tampere, on "the world's first anti-gravity device." In the ensuing publicity—which included confusion over the iden-

tity of a co-author—Podkletnov withdrew his paper, scheduled to appear in the *British Journal of Physics D*.

But NASA has already sunk several hundred thousand dollars into the anti-gravity game, according to L. Whitt Brantley, chief of the Advanced Concepts Office at Marshall Space Flight Center in Huntsville, Alabama. Brantley says physicist Ning Li of the University of Alabama approached NASA several years ago with a theory postulating that there are connections between gravity and electromagnetism that could be manipulated by superconduc-

tors. NASA was also spurred by a 1992 paper by Podkletnov that "seemed to exhibit the effect she [Li] was predicting," says Brantley.

Li currently heads a project to build Podkletnov's device. At its heart is an electromagnetically suspended, rapidly rotating ring of superconducting ceramic, 275 millimeters in diameter, that is cooled in liquid nitrogen. In the paper Podkletnov withdrew, he reports that when various substances were suspended in a balance scale over the ring, they lost about 2% of their weight. Engineer Ronald Koczor of the Marshall Center, who readily admits that "this is far-out stuff,"

says that they ought to get results from the experiment within a year.

Koczor says the project has not been reviewed by scientists outside of NASA. But Paul Chu of the superconductivity center of the University of Houston isn't expecting it to push back the frontiers of physics anytime soon. Chu says he heard a presentation by Li, but neither he nor his colleagues fully understand "what Li was trying to say."

Podkletnov could not be reached for comment. Indeed, NASA officials don't seem to know how to reach him. But he's been assured, says Koczor, that "if we got results ... he'd be the first to know."



R. A. MITTERMEIER

Going, going... Radiated tortoise.

Endangered Species List Updated

The radiated tortoise from Madagascar, besieged by collectors as well as habitat loss, is one of the 5205 species at risk of extinction, according to the 1996 World Conservation Union's (IUCN's) Red List. The latest version of the biennial report is compiled with contributions from some 500 scientists by IUCN's Species Survival Commission (SSC). It uses new, more rigorous criteria to assess species as Critically Endangered, Endangered, or Vulnerable, and, as a result, leaves out many for which there are not enough data, says Russell Mittermeier, president of Conservation International, who heads the SSC's primate group. Also in this year's report is the first analysis of threats to all 4600 known species of mammals, one-quarter of whom are at risk of ex-

tinction. Leading the list are monkeys and apes (46%); shrews and moles (36%); and pigs, antelope, and cattle (33%). Birds were also assessed in their entirety, and 11% are at risk. So are 25% of amphibians and 34% of fishes. Says SSC Chair George

Rabb, director of Chicago's Brookfield Zoo, "it's pretty scary stuff."

The report can be accessed at <http://www.iucn.org/themes/ssc/index.html>.

Downer for Electric Cars

Electric cars have enough problems as it is. Existing models are expensive, slow to recharge, and have only about half the range of a gas guzzler with a full tank. Now even their clean-air advantage seems to be evaporating.

In last month's issue of *Environmental Science and Technology*, environmental engineer Ted Russell at Georgia Tech University and colleagues at Carnegie Mellon University report that putting 500,000 electric cars on the road today in smog-bound

cities like Los Angeles would probably reduce ozone by only 0.5%, or from 200 parts per billion (ppb) to 199 ppb. That's less than half an earlier estimate of 1.3% made by Russell and colleagues at Resources for the Future in 1990.

What's happening, Russell explains, is that gas-powered cars have been forced to clean up their act faster than most experts expected even 5 years ago, thus narrowing the advantage held by zero polluters. State agencies, such as California's powerful Air Resources Board (ARB), have hastened the cleanup by mandating that cars reduce emissions of some compounds to as little as one-third of federal ceilings. As a result, today's cars emit on average only about 13% of the ozone-causing nitrogen oxides, for example, that they gave off before emissions controls began in the late 1960s. Although electric cars are cleaner still—the power-plant pollutants that would accompany energy production for electric cars are about one-tenth of what an equivalent amount of gasoline would produce—the gap continues to narrow. Another factor, Russell says, is that

ON POSTMODERN CRITIQUES OF SCIENCE

"The dictum that everything that people do is 'cultural' ... licenses the idea that the cultural critic can meaningfully analyze even the most intricate accomplishments of art and science. ... It is distinctly weird to listen to pronouncements on the nature of mathematics from the lips of someone who cannot tell you what a complex number is!"

—Rutgers University mathematician Norman Levitt, in *The Flight from Science and Reason*, a collection of essays recently published by the New York Academy of Sciences.

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several years ago most experts assumed that electric vehicles would mainly replace gas-fueled vans and delivery vehicles. But today, it looks as if most electric vehicles will replace cars, which are less polluting than vans anyway. The upshot, says Russell: "You don't see electric vehicles giving you as much bang for the buck."

Is it still worth following through with electric vehicle mandates, such as ARB's requirement that "zero emission" cars compose at least 10% of those sold in California in 2003? Absolutely, says California's ARB chief deputy executive officer, Tom Cackette. Cities like Los Angeles still need to pull all the stops to lower ozone levels from 200 ppb to the federally mandated 120 ppb. "There isn't anything out there that provides a 2%, 5%, or 10% ozone reduction," he says. "All the easy stuff was done 20 years ago."

DNA Dumping

Cells that can selectively prune their own DNA are finally yielding some of their secrets. Scientists at the University of Rochester in New York and the Fred Hutchinson Cancer Research Center in Seattle have found evidence for a protein-and-DNA structure that seems to play a role in the selective degradation of chromosomes in a single-celled organism, *Tetrahymena*. They have also identified a protein that may help create the structure. This marks the first time a specific protein has been linked to targeted DNA degradation—a finding that could shed light on the mysteries of programmed cell death (apoptosis), a hot topic in studies of development, aging, and cancer.

The protein in question, called Pdd1p, associates with the DNA that is to be eliminated and seems to cluster into spherical structures in the cell's nucleus. Rochester cell biologist David Allis and his colleagues, reporting in the 4 October *Cell*, speculate that these structures help dispose of unneeded DNA during certain stages of reproduction. Suitably, Allis likes to refer to the structures as "dumposomes."

In previous studies, the researchers identified several proteins—including Pdd1p—that appear as the cell rids itself of DNA. Now they have zeroed in on Pdd1p and found it in developing nuclei where the cell selectively deletes about 15% of its DNA. The protein also showed up in old nuclei being destroyed during *Tetrahymena* reproduction—a process the researchers say is analogous to apoptosis in multicellular organisms.

Allis has speculated that the structure may be a new organelle, on a par with ribosomes or mitochondria, but Lawrence Klobutcher, a molecular biologist at the University of Connecticut Health Center, says it's far too early to rewrite any textbooks. To be a true organelle, he says, the structure would have to be shown to serve as a "garbage disposal"

that actually degrades DNA, rather than a wastebasket that simply gathers the nucleotides together so another process can destroy them. However, Klobutcher says, spotting the protein should help scientists understand more about how some organisms selectively remodel their genomes, and perhaps apoptosis as well.

Glenn Herrick, a geneticist at the University of Utah, says scientists will now be able to genetically manipulate *Tetrahymena*, knocking out the gene that codes for the dumposome-forming



Nuclear dump. *Tetrahymena* with Cheerio-shaped "dumposomes" in nuclei.

Flagging Malaria Research

The world's big killers still aren't attracting the big research dollars, according to a survey by Britain's Wellcome Trust. According to a report* issued last month by the trust, the world's largest philanthropy, the world is giving "markedly higher" support to work on AIDS and asthma than to diseases such as malaria and tuberculosis (TB) which kill many more people annually. Wellcome has decided that malaria research is being neglected, and as a result has quadrupled its spending on the disease over the past decade to \$10 million a year. But over the same period the field's main source of funding, the United States, has nearly halved its contribution, from about \$65 million to about \$35 million, mainly because of congressional cuts in foreign-aid spending. Funding from the World Health Organization and allied agencies has hovered at \$9 million to \$10 million.

* *Malaria Research: An Audit of International Activity*. For information e-mail prism@wellcome.ac.uk.

GLOBAL RESEARCH EXPENDITURES FOR FOUR DISEASES, 1990		
	Mortality (thousands)	Research \$ per fatality
HIV/AIDS	290.8	3274
Asthma	181.3	789
Malaria	926.4	65
TB	2015.5	13

protein and determining its specific function—and perhaps open a window to the DNA elimination mechanism. "There must be other proteins involved," he says. "This might be a hook that will enable us to get hold of them."

A Gene That Controls Herpes

Before there was HIV and AIDS to worry about, there was herpes. Magazine cover stories warned of the spreading epidemic, and experts decried the lack of effective treatments. Even today, there's no cure for the herpes simplex viruses (HSV), which periodically cause painful cold sores or genital lesions and then spend the rest of their time hiding in nerve cells, unreachable by either the immune system or medication.

Now a team of virologists has identified a single gene that determines where the sores recur, a finding that could be a first step toward preventing reactivation. Philip Krause of the U.S. Food and Drug Administration and his colleagues have found a key difference between DNA in HSV-1, which commonly causes facial cold sores, and HSV-2, the usual culprit in genital herpes, which infects one in five adults. That DNA includes the gene for the "latency-associated transcripts," the only RNA found while the virus is hiding out.

Initially both viruses can infect either the mouth and eyes or the genital tract, but afterward, "there's something which causes them to recur differently," says Krause. That something is the gene that encodes the latency-associated transcripts. First identified in 1987, that gene seemed like a good candidate to find differences because of its activity during latency. When the researchers removed this DNA from HSV-2, replaced it with the same DNA from HSV-1, and exposed this mutant virus to guinea pigs and rabbits, HSV-2 started behaving just like HSV-1: showing up mainly in the eyes rather than the genitals, they reported late last month at a microbiology meeting in New Orleans.*

Virologist Stephen Straus of the U.S. National Institute of Allergy and Infectious Diseases notes that the prominent role of a single gene sets HSV apart from other viruses, whose manifestations are controlled by many genes. That this gene "has such a strong influence on site-specific rates of reactivation is a surprise." It may also aid efforts to develop treatments, says Krause, because it gives scientists a way to figure out the mechanism of reactivation.

*The 36th Interscience Conference on Antimicrobial Agents and Chemotherapy, 15–18 September.