NOVEMBER 11, 2006 PAGES 305-320 VOL. 170, NO. 20

SCIENCE NEWS

THE WEEKLY NEWSMAGAZINE OF SCIENCE

vitamin d wards off germs

malaria: old drug is new again

peppers, spider share bite

making blind mice see

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THE WEEKLY NEWSMAGAZINE OF SCIENCE

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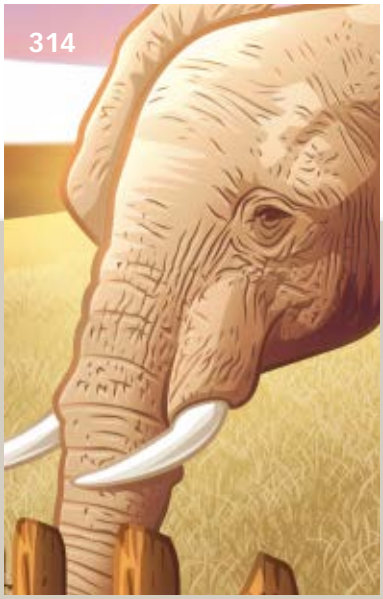

Features

This Week

Of Note

Meetings

Departments

Cover A new conservation plan called Pleistocene rewilding suggests reintroductions of megafauna species that lived in North America thousands of years ago. (Dean MacAdam) Page 314

Science News (ISSN 0036-8423) is published weekly on Saturday, except the last week in December, for $54.50 for 1 year or $98.00 for 2 years (foreign postage is $18.00 additional per year) by Science Service, 1719 N Street, N.W., Washington, DC 20036. Preferred periodicals postage paid at Washington, D.C., and an additional mailing office.

POSTMASTER Send address changes to **Science News**, P.O. Box 1925, Marion, OH 43306. Two to four weeks' notice is required. Old and new addresses, including zip codes, must be provided.

EDITORIAL, BUSINESS, AND ADVERTISING OFFICES 1719 N St. N.W., Washington, D.C. 20036 202-785-2255; scinews@sciencenews.org.
LETTERS editors@sciencenews.org

SUBSCRIPTION DEPARTMENT P.O. Box 1925, Marion, OH 43306. For new subscriptions and customer service, call 1-800-552-4412.

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Malaria Reversal

Drug regains potency in African nation

An inexpensive drug that has lost much of its punch against malaria over the past 20 years is showing signs of regaining its strength in the African nation of Malawi. But researchers warn that the entire continent would have to coordinate its fight against the disease in order for the drug to regain a prominent place among malaria fighters.

Doctors have used the drug, chloroquine, to treat malaria for 60 years, but *Plasmodium falciparum*, the protozoan that causes severe malaria, has become increasingly resistant. Malawi abandoned the drug in 1993, and doctors there replaced it with an inexpensive combination pill containing sulfadoxine and pyrimethamine. Scientists took note.

The Malawi strategy worked for several years. But eventually, the combination drug became less and less effective against *P. falciparum*. The emerging therapy of choice in Africa is now a more expensive, more complex treatment that includes derivatives of artemisinin—an extract of the Chinese herbal remedy called sweet wormwood (*SN: 2/7/04, p. 94*).

Meanwhile, scientists working in Malawi have found that the malaria protozoan recovered from patients no longer harbored a mutation that makes it resistant to chloroquine, says Miriam K. Laufer, a pediatric infectious-disease physician at the University of Maryland School of Medicine in Baltimore.

To find out whether the drug might again be effective, Laufer and her colleagues last year identified 210 Malawian children with malaria and gave half of them chloroquine and half of them the sulfadoxine-pyrimethamine combination.

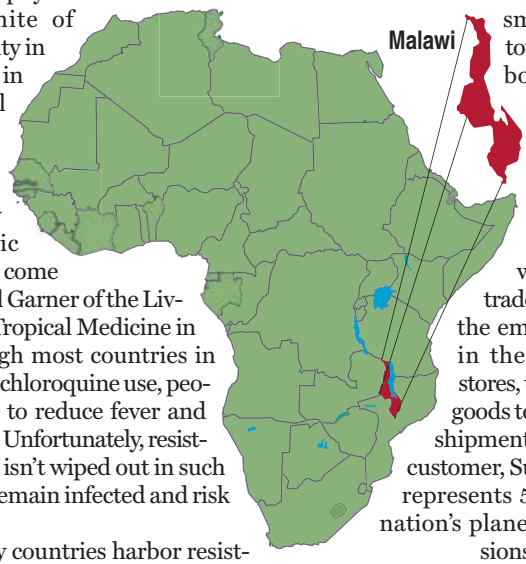
Of 80 children who had received chloroquine and were monitored over 4 weeks, all but one cleared the parasite from their blood. In contrast, 71 of 87 children who were reexamined after getting the sulfadoxine-pyrimethamine combination failed to improve and received other drugs, the scientists report in the Nov. 9 *New England Journal of Medicine*. Some children weren't available for follow-up.

The Malawi experience establishes that *P. falciparum* can become susceptible again to chloroquine after the drug has been absent, says physician Nicholas J. White of Mahidol University in Bangkok, writing in the same journal issue.

"It's a fascinating finding, but it's too early to say whether a public health policy will come out of it," says Paul Garner of the Liverpool School of Tropical Medicine in England. Although most countries in Africa discourage chloroquine use, people take the drug to reduce fever and feel better, he says. Unfortunately, resistant *P. falciparum* isn't wiped out in such patients, so they remain infected and risk the fever's return.

Because nearby countries harbor resistant malaria strains that could reenter Malawi, Laufer says, reintroducing chloroquine as a standard treatment is unlikely. Any push to stop chloroquine use "would need to be continentwide," she says.

If chloroquine were to replace failing sulfadoxine-pyrimethamine therapy across Africa before chloroquine resistance had disappeared from *P. falciparum*, the result could be a parasite impervious to both treatments, warns Toshihiro Mita, a physician at Tokyo Women's Medical University. —N. SEPPA



SWITCHING
Malawi raised eyebrows in 1993 when it banned use of chloroquine against malaria.

E. ROELL

Not So Clean

Service industries emit greenhouse gases too

In recent decades, a large part of the U.S. economy has shifted to providing services rather than manufacturing products. Despite the presumption that the change bodes well for the environment, service industries such as the retail trade are creating just as much planet-warming carbon dioxide as the manufacture and operation of motor vehicles do, a new analysis suggests.

Industrial ecologist Sangwon Suh of the University of Minnesota in St. Paul scrutinized the movement of energy, raw materials, and products through various sectors of the economy. In such an analysis, emissions "that happen behind the scenes can then be taken into account," he notes.

In aggregate, all the companies that provide services are directly responsible for less than 5 percent of U.S. greenhouse-gas emissions, says Suh. However, when researchers also account for emissions that are generated in supporting activities such as the manufacture of equipment and supplies for service industries, some of them don't look so green.

For example, the retail trade—everything from large department stores on New York City's Fifth Avenue to small shops on any town's Main Street—boosts greenhouse gases annually in amounts that warm Earth as much as 326.8 million tons of carbon dioxide would. That retail-trade estimate includes the emissions generated in the construction of stores, the manufacture of goods to be sold, and their shipment to the retailer or customer, Suh notes. The total represents 5.4 percent of the nation's planet-warming emissions.

Similarly, restaurants, bars, and similar establishments each year contribute 5.0 percent. For comparison, the manufacture and operation of motor vehicles is responsible for 5.1 percent. However, greenhouse-gas emission from inefficiencies during generation of electricity and construction of those facilities is a whopping 16.2 percent.

When the emissions from supply manufacture and transport are included, hospitals account for 4.4 percent of U.S. greenhouse-gas emissions, Suh estimates. Real estate services contribute 2.1 percent, which is more than that generated by air transportation. Doctors' and dentists' offices are responsible for just under 1 percent, slightly exceeding that generated by trucking and courier services.

Suh describes his analysis in the Nov. 1 *Environmental Science & Technology*.

"People have forgotten that services rely on a background of physical products," says Reid J. Lifset, an industrial ecologist at Yale University. The type of analysis that Suh has conducted is "straightforward yet often neglected," he notes.

Suh's analysis may better apportion the responsibility for greenhouse gas emissions among various industries, says Brad Allenby, an industrial ecologist at Arizona State University in Tempe. However, it doesn't account for reductions in greenhouse-gas emissions that may come from opportunities, such as telecommuting, that a service-based economy offers.

"It's often tempting to look at a product [or service] and claim it's green," says Lester B. Lave, an economist at Carnegie Mellon University in Pittsburgh. "But what looks good for you may not be good for the Earth overall." —S. PERKINS

Sick and Tired

Tracking paths to chronic fatigue

Stressful experiences and a genetic predisposition toward emotional turmoil contribute to some cases of chronic fatigue syndrome, two new studies indicate.

The investigations, published in the November *Archives of General Psychiatry*, add to growing evidence that several varieties of chronic fatigue syndrome (CFS) occur, each with distinct causes.

CFS affects roughly 800,000 people in the United States. It's characterized by disabling fatigue lasting 6 months or more and at least four of eight other symptoms: muscle pain, joint pain, memory or concentration loss, unusual fatigue after exercise, unrefreshing sleep, tender lymph nodes, headaches, and sore throat.

In the first of the new studies, psychologist Christine Heim of Emory University School of Medicine in Atlanta and her coworkers found that adults with CFS report a greater number of traumatic circumstances in their childhoods than other adults do.

From a representative sample of Wichita, Kan., residents, the researchers identified 43 individuals with CFS and 60 others with no fatigue problems. Two-thirds of the CFS group reported childhood experiences of emotional, physical, or sexual abuse or emotional or physical neglect. Only one-third of the nonfatigued group reported such traumas.

CFS rates were highest for individuals who cited more than one type of childhood trauma and for those who endured especially severe ordeals, Heim's team says. Depression, anxiety, and post-traumatic stress disorder also appeared frequently in participants with childhood traumas.

In some people, profound stress during childhood impairs the brain's responses to new challenges, the researchers propose. This process sets the stage for a number of ailments, including CFS, in their view.

The second new study, led by epidemiologist Nancy L. Pedersen of the Karolinska Institute in Stockholm, included 19,192 Swedish twins born between 1935 and 1958 and interviewed from 1998 to 2002. In that population of identical and fraternal twins, 447 individuals had CFS and another 1,120 reported disabling fatigue without four of the other symptoms.

Nearly one-fourth of these fatigue-troubled participants described their daily lives as "very stress filled," compared with 13 percent of the others. High stress levels contributed to CFS and persistent fatigue independently of each twin's genetic makeup, the scientists found.

The team also considered a personality tendency to experience emotional distress. Twins who scored high on this measure displayed a higher CFS rate than those with low scores did. The study revealed that genetic influences on this trait promote "chronic fatigue–like illness," the researchers speculate.

Since many adults with CFS in the new studies didn't report childhood traumas or severe daily stress, the new findings don't support the controversial notion that CFS is a by-product of depression, comments psychologist Leonard A. Jason of DePaul University in Chicago.

In a related study described in the Sept. 16 *British Medical Journal*, a team led by psychiatrist Ian Hickie of Sydney (Australia) University reports that 29 of 253 people who became infected with any of three viruses, including Epstein-Barr virus, developed CFS soon afterward. Preexisting stress or psychological ailments played no role in these CFS cases, the authors say.

Several genes that contribute to the body's stress-response system have also been linked to CFS. —B. BOWER

STATS

800,000

Estimated number of people in the United States with chronic fatigue syndrome

See How They See

Immature cells boost vision in night-blind mice

Transplanted retinal cells can restore some vision in mice with degenerative eye disease, experiments show. The new findings could point the way toward treatments for several forms of progressive blindness, including macular degeneration, which affects an estimated 6 million people nationwide.

For years, researchers have aimed to transplant stem cells to replace light-sensitive rod cells that had degenerated. Rods provide vision in near darkness and are usually the first photoreceptor cells to die in blindness.

Animal experiments, however, have shown limited success, in part because stem cells tend not to develop into rods after transplantation. Mature rod cells, by contrast, haven't integrated well with existing tissues.

In the new study, researchers transplanted retinal cells from fetal mice, newborns, or adults to mice that, because of genetic defects, had lost their rods and thus their night vision.

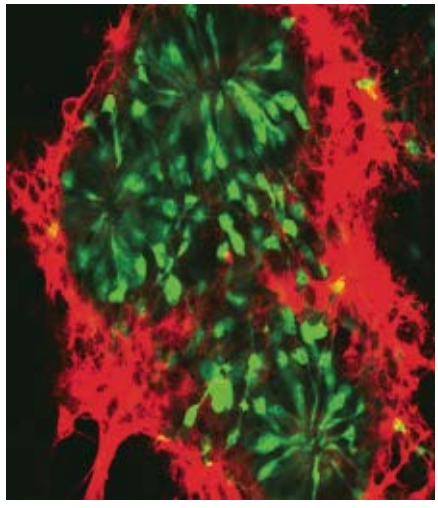
Some retinal cells from the newborns became functioning rods after transplantation. By contrast, only a few of the cells from the fetal mice and none of those from the adults became functional.

To determine whether transplanted cells were working, the scientists exposed the recipient animals to light. Pupil dilation in response to low light demonstrated that the eye's neural circuits were intact in the majority of the animals that received immature photoreceptor cells, reports retinal surgeon and study coauthor Robert E. MacLaren of Moorfields Eye Hospital in London. Electrical impulses recorded in the visual area of the brain also indicated that the transplants had restored some vision.

The developmental stage of the transplanted cells was the crucial factor, MacLaren, neurobiologist Anand Swaroop of the University of Michigan in Ann Arbor, and their colleagues conclude in the Nov. 9 *Nature*.

The new study may be the first to systematically compare nervous system cells transplanted at different stages of maturity, says Thomas A. Reh, a retinal-developmental biologist at the University of Washington in Seattle. Similar rigor could aid researchers seeking to transplant brain cells into people with Parkinson's disease and other neurological problems, he says.

For vision repair, Reh adds, a surprising



SIGHT LINE **These rod photoreceptor cells, shown in green, are at the optimal developmental stage for transplantation into blind mice. Other cells appear red.**

SWAROOP LAB./UNIV. MICHIGAN KELLOGG EYE CENTER

aspect of the new report is that cells well on the path to becoming rods—rather than stem cells with more developmental options—appear to be the most promising transplant candidates.

The maturity of the successfully transplanted mouse cells corresponds to that of human retinal cells late in the first trimester of pregnancy, Swaroop says.

Says MacLaren, "At the moment, we simply don't have a source of the immature photoreceptor cells."

However, scientists might someday convert stem cells from either adults or embryos into cells that would succeed as transplants. Last year, Reh and his team reported coaxing embryonic stem cells to resemble immature rod cells.

Using those ripened stem cells, the team is now attempting to replicate MacLaren's results. The experiments could lead to trials that use embryonic stem cells in people with progressive blindness. —B. HARDER

Hot, Hot, Hot

Peppers and spiders reach same pain receptor

The burn of hot peppers and the searing pain of a spider bite may have a common cause. New research suggests that molecules in hot peppers and in a certain spider's venom target the same receptor on nerve cells.

Several years ago, scientists identified a channel on neurons that's opened by capsaicin, the molecule responsible for peppers' burn. Follow-up research showed that this channel is a member of a family of cell-surface receptors that sense both chemicals and temperature. When these channels are activated, ions flood into nerve cells and cause them to fire.

Although scientists have already studied components of spider venom that cause shock, paralysis, and death, little is known about the molecules that cause the pain. David Julius of the University of California, San Francisco and his colleagues wondered whether pain-inducing venom ingredients might activate the dual-purpose cell-surface channels.

The team purchased venoms collected from a variety of spider, scorpion, and snail species known to deliver painful bites. The researchers diluted the venoms and added them to dishes containing human-kidney cells that had been genetically altered to carry various types of channels.

Only the venom of one West Indian tarantula species, *Psalmopoeus cambridgei*, sent a flood of ions into cells that sported the same receptor that's sent by capsaicin. When the scientists broke down that venom, they identified three component molecules responsible for the rush of ions.

To confirm that these molecules opened the capsaicin-responsive channel, the researchers added each compound separately to dishes containing nerve cells from normal mice or from mice engineered to be missing just that channel. The team found that ions entered only the cells from normal mice. Furthermore, only animals with the capsaicin-responsive channel appeared to feel pain in response to any of the molecules.

The team reports its results in the Nov. 9 *Nature*.

Julius notes that because triggering the receptor produces such strong pain sensations, it's not surprising that organisms as distantly related as pepper plants and tarantulas use the same defensive mechanism.

"Different organisms have figured out how to tap this site as a way of telling predators, 'You won't be comfortable if you mess with me,'" he says.

Michael Caterina, who studies this family of dual-purpose cell-surface channels at Johns Hopkins University School of Medicine in Baltimore, notes that spider venom could eventually become a powerful tool for researchers to use in investigating channels active in several types of chronic pain.

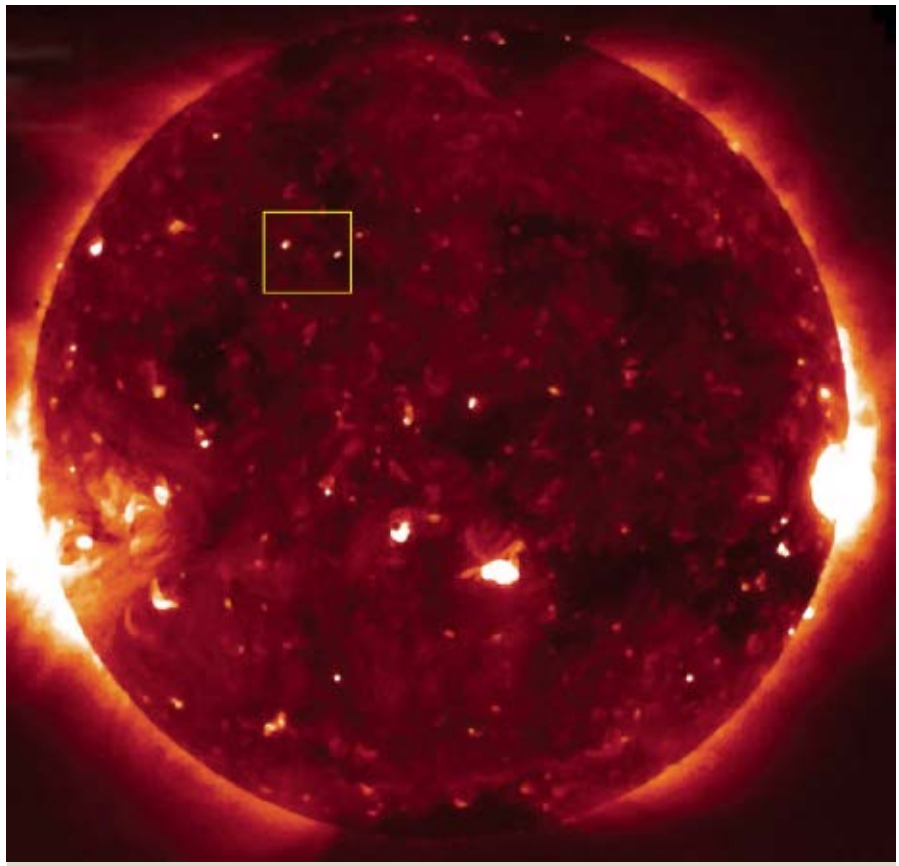
"Anything that helps us understand how these channels are activated will facilitate [development of] drugs that block these channels," he says. —C. BROWNLEE

Birds Beware

Several veterinary drugs may kill scavengers

Scavenging birds worldwide could be at risk of accidental poisoning from carcasses of livestock that farmers had dosed with certain anti-inflammatory drugs, according to a survey of veterinarian records.

The work grows out of discoveries in the



JAXA

New eye on the sun

The recently launched Hinode spacecraft made this X-ray portrait of several-million-degree gas in the sun's outer atmosphere on Oct. 28. The test image reveals that features known as X-ray bright points (two examples are in box) are simple magnetic loops entraining hot gas. Astronomers will monitor the bright points to find out why the sun's atmosphere, or corona, is so much hotter than its interior. In addition to an X-ray telescope, Hinode carries a visible-light telescope to study the sun's surface and a magnetograph to monitor magnetic fields associated with sunspots. The craft also has an extreme-ultraviolet imaging spectrometer to track hot gas in the corona (*SN: 8/19/06, p. 120*). After further testing, Hinode, which means sunrise in Japanese, will begin its 3-year mission in December. —R. COWEN

past 2 years that several *Gyps* vulture species have almost vanished from India and Pakistan because residues of the anti-inflammatory drug diclofenac in dead farm animals ruin the kidneys of the scavenger birds (*SN: 2/4/06, p. 70*).

To estimate sensitivities to diclofenac and related drugs, researchers combed veterinary records around the globe for unexpected deaths of captive birds treated with nonsteroidal anti-inflammatory drugs (NSAIDs). Suspicious deaths turned up in 11 species, including 7 not closely related to the Asian vultures, says Richard Cuthbert of the Royal Society for the Protection of Birds in Sandy, England.

The study associated diclofenac with more than two dozen bird deaths and carprofen and flunixin with several deaths each, Cuthbert and his colleagues report in an analysis now online for *Biology Letters*.

Because birds scavenge dead farm animals, "any NSAID that is used without testing [on birds] is a real cause of concern," Cuthbert says.

The survey team collected records of NSAID treatment for nearly 900 birds in 79 species, mostly scavengers. The researchers looked for fatalities in cases such as minor surgery, where "the bird shouldn't have died," says Cuthbert.

Out of 40 birds treated with carprofen, 5 died, as did 7 out of 24 treated with flunixin. Deaths of two other birds—a Eurasian black vulture, which was the only bird in the study treated with ibuprofen, and one of Africa's lappet-faced vultures, the only bird that got phenylbutazone—caused the researchers to call for more investigation. In contrast, meloxicam seemed safe for birds.

The deaths documented in birds receiving carprofen and flunixin included three black vultures and a spoonbill from Eurasia, a Marabou stork from Africa, and, from the Americas, two Harris's hawks, a red-legged seriema, and two northern saw-whet owls.

Cuthbert worries about vultures that dine where conservationists in Africa set out dead livestock. Vets there use several NSAIDs that the new study links to bird deaths.

The world presents a vast patchwork of NSAID usage. India and Nepal recently banned the importation and manufacture of diclofenac. However, vets in South America use it, notes one of the paper coauthors, captive-raptor specialist Jemima Parry-Jones of the National Aviary in Pittsburgh.

U.S. vets don't administer diclofenac to any farm animals, says pathologist Carol U. Meteyer of the National Wildlife Health Center in Madison, Wis. She says that the center hasn't noted fatal kidney damage in any scavenging birds. However, she welcomes the new report as a good indication that farm veterinarians need to be very careful about pharmaceuticals. —S. MILIUS

The Little Chill

Tiny wind generator to cool microchip hot spots

Technologists cramming more and more transistors onto microchips face a common problem: too much heat. To make computers chill, manufacturers typically outfit hot chips with heat sinks, whose fins release heat into a stream of air.

Now, a team of university and industrial engineers has created a prototype, microscale air pump that they say could be fabricated with the techniques used to mass-produce microelectronic components such as transistors. The pump would generate cooling breezes right where the heat is being created.

The new work shows for the first time that "the principle works at the scale and the rate needed," says electrical engineer Alexander V. Mamishev of the University of Washington in Seattle, who leads the group that came up with the device.

To make the pump, the researchers used a beam of high-energy ions to carve a slender needle jutting out from the edge of a piece of silicon. When mounted just above a pad of electrically conductive foam and energized with thousands of volts, the needle produces a powerful electric field that ionizes the surrounding air, says University of Washington team member Nels E. Jewell-Larsen.

Propelled by the electric field, ionized gas molecules rush toward the pad below. On their way, they run into gas molecules and push them along. The minuscule wind, which reaches about 25 kilometers per hour, cools the pad's surface, Jewell-Larsen says.

In recent tests of the prototype micropump mounted above a pad heated to roughly 50°C, the microwind cooled a fingerprint-size patch of the pad by as much as 25°C, Chi-Peng Hsu of the University of Washington reported on Tuesday in Chicago at a mechanical engineering conference.

The tests show that the pump's underlying principle, "which has been known for decades, can be applied at the chip level," comments thermal engineer David A. Rosato of the Canonsburg, Pa., company Ansys. Still, some further cooling mechanism must remove the heat picked up by the microwind and generated by the pump itself, he cautions.

Researchers at Purdue University in West Lafayette, Ind., have developed related technology also aimed at cooling chips by means of ion-propelled winds and have formed a company to commercialize the technology.

The high voltages employed by the Washington group may require precautions, such as isolating the microchip's sensitive electronic components from the pump. However, Mamishev notes that the power levels in the cooling devices don't pose a safety hazard.

Before the new cooling gadget begins serving on chips, it might show up between heat-sink fins, where air tends to stagnate, says Jewell-Larsen.

The Belmont, Mass.–based company Kronos Air Technologies plans to commercialize the Washington team's micropumps, he adds. The researchers have also been working with chip maker Intel Corp. on the technology. (Intel sponsors some of the educational programs of Science Service, the publisher of *Science News*.) —P. WEISS

ISTOCKPHOTO

SENSITIVE The Harris's hawk, native to the Americas, is one of the species whose members have died after veterinary treatment with anti-inflammatory drugs.

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THE ANTIBIOTIC VITAMIN

Deficiency in vitamin D may predispose people to infection

BY JANET RALOFF

In April 2005, a virulent strain of influenza hit a maximum-security forensic psychiatric hospital for men that's midway between San Francisco and Los Angeles. John J. Cannell, a psychiatrist there, observed with increasing curiosity as one infected ward after another was quarantined to limit the outbreak. Although 10 percent of the facility's 1,200 patients ultimately developed the flu's fever and debilitating muscle aches, none did in the ward that he supervised.

"First, the ward below mine was quarantined, then the wards on my right, left, and across the hall," Cannell recalls. However, although the 32 men on his ward at Atascadero (Calif.) State Hospital had mingled with patients from infected wards before their quarantine, none developed the illness.

Cannell's ward was the only heavily exposed ward left unaffected. Was it by mere chance, Cannell wondered, that his patients dodged the sickness?

A few months later, Cannell ran across a possible answer in the scientific literature. In the July 2005 *FASEB Journal*, Adrian F. Gombart of the University of California, Los Angeles (UCLA) and his colleagues reported that vitamin D boosts production in white blood cells of one of the antimicrobial compounds that defends the body against germs.

Immediately, Cannell says, the proverbial lightbulb went on in his head: Maybe the high doses of vitamin D that he had been prescribing to virtually all the men on his ward had boosted their natural arsenal of the antimicrobial, called cathelicidin, and protected them from flu. Cannell had been administering the vitamin D because his patients, like many other people in the industrial world, had shown a deficiency.

The *FASEB Journal* article also triggered Cannell's recollection that children with rickets, a hallmark of vitamin D deficiency, tend to experience more infections than do kids without the bone disease. He shared his flu data with some well-known vitamin D researchers, and they urged him to investigate further.

On the basis of more than 100 articles that he collected, Cannell and seven other researchers now propose that vitamin D deficiency may underlie a vulnerability to infections by the microbes that cathelicidin targets. These include bacteria, viruses, and fungi, the group notes in a report available online for an upcoming *Epidemiology and Infection*.

This is only a hypothesis, "but a very credible one" that deserves testing, says immunologist Michael Zasloff of Georgetown University in Washington, D.C.

Behind the hypothesis are recent studies that link vitamin D intake to revved-up cathelicidin production. These investigations point to an infection-fighting role for vitamin D, which is produced in skin exposed to sunlight but is present in few foods.

A study published earlier this year that investigated the relationship between vitamin D and susceptibility to tuberculosis also bolsters the idea proposed by Cannell's team. Scientists have already planned a handful of clinical trials to evaluate the antimicrobial benefits of vitamin D supplementation.

Zasloff argues that if studies support the hypothesis, "we can imagine one day treating infections not by giving somebody a drug, but by giving them safe and simple substances—like a vitamin."

WINTER WOES — Cold-weather wear and the sun's angle in the winter sky limit how much ultraviolet light reaches the skin. This can add up to a deficiency in production of vitamin D, which might explain why respiratory infections are common and severe in winter.

ISTOCKPHOTO

INNATE PROTECTION Legions of germs come into contact with our bodies every day. Each microbe seeks a host in which it can multiply. Most would-be invaders, however, don't succeed; if not barred entry outright, they're destroyed by cellular recruits called up to participate in local immune militias.

Scientists hadn't been sure what serves as the call to arms for these immune cells and what triggers the production of their antibiotic arsenal, which includes several chemical weapons.

Over the past 5 years, a spate of studies began to shed light on the rollout of one of those munitions—cathelicidin. Dermatologist and immunologist Richard L. Gallo of the University of California, San Diego, a coauthor of many of these studies, explains that cathelicidin "targets the bad guys." It kills invaders by punching holes in the external membrane of a microbe, permitting its innards to leak out.

Molecular geneticist John H. White of McGill University in Montreal and his colleagues were the first to observe that cathelicidin production is ramped up by vitamin D—or, more specifically, by the hormone 1,25-D, the vitamin's active form (*SN: 10/9/04, p. 232*). Through a cascade of events, vitamin D transforms into a compound, called a prehormone, that circulates in blood and then is converted locally, as needed, into 1,25-D.

In the nucleus of cells, 1,25-D binds to short sequences of DNA. Known as response elements, these sequences switch on the activ-

(continued on page 317)

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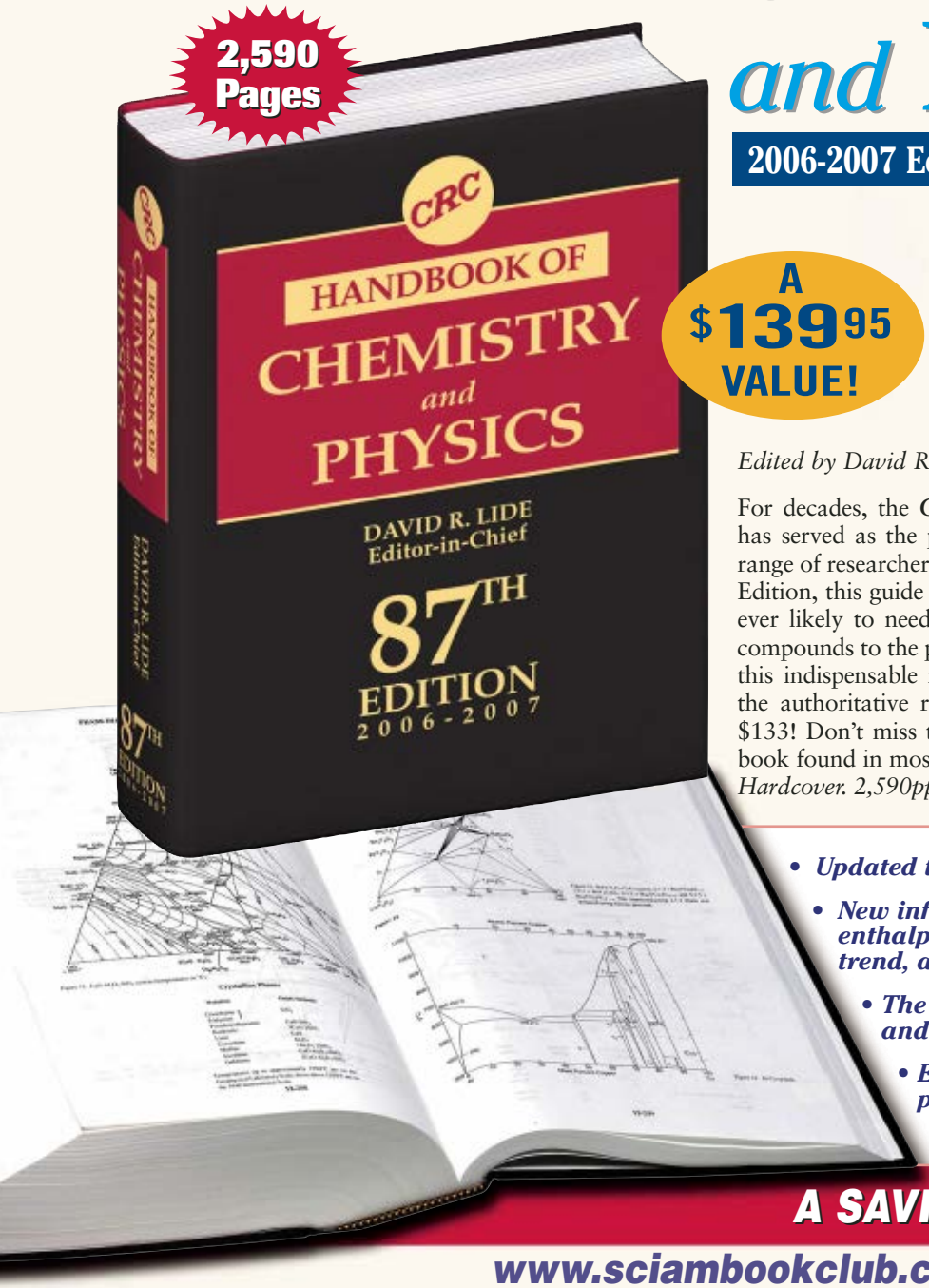

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Science News 11/11/06 LSC0611PT-01/94

BRAVE OLD WORLD

The debate over rewilding North America with ancient animals

BY ERIC JAFFE

For the first time in several thousand years, a lion's roar reverberates through the Grand Canyon. California condors descend into that chasm as though sliding down a spiral staircase. Bolson tortoises creep through spiky yucca plants in the Chihuahuan Desert in New Mexico. Nearby, camels and elephants munch woody shrubs. A cheetah, chasing a pronghorn toward a deep ravine, proves that you can in fact come home again.

If one group of conservation biologists has its way, this is how the western United States could look within the next century: filled with megafauna, including carnivores and herbivores imported from Africa, Asia, and other parts of the world. These animals would repopulate the area where they lived until about 13,000 years ago, when the arrival of people in the region caused them to go extinct.

The plan, called Pleistocene rewilding, suggests reintroducing into Arizona, the Great Plains, and elsewhere various species—such as Bactrian camels, peregrine falcons, and Old World cheetahs—that were once native to North America. If all goes well, these species could reestablish ecosystems that thrived in ancient times, before people began affecting the environment.

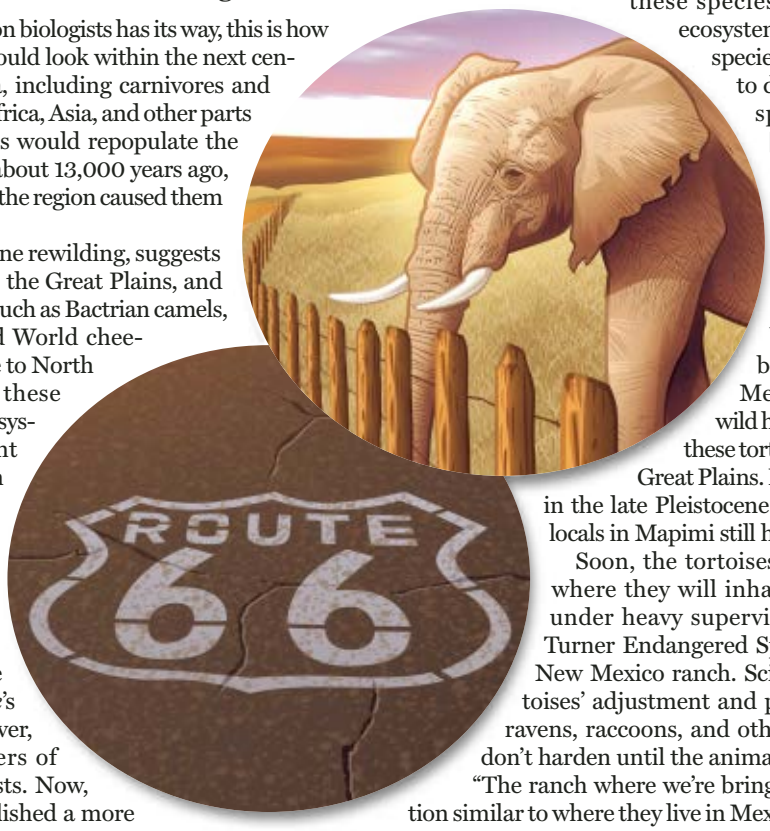
When first proposed as a brief commentary in the Aug. 18, 2005 *Nature*, the idea tickled the imaginations of many journalists. It even earned mention in the *New York Times Magazine*'s "Year in Ideas" issue. However, it also aroused the tempers of some conservation biologists. Now, the same authors have published a more comprehensive follow-up, which appears in the November *American Naturalist*. The new version presents some compelling reasons to take the plan seriously: Pleistocene rewilding could restore lush ecosystems, curb Lyme disease, and provide a bold alternative to failing models of species conservation around the world.

"We might partially restore these lost taxa and the ecological functions that go with them," says coauthor Harry Greene of Cornell University. "One could imagine, 100 years from now, the American Great Plains turned into an ecological reserve."

But another group of researchers counters that vision. In the October *Biological Conservation*, a team led by Dustin R. Rubenstein, now at the University of California, Berkeley, challenges the tenets of Pleistocene rewilding, calling it only "slightly less sensational" than Michael Crichton's 1990 novel *Jurassic Park* (Knopf).

Rewilding could disrupt modern ecosystems just as easily as it could restore historic ones, Rubenstein argues. Once brought from Africa and Asia, genetic relatives of former inhabitants might behave differently than the original species did. A better plan would be to preserve these animals, many of which are endangered, in their native habitats, Rubenstein and his colleagues propose.

"You're putting back species that might be genetically different, and in most cases are, into ecosystems where there haven't been these species in 10,000 years, and the ecosystems have evolved without the species," says Rubenstein. "We need to do something to preserve the species on this planet, but [Pleistocene rewilding] is so bold that it requires different perspectives."



DEAN MACADAM

BORN TO REWILD At a New Mexico ranch, Pleistocene rewilding has already begun. Bolson tortoises have been moved there from Mapimi, Mexico—their only remaining wild habitat. During the Pleistocene, these tortoises lived in what's now these Great Plains. People who entered the region in the late Pleistocene preyed on the tortoises, and locals in Mapimi still hunt the 100-pound animals.

Soon, the tortoises will be moved to Arizona, where they will inhabit two 8.5-acre enclosures under heavy supervision, says Joe Truett of the Turner Endangered Species Fund, which runs the New Mexico ranch. Scientists will monitor the tortoises' adjustment and protect young tortoises from ravens, raccoons, and other predators. Tortoise shells don't harden until the animals are about 6 years old.

"The ranch where we're bringing the tortoises has vegetation similar to where they live in Mexico, and the climate's not that much different, either," says Truett. "If they survive the first winter, they'll be fine."

The Bolson project emerged from a meeting held at the New Mexico ranch in September 2004. Ecologist Josh Donlan of Cornell, Greene, and 10 other conservation biologists have summarized that meeting and their plan for Pleistocene rewilding in *Nature* last year and in the upcoming *American Naturalist* article.

Most conservation and rewilding efforts focus on animals that went extinct after Columbus came to America, but that approach is flawed, argue Donlan and his coauthors in those publications. It's more logical to use the Pleistocene as a benchmark for conser-

vation, Donlan says. That's when people moved into North America across the Bering land bridge that connects Asia and Alaska. Once they arrived, people began altering habitats and exploiting natural resources. These activities eventually eliminated many species of megafauna.

Archaeological evidence showing that people directly caused these extinctions is scarce, says Paul Martin of the University of Arizona in Tucson, who is part of Donlan's team. But fossilized bones and dung, as well as remains from hunters who lived during the late Pleistocene, suggest that the disappearance of megafauna in what's now the southwest United States coincided with the arrival of people, he says.

"It's false logic to insist that if people caused these extinctions, there ought to be abundant kill-sites," says Martin. "People arrive and the extinctions occur. The field evidence is frustrating, but I think it's more than sufficient as far as ecologists are concerned."

The loss of large animals set off a chain reaction of ecological changes, Donlan's team states. It's not possible to know exactly what changes occurred, but dwindling populations of contemporary animals can show how the process might have played out.

Consider, for example, North American gray wolves. As the wolf population diminished in the 20th century, their herbivorous prey such as deer and cattle flourished, reducing the prevalence of aspen trees and other plant species throughout North America.

Reintroducing top predators from the Pleistocene, such as lions and cheetahs, could keep herbivores in check and restore lush forest structures and biodiversity that once existed in the western United States, Donlan's team argues.

This trickle-down effect on vegetation is a reasonable idea, says ecologist Deborah Letourneau of the University of California, Santa Cruz. Top predators reduce the dominance of any particular herbivore, which creates a habitat amenable to plant diversity, she explains.

If proponents of Pleistocene rewilding choose areas where a good amount of plant diversity already exists, reintroducing megafauna could shift vegetation back toward its previous state, she says. "In a desert climate, you won't get a rain forest," says Letourneau. "But it might be surprising what you do get, in terms of diversity and lushness." Moreover, she adds, the opposite technique of introducing only vegetation—a "bottom-up" approach—would likely promote less biodiversity than rewilding fauna would.

Some reintroduced megafauna could have a more direct benefit on human health. For example, forest animals that prey on deer might curb rising rates of Lyme disease. The reintroduced animals might deter deer from entering deep into the forests and picking up the ticks that convey Lyme disease to people.

Rewilding can begin right away, says Donlan, though that doesn't mean that he'll soon drive into the Colorado River Canyon with a truck full of cheetahs and pop the tailgate. The plan should progress in phases and be continually evaluated.

Start with a few Bolson tortoises heavily monitored on a ranch. Perhaps next, larger carnivores can be supervised in fenced-in areas. Down the line, the animals might coexist in an ecological history park, Donlan says, "where there's an economic revenue and science going on."

SLOW TRAIN — Conservationists are moving Bolson tortoises from Mexico to New Mexico, where the species lived during the Pleistocene.

MYLES TRAPHAGEN

CRAZY LIKE A RED FOX In the 1850s, the European red fox was deliberately brought to Australia for recreational hunting. Since then, it has spread throughout the country, reduced bird and green turtle populations, and ravaged farmlands. Now, hunting a red fox is no longer recreational: Bounties are often placed on the species—to no avail. The animals remain largely uncontrolled.

Plenty of animal introductions have ended just as poorly, says invasive-species expert Dan Simberloff of the University of Tennessee in Knoxville. Consider reindeer in South Georgia, sea lampreys in the Great Lakes, wild boars in Hawaii, or goats in the Galápagos.

"So many different things can go wrong," Simberloff says. "It's often said that bioinvasions are inherently unpredictable. That's an overstatement, but there are an awful lot of factors to consider."

Rubenstein's group wants to avoid unpredictable outcomes. It's exciting to think that Pleistocene rewilding could reestablish lost ecosystems, says Paul Sherman of Cornell, one of Rubenstein's coauthors, but reintroducing megafauna could just as easily disrupt contemporary ones.

For example, Donlan's team argues that there haven't been any plant extinctions in North America since the Pleistocene, which implies that the tools for a reestablished ecosystem are in place. But even if Pleistocene vegetation remains extant, these plant species could be different chemically from their ancient forms or dispersed in different regions, Sherman says.

"It's not clear that you can argue that nothing's changed," he says. "Nobody really knows what things were like back then."

Moreover, he says, plant life in the western United States differs significantly from African vegetation.

The only way to ensure that an ecosystem or an animal species hasn't evolved dramatically is to introduce fauna that had gone extinct in a region in the past couple hundred years, says Sherman. This traditional type of rewilding, which is a common conservation technique, differs greatly from Pleistocene rewilding, he says.

In the traditional model, reintroducing peregrine falcons, for example, makes sense because the animal just recently became threatened by heightened amounts of the pesticide DDT in the environment, says Sherman. The Bolson tortoise passes muster, too, because it's only making a 600-mile trip to an environment similar to its current habitat.

Animals shipped from Africa and Asia, however, could complicate the situation. "Traditional rewilding has the advantage of working with the exact species that has been lost," says Ross Barnett of the University of Oxford in England, who studies cat evolution. "With Pleistocene rewilding, you are talking about taking analogous species, which have different ecological requirements, into an environment which they have not evolved to cope with."

For example, says Barnett, the closest genetic relative to the American cheetah is actually a puma—not an African cheetah. "If you bring in African cheetahs, aesthetically it seems like the same thing," he says, "but they're fundamentally not the same."

Given the uncertainties surrounding Pleistocene rewilding and the limited money that goes toward species conservation, efforts should be focused on traditional rewilding, Rubenstein says.

"We don't believe in moving species to habitats where they never existed," says Rubenstein. "We take the line that it may be better to try to preserve species facing extinction in their native habitats."

Donlan and Greene insist that they don't want Pleistocene rewilding to become a priority over on-the-ground conservation in Africa or Asia. Rather, rewilding is a chance to redress the mistaken

elimination of these species and to improve biodiversity in the process.

"We're already playing God," says Donlan. "There isn't a single meter of this planet that isn't affected directly by humans. We're affecting biodiversity either by default or by design. [People] need to decide what kind of world they want to live in and how much biodiversity they're willing to coexist with."

PLEISTOCENE PARK As unique as Pleistocene rewilding sounds, Donlan's team isn't the only one to propose such a plan. Since the early 1990s, Sergey Zimov of the Northeast Science Station in Russia has discussed his version of rewilding, called Pleistocene Park.

Zimov's project takes place in the northern Siberian region of Yacutia, but his main goal is similar to that of Donlan's group: reverse a series of negative ecological changes by reintroducing Pleistocene megafauna.

At the current rate of climate change, carbon trapped in Yacutian soil will be released into the atmosphere as carbon dioxide, accelerating global warming, Zimov said in the May 5, 2005 *Science*. The reintroduction of megaherbivores—particularly bison from Canada—could return this terrain to a grassy ecosystem that retains carbon. So far, Zimov has introduced moose, musk oxen, and reindeer, which are being used in experiments.

A favorable environmental change in Yacutia could take place in as few as 5 to 10 years, says ecologist F. Stuart Chapin of the University of Alaska in Fairbanks, who has worked with Zimov on Pleistocene Park. The trick is not only having a high density of animals but also giving them enough room to roam comfortably.

"Human hunting contributed to the disappearance of megafauna and the difference in ecosystems that appear today," says Chapin. "Should we sit back and accept that, or are there other things to consider doing? It is a good time to open this debate and think about things in that broader context."

In the end, the debate over rewilding might elicit sympathy from its challengers, says conservation biologist Martin Schlaepfer of the University of Texas at Austin.

"It will have a positive effect of getting the public, including conservation biologists, to ask themselves what we want our wilderness to look like in the future," Schlaepfer says. "Right or wrong, Pleistocene rewilding has brought that to the forefront, and that's a very positive thing."

Even the scientists who voiced a concern about rewilding when interviewed for this article also gave reasons to try it. Simberloff likes the idea's vision and says that even if the animals did become problematic, they could be easily controlled through hunting.

Barnett says that the African lion could do well in North America, as long as it had enough space.

Rubenstein and Sherman also admit that Pleistocene rewilding is an exciting concept. The aim of their paper wasn't to silence rewilding but to incite scientific discussion, Sherman says.

"It's not the usual scientific debate," he says. "It's about a future direction of conservation biology, so you take it in the spirit of trying to figure out what's the best direction, and you go forward from there." ■

DEAN MACADAM

OF NOTE

FOOD & NUTRITION

Curry may counter cognitive decline

A chemical found in turmeric, an ingredient in curry, may prevent cognitive impairment, a study of Singaporeans suggests.

The chemical, called curcumin, has anti-inflammatory and anticancer properties, past research in animals had suggested (*SN: 12/8/01, p. 362*). To explore its potential effects in people, medical researchers in Singapore analyzed a database of some 1,000 elderly, mentally sound adults living in that ethnically diverse city-state in Southeast Asia.

The database, originally collected for diet and health studies, provided a measure of cognitive function for each volunteer and information on, among other things, ethnicity and curry consumption.

Compared with people who reported rare or no curry consumption, those who said they ate curry often or occasionally had slightly higher scores on the cognitive-function tests. Researchers led by Tze-Pin Ng of the National University of Singapore report the finding in the Nov. 1 *American Journal of Epidemiology*.

Once the researchers took education and other factors into account, the results were strongest in people of Indian ethnicity. Indian curry recipes rely heavily on turmeric. —B.H.

NEUROLOGY

Dementia warning

Aging populations of the world, beware: Disorders of memory and thinking will become much more common with more people living into their 90s, according to a long-term study in England and Wales.

Even people who reach age 80 free of Alzheimer's disease or other dementias stand a good chance of later developing some form of mental incapacity, says a team led by epidemiologist Carol Brayne of the University of Cambridge in England.

From 1990 to 1992, Brayne and her coworkers assessed the mental status of 13,004 people age 65 and older at six rural or urban sites. All were receiving medical care unrelated to mental problems. Of 12,387 individuals who died by 2004, there were 2,566 who had developed dementia. A comparable number had developed severe memory and thinking impairments that fell short of dementia.

Dementia rates rose sharply as people got older, the researchers report in the October *PLoS Medicine*. Prevalence rates ranged from 6 percent for those who died between ages 65 and 69 to 58 percent for those who died at age 95 or older.

Overall, dementia affected 38 percent of women and 22 percent of men, a significant disparity even after accounting for the tendency of women to live longer than men, the scientists say. —B.B.

(continued from page 312)

ity of adjacent genes. "We wanted to find out what genes were next to the vitamin D response elements," White recalls.

Two of these response elements proved to be neighbors of genes that make antimicrobial peptides, cathelicidin and beta-defensin 2, the researchers reported in 2004. When the researchers administered 1,25-D to a variety of cells, production of beta-defensin 2 increased "modestly," White told *Science News*. In contrast, he says, the gene for making cathelicidin "went boom! Its induction was very, very strong."

Almost a year later, while hunting for triggers for cathelicidin production, Gombart confirmed the McGill finding. His group had been administering various natural signaling agents to white blood cells, which the immune system sends out to vanquish germs.

In these cells, "nothing turned on the cathelicidin gene to any degree except vitamin D. And it really turned that gene on—just cranked it up," Gombart says. "I was completely surprised."

Independently, dermatologist Mona Ståhle of the Karolinska Institute in Stockholm reached a similar conclusion when she realized that both vitamin D and several antimicrobials, including cathelicidin, are produced in the skin. She says, "It just came to me—an intuitive thought—that maybe the sun, through vitamin D production, might help regulate the skin's antimicrobial response."

So, her team administered an ointment containing a drug mimic of 1,25-D to the skin of four healthy people. The salve hit "the jackpot, right away," Ståhle says. In the May 2005 *Journal of Investigative Dermatology*, her team reported that where the ointment had been applied, cathelicidin-gene activity skyrocketed as much as 100-fold. The team also found evidence of a localized increase in the concentration of cathelicidin.

TACKLING TB AND MORE Those studies, though suggestive, didn't reveal whether vitamin D directly reduced infection risk in people. Together with Gallo, microbial immunologist Robert Modlin of UCLA and his colleagues moved closer to that goal: They evaluated the vitamin's role in fending off the tuberculosis (TB) germ *Mycobacterium tuberculosis*.

This group, working independently of Gombart's team, had been focusing on macrophages, a type of white blood cell deployed by the immune system to gobble up and destroy germs. These defense cells have features, called toll-like receptors, that identify biochemical patterns characteristic of invading microbes. If the receptors sense an invader, they can trigger cathelicidin production.

Modlin's team showed that before making that antibiotic, those cells briefly boosted their production of vitamin D receptors and of an enzyme that converts the vitamin D prehormone into 1,25-D. However, the data suggested that significant concentrations of 1,25-D would develop only in the presence of the TB bacteria. This indicated that the microbe, and perhaps other germs, must be present for the enzyme to maximize its production of 1,25-D, Modlin says.

His group then tested whether people's blood concentrations of the prehormone are high enough to drive the production of germ-killing concentrations of cathelicidin. Black people, because of the sun-filtering effect of dark pigments in their skin, are far more likely than whites to be vitamin D deficient (*SN: 10/16/04, p. 248*). Furthermore, blacks tend to be more susceptible to TB than whites and to develop a more severe illness when infected.

The team collected blood serum from white people and from blacks. When the researchers added TB bacteria, macrophages in the serum from black participants produced 63 percent less cathelicidin—and were less likely to kill the germs—than were macrophages incubated in serum from whites.

The scientists then added vitamin D to the serum from blacks until concentrations of the prehormone matched those in the serum from whites. This boosted the macrophages' cathelicidin production and rates of TB-microbe killing to those seen when such cells were incubated in serum from whites. Modlin's group reported its findings in the March 24 *Science*.

The new data may explain the difference between blacks and whites in TB susceptibility. Modlin says, "We showed that serum from African American individuals did not support the production of the antibiotic by immune cells, until the serum received supplemental vitamin D."

"We're now planning to do a clinical trial and treat African Americans who are deficient with vitamin D to correct their serum levels [of the prehormone] and see if this will change their antimicrobial response," Modlin says.

> "We can imagine one day treating infections … by giving safe and simple substances—like a vitamin."
>
> — MICHAEL ZASLOFF, GEORGETOWN UNIVERSITY

Gallo is also planning a new trial. His group will compare the effectiveness of supplemental vitamin D in elevating cathelicidin concentrations when administered as oral supplements or as a skin treatment.

The team expects to see the biggest benefit in skin wounds. However, Gallo predicts that even healthy skin will exhibit somewhat elevated antimicrobial concentrations, signaling an improved resistance to infection.

Sun exposure—in moderation—might also prove therapeutic, Ståhle's team suggested in the November 2005 *Journal of Investigative Dermatology*. The scientists showed that in eight fair-skinned people, a single dose of ultraviolet-B radiation—just enough to evoke some skin reddening the next day—activated the vitamin D receptor and the cathelicidin gene in the exposed skin.

Ståhle is now beginning a trial of people with skin infections. A drug analog of 1,25-D will be applied to see whether it speeds wound healing.

FLU TOO? Many other findings also suggested to Cannell's team that flu vulnerability might be tempered by adequate vitamin D intake. The researchers have marshaled data, gleaned from 120 or so reports over the past 70 years, suggesting a link between vitamin D and resistance to infections.

For instance, the researchers point to studies showing that in winter, colds, flu, and other respiratory diseases are more common and more likely to be deadly than they are in summer. During winter, ultraviolet-light exposure tends to be low because people spend more time indoors and the atmosphere filters out more of the sun's rays, especially at mid and high latitudes.

Cannell's group cites a 1997 study showing that the rate of pneumonia in Ethiopian children with rickets, and therefore a likely vitamin D deficiency, was 13 times as high as in children without that disease. The researchers also point to five studies since the 1930s that have linked reduced risks of infectious disease to dietary supplementation with cod liver oil, a rich source of vitamin D.

Although the arguments in the paper by Cannell's group "are provocative," White says, "I find them believable."

So does Gallo. "There are many microbes out there that rarely-to-never cause disease in immunocompetent individuals. It's not because the microbes don't choose to infect us," he notes. "It's because the body's immune defense against the microbes is sufficient to control their proliferation."

It's possible, he says, that a shortfall in vitamin D might seriously compromise that defense.

Gombart's group is developing rodents in which vitamin D modulates cathelicidin.

Until such lab animals are available, vitamin D's impact—even on flu risk—"should be explored in clinical trials," Zasloff says, because the treatment poses little risk to people.

Moreover, he argues, the payoff from any positive finding "would be amazing. Imagine being able to block the spread of epidemic flu with appropriate doses of this vitamin." ■

PALEOGEOGRAPHY

Rodents tell a geologic tale

The discovery of previously unknown rodent species that lived in Chile millions of years ago suggests that mountains in the southern Andes first rose to significant heights at least 18 million years ago.

By measuring the proportions of radioactive isotopes in ash deposits, scientists can estimate the date but not the height of a particular volcanic eruption. Therefore, geologists haven't been able to determine when the southernmost portions of South America's Andes, as a whole, rose to their current heights, says Jill Wertheim, a paleontologist at the University of California, Santa Barbara. Fossils could provide the answer.

Wertheim and her colleagues studied fossils of small animals from a part of that area that's now hundreds of meters high. More than 20 million years ago, however, the region would have sat close to sea level along the Pacific coast, she notes.

In rocks laid down as sediments as early as 18 million years ago, the researchers found the fossils of 20 species of rodents that haven't been reported anywhere else in South America. "That's a large number of new species," says Wertheim, who notes that 10 of those species belong to new genera. While some of the species that suddenly appeared are related to the agoutis and spiny rats that live in South America today, others belong to extinct lineages, says Wertheim.

Geographic isolation by the growing mountains would be a likely explanation of rapid evolution of rodent species along the coast. So, the appearance of the new rodent species about 18 million years ago indicates that the southern Andes at that time became too tall for the animals to cross, Wertheim speculates. —S.P.

ICE AGE MAMMALS

DNA analysis reveals extinct type of wolf

Many species of large mammals went extinct when the last ice age ended about 12,000 years ago. But *Canis lupus*, the gray wolf, survived that wrenching period unscathed—or so scientists thought. New genetic analyses of the remains of gray wolves found in Alaska indicate, however, that a distinct subspecies of *C. lupus* disappeared at that time, possibly because of its dietary habits.

Blaire Van Valkenburgh of the University of California, Los Angeles and her colleagues conducted a genetic study of living gray wolves and also samples of mitochondrial DNA recovered from wolf bones found in Alaskan permafrost. The remains of those 21 animals ranged in age from 12,600 years to at least 47,000 years.

The team's analyses revealed 15 combinations of genetic variations in the Alaskan wolves that didn't match any of those in 126 modern gray wolves. "This was surprising, so then we looked at the bones," says Van Valkenburgh. They found that the ancient gray wolves had broader snouts, larger teeth, and deeper jaws than their living cousins.

Overall, the findings suggest that the ancient gray wolves belonged to a subspecies adapted to consume bones and carcasses more thoroughly than living wolves do.

The gray wolf subspecies might not have survived the end of the ice age because it depended on a steady supply of large carcasses, says Van Valkenburgh. As the populations of mammoths, mastodons, and other large mammals dwindled, the wolves' food supply would have disappeared, she notes. —S.P.

DINOSAUR BEHAVIOR

Society sans frills

Past studies suggest that horned dinosaurs such as *Triceratops* and their relatives, a group known as ceratopsians, lived in herds and used the frills on their skulls and other ornamentations to identify members of their own species, as did many other dinosaurs (*SN: 8/13/05, p. 103*). Now, the discovery of the fossils of several young dinosaurs in one small space suggests that an ancestor of ceratopsians exhibited social behavior millions of years before their group gained large distinctive decorations.

Researchers found the remains of six juvenile psittacosaurs in 128-million-year-old rocks in northern China. Those rocks appear to have been laid down quickly as a cementlike mixture of water, fresh volcanic ash, and eroded soil, says Paul Barrett of the Natural History Museum in London.

All of the house cat–size psittacosaurs were intact, and they were lying in the same orientation in an area measuring about 0.5 square meter, suggesting that the youngsters were trapped together, he notes. But individual body measurements indicate that members of the group would have ranged in age from about 18 months to 3 years, says Barrett.

Psittacosaurs didn't have horns and broad skull frills like those on *Triceratops*, a distant relative that lived about 60 million years later. However, the new finding hints that psittacosaurs traveled in groups that included individuals from several different clutches of eggs, says Barrett. Previously, researchers had discovered a fossilized nest that included almost three dozen psittacosaur hatchlings, possibly from several mothers. Together, this evidence of gregarious behaviors suggests that ceratopsians developed complex social behaviors long before they possessed extreme cranial ornamentations, says Barrett. —S.P.

PALEOBIOLOGY

Early tetrapod likely ate on shore

The skull structure of *Acanthostega*, a semiaquatic creature that lived about 365 million years ago, suggests that although the creature spent most of its time in the water, it fed on shore or in the shallows rather than in deep water.

Molly J. Markey, a paleontologist at Harvard University, examined the pattern of boundaries between skull bones in *Acanthostega*. The boundaries, called sutures, can have straight edges or jagged, interlocking edges.

The pattern of skull sutures in *Acanthostega* doesn't match the one found in *Polypterus*, a modern fish that, like most fish today, captures its prey by slurping it in (*SN: 4/24/04, p. 264*), or the pattern found in *Eusthenopteron*, a fish that lived about 385 million years ago and seems to have been a suction feeder. Both creatures had a straight-edged suture running along the top center of its skull.

In contrast, the suture layout in *Acanthostega*'s skull closely resembles the pattern found in the skull of *Phonerpeton*, an amphibian that lived and presumably fed on land around 300 million years ago. Markey has measured the strains across straight and jagged sutures in Polypterus skulls as the fish ate. From her results, she speculates that the *Polypterus* and *Eusthenopteron* pattern successfully resists the stresses that arise during slurping, while the *Phonerpeton* and *Acanthostega* pattern would better resist the stresses during grab-and-bite dining.

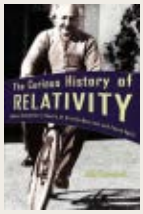
Therefore, Markey suggests that *Acanthostega* fed on shore or in shallow water. —S.P.

Books

A selection of new and notable books of scientific interest

THE CURIOUS HISTORY OF RELATIVITY: How Einstein's Theory of Gravity Was Lost and Found Again

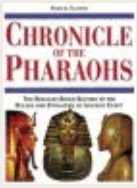
JEAN EISENSTAEDT



Albert Einstein's general theory of relativity was met with widespread acceptance and acclaim, although most people didn't understand it. Then, the theory languished for years, as some physicists became fascinated with quantum mechanics and others turned against Einstein's difficult-to-comprehend notion of relativity. In this English translation, Eisenstaedt, a senior researcher at France's National Center for Scientific Research, reviews how Einstein developed the theory that would supplant Newton's principles of gravity. The author reviews the period from the 1920s to the 1950s, during which Einstein confronted his critics. Finally, Eisenstaedt ponders what will become of general relativity as today's physicists search for a unifying theory of the quantum and gravitational domains. ***Princeton, 2006, 363 p., b&w photos and illus., hardcover, $29.95.***

CHRONICLE OF THE PHARAOHS: The Reign-by-Reign Record of the Rulers and Dynasties of Ancient Egypt

PETER A. CLAYTON



Pharaohs led a powerful Egyptian civilization that lasted more than 3,000 years. In this vivid guide containing more than 300 images, archaeologist Clayton details the reigns of nearly 200 pharaohs, including Amenhotep, Ramses the Great, Tutankhamen, and Cleopatra. For each dynasty, Clayton includes tables that provide royal names, places of burial, genealogies, and main achievements. Sidebars include further details, such as the rags-to-riches story of Weni, the ambiguous gender of Akhenaton, and further information about texts found in the pyramids, royal mummies, and tomb robberies. Among the images are portraits of all the major rulers, diagrams of royal tombs and monuments, and photos of artwork and artifacts. ***Thames & Hudson, 2006, 224 p., b&w and color images, paperback, $24.95.***

OUT OF THIN AIR: Dinosaurs, Birds, and Earth's Ancient Atmosphere

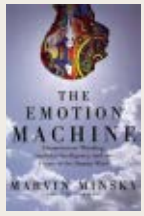
PETER D. WARD

Dinosaurs were the dominant life form on Earth for more than 150 million years. In this book, Ward, a professor of biology and Earth and space studies, proposes an unusual thesis to account for this incredible longevity. At the core of Ward's theory is oxygen, in its unique role in shaping life on Earth. As Ward explains, the atmosphere of ancient Earth was constantly changing, with oxygen concentrations fluctuating in sync with the appearance and disappearance of certain animals. The author traces the critical geological periods during which life emerged, examining each period's fossil record for clues to the evolution of certain body plans. He explains how dinosaur metabolism, form, and reproduction were uniquely suited for this period in Earth's history. Ward asserts that modern bird flight is a vestige of the dinosaurs' respiratory system becoming adapted to low-oxygen air. Finally, the author ponders what role Earth's ever-changing atmosphere could have on life in the future. ***Joseph Henry Press, 2006, 282 p., b&w illus., hardcover, $27.95.***

THE EMOTION MACHINE: Commonsense Thinking, Artificial Intelligence, and the Future of the Human Mind

MARVIN MINSKY

The human mind is constantly processing information, even when it's unaware of it. Thinking goes beyond deliberation to include emotions, gut instincts, and self-reflection. Minsky, a computer scientist, proposes that thinking can be understood as a series of machinelike processes. Infants react to what's around them as if through the action of a switch. As people mature, their thinking becomes more critical but is still based on machinelike processes. From this foundation, the author analyzes how the brain deals with information, such as the experience of pain and moods. Using computation diagrams, Minsky ponders how people develop common sense and how they use elements of their environments as resources for learning and adaptation. Finally, he suggests how the brain develops a flexible sense of self. ***Simon & Schuster, 2006, 387 p., b&w illus., hardcover, $26.00.***



JANE GOODALL: The Woman Who Redefined Man

DALE PETERSON

In her youth, Jane Goodall impressed people mainly as an extroverted and attractive woman. She graduated from secretarial school in London and became a secretary at Oxford University. Then, in 1957, at the invitation of a vacationing friend, she made her first visit to Africa. Little did her hosts know that Goodall would become the woman whose work with chimpanzees would forever change the way in which scientists view primates. In this lengthy biography, Peterson, who has collaborated with Goodall on several books, explains how Goodall became an icon. Despite Goodall's lack of science education, Louis Leakey hired her to help him study the social life and behavior of chimpanzees. What made her famous was her unique style of observation of the animals. She was the first person to witness chimps eating meat, using tools, and behaving as individuals with different temperaments. Peterson also chronicles Goodall's life as a wife and mother—she raised a son in East Africa—and her eventual transition into an animal-welfare activist. ***Houghton Mifflin, 2006, 740 p., b&w plates, hardcover, $35.00.***

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LETTERS

The Carolinas to New Jersey

"Bad-News Beauties: Poison-spined fish from Asia have invaded U.S. waters" (*SN: 9/9/06, p. 168*) cites evidence of a severe genetic bottleneck, suggesting that perhaps no more than three pregnant females launched the expanding western Atlantic red lionfish population. How can there be "pregnant females" in an animal with the external fertilization described in the article? Do you mean a founder population of as few as three individuals? Please clarify.

LYNN LOZIER, FAIRFAX, CALIF.

Three females, which release their eggs, could be the maternal ancestors of the red lionfish now breeding in the west Atlantic. There would also have had to be at lease one fertilizing male present in that ancestral brood stock. —J. RALOFF

I just wanted to report catching a 1½-inch lionfish behind Strathmere near Corson Inlet off New Jersey. I have been catching fish for my aquariums since 1961, and this is my first-ever lionfish. I read about some being caught near Long Island and Rhode Island and many living off the Carolinas. I thought I might capture one some day in the future but was much surprised to see this one in my trap.

BOB SEABROOK, ABSECON, N.J.

Bugged

If your cell phone battery is depleted for no obvious reason ("Cyber attack depletes cell phone batteries," *SN: 9/16/06, p. 190*), another possibility is an attack by law enforcement. Special firmware may have been surreptitiously downloaded into your phone, turning it into a bug that operates even if the phone appears to be off.

STEVEN R. NEWCOMB, BLACKSBURG, VA.

Bright idea

"Enigmatic Eruptions: Gamma-ray bursts lack supernova fireworks" (*SN: 9/23/06, p. 196*) states that gamma-ray bursts are "a million trillion times as bright as the sun." The sun is so bright that humans can't look directly at it from 93 million miles away. How can we possibly wrap our minds around something a million trillion times brighter? Astronomy is great.

DONALD KAUFMANN, PHILADELPHIA, PA.

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