SCIENCE NEWS

THE WEEKLY NEWSMAGAZINE OF SCIENCE

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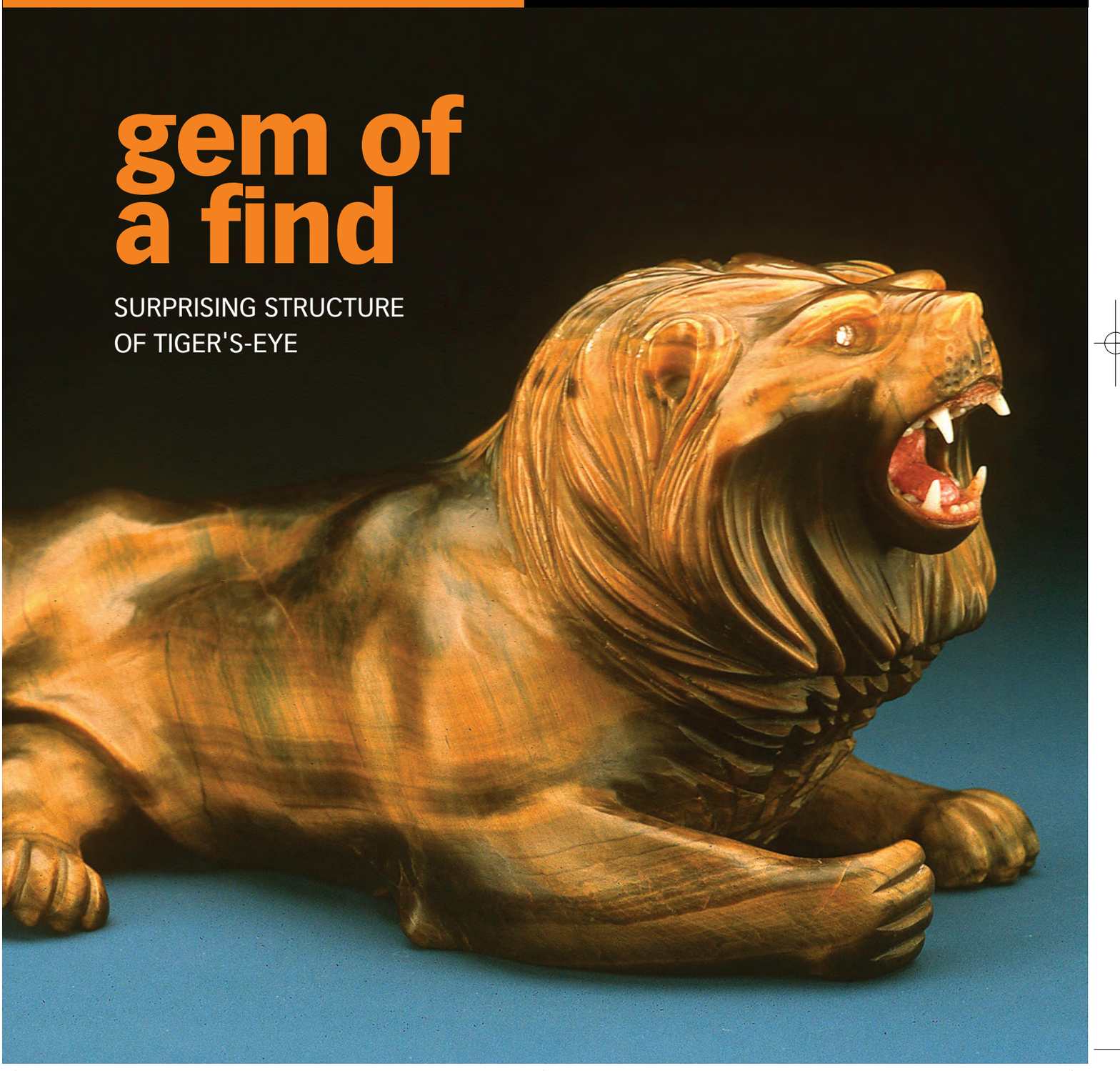
gem of a find

SURPRISING STRUCTURE OF TIGER'S-EYE



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Cover A discovery about the crystal structure of tiger's-eye quartz suggests that the shimmering gemstone isn't formed the way that scientists have assumed for the past 130 years. (C. Clark/National Museum of Natural History) Page 263

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Spheres in Disguise

Solid proof offered for famous conjecture

A Russian mathematician may have finally cracked one of the most famous problems in mathematics: the Poincaré conjecture, a question about the shapes of three-dimensional spaces. If his work is correct, it will make him eligible for a $1 million prize from the Clay Mathematics Institute in Cambridge, Mass., which has declared the conjecture one of the seven most important mathematical problems of the new millennium.

More than 100 mathematicians packed a lecture hall at the State University of New York at Stony Brook this week to hear Grigori Perelman of the Steklov Mathematical Institute in St. Petersburg, Russia, describe his work. Last week, Perelman told an equally attentive audience at the Massachusetts Institute of Technology (MIT) that he has proven the conjecture together with a broader problem called the Thurston geometrization conjecture. This second problem proposes that any three-dimensional space can be chopped in a standard way into pieces, each of which has a simple geometric structure.

Perelman has posted two papers about his research on the Internet (*http://xxx.lanl.gov/abs/math.DG/0303109* and *http://xxx.lanl.gov/abs/math.DG/0211159*). Mathematicians are now scrutinizing every line of the work to verify its correctness.

"We're all waiting with bated breath," says Yair Minsky of Stony Brook, who attended the lecture there.

The Poincaré conjecture belongs to the field of topology, which studies properties that are preserved when a shape is stretched or twisted without tearing. Topologically speaking, the surfaces of a doughnut and of a coffee cup are the same, but they're different from the surface of a ball.

Mathematicians have established criteria for distinguishing among types of surfaces. For example, consider a loop of string lying on a closed surface. More than a century ago, mathematicians proved that if every such loop can be shrunk to a single point without leaving the surface, the object is a sphere. On a doughnut, by contrast, a loop that encircles the hole can't be shrunk to a point.

The traditional, or two-dimensional, sphere is the set of all points in three-dimensional space that are a given distance from a fixed center. Mathematicians also study what they call the three-dimensional sphere—the set of all points a given distance from a center in four-dimensional space. French mathematician Henri Poincaré conjectured 99 years ago that, just as in the case of surfaces, any closed three-dimensional space in which loops can be tightened to a single point is really a three-dimensional sphere.

In the intervening years, dozens of mathematicians have put forth mistaken proofs, often very subtly in error. For that reason, mathematicians are hesitant to declare the conjecture settled until Perelman's proof has been thoroughly checked. But they agree that, unlike most previous attempts, Perelman's papers contain a wealth of important ideas that will be valuable even if his work turns out to fall short of proving the full Poincaré conjecture.

"The first paper is already amazing," says Jeff Viaclovsky of MIT. "It's a major breakthrough." —E. KLARREICH

Fig-Wasp Upset

Classic partnership isn't so tidy after all

Textbooks that marvel over an extreme example of the buddy system—fig species that supposedly each pair up with a lone pollinating wasp species—may need rewriting, according to a new genetic analysis.

In four out of eight fig species tested in Panama, genetic markers reveal that the supposedly single type of wasp living in the flower turns out to be two species, reports Drude Molbo of the Smithsonian Tropical Research Institute (STRI) based in Balboa, Panama. Fig partnerships with multiple wasps may turn out to be "routine," Molbo and her colleagues suggest in an upcoming *Proceedings of the National Academy of Sciences*. They also have evidence of a single wasp species teaming up with different figs.

Another pollination biologist, Olle Pellmyr of the University of Idaho in Moscow, welcomes the new study as "nice work." The old idea that, except for a few oddballs, each of the world's 800 fig species has an exclusive partnership with a wasp has been "dogma," he says.

Pellmyr points out that biologists have long used fig wasps to study big questions, such as sex ratios, cheating in partnerships, and formation of new species. Molbo's coauthor Allen Herre, also of STRI, says that the team's findings will require some rethinking across a wide range of work, including his own.

The wasps, usually only a few millimeters long, make epic flights of up to 20 kilometers to find the right species of fig in bloom. The female wriggles into the flask-shaped flower, lays eggs, and dies there. Her offspring hatch and mate inside the fig flower. Each daughter then sets off to find a new fig plant in which to lay eggs. When she arrives inside a flower, she deposits her natal fig's pollen.

Scientists have known that several female wasps can converge on the same flower. To sort out batches of offspring, Molbo identified DNA markers that distinguish the offspring of these females. As she analyzed populations in hundreds of fig flowers, some combinations of markers never showed up. The researchers began to suspect that the figs held pairs of wasp species.

To check their results, they turned to Carlos Machado, now of the University of Arizona in Tucson. He has identified DNA markers not from the cell nucleus, as Molbo does, but from mitochondria, the cell powerhouses. The mitochondrial markers displayed the same patterns.

"Drude was looking for one thing and found something very surprising and different," says Herre.

One of the wasp pairs working the same fig species seems to have evolved from a shared ancestor within the past few million years, says Herre. Pellmyr highlights this finding as a possible example of a species that split despite close quarters (*SN: 7/21/01, p. 42*).

Rethinking wasps and figs may rock

H. STADELMANN AND MOLBO

ROOM IN BLOOM Inside the developing green fruit of a Panamanian fig, a wasp generation hatches and mates. Inset shows male (wingless, left) and female (right).

some established ideas, but Herre says the finding does solve some puzzles in theories of resource allocation between sons and daughters. Now that Herre can tell the size of wasp broods that each mother provides, the male-female ratios better fit some earlier predictions. —S. MILIUS

Feel the Heat

Rain forests may slow their growth in warmer world

During a long-term research project in a Central American rain forest, mature trees grew more slowly in warm years than they did in cooler ones. This observation hints that tropical forests may become less efficient at removing planet-warming carbon dioxide from the atmosphere if global temperatures continue to rise.

From 1984 to 2000, scientists studied the old-growth forest at La Selva, Costa Rica. Annually, the team measured the diameter of all mature trees within a 2-square-kilometer area. They found that diameter growth varied significantly from year to year and was related to average daily temperature. The annual tree growth from 1984 to 1986, the coolest interval during the period, averaged 81 percent greater than the growth tallied during the record hot spell related to the El Niño that began late in 1997. The average daily temperature difference between the two periods was about 1.4°C.

Tree growth in the forest was also particularly slow during the El Niño year of 1987, says Deborah A. Clark, a biologist at University of Missouri–St. Louis. Clark and her colleagues report their results in an upcoming *Proceedings of the National Academy of Sciences.*

Looking at global carbon dioxide measurements during the same period, the researchers noticed that quantities of the gas attributable to land plants in tropical regions increased during warm years. That phenomenon could stem from typical plant-growth characteristics, the researchers say.

Plants use photosynthesis to convert sunlight, carbon dioxide, water, and nutrients into carbohydrates. When the plants tap into their stores of carbohydrates for chemical energy, however, they return carbon dioxide to the atmosphere—just as animals do—in the process called respiration. Although a plant's rate of photosynthesis begins to drop off above a temperature that's characteristic of its species, its rate of respiration continues to rise with increasing temperatures, says Clark.

Most of the observed global spikes in carbon dioxide during warm years probably stemmed from the increased respiration of tropical land plants, but some may have been produced by other sources, such as forest fires or agricultural burning, says Stephen C. Piper, a biogeochemist at Scripps Institution of Oceanography in La Jolla, Calif., and a coauthor of the team's report.

The growth rate of mature trees can be a useful indicator of the climate's effect on the rest of an ecosystem, says David S. Schimel of the National Center for Atmospheric Research in Boulder, Colo. The link that Clark's team discovered between slow growth rates in Costa Rican trees and increases in the atmospheric carbon dioxide traceable to tropical plants is "an innovative result that's hard to argue with," he says. —S. PERKINS

D.B. CLARK

STEAM HEAT Measurements show that mature trees of several species grow less during warmer years in the old-growth rain forest at La Selva, Costa Rica.

Genetic Clue to Aging?

Mutation causes early-aging syndrome

Why does the human body deteriorate as a person ages? Two research teams have found a new clue to this longstanding mystery. Both groups have identified a mutation that causes children to suffer a form of accelerated aging that usually results in death in their teens from heart attack, stroke, or other problems more typically associated with elderly people.

While investigators continue to debate exactly how well this so-called Hutchinson-Gilford progeria syndrome mirrors normal aging, many are convinced that the newly discovered mutation could provide insight into the process.

This finding "will help scientists across the globe to explore the fundamental mechanisms that drive human aging. We hope this will also lead to treatment and an eventual cure for progeria," says Leslie Gordon of Tufts University School of Medicine in Boston.

Hutchinson-Gilford progeria syndrome, also known simply as progeria—Greek for early aging—affects only an estimated 1 in 4 million children. "It's been a very difficult disease to get a handle on. There's no more than 100 case reports in the literature," says W. Ted Brown of New York State Institute for Basic Research in Developmental Disabilities in Staten Island.

Children with progeria are usually diagnosed 6 months to a year after birth, when their physical development starts to lag. They rarely grow taller than 4 feet, and their heads are oversized for their bodies. The children become bald and have skin problems such as scleroderma. While their mental development is normal, children with progeria rapidly develop atherosclerosis and die, on average, at the age of 13.

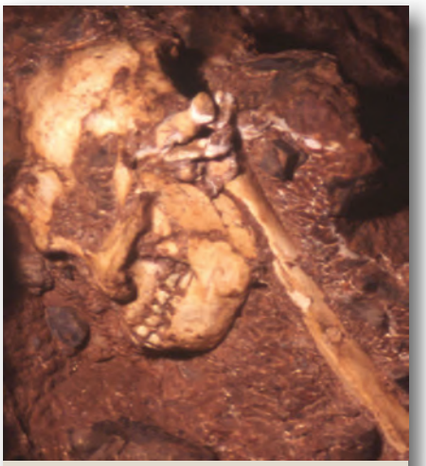
In a report to appear in an upcoming *Science*, Nicolas Lévy of Hôpital de la Timone in Marseille, France, and his colleagues identify a subtle but identical mutation in two kids with progeria. The defect is in a gene that encodes two proteins called lamin A and lamin C. Strengthening the case against this gene, *Nature* last week released a similar report from a group of researchers including Gordon and Brown. This second team, led by Francis S. Collins of the National Human

Genome Research Institute in Bethesda, Md., found the same change in the gene's DNA sequence in 18 of 20 children with progeria that the team studied.

The lamin proteins are the main structural elements forming the envelope of a cell's nucleus, where DNA resides. Although it doesn't alter lamin C, the progeria mutation shortens lamin A. As a result, the majority of cells from children with the progeria mutation have abnormally shaped nuclei, both research teams found.

It's still unclear how a mutant lamin A and the misshaped cell nuclei lead to the symptoms of progeria, but Huber Warner of the National Institute on Aging in Bethesda speculates that such a defect could prevent so-called stem cells from replacing worn-out or damaged cells and tissues.

"This discovery opens one or two doors for aging research," he says. "Nuclear structure and function now become targets of research."



SCIENCE: F.E. WEBER/UNIVERSITY HOSPITAL ZURICH

Ancestors Go South

Some of the oldest fossils in the human evolutionary family—including the skull and upper-arm bone shown above—come from South Africa, according to a study in the April 25 *Science*.

A mix of new and already excavated *Australopithecus* fossils, found in two caves, date to 4 million years ago, reports a team led by Timothy C. Partridge of University of the Witwatersrand in Johannesburg. That's as many as 1 million years earlier than previous estimates for South Africa. The scientists suspect that the bones represent two forms of *Australopithecus*. An *Australopithecus* species of comparable age lived in eastern Africa (*SN: 5/15/99, p. 315*).

The age estimates of the South African fossils hinged on measurements of the decay of radioactive isotopes in cave sediments. —B. BOWER

In terms of progeria, Gordon and Collins predict that within a year, there will be a genetic test that can confirm a diagnosis. Correcting the genetic defect through gene therapy, drugs, or other means is much further off but is now a possibility, say these researchers.

"This is a great springboard," says Gordon. Collins adds that researchers will quickly introduce the progeria mutation into mice to create a model for testing treatments for the syndrome.

In previous studies, researchers have attributed a form of muscular dystrophy and five other human disorders to defects in the same lamin gene. Now with the addition of progeria, Collins calls that tally a record for one gene. —J. TRAVIS

Bone Fix

New material responds to growing tissue

Surgeons routinely harvest fragments from a healthy part of a patient's skeleton to repair wrecked bones elsewhere. This surgical step causes pain and expense that some researchers aim to eliminate by using a new bone-forming strategy that has shown promise in animal studies.

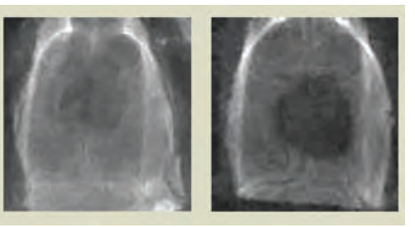
For years, scientists have been devising scaffold materials—both natural and synthetic—that encourage the growth of bone tissue. But the natural materials, primarily cow collagen, have raised worries of disease transmission. On the other hand, synthetic polymers can cause inflammation, and it's difficult to engineer the material to break down in synchrony with bone regrowth.

In the May *Nature Biotechnology*, Jeffrey Hubbell and his coworkers describe a material that could amount to a happy medium: scaffolding that's synthetic yet responds to the body's bone-making cues.

"We were trying to look at how nature evolved [bone growth] and copy that in synthetics," says Hubbell, a researcher at the Swiss Federal Institute of Technology in Zurich and the University of Zurich.

He and his colleagues built their scaffolding with star-shaped poly(ethylene glycol) molecules and added special properties. They adorned the scaffolding with synthetic peptides, or protein fragments, that help bone-forming cells called osteoprogenitor cells adhere to the framework. They also glued together the branched arms of the polymer with other peptides that are susceptible to enzymes secreted by the osteoprogenitor cells. In their three-dimensional mesh, the researchers trapped proteins called BMPs that stimulate the cells to begin bone regeneration.

When osteoprogenitor cells attach to a scaffold implanted at the injury site, they release enzymes that snip through the mesh, destroying the scaffolding. BMPs spill out just where and when they're needed.



BUILDING BONES A rat's skull regenerates better with a new bone-promoting scaffold (left) than with a less-sophisticated scaffold (right).

Hubbell and his coworkers tested their scaffolding on rats from which they'd removed small circular sections of skull. Disks made of the scaffold material stimulated bone regeneration as well as cow collagen does.

The system is "a clever and unique approach to develop synthetic bone scaffolds that are cell-responsive," comments Kristi S. Anseth of the University of Colorado in Boulder. The work "has wide implications in numerous wound- healing and regenerative medicine applications," she adds.

In the future, Hubbell says, doctors could make the repair less invasive by applying the scaffolding as a liquid—perhaps through a syringe or a small incision—that hardens once it's in place. —J. GORMAN

Blunt Answer

Cracking the puzzle of elastic solids' toughness

Soft, springy materials like rubber and skin don't tear easily because they stretch before breaking apart. For decades, however, detailed understanding of how that stretchiness toughens these materials has eluded researchers.

Now, researchers from the DuPont Company in Wilmington, Del., and Cornell University have come up with a comprehensive explanation—based on both experiments and computer modeling—for the toughness of soft solids.

The new insights have "massive implications," says team member Stephen J. Bennison of DuPont. The findings could potentially influence development of materials ranging from adhesives and solid rocket propellants to artificial biological tissues and foods, he says.

The essence of what the team presents in an upcoming *Proceedings of the Royal Society of London A* is this: Cracks that begin in soft solids, having a ratio of stiffness to strength of about one-third or less, stop moving forward through the material. Instead, says Cornell theorist Chung-Yuen (Herbert)

Hui, the crack "just sits there and opens up"—a phenomenon known as blunting.

While materials investigators had previously noted blunting at crack tips in some soft solids, the DuPont-Cornell team has identified a new mechanism of crack propagation that incorporates blunting. "It's really a fundamental change in [comprehending] the way a crack runs in a material," Bennison says.

Kenneth R. Shull of Northwestern University in Evanston, Ill., agrees. "I really do think it is potentially one of the key ideas . . . that's been largely missed before," he says.

In materials-speak, a tough substance is one that absorbs a lot of energy as it breaks apart. Consider the transparent polymer called plasticized polyvinyl butyral, or PVB, that DuPont makes to cement sheets of glass together in car windshields. PVB puzzled DuPont scientists, Bennison notes, because it was tougher than could be accounted for by the prevailing ideas of how materials fail.

In studies that began in 1995, the DuPont-Cornell team made the startling experimental observation that a slit the length of a paperclip in stretched PVB would open into a hole rather than tear the fabric further. Adding to this unexpected, macroscopic demonstration of crack blunting, computer simulations of fracturing in even more elastic materials pointed to blunting as a barrier to crack growth on a microscopic scale.

To understand what makes these materials give way when they finally do, the team found X-ray evidence that thousands of minuscule voids—the result of blunting of submicroscopic cracks—set the stage for the failure. The scientists contend also that stretching around blunted microcracks eventually stiffens the material to the point that it can't deform any more, leading to the massive breaking of chemical bonds that sums into ordinary fractures (*SN: 1/4/03, p. 3*).

Manoj K. Chaudhury of Lehigh University in Bethlehem, Pa., calls the new work the first quantitative theory to show that blunting can account for the toughness of rubbery materials. Nonetheless, cautions Shull, many more experiments are needed to see if it's possible to poke holes in this new theory. —P. WEISS

Out of China

SARS virus' genome hints at independent evolution

The newly deciphered genome of the pathogen responsible for severe acute respiratory syndrome (SARS) suggests that the virus is the product of a long and private evolutionary history.

Since emerging from southern China in February, SARS has struck at least 4,000 people worldwide and killed more than 200. Disease researchers have launched a massive effort to understand the pathogen and control the epidemic.

On April 16, European scientists announced that they had demonstrated that the agent responsible for SARS is a coronavirus never detected before the current outbreak. In experiments on monkeys at the Erasmus Medical Center in Rotterdam, the Netherlands, researchers showed that the new coronavirus alone can cause SARS. Earlier in the outbreak, a member of a separate viral family was also a suspect (*SN: 3/29/03, p. 198*).

On April 12, researchers at the British Columbia Cancer Research Centre in Vancouver reported that they had completely sequenced the virus' genome. Scientists in the United States presented nearly identical findings on April 14. Chinese researchers also sequenced the SARS virus and found that some samples differ considerably from those decoded in North America, which suggests that the virus mutates rapidly.

Comparisons among the newly sequenced genome and other coronaviruses' genomes indicate that SARS virus doesn't belong to any of the three known clusters of related coronaviruses. The 10 coronaviruses that infect mammals fall into two clusters, each of which contains one virus that causes colds in people. The third cluster contains two bird pathogens.

Coronaviruses readily swap genetic material with each other in a process known as recombination. This creates new viruses that share some genetic similarities with each parent virus and occasionally have novel capabilities to cause disease or to infect different hosts.

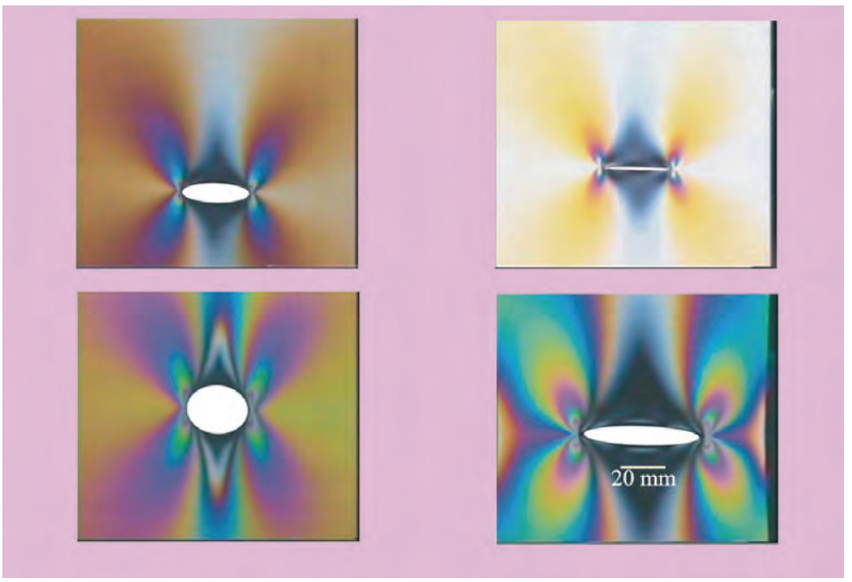
The largest SARS-virus gene, which makes up a whopping two-thirds of the pathogen's genome, appears to be distantly related to the corresponding gene in a mouse coronavirus. Another region of the SARS genome shares a few similarities with the avian coronaviruses.

Those observations suggest that recombination may have given rise long ago to an ancestor of SARS virus, says Michael M.C. Lai, a virologist at the University of Southern California in Los Angeles. If recombination had occurred more recently, the genetic similarities would be more striking, he says.

By itself, Lai notes, "recombination was not responsible for [SARS virus' recent] emergence as a human pathogen."

The SARS virus may have long had the capacity to infect people but only recently encountered conditions that facilitated its spread, says virologist Shinji Makino of the University of Texas Medical Branch in Galveston. Alternatively, it may have derived from one or more unidentified animal coronaviruses that only recently mutated or recombined to create a human pathogen, he says.

The new pathogen "is very likely from a wild animal," argues Lai. Future investigations in the region of China that seems to harbor the virus might eventually turn up the mysterious animal host, he says. —B. HARDER



HUI ET AL./PROCEEDINGS OF THE ROYAL SOCIETY OF LONDON A

TENSE SCENE In the polymer PVB (left), a horizontal crack transforms into a hole (white). In a similarly elastic but weaker polymer (right), the crack elongates. Colors indicate stress levels.

EYE OF THE TIGER

Discovery about gem's structure overturns old theory

BY SID PERKINS

In London in the mid-1870s, 25 shillings—about $85 in today's terms—went a long way. You could buy 7 grams of gold, 40 liters of rum, or about a half kilogram of opium. Where you couldn't get a bargain, however, was the jewelry store. That same amount of money bought just 1 carat, or 0.2 gram, of a gem called tiger's-eye. When rich sources of that precious stone were found in western South Africa in the 1880s, prices plummeted. By 1900, tiger's-eye was considered merely semiprecious. Today, a savvy shopper can purchase the gem for about $1.50 per carat.

The passage of time has transformed more than the gem's price. Recent research has upended a 130-year-old theory about how tiger's-eye forms. As a result, scientists soon will be scrambling to update everything from mineralogy textbooks to museum displays.

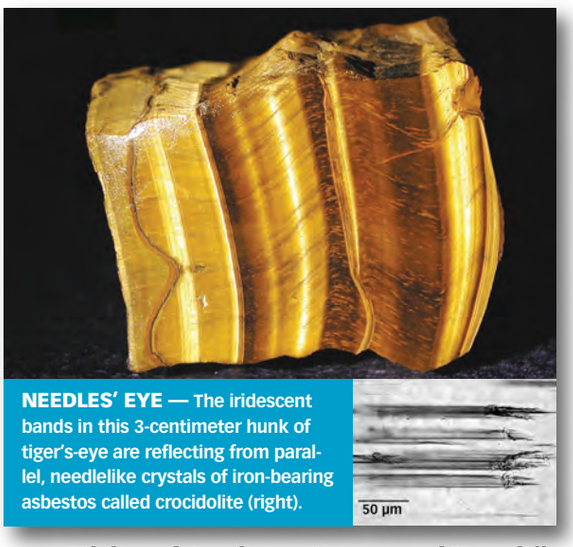
SHINING BRIGHT In its natural state, tiger's-eye is an unremarkable rock with a dull sheen. When polished and illuminated, however, the stone reflects a narrow band of light that changes position as the gem is turned back and forth. This effect, called chatoyancy, gets its name from the French phrase for "cat's eye" because of its resemblance to a feline's slitted pupil. Chatoyancy occurs when light reflects from minute, parallel ridges, fibers, or tubes within a transparent material.

Early in the 1800s, mineralogists recognized that tiger's-eye was a fibrous variety of quartz, or silicon dioxide. In 1873, the German mineralogist Ferdinand Wibel learned more. While studying the chemistry of hawk's-eye, a blue form of tiger's-eye, he found that the gem was almost entirely quartz but that it also contained fibers of crocidolite, an often bluish, iron-bearing form of asbestos. Wibel proposed that hawk's-eye forms in Earth's crust when quartz dissolved in hot water infiltrates spaces between crocidolite fibers and then slowly replaces the asbestos' molecules. Brown tiger's-eye, Wibel said, comes after yet another step. It results when chemical reactions transform some of the iron in the bluish crocidolite into brownish iron oxide.

The idea that tiger's-eye is a pseudomorph—a mineral in which crystals of one material take on the form of another, which it replaces atom by atom—held sway for more than 125 years. In fact, tiger's-eye is cited in many textbooks as a classic example of a pseudomorph, says Peter J. Heaney, a mineralogist at Pennsylvania State University in University Park. During his own efforts to understand the processes underlying pseudomorphism, Heaney examined thin samples of tiger's-eye under a microscope and realized that Wibel was wrong.

Heaney expected to find that the quartz in tiger's-eye is chalcedony, a form that typically consists of fibrous, defect-riddled crystals less than 1 micrometer in diameter. Instead, Heaney was surprised to discover relatively fault-free, column-shaped quartz crystals that measured more than 100 micrometers across and up to 10 millimeters in length. Pseudomorphism doesn't produce such a uniform crystal form.

Heaney and his Penn State colleague Donald M. Fisher suggest that the crystal structure of tiger's-eye forms via a so-called crack-seal mechanism. In such a process, quartz and crocidolite crystals simultaneously condense from hot, mineral-rich fluids coursing through a tiny crack in a rock and grow to fill it. Repeated episodes of fracturing lead to more cycles of simultaneous, crack-filling growth of the two crystals.



NEEDLES' EYE — The iridescent bands in this 3-centimeter hunk of tiger's-eye are reflecting from parallel, needlelike crystals of iron-bearing asbestos called crocidolite (right).

HEANEY

In the tiger's-eye samples that Heaney studied, crocidolite fibers often ran parallel to the quartz columns. In some cases, however, the angle between the crocidolite and quartz was as much as 30°. Because, in those instances, the reflected cat's-eye bands of light were perpendicular to the crocidolite fibers, the scientists conclude that in tiger's-eye the chatoyancy arises from the crocidolite fibers, not the quartz. The researchers report their findings in the April *Geology*.

LONG TIME COMING So, why did it take 130 years for scientists to replace Wibel's tiger's-eye theory? After all, the techniques that Heaney used—optical and electron microscopy and X-ray diffraction—aren't new. The short answer, says Heaney, is that nobody had bothered to look. "Scientists merely accepted the old explanation, as I had," he explains. Also, because tiger's-eye is only a semiprecious stone, it hadn't attracted enough attention to merit a detailed investigation, he notes.

"It tickles me how [this finding] counters the longstanding assumption about how tiger's-eye forms," says Jeffrey E. Post, curator of gems and minerals at the Smithsonian's National Museum of Natural History in Washington, D.C. Says Post, who supports the new interpretation: "Sometimes an explanation is so pat that no one thinks to challenge it."

Tickled or not, Post joins the legion of curators in museums worldwide who will need to revise their mineralogy displays. But that's okay, he quips, because it's going to be even tougher for all those textbook editors. ■

A ROCKY START

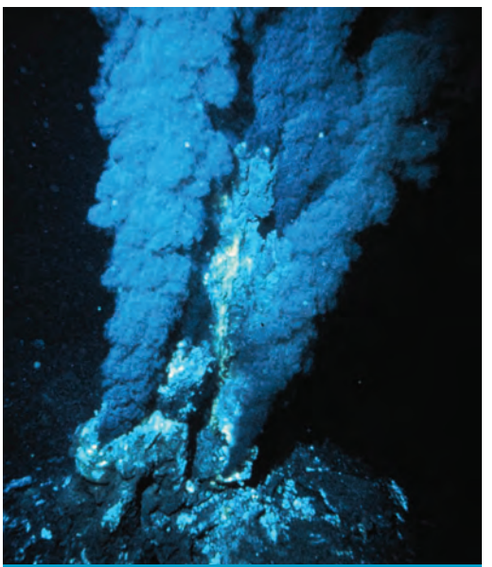
Fresh take on life's oldest story

BY KENDALL MORGAN

In the dark ocean depths, kilometers beneath the waves, scalding water spews from hydrothermal vents as it has for billions of years. Bubbling up at the breaks between Earth's plates, that water is a searing brew of minerals dominated by black iron sulfide. As it billows upward in vast quantities, the minerals roil like smoke from a raging fire. It looks like a place that ought to be dead as stone. Yet on the ancient Earth, that abundant black mineral might have been the crucial ingredient that first sparked all life, some scientists say. As they see it, the simplest life forms got their start within tiny cell-like chambers in iron sulfide rock that settled out from the hydrothermal vents' exhalations.

What's more, these origin-of-life researchers suspect that the two major groups of bacteria, known as archaebacteria and eubacteria, originated on two separate occasions about 3.8 billion years ago. Only much later, the scientists propose, did these original microbes join forces to create the first eukaryotes, the group that includes plants and animals.

The first complete synthesis of what might be called the iron sulfide theory for the origin of life appeared in the January *Philosophical Transactions of the Royal Society of London B*. Nearly 15 years in the making, this portrait of life's start, by microbiologist William Martin of the Heinrich-Heine Universitaet Düsseldorf in Germany and Michael J. Russell of the Scottish Universities Environmental Research Centre in Glasgow, is stirring up fellow origin-of-life researchers. Some of them describe the theory as speculative, while others call it ingenious.



SMOKIN' — Iron sulfide spewing from hydrothermal vents like this one may have sparked the first life on Earth nearly 4 billion years ago, according to a new theory on life's origins.

NOAA

But at the most basic level, says Martin, it's simple. "All you need is rocks and water, and everything else happens by itself," he says. "There's no magic here."

Where most versions of life's origins are "fuzzy around the edges," the new theory is explicit, Martin says. It traces life's opening chapters from the beginnings of biochemistry to the emergence of cells that look much like modern-day bacteria.

The theory also presents a notably wide target for anyone looking to criticize it, says chemist David W. Deamer of the University of California, Santa Cruz. So far, however, it's drawing more praise than flak as scientists agree that Martin and Russell's bold outlook and interdisciplinary approach promise to launch a new prong of research aimed at one of the biggest questions there is: How did life begin?

IRONCLAD BEGINNING Russell began imagining that rocks might have been the spawning ground of life itself while studying iron sulfide mineral deposits collected from old hydrothermal vents.

When hot iron sulfide–containing water meets the cooler ocean, some of the mineral forms into chimneys, which now can be found in many places underneath the Pacific and Atlantic Oceans and elsewhere. Some of the rock spires have reportedly grown at rates up to 1 meter every 2 months. One off the coast of Oregon reached the towering height of a 15-story building before toppling over. Researchers often find thriving communities of creatures around these vents, some of the animals specially adapted to the intense chemical environment.

What fascinates Martin and Russell most, however, is the internal structure of the chimneys themselves. Far from being solid lumps of stone, they have a "highly compartmentalized internal fabric," the scientists say.

In 1997, while working with another colleague, Russell simulated formation of these rock structures by injecting a warm, alkaline solution of sodium sulfide into a cooler, iron-rich solution in the lab.

Immediately after the injection, iron sulfide bubbles spontaneously began to form, Russell recalls. Just a minute later, an iron sulfide structure several centimeters high had formed. The resulting mineral construction contained a honeycomb of tiny compartments.

Russell proposes that at undersea hydrothermal vents around 4 billion years ago, such compartments acted as an incubator in which life's basic ingredients concentrated and the first cells were born. In this model, the walls of the compartments serve as the first cellular membranes.

Like the age-old chicken-and-egg question, the source of the first cell membrane has been a major hurdle for the theories about the origin of life. Today's cell membranes are made of long, oily, lipid molecules that form into pliant fluidlike films surrounding a cell's biomolecular machinery. Such walls concentrate the molecules of life into a small space in which they can work together. "Without a membrane, a cell bleeds to death," Russell says.

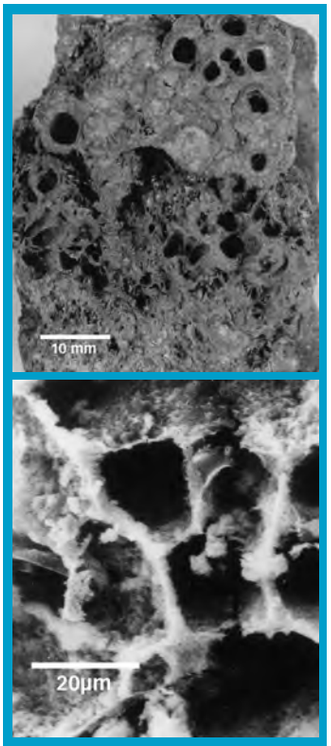
Decades ago, Deamer proposed a solution to the problem. He suggested that lipid membranes, similar to those that envelop cells today, might have preceded other complex molecules of life. After

all, he says, chemists have shown that under the right conditions, cell-like membranes will form spontaneously from simple chemical ingredients (*SN: 2/3/01, p. 68*).

Lipid membranes may have self-assembled on the early Earth, acknowledge Martin and Russell. However, they question how those first lipid droplets could have contained precisely the right mix of ingredients for life. Perhaps more importantly, they ask, how would such protocells capture the energy required to create more of themselves?

"It's a huge stumbling block," Deamer agrees.

JUMP-STARTED The rock compartments nestled within the hydrothermal chimneys provide a possible answer. When rock membranes form in the laboratory, they create a voltage of 600 millivolts as their thin walls separate the simulated hydrothermal and ocean solutions, which have different ion concentrations. The voltage lasts for several hours, says Russell, and is comparable to that across the membranes of today's living cells.



ROCKY CRADLE — Hunks of iron sulfide rock (top) from undersea chimneys that surround hydrothermal vents are made up of cell-like compartments that may have housed the first life, scientists say. A micrograph (bottom) of similar structures recreated in the lab reveal some compartments the size of cells.

"That energy would be sufficient to drive a putative metabolism," Russell notes.

Deamer remains skeptical, but he's intrigued enough that he's planning to conduct laboratory tests of his own to see whether iron sulfide structures can sustain voltages sufficient for catalyzing reactions that help form, for example, ATP—the cell's biochemical fuel.

Martin and Russell point to previous evidence, such as that amassed by organic chemist Günter Wächtershäuser. He was one of the first scientists to suggest that iron sulfides and nickel sulfides might have held an important role in early life. He suspects that the flat surfaces of such minerals could have served catalytic roles similar to those of a modern cell's enzymes.

Wächtershäuser and his colleagues showed that metal sulfides can catalyze the formation of a so-called activated thioester from simple ingredients (*SN: 1/9/99, p. 24*). Some scientists suspect that thioesters may have preceded ATP as carriers of biochemical energy.

Later, Wächtershäuser's team showed that when catalyzed by iron sulfide, amino acids link into short peptide chains, the beginnings of proteins. Two years ago, geophysicist George D. Cody of the Carnegie Institution of Washington, D.C., and his colleagues added another piece of the puzzle: Iron sulfide leads to the synthesis of pyruvate, a molecule involved in many metabolic reactions.

In a series of lab experiments that simulate the hot, highly pressurized conditions found in deep hydrothermal vents, Cody's team found that fundamental chemical ingredients, including carbon dioxide and hydrogen, in the presence of iron sulfide, "enter into cycles that look a lot like metabolism." Says Cody: "It's hard to imagine a better catalyst [than iron sulfide], which we know was there in abundance" in the early ocean. "It's guaranteed that on the early Earth, all sorts of organic chemistry was happening," he adds.

Russell and Martin offer yet another piece of circumstantial evidence that life may have emerged from iron sulfide–catalyzed chemistry: Many of the large proteins that drive basic

RUSSELL AND A. HALL

First Couple?

Bacterial partners might have spawned a new life-form

Although archaebacteria and eubacteria, Earth's two main bacterial groups, diverged almost from their inception nearly 4 billion years ago, they came back together about 1.5 billion years later to form the third branch of life, the eukaryotes, according to William Martin of the Heinrich-Heine Universitaet Düsseldorf in Germany and Michael J. Russell of the Scottish Universities Environmental Research Centre in Glasgow. They conjecture that an evolutionary quantum leap happened after an archaebacterium swallowed a eubacterium.

Other scientists are now discovering that the deep ocean is a hotbed of unconventional symbiotic relationships that they say may yield clues about eukaryotes' oldest ancestor. Microbiologist Joan M. Bernhard of the University of South Carolina in Columbia has found a diversity of single-celled eukaryotes, called protists, bearing bacterial partners. She describes the deep-sea environment as a "symbiotic oasis." Some of the critters, including members of two major protist groups—the whip-tailed flagellates and hairy ciliates—harbor bacteria internally. Still others are coated in bacterial partners.

The inner workings of these pairings haven't yet been defined, but their abundance suggests that teamwork is a useful solution to the stark ocean environment, Bernhard says. It's possible that the first eukaryotes originated in similar communities, she adds.

The first examples of bacterium-bacterium collaborations have begun to surface. Two years ago, Antje Boetius of the Alfred Wegener Institute for Polar and Marine Research in Bremen, Germany, and her colleagues found clumps of archaebacteria surrounded by a rind of sulfate-reducing eubacteria—the first example of a pairing between the two bacterial groups (*SN: 10/7/00, p. 232*). The duo apparently feeds on methane in the oxygen-depleted ocean.

These and more recently discovered bacterial assemblages account for the "massive biomass" at the seafloor, forming mats up to 4 feet deep, Boetius' team reports in the August 2002 *Nature*. These organisms might represent the kinds of associations that led to the first eukaryote, Boetius says.

The only known instance of a bacterium within a bacterium—the structure proposed as the origin of eukaryotes—has turned up inside abdominal cells of a mealybug insect (*SN: 7/28/01, p. 53*).

So, why haven't such species collaborations more often led to new life-forms? Martin suspects that the shift from symbiosis to wholesale melding of the partners' genomes only rarely proves possible, let alone viable.

However, he admits, any theory of eukaryotic origins faces a grand challenge. "It has to be plausible enough to have happened once, but not so easy that it happens a thousand times," he says. —K.M.

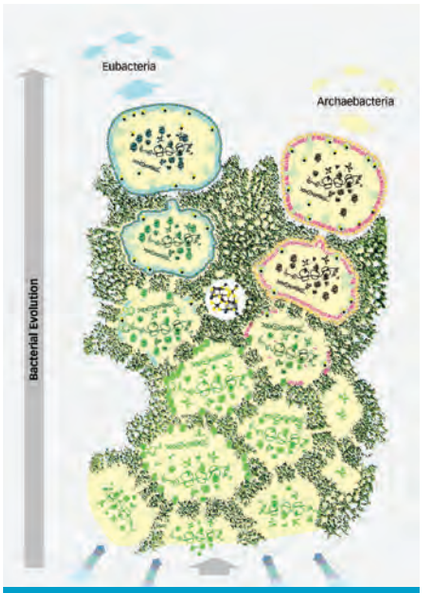
biochemical reactions today—such as ferredoxin, a protein that mediates metabolic reactions—rely on smaller iron sulfur cofactors. "It's a little bit of rock [in cells] that reminds us where we came from," Russell says. With these iron sulfide–based cofactors, proteins spur chemical reactions similar to the ones that the mineral itself can drive, Martin adds.

BREAKING OUT If Russell and Martin's theory has any chance of being right, naked, rock-cradled life-forms must at some point have invented the biochemistry required to produce their own membranes.

Once the ingredients for making lipids found their way inside the catalytically active iron sulfide compartments, the soft membranes emblematic of living cells could have formed. The microbes, now equipped with their own working membranes, could have begun emerging from their iron microwombs to colonize the early biosphere.

Actually, Russell and Martin say, this crucial evolutionary leap may have happened in two different ways that correspond to what subsequently became archaebacteria and eubacteria.

The lipid molecules that build into the membranes of archaebacteria and eubacteria bear a subtle difference: One is the mirror image of the other. Although the difference between the forms carries no known consequence in terms of survival, it has major implications for the membranes' origins, says Yosuke Koga of the University of Occupational and Environmental Health in Kitakyushu, Japan.



TWIN BIRTH — In this model, chemicals concentrate and react inside hydrothermal rock, forming essential life ingredients. Eventually, the two bacterial groups—archaebacteria and eubacteria—separately devise cell membranes and emerge.

MARTIN/ROYAL SOC. LONDON

Koga and his colleagues examined the genetic makeup of the key enzyme—(G-1-P) dehydrogenase—responsible for the formation of archaebacterial lipids. In 1998, the group reported that the genetic sequence encoding this membrane-building enzyme bore no resemblance to the corresponding enzyme in *Escherichia coli*, a representative eubacterium.

This genetic difference is too gaping for one type of membrane biochemistry to have evolved from the other, Koga argues. Therefore, he says, the two membrane types must have arisen independently, back when the first living cells emerged.

Martin and Russell conjecture that the bacterial ancestors living within their rock shelters cooked up two separate biochemical recipes for membranes. Then, with their distinctive membranes, the two types of cells presumably left their rocky starting places to begin paving their own evolutionary ways (*see box, page 265*).

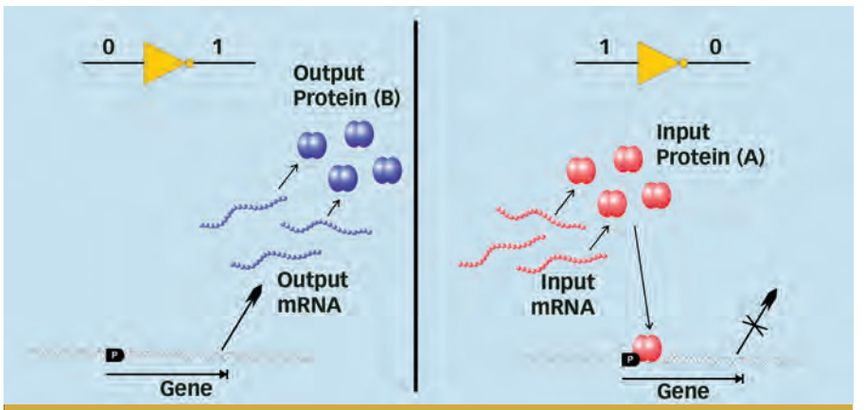
For people hoping to find life on other planets, the iron sulfide theory's version of earthly events should come as good news. The environment that Russell and Martin propose as the birthplace of life requires only rocks, water, and the most basic of chemical ingredients. Given that there likely are billions of venues like that throughout the universe, says Russell, "life can't help but happen." ■

DIGITAL CELLS

Computer circuits made of genes may soon program bacteria

BY ERICA KLARREICH

Imagine a future in which a single drop of water holds a veritable army of living robots; in which people download software updates not for their computers, but for their bacteria; and in which specially programmed cells course through a person's arteries, monitoring blood sugar concentrations and keeping an eye out for cholesterol buildups. These scenarios still belong to the realm of science fiction—but implanting computer programs into living creatures may not be far away. In the past few years, scientists have taken the first steps towards creating a host of cellular robots that are programmed to carry out tasks such as detecting and cleaning up environmental pollutants, tracking down cancer cells in a body, and manufacturing antibiotics or molecular-scale electronic components. These researchers have imported notions of electrical engineering—digital logic, memory, and oscillators—into the realm of biology. Their plan: to create cells with computer programs hardwired into the DNA.



INVERSE LOGIC — A digital inverter that consists of a gene encoding the instructions for protein B and containing a region (P) to which protein A binds. When A is absent (left)—a situation representing the input bit 0—the gene is active. and B is formed—corresponding to an output bit 1. When A is produced (right)—making the input bit 1—it binds to P and blocks the action of the gene—preventing B from being formed and making the output bit 0.

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"Eventually, the goal is to produce genetic 'applets', little programs you could download into a cell simply by sticking DNA into it, the way you download Java applets from the Internet," says Timothy Gardner, a bioengineer at Boston University.

The goal is not to produce a Pentium in a test tube. Cellular computers will probably never rival silicon chips in speed and reliability. "We don't use cells because they're a good medium for computation but because they can actually do stuff for us," says Adam Arkin, a bioengineer at the University of California, Berkeley.

Scientists intend to harness the multitude of cellular activities, which go beyond the capacity of silicon devices. Living cells can survive on the flanks of undersea volcanoes and in acidic mine drainage. They operate amazingly efficient factories for producing antibiotics, enzymes, and other useful chemicals, and they generate numerous copies of themselves. Cells can detect minute changes around them, and perhaps most crucially, interact with their environment.

By cutting and pasting pieces of genetic material, and most recently using artificial evolution as a design tool, engineers are starting to program microbes to carry out behaviors that nature never dreamed of. "We're basically hacking DNA instead of software," says Ron Weiss of Princeton University.

CELLULAR LOGIC Digital circuits, the building blocks of modern computers, encode bits of information in zeros and ones and then manipulate them in exact, controlled ways. Cells, which are basically bags of organelles, proteins, and small molecules, might not at first glance seem promising for such computations.

However, as cells regulate their activities and respond to the environment, they use many of the same tricks that go into digital circuits, such as on-off switches and feedback loops. What's more, cells house one of the richest known information-processing systems. Their strands of DNA include detailed instructions on how and when to build each of thousands of proteins. A control center for each gene turns it on or off according to the cell's changing needs.

Just as electrical engineers wire together transistors—the basic on-off switches of silicon chips—into complicated circuits, researchers are now stringing together genes and control centers in novel combinations to build what they call genetic circuits, in which the output protein of one gene regulates the next gene.

Silicon circuits perform complex operations using a handful of simple components known as logic gates. Genetic-circuit engineers are now building the same devices inside cells. One such logic gate is the inverter, which outputs a 1 if the input is 0, and a 0 if the input is 1. Another is the AND gate, which takes two inputs, and outputs a 1 only if both inputs are 1s. Amazingly, as simple as these two gates sound, mathematicians can construct any logical operation by hooking up enough of them.

Between 1998 and 2001, Weiss took some of the first steps toward building cellular logic gates when he modeled and built a cellular inverter, an AND gate, and two other gates. In Weiss' inverter, the input bit and output bit are encoded in the concentrations of two proteins—for simplicity's sake, call them protein A and protein B. If the concentration of protein A is high,

the input bit is a 1; if the concentration is low, the input bit is a 0. Similarly, the concentration of B corresponds to the output bit. Weiss constructed in *Escherichia coli* bacteria a loop of DNA containing two important pieces: the gene with the instructions for building protein B, and near that, a segment of DNA to which protein A binds.

To make protein B, a special molecule called messenger RNA assembles itself along the DNA, copying the gene's instructions and carrying them to the cell's protein-making factory. If the concentration of A is high, molecules of protein A will bind to the DNA loop and block the messenger RNA from attaching to the DNA. This prevents the cell from building protein B. If, on the other hand, the concentration of A is low, then protein B will be built in abundance. Thus, in Weiss' circuit, B is high when A is low, and vice versa. Weiss' other gates are constructed in similar, slightly more complicated ways.

By hooking together inverters, engineers can create a wide variety of interesting devices. In 2000, Gardner and his colleagues James Collins and Charles Cantor, both also of Boston University, built a memory device in *E. coli* out of two inverters for which the output protein of one is the input protein of the other, and vice versa.

HIGH AND LOW — With novel circuits genetically engineered into the bacteria, these *Escherichia coli* detect the presence of a substance called a quorum-sensing chemical, which is released by other cells (in a disk at right in these images). The bacterial cells produce a fluorescent protein when the concentration of the quorum-sensing chemical is above a set threshold (top) or below a set threshold (right). The team more recently constructed a circuit that detects the chemical when its concentration falls within pre-selected boundaries.

In the same year, Michael Elowitz and Stanislas Leibler of Rockefeller University in New York City made an oscillator in which three inverters are connected in a loop so that the output protein of each inverter is the input protein of the next inverter. In one test of their system, a fluorescent protein became active whenever one of the proteins was in its low state. The result was a population of gently twinkling cells like flashing holiday lights, Elowitz says. "It was very beautiful," he says.

Weiss' team has just put the finishing touches on a five-gene circuit in *E. coli* that can detect a particular chemical in its surroundings and turn on a fluorescent protein when the chemical concentration falls within preselected bounds. Such circuits could eventually be used to detect environmental toxins, Weiss notes.

"If you spread cells around . . . they will form a fluorescent ring around the [chemical], and the middle of the bull's eye is where the bad guys are," Weiss says.

Ultimately, he says, different cells could be programmed to respond to different concentrations of a toxic chemical and to fluoresce in different colors, so that the cell population would generate a color-coded topographical map of the toxin concentrations.

A DASH OF SPICE It's far easier to describe the schematics of these circuits than to build them for operation inside a cell. For instance, to hook up one gate to the next, the amount of protein produced by the first gate must be the right amount to activate the next gate. And at every step, the output protein must be either very high or very low, to avoid false positives or negatives. It's also necessary to tweak many parameters, such as the strength with which the various proteins and the messenger RNA bind to different parts of the DNA sequence.

Making such adjustments via trial and error is prohibitively expensive, Gardner says. Genetic-circuit engineers usually use enzymes to cut pieces of DNA out of one organism's genome, glue the pieces together in different combinations using other enzymes, and then put them in another organism. "It's as if you had to write an article by cutting words out of magazines and pasting them together," Gardner says.

To get around this difficulty, genetic-circuit engineers have turned to mathematical modeling to predict how a circuit will behave before they build it. Together with biologists, mathematicians, and other researchers, they have collectively developed a computer-aided design tool called BioSPICE, named after SPICE, the program that engineers have used for decades to test electronic-chip layouts. BioSPICE simulates a genetic circuit by modeling the speed of the biochemical reactions between the proteins, genes, and other molecules.

Because the circuits generally include complicated feedback loops, the equations that model the reactions tend to be nonlinear. If the quantity of an input protein doubles, the quantity of output protein won't necessarily double. In nonlinear equations, which come up in situations such as weather prediction and population dynamics, tiny changes in parameters can produce large swings in the behavior of a system. This makes the system difficult to analyze. At the same time, nonlinearity is the source of much of the wealth of possibilities for genetic circuits, says Jeff Hasty, a bioengineer at the University of California, San Diego.

"Even in small gene networks with just three or four genes, there is a whole zoo of potential behaviors," he says.

Because many of the parameters of the biochemical reactions are only partly understood, and because the random jiggling of molecules complicates the picture, mathematical simulations give only partial information about whether a circuit will work. Consequently, it's quite common to build a circuit only to find, for instance, that it produces 50 protein molecules when you really need 500, Weiss says. "Then you go back to the model and ask it what parameters to change to get 500 instead of 50," he says. "It's a continual process of simulation, refinement, simulation, refinement, until it works," Weiss says.

Weiss is now bringing another tool—evolution—into the design process. Once a circuit is working almost as he wants, instead of the engineers' refining the design again and again, Weiss permits the DNA to mutate and lets a lab-made version of natural selection do the hard work. In the Dec. 24 *Proceedings of the National Academy of Sciences*, Weiss and Frances Arnold of the California Institute of Technology in Pasadena report their team's work that used evolution to fix up a faulty inverter that Weiss had previously built.

The team took copies of the inverter and introduced small ran-

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dom changes, then put the mutated DNA circuits into cells and measured how well they performed. The engineers kept the circuits that worked better than the original and threw away the others. After only two rounds of mutation, the team had a working inverter.

"Without feeding the circuit any information, we evolved the original protein into a non-natural protein that acts as a very good digital logic inverter," Weiss says.

BRICK BY BRICK As with electronics, the real power will come from assembling cellular logic gates into large circuits. To do that, many technical challenges must be overcome. For example, each new gate in a circuit must usually be turned on and off by different proteins than those that control the previous gates. What's more, the DNA must be carefully designed so that the proteins produced at each stage don't accidentally interfere with other parts of the circuit. Right now, only a few proteins are understood well enough to be useful to genetic circuit engineers, says Drew Endy of the Massachusetts Institute of Technology (MIT).

"There are only about half a dozen parts that have been captured from the wilds of nature," he says.

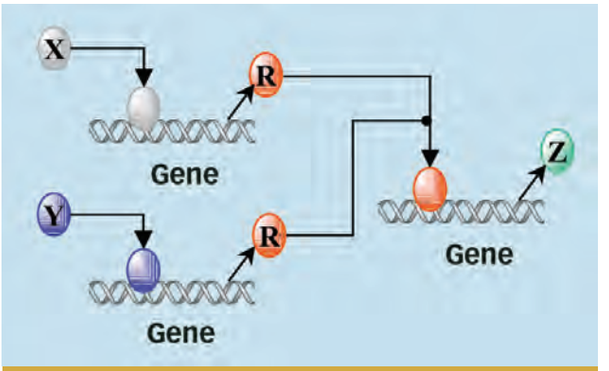
Endy and Thomas Knight of MIT are working to create a library of what they call BioBricks, standardized building blocks that genetic circuit engineers could link. "In electrical engineering, there's the idea of the specification sheet, which tells all the important properties of a component, like the environment in which it will work, the extreme conditions in which it will break, its size, accuracy, reliability, and so on," Knight says. "We'd like to make a similar set of biological components.

"We want to move away from the situation where you build the system and pray that it will work, toward the situation where you build the system and, unless you've done something stupid, it will work," he says. "It's going to be a long road, but we've got to get started."

Building circuits with many components will probably take years of work, researchers agree. "We're still making baby steps, and it's not clear just what we'll achieve," Arkin says. "But . . . if we keep pushing the boundary, we're going to get someplace."

Genetic-circuit engineers can look for inspiration in the shining example set by natural cells themselves. "Obviously, evolution has been able to figure things out to the point where it can get really complicated behavior from biological systems," says Michael Simpson of Oak Ridge National Laboratory in Tennessee.

If researchers can figure out how to tap into this richness, cellular robots may not be far away. ■



AND MORE — In this biological AND gate, the input proteins X and Y bind to and deactivate different copies of the gene that encodes protein R. This protein, in turn, deactivates the gene for protein Z, the output protein. If X and Y are both present, making both input bits 1, then R is not built but Z is, making the output bit 1. In the absence of X or Y or both, at least one of the genes on the left actively builds R, which goes on to block the construction of Z, making the output bit 0.

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OF NOTE

ZOOLOGY

Chicks open wide, ultraviolet mouths

The first analysis of ultraviolet (UV) reflections from the mouths of begging baby birds has revealed a remarkable display that birds can see but people can't.

The colors of chick mouths have attracted much scientific interest, says Sarah Hunt of the University of Bristol in England. An old theory held that the bright yellows and reds create conspicuous targets for parents delivering food in dim nests. Newer evidence shows that health influences mouth color, which suggests that various shades give parents a quick clue to a chick's condition and need for food.

To get a better idea of what the birds are seeing, Hunt and her colleagues measured UV reflection from chick gapes and nests for barn swallows, blackbirds, house sparrows, and five other European species. The gapes reflect a lot of ultraviolet, but the nests don't, Hunt and her colleagues report in an upcoming *Proceedings of the Royal Society of London B*. The big UV difference between nest and chicks suggests that it's time to dust off the old conspicuous-target theory, says Hunt. —S.M.

ENVIRONMENT

Traces of lead cause outsize harm

Minute amounts of lead in blood are worse for children than scientists had realized, according to new research. Data now suggest that lead affects development of kids' thinking skills at concentrations below 10 micrograms per deciliters (μg/dl) of blood. Higher concentrations had previously been recognized generally as harmful to the brain.

In fact, microgram for microgram, lead may pack more punch below 10 μg/dl than it does at higher concentrations, according to Richard L. Canfield of Cornell University and his colleagues. They periodically measured blood-lead concentrations in 172 children beginning when the kids were 6 months old and continuing until they were 5 years old. They gave each child an intelligence test at age 3 and at the end of the study.

Putting all these data together, the researchers found that for blood-lead concentrations between 1 and 10 μg/dl, the average effect of each additional 1 μg/dl was a drop of 0.82 IQ point. However, each 1 μg/dl blood lead above 10 μg/dl translated into only a 0.13-point loss, the researchers report in the April 17 *New England Journal of Medicine*.

A recent study found that 2.2 percent of U.S. children age 1 to 5 have blood-lead concentrations greater than 10 μg/dl, nearly 10 percent have concentrations of at least 5 μg/dl, and 90 percent have at least 1 μg/dl (*SN: 2/22/03, p. 120*). —B.H.

BIOMEDICINE

Little vessels react to magnetic switch

In laboratory animals, a magnet can act like a switch to either open or constrict tiny blood vessels, researchers report. Although preliminary, their study suggests the prospect of using magnets to alter blood flow in damaged tissue.

To look for vascular responses to magnets, biomedical engineer Thomas Skalak of the University of Virginia in Charlottesville obtained a grant from the National Institutes of Health and recruited electrical engineer Cassandra E. Morris, also at U. Va. "I was initially quite skeptical" of finding an effect, says Morris.

In the experiments, she cut thin layers of rat muscle and folded them away from the body so that blood vessels, just 10 to 100 micrometers in diameter, continued to nourish them. Next, she measured the blood vessels' diameters before and after 15-minute exposures to a static, 700-gauss magnetic field.

Initially, "it looked like nothing happened," Morris says, because the overall blood flow didn't appear altered. On closer inspection, however, she found that vessels that had initially been dilated became constricted, and those that had been constricted were dilated. Her team is now trying to find the mechanism for the switch. —J.R.

ENVIRONMENT

Prenatal nicotine: A role in SIDS?

Babies whose moms smoke during pregnancy are five times as likely to die from sudden infant death syndrome (SIDS) than are nonsmokers' infants, notes Ralph E. Fregosi of Arizona State University in Tucson. In studies with rodents, he and Zili Luo have now identified a possible explanation: Nicotine exposure in the womb may slow or even stop the firing of respiratory nerves that trigger breaths.

Earlier studies linked nicotine to SIDS (*SN: 9/14/02, p. 163*). To explore what the stimulant might be doing, the researchers implanted tiny pumps under the skin of female rats on the third day of their 3-week pregnancies. The pumps delivered either saline or nicotine—the latter, in amounts that yielded blood concentrations comparable to those in people who smoke two packs of cigarettes per day.

As each pup was born, the researchers removed the animal's brainstem and spinal cord and kept that tissue alive for 3 days. The nerves continued to fire signals that would normally trigger a newborn rat's diaphragm to contract, thereby initiating breaths. Luo recorded these signals before and after administering a drug that mimics gamma-aminobutyric acid (GABA), a natural brain chemical that keeps nerves from overfiring.

In the tissue from pups that had received saline, the drug blunted the breathing system slightly. In the tissue from nicotine-exposed newborns, neural activity dropped by 20 to 30 percent with the drug. In some instances, the breathing signals ceased.

Fregosi and Luo then counted the sites on brain cells where GABA would typically attach. The brainstems from nicotine-exposed pups had far more of these GABA receptors than the tissue from saline-exposed rats did. The researchers are now studying how long the prenatal nicotine renders nerves oversensitive to GABA. —J.R.

PHYSIOLOGY

Athletes develop whey-better muscles

Shops that cater to body builders sell large volumes of dietary supplements, especially products that combine the natural compound creatine with whey protein, a waste product of cheese making. Despite the supplements' popularity, "no study had actually examined [their] impact on muscle-fiber characteristics," notes Paul Cribb, of Victoria University in Melbourne, Australia.

His team recruited 33 men in their mid-20s, all highly trained bodybuilders, for a 13-week dietary trial. New data from the study confirm muscle-building benefits from the supplement combo.

The researchers divided their volunteers into four groups, giving each man the same caloric bonus per day: a flavored drink containing a gram of supplement per kilogram of bodyweight. One supplement contained just carbohydrates, another just whey powder, and the last two a mix of creatine with either carbohydrates or whey. Neither the athletes nor the scientists knew which supplement any volunteer received until the trial was over.

Throughout, the men performed supervised resistance training three times a week, and all experienced strength gains. However, men supplemented with whey made bigger gains than those getting just extra carbohydrates did. Adding creatine further boosted gains, with the whey-creatine supplement offering the biggest strength enhancement. The gains roughly correlated with increases in the cross-sectional area of type-II muscles—those that bulk up in response to exercise and produce maximum force, Cribb notes. In the men taking whey-creatine supplements, the cross-sectional area of such muscles increased 12 times as much as it did in the men getting just a carbohydrate drink.

Whey's bodybuilding benefits might also help elderly people, who typically lose muscle and strength over time, says Cribb (*SN: 8/10/96, p. 90*). Indeed, his colleagues are now investigating whey supplementation for the geriatric set. —J.R.

EPIDEMIOLOGY

Teen taters, too

Plenty of recent studies have chronicled an epidemic of obesity in the United States. Although adults have been chided for not exercising enough, many researchers have blamed the growing girth of adolescents on their love of junk food and soft drinks. Now, an analysis of government data on teens' health concludes that most obesity in adolescents, as in their elders, traces to insufficient physical activity.

Before conducting the study, nutritionist Lisa A. Sutherland of the University of North Carolina in Chapel Hill was puzzled, she says. Although national data indicated runaway obesity in young people, several studies suggested that calorie intake among teens has remained fairly constant. So, she plumbed 20 years of data on some 3,400 children, age 12 to 19, from a U.S. Department of Agriculture program known as the Continuing Survey of Food Intakes by Individuals. The results confirmed that over the past 2 decades, teens' calorie consumption has risen only 1 percent. Over that same period, others have reported, the prevalence of overweight U.S. teens has nearly tripled—to 15 percent.

For a measure of youthful activity, Sutherland consulted data on 12,400 teens collected by the Centers for Disease Control and Prevention. On the basis of self-reports, the numbers showed a 13 percent drop in physical activity during the past 2 decades. By 2000, Sutherland found, just 29 percent said they exercise regularly.

She now plans to probe gender differences in teens' exercise and eating habits. She hopes to make people aware of the implications of cutbacks by schools in physical education requirements and to figure out what interventions might be needed to compensate for teens' reduced activity. —J.R.

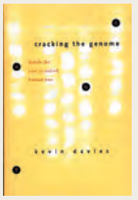
Books

A selection of new and notable books of scientific interest

CRACKING THE GENOME: Inside the Race to Unlock Human DNA

KEVIN DAVIES

As founding editor of the journal *Nature Genetics*, Davies has tracked one of the most important scientific stories ever to unfold, the mapping of the human genome. Firsthand accounts of work in the government program run by Francis Collins and in the private company Celera Genomics that was headed by Craig Venter provide historical as well as financial perspectives on the projects and the conflict between the public and private ventures. Commentary from leading scientists in the field helps readers understand how deciphering our genetic code will revolutionize medicine while posing ethical dilemmas. Originally published in hardcover in 2001. ***Johns Hopkins, 2002, 327 p., paperback, $17.95.***



DR. TATIANA'S SEX ADVICE TO ALL CREATION: The Definitive Guide to the Evolutionary Biology of Sex

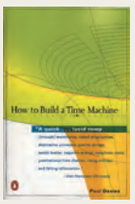
OLIVIA JUDSON

Move over, Ruth Westheimer and Joyce Brothers. Unlike the advice from those popular sex counselors, Dr. Tatiana isn't limited to the human lovelorn. She helps creatures—be they insect or mammal—through any number of sexual difficulties by applying her extensive knowledge in evolutionary biology. She fields questions from a stick insect whose lover is obsessed with her, a fig wasp that laments that all the males she knows bite their girlfriends in half, and a lion bemoaning the fact that his mate is a nymphomaniac. While the presentation is parodistic, the information Judson provides is a rigorous overview of the reasons different animals mate as they do and how their sex acts figure into the evolutionary process. Originally published in hardcover in 2002. ***Owl Bks, 2003, 308 p., paperback, $14.00.***

HOW TO BUILD A TIME MACHINE

PAUL DAVIES

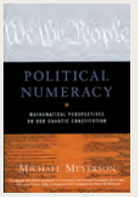
If you want to make your way to the past, then your best bet is to find a black hole conveniently equipped with a traversable wormhole. If the future is more your style, then get on a spaceship that travels just under the speed of light. With a tongue-in-cheek approach, renowned astrophysicist and science popularizer Davies shows how "a limited form of time travel is certainly plausible." Through lots of illustrations and straightforward text, he illustrates just how time-traveling contraptions would be built and what scientific principles would fuel them. Originally published in hardcover in 2001. ***Penguin, 2003, 131 p., b&w illus., paperback, $12.00.***



POLITICAL NUMERACY: Mathematical Perspectives on Our Chaotic Constitution

MICHAEL MEYERSON

How do the logical paradoxes revealed in Kurt Gödel's incompleteness theorem explain Kenneth Starr's investigation of President Bill Clinton? How does chaos theory provide insight into rulings handed down by the Supreme Court? Meyerson answers such questions by examining the Constitution and our laws through the lens of modern mathematics. While most people wouldn't compare politics to mathematics, Meyerson proves that the parallels are many and that math influences government. He considers, for instance, how the Electoral College functions. Even before the 2000 presidential election, many people judged this method for electing our leaders as severely flawed. However, Meyerson's mathematical analysis of other methods of election shows them to be flawed as well, and he argues that our current system is as good as any. Originally published in hardcover in 2002. ***Norton, 2002, 287 p., paperback, $14.95.***



PREHISTORIC AMERICA: A Journey Through the Ice Age and Beyond

MILES BARTON, NIGEL BEAN, ET AL.

Journey back in time 13,000 years and imagine the landscape and animals that the first North Americans encountered upon arrival. From the icy arctic waters to the steamy swamps of the Everglades, this book examines the continent region-by-region and introduces extinct creatures. Ground sloths rear their heads as high as a giraffe to graze on trees. The 5-foot-tall, almost 1-ton glyptodont looks like a rat covered in armor. Computer-generated images bring these animals to life alongside those that have survived, including cheetahs, salmon, and caribou. This is the companion book to a Discovery Channel television show. ***Yale U Pr, 2002, 192 p., color photos/illus., hardcover, $29.95.***

SECRET AGENTS: The Menace of Emerging Infections

MADELINE DREXLER

While the public consciousness is saturated with fears of bioterrorism, Drexler tries to shift the focus toward emerging natural pathogens. She points to diseases carried by animals and insects, antibiotic-resistant bacteria, foodborne pathogens, and lethal influenza as dangerous weapons in Mother Nature's arsenal. Chapters devoted to these subjects document occurrences of and responses to threats. Drexler reflects on outbreaks of swine flu, legionnaire's disease, and AIDS that took people by surprise in the past few decades. If we aren't better prepared, such history is certain to be repeated, she contends. Drexler discusses sophisticated epidemiological tools that are helping to identify some new infections before health emergencies occur. Originally published in hardcover in 2002. ***Penguin, 2003, 320 p., paperback, $15.00.***

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LETTERS

Know your limits

Phthalates ("Proof of Burden," *SN: 2/22/03, p. 120*) have been subject to significant regulatory scrutiny by governmental agencies. Centers for Disease Control and Prevention data show exposure levels well within the safety levels established by federal regulators. Further, a growing body of evidence indicates that studies using rodents may not be relevant to humans.

MARIAN K. STANLEY, AMERICAN CHEMICAL COUNCIL, ARLINGTON, VA.

Live wire

In reference to the hands-free headsets ("Hold the Phone? Radiation from cell phones hurts rats' brains," *SN: 2/22/03, p. 115*), it has been shown that on the wire to a cell phone, a standing wave can exist that can penetrate deep into the brain.

PETER L. NELSON, AVOCA BEACH, AUSTRALIA

Elephants, donkeys, and rats

"Dirty RATS: Campaign ad may have swayed voters subliminally" (*SN: 2/22/03, p. 116*) fails to mention the very plausible explanation that was offered at the time by those who produced the Republican campaign ad. Namely, that they were using a graphics-software program that produces the effect in question for any word. The validity of the research isn't affected by whether the appearance of "rats" in this manner was deliberate or not, but the validity of statements made in presenting this research certainly is. Talk of "negative campaigning" implicitly accuses the Republicans of deliberate use of subliminal messages, but that has never been established

VINCENT FITZPATRICK, MANVEL, N.D.

In the article, the string XXXX is called "a presumably neutral nonword." But the string XXX is standard for "poisonous," and a string of Xs is symbolic for what is crossed out or forbidden. Another example is the X- to XXXX-rated movie.

ELIHU LUBKIN, SHOREWOOD, WIS.

Researcher Joel Weinberger says that XXXX has been used in previous studies by many researchers. He admits, however, that he didn't think of the adult-movie connotation of XXXX. Future research may instead use strings of random letters. —S. PERKINS

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All letters subject to editing

CAN HONEY REPLACE DRUGS?

In 1989, an editorial in the respected British publication, the *Journal of the Royal Society of Medicin*e stated:

> The therapeutic potential of uncontaminated, pure honey is grossly underutilized. It is widely available in most communities and although the mechanism of action of its properties remains obscure and needs further investigation, the time has come for conventional medicine to lift the blinds off this traditional remedy and give it its due recognition. (Vol. 82, pp.384-385).

In the 14 years since this editorial appeared, there has been an outpouring of work on honey—not anecdotes, but solid scientific studies—that document the medicinal value of honey.

The medicinal benefits of honey are due to honey's antibacterial properties and to its moisture retaining properties.

Honey has been show effective against a wide range of bacteria, including Helicobacter pylori (the main cause of stomach ulcers, and implicated in a number of other maladies) and Staphylococcus aureus, aka Staph (drug resistant strains of this pernicious bacterium are causing problems in hospitals across the country).

Honey has been shown to provide relief for, or to cure, a number of different disorders, including, but not limited to the following:

Diarrhea, ulcers, infections, irritable bowel syndrome (IBS), gastrointestinal problems, and staphylococcus (staph) infections.

Infectious diseases caused by bacteria that are sensitive to Honey include the following:

Anthrax, diphtheria, urinary tract infections, ear infections, meningitis, respiratory infections, sinusitis, pneumonia, tuberculosis, infected animal bites, typhoid, dysentery, abscesses, boils, carbuncles, impetigo, tooth decay, puerperal fever, rheumatic fever, sore throat and cholera.

A number of different types of wounds have been successfully treated with Honey, including:

abrasions, amputations, abscesses, bed sores, burns, burst abdominal wounds following cesarean delivery, cancrum, cervical ulcers, chilblains, cracked nipples, cuts, diabetic foot ulcers and other diabetic ulcers, a fistula, foot ulcers in lepers, infected wounds arising from trauma, large septic wounds, leg ulcers, malignant ulcers, sickle cell ulcers, skin ulcers, surgical wounds, wounds to the abdominal wall and perineum, varicose ulcers.

The drug industry spends billions on advertising and promotion—$7 billion on sales representatives alone (*The New Republic*, December 16, 2002), billions more on print and TV ads. In contrast, the total sales of honey in the U.S. are miniscule—well under a billion dollars. The drug industry has a powerful lobby in Washington D.C., the honey industry has none. The "side effects" portion of drug information often runs into thousands of words; there are no side effects for honey. The advertising budget for honey is next to nothing. The positive results of clinical studies on honey are truly amazing—if drug companies had results like this you'd be bombarded with the data. Now, a new book details the amazing, proven benefits of honey.

HONEY *The* Gourmet Medicine

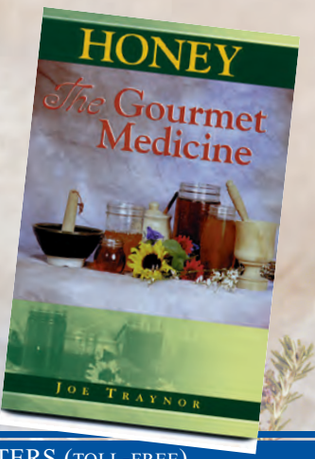
by Joe Traynor

"...an eye opener...especially pertinent in this time of multidrug-resistant bacteria."

—Daniel Blodgett, M.D.

"…very informative and well-written. I highly recommend this book for everyone, especially medical professionals."

—Christopher M. Kim, M.D.



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