

# NATURAL HISTORY

6/01



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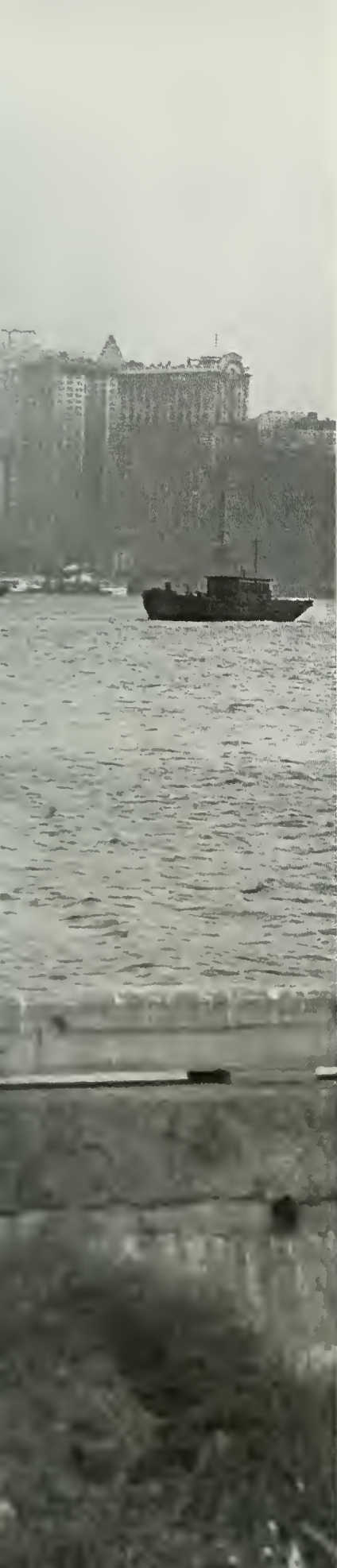
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# NATURAL HISTORY

JUNE 2001

VOLUME 110

NUMBER 5

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**COVER** Our bodies have two kinds of genomes. The larger one (recently sequenced) is found in the center of each of our cells. But a different set of genes, of bacterial ancestry, dwells in the mitochondria, the cells' powerhouses. Our innards and skin also host hundreds of microbial species, each with its own genome. Artist **Alexis Rockman** adds mitochondria (green ovals) and bacteria (spirals and rods) to a fanciful "portrait" of our species.



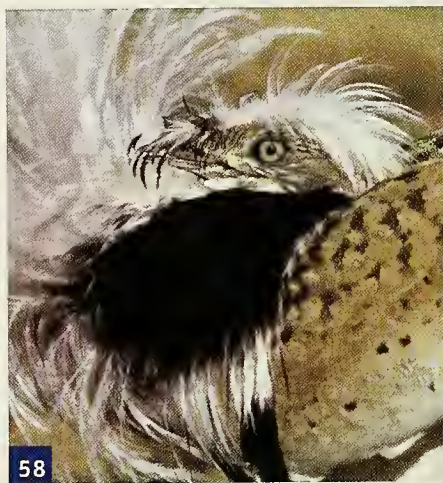




## A NOSE FOR ALL REASONS

A sampler of various and versatile snouts

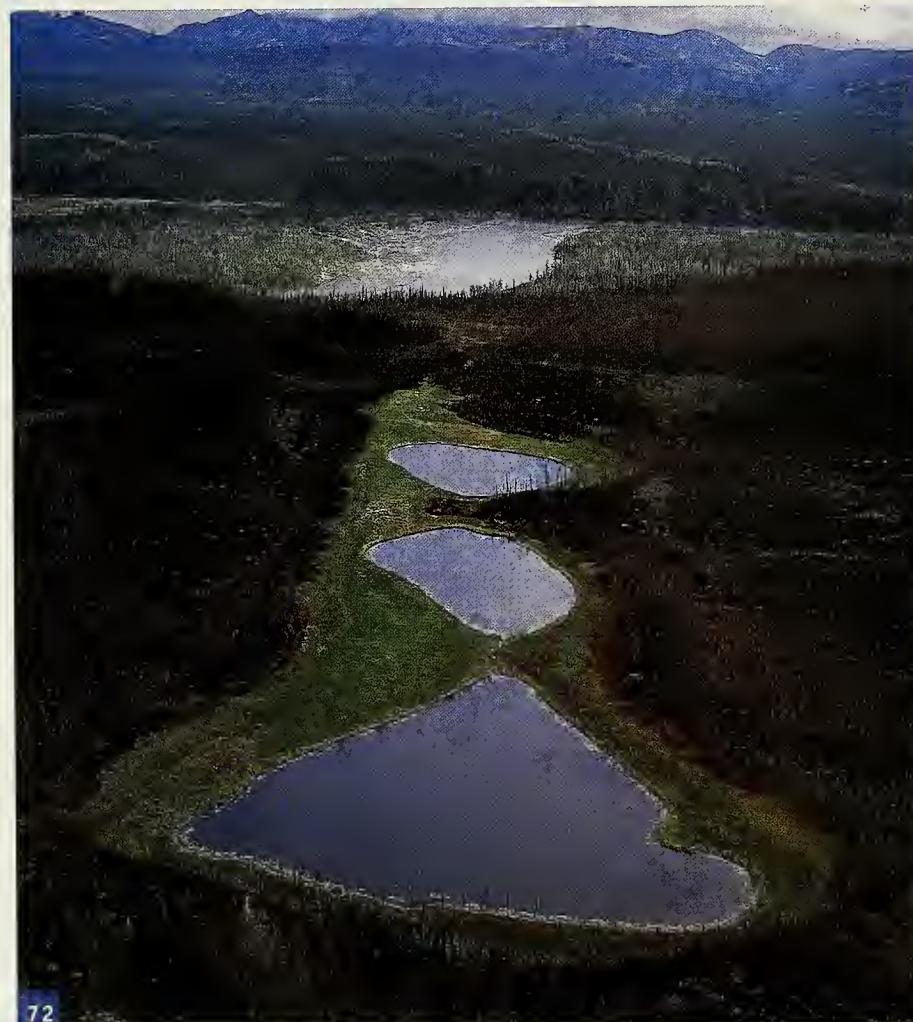
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Trusting, hand-raised birds go to desert survival school.

YOLANDA VAN HEEZIK AND  
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## UP FRONT

# Lifestyles of the Small and Obscure

Science, a product of the human mind, has delivered repeated blows to the human ego. Copernicus removed us from the center of the solar system. Darwin displaced us from near-angelic status by sticking us on a quite ordinary, though relatively new and green, branch of the animal family tree. Edwin Hubble informed us that the Milky Way is an undistinguished galaxy in a nondescript corner of the universe. Then came the Human Genome Project. When a draft of our genome was published in February of this year, we learned that we don't even have as many genes as we thought we did. The estimated 100,000 was pared down to a mere 33,000—not all that much bigger than the genome of a mouse (28,000), a fruit fly (23,000), or a nematode (22,000).



Of course, numbers aren't everything, but these findings do bruise our vanity.

So far, gene-sequencing professionals have devoted most of their attention to bacteria, whose genomes typically have between 1,500 and 4,000 genes. And as the most abundant (and arguably the most influential) organisms on Earth, bacteria deserve the limelight. About 2 billion years ago, they made complex life possible by getting themselves incorporated

into larger cells and going on to do the jobs of respiration and photosynthesis in animals and plants. (Plants and animals, including humans, still carry bacterial descendants in each cell, as Lynn Margulis and Dorion Sagan explain in "The Beast With Five Genomes," page 38.)

Genomic studies can tell us something about how bacteria behave in human bodies and other habitats. Turn to "Bacterial Revelations" (page 52) for new insights into the obscure lifestyles of several germs—including the tuberculosis bacillus, the typhus pathogen, and the sea-dwelling *Prochlorococcus marinus*, the world's smallest and most common photosynthetic bacterium.—Ellen Goldensohn

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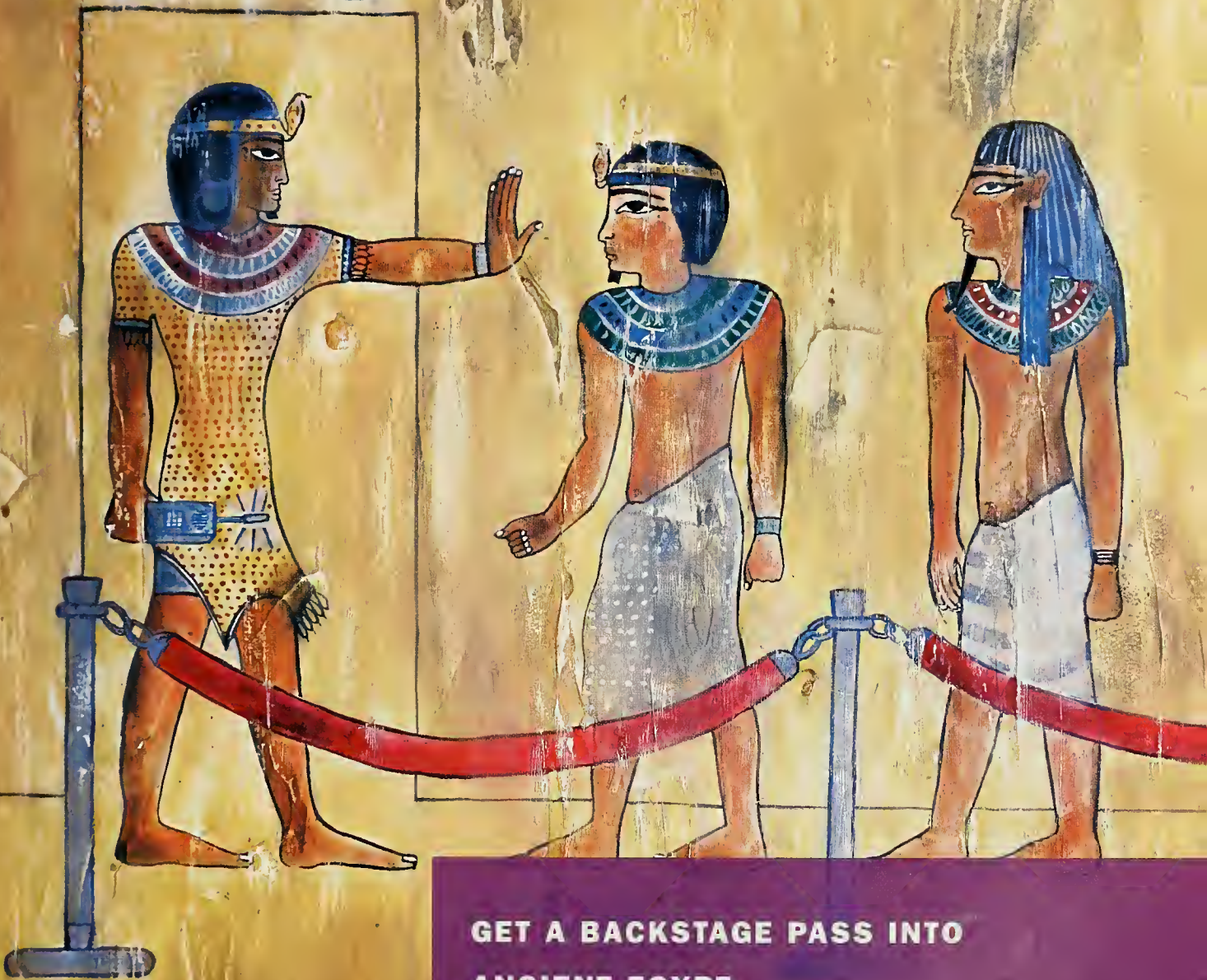
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## LETTERS

### O Pioneers!

"The Scavenging of 'Peking Man,'" by Noel T. Boaz and Russell L. Ciochon (3/01), was fascinating. However, Helmuth Zapfe's studies on cow bones fed to captive hyenas were not, as the article states, pioneering. William Buckland, the first professor of geology at the University of Oxford, conducted the same experiments in Oxford in 1821 by feeding ox bones to a hyena in a traveling menagerie. He compared the fragmented bones with bones found in various caves in England, which he interpreted as having been ancient hyena dens—at the time, a most controversial view. Buckland published detailed comparative engravings of the bones. His work and experimental approach were truly pioneering.  
*Neville Haile*  
*Oxford, England*

### Heaven ...

Could authors Scott L. Wing ("Hot Times in the Bighorn Basin," 4/01) and Kenneth D. Rose ("Wyoming's Garden of Eden," 4/01) be a little more specific as to where in the Bighorn Basin their studies take place? The basin is paleontology heaven, with excellent outcroppings of everything from Precambrian to Tertiary age in the Wind River Canyon, at the southernmost part of the basin, to huge dinosaur finds in Thermopolis and

outstanding petroglyphs at Cottonwood Creek near Hamilton Dome.

*Steve Leece*  
*Baguio City, Philippines*

SCOTT WING REPLIES: "Paleontology heaven" is the best two-word description I have heard for the Bighorn Basin. The Paleocene and Eocene fossils and rocks that we study are near Cody and other towns in the central portion of the basin. Within that area are literally thousands of fossil locations. Almost all of these are away from major rivers, dry creeks, and sagebrush flats, where recent sediments tend to cover the older rocks.

### ...and Hell

Scott Wing matter-of-factly writes of the Eocene global warming as being "perhaps as rapid as the one we humans are about to cause and experience." I don't believe there is enough evidence to support such a strong indictment of humanity. There have been many other documented ice ages in the last 100,000 years and rapid warming and cooling spells in the interglacial periods. The fact is that little, if anything, is known about what causes climatic change on a large scale. To blame humanity is harsh and premature.

*Kent K. Smith*  
*via e-mail*

SCOTT WING REPLIES: Kent Smith correctly states that

rapid climate change has occurred in the absence of human activity. That was what my article was about, so I clearly do not "blame humanity" for all climate change. That said, the addition of CO<sub>2</sub> and other greenhouse gases to the atmosphere through human activities is well measured and documented, and I am not aware of any scientists who dispute that these gases lead to an increase in temperature near the earth's surface. The exact amount of warming that will be generated, the possible role



KENNETH D. ROSE

of countervailing factors, and the effects of climate change on plants and animals are poorly understood at present. That's one reason why research into the history of climate change is important and why working on these problems is prudent planet management—the business we humans are getting into, whether we admit it or not.

### Interesting Point

Steven N. Austad's review of *The Quest for Immortality: Science at the Frontiers of Aging*, by S. Jay Olshansky and Bruce A. Carnes

(4/01), does a good job of highlighting the two sides of the anti-aging debate, but his description of a bet with Olshansky overstates the miracle of compounding. The author reckons that their \$300 bet will have become \$500 million in the year 2150. Not likely. Eight percent rather than 10 percent annual interest is probably more realistic, and that yields a more modest figure of \$30 million. Figuring in 3 percent inflation, we wind up with a much less impressive \$450,000 at the end of those 150 years.

*Stuart Robinson*  
*via e-mail*

### STEVEN AUSTAD REPLIES:

The miracle of compound interest is pretty evident, given that a mere 2 percent difference in our interest-rate assumptions leads to Stuart Robinson's calculation of my wager as yielding a mere \$30 million, compared with my estimate of \$500 million (or more than \$7 billion if the interest rate is 2 percent higher). I trust that my guess, using seventy years of history as a guide, has as much credibility as Robinson's—that is, very little. Regardless of inflation, \$30 million or \$500 million or \$7 billion ought to at least buy my descendants a good night on the town to celebrate their farsighted ancestor.

*Natural History's e-mail address is nhmag@amnh.org.*



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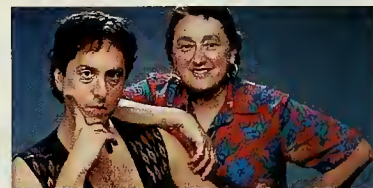
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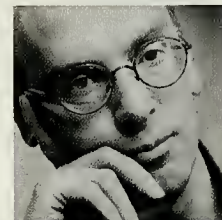


## CONTRIBUTORS

As a graduate student in genetics at the University of Wisconsin, **Lynn Margulis** ("The Beast With Five Genomes," page 38) learned about "cytoplasmic genes"—such as those that determine green color in plants—and recognized that they belonged to microbes that were once free living. Now a Distinguished University Professor at the University of Massachusetts Amherst, she is still exploring the implications of such ancient mergers. Margulis has collaborated with writer **Dorion Sagan** on numerous books, including *Slanted Truths: Essays on Gaia, Symbiosis and Evolution* (Springer-Verlag, 2001). Their collaborative work is accessible at [www.sciencewriters.org](http://www.sciencewriters.org). Sagan is now coauthoring a book on the evolution of intelligence.



**Mark Ridley** ("Sex, Errors, and the Genome," page 42) started gravitating toward a career in the natural sciences in his midteens, after hearing a lecture by famed marine biologist Alistair Hardy. Ridley's fascination with genes and evolution developed at the University of Oxford, where he first encountered the ideas of Richard Dawkins. Now himself a researcher in Oxford's zoology department, Ridley is the author of several books, including *The Cooperative Gene* (Free Press, 2001). Despite his penchant for theory, Ridley still enjoys old-fashioned natural-history pursuits such as identifying birds, bugs, and flowers.



**Roberta Friedman** ("Bacterial Revelations," page 52) grew up in Queens, New York City. She received her Ph.D. in pharmacology from Vanderbilt University in Tennessee, where she researched the neurotransmitter serotonin, but realized that she much preferred writing about research to carrying it out. For the past fifteen years, she has written on science and medicine for a variety of publications. A resident of Santa Cruz, California, Friedman has also written on science for children, inspired lately by her three boys. Now ready for a third career, she makes pottery and hopes to set up her own studio soon.



**Yolanda van Heezik** and **Philip Seddon** ("Born to Be Tame," page 58), coauthors and spouses, spent nine years at the National Wildlife Research Center in Taif, Saudi Arabia, helping to reintroduce houbara bustards into the desert. The pair met at the University of Otago in New Zealand, where both did their doctoral work. They wrote about jackass penguins in South Africa for the November 1997 issue of *Natural History*. Now back in New Zealand with their four-year-old son, Connor, Heezik (left) is a lecturer in zoology and Seddon (see page 61) is director of the wildlife management program at the University of Otago. Sophie the fox now lives "in a state-of-the-art captive breeding facility in the United Arab Emirates and may soon embark on a new career as a mother."

Associate professor of anatomy at the Ohio University College of Osteopathic Medicine in Athens, **Lawrence M. Witmer** ("A Nose for All Reasons," page 64) has "one foot in the Mesozoic and the other in the present, studying living species to learn about dinosaurs and other extinct beasts." To that end, he has made CAT scans of everything from tapirs to *Allosaurus* and, with his students, dissected all sorts of animals (currently in the freezer are two rhinoceros heads, two manatee heads, monitor lizards, seals, and birds). To colleagues who complain about the odor coming from his lab, he replies, "Sometimes science stinks."



After two decades of traveling on assignment for *National Geographic*, photographer **Jim Brandenburg** ("The Natural Moment," page 84) decided to settle down next to a million-acre wilderness in his native Minnesota. Coming upon the tracks of a wolf or a lynx gives him even more pleasure, he reports, than did his former adventures in the Namib Desert or the forests of Manchuria. Brandenburg's books include *White Wolf: Living With an Arctic Legend* (Northword Press). He has established a nonprofit gallery of his work in his hometown of Luverne; the proceeds go to acquiring and preserving the tallgrass prairie near his boyhood farm there (see [www.jimbrandenburg.com](http://www.jimbrandenburg.com)).



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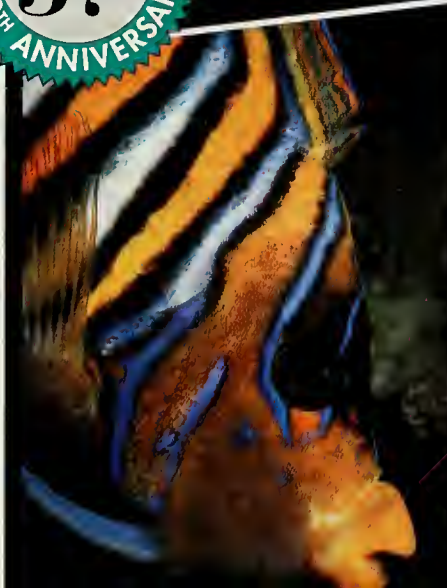
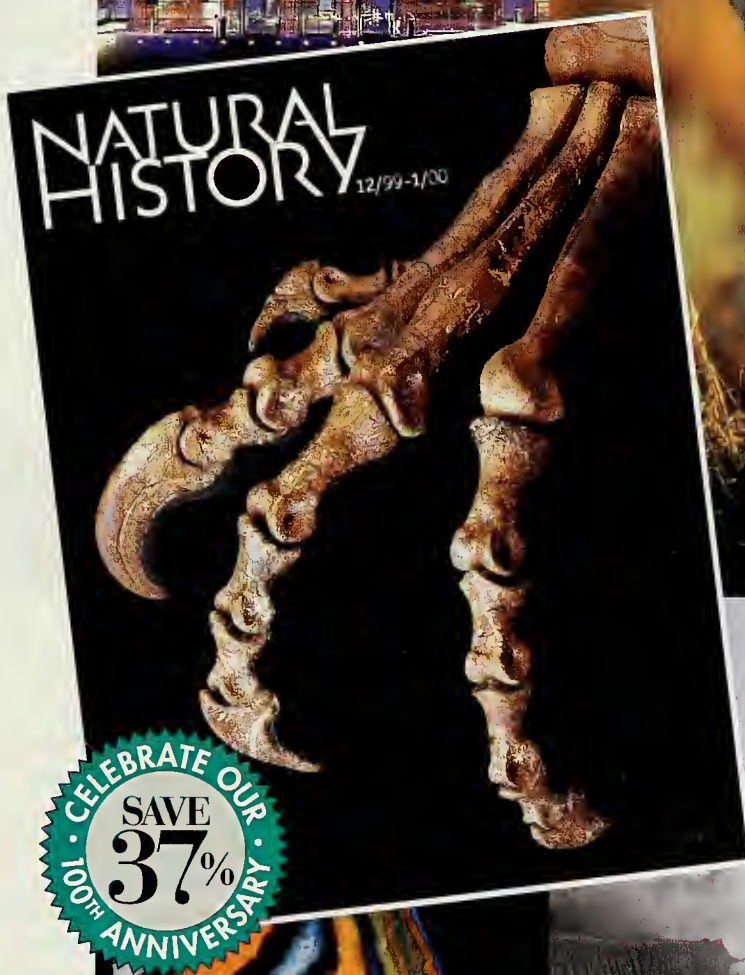
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## FINDINGS

# THE CHIMERIC SELF

Cellular traffic between mother and fetus raises questions about the causes of autoimmune disease.

By J. Lee Nelson

One of the unsolved mysteries of immunology is why the body of a pregnant woman doesn't reject her fetus. After all, our immune system evolved to keep foreigners out, to maintain a clear distinction between self and other. In recent years, the mystery deepened as researchers learned that fetal cells get into the maternal bloodstream during pregnancy and, what's more, may stay there for decades, perhaps indefinitely. What might this mean for how we think about autoimmune disease?

The traditional view of autoimmunity is that it is a case of mistaken identity: a body, often for no apparent reason, fails to recognize some of its own tissues and mounts an attack against it-

self. There is no evidence, for example, that rheumatoid arthritis sufferers had anything wrong with their joints, or with the tissues lining the joints, that might have led to the onset of the disease; their bodies appear to have simply turned on themselves. The indefinite persistence of fetal cells in a woman's body, however, led me to ask if some so-called autoimmune diseases may be triggered by foreign cells, specifically by fetal cells present in the mother's body. After several years of looking into this question, I recently proposed that what we may be dealing with, in fact, is a kind of chimerism, though on a microscopic scale.

In Greek mythology, the chimera was a fire-breathing creature with the

head of a lion, the body of a goat, and the tail of a serpent. In modern medicine, the term "chimerism" refers to an organism whose body contains populations of cells derived from another individual—a less grand but, to researchers like myself, equally compelling notion. Microchimerism is said to exist when the number of nonhost cells is very low (for example, fewer than one foreign cell for every million host cells).

One observation that led me to suggest a connection between microchimerism and autoimmune diseases was that these illnesses—among the most familiar of which are rheumatoid arthritis, systemic lupus erythematosus, and multiple sclerosis—usually afflict







ARCHAEOLOGICAL MUSEUM, FLORENCE; SCALA/ART RESOURCE, NY

Arezzo chimera, bronze, sixth century B.C.

nancy was an obvious one, and I was intrigued by the possibility that women are uniquely subject to a kind of reverse inheritance from their children.

Men and children, as well as women who have never been pregnant, also develop autoimmune diseases, but microchimerism could play a role in these populations as well, because there are several ways people may wind up, perhaps indefinitely, with cells that aren't their own. During pregnancy, for instance, the mother's cells can also pass into the developing fetus. In addition, cell transfer can occur between twins in utero. Blood transfusions are another pathway for cell transfer: although the donor's red blood cells—which have no nucleus and are short-lived—are soon cleared from the recipient's circulation, it turns out that the donor's longer-lived, nucleated white blood cells can persist within the recipient.

Further sparking my interest was the knowledge that leukemia and lymphoma patients who undergo bone marrow (stem cell) transplantation often develop a syndrome known as chronic graft-versus-host disease, with symptoms very much like those of certain autoimmune diseases, especially those of scleroderma. This syndrome occurs most often when the donor's cells are not perfectly matched to the recipient's.

Much of my own research—done in collaboration with colleagues at the Fred Hutchinson Cancer Research Center in Seattle and with Diana Bianchi and her team at the New England Medical Center in Boston—has focused on scleroderma. This insidious illness begins with hardening of the skin on the fingers and toes and slowly marches to the arms, legs, face, and trunk. Advanced hardening can result in skin ulcers and the loss of fingers or toes. No treatment exists to reverse scleroderma, and when it moves to the digestive system, kidneys, heart, and lungs, it is often fatal.

To investigate the possible role of microchimerism in scleroderma, we

took blood samples from two groups of women: scleroderma patients and healthy “controls.” All the women had previously given birth to a son (mothers of sons were chosen because of the availability of a simple test to detect male DNA in a female host). We found that the amount of male DNA in a tablespoon of blood was significantly greater in the scleroderma patients—equivalent to as many as sixty-one male cells, compared with a maximum of two in the healthy women. The women in our studies had borne their sons many years earlier, yet the levels of male DNA in some of the scleroderma patients exceeded those found in pregnant women carrying a normal male fetus. Working with skin biopsy samples, other investigators subsequently produced similar results.

As exciting as these results were, the mere presence of foreign DNA is not sufficient to explain scleroderma. By using very sensitive techniques, we were able to detect fetal cells in the immune systems of more than 60 percent of the healthy women we tested—present in smaller numbers than in the sick women, but there nonetheless. Why, then, do I think that such cells may be implicated in some instances of disease but not others?

The answer, I believe, lies in the genetic relationship between a mother and her child, specifically in how closely matched their *HLA* genes are. Present in almost all the nucleated cells in our body, *HLA* genes are a crucial part of our immune system, vital to the body's ability to distinguish self from other. A clear mismatch between the *HLA* genes of a pregnant woman and her fetus appears to be a good thing, because the mother's immune system will then have no trouble recognizing as foreign any fetal cells that may cross over into her bloodstream. If maternal and fetal *HLA* genes are a perfect match, cell movements from one to the other will also not be a problem, since there would in

more women than men, sometimes at ratios of greater than ten to one. Furthermore, many of these diseases strike most often in late middle age. Three such examples are thyroiditis (an inflammation of the thyroid gland), primary biliary cirrhosis (inflammation and destruction of the liver), and scleroderma (progressive hardening of the skin and internal organs). If these diseases hit earlier in life, when hormonal differences between men and women are so striking, the logical assumption would be that sex hormones were somehow implicated. But in part because they tend to come on later in life (often not developing until after menopause), I looked for other differences between males and females. Preg-



fact be nothing foreign to worry about. But what if, my colleagues and I wondered, the *HLA* genes of mother and child are not identical but very similar? Might this make it easier for the foreign cells to slip past the mother's immune system undetected and remain active? If so, might this somehow put the mother at risk of developing an autoimmune disease later in life?

To find out, we looked at the *HLA* genes of women with scleroderma and those of their children.

Such studies can be confusing because of the many variations on the theme of matched and mismatched genes. "Foreignness" quickly be-

comes a matter of perspective. For illustration's sake, imagine a mother who has *HLA* genes *A* and *B* and a father who has *A* and *C* (in reality, of course, many more genes than this are involved). If both the mother and the father happen to pass on the *A* gene, then the fetus—with its two *A* genes—will not seem foreign to the mother, who has an *A* gene of her own. From the point of view of the fetus, however, the mother's *HLA B* gene will register as foreign.

In this study, we concentrated on *HLA-DRB1*, a gene known to be involved in scleroderma and in many other autoimmune illnesses. What we found was striking: scleroderma patients were nine times more likely than were healthy mothers to have given birth to a child whose *HLA-DRB1* genes were very similar to theirs. And when, as in the hypothetical example above, foreignness existed only from the perspective of the child, the mother then appeared to run as much as a nineteen-fold risk of developing this devastating disease.

Fetal cells and fetal DNA have been found by researchers investigating other autoimmune diseases as well—for example, according to one

preliminary report, in the salivary glands of some patients with Sjögren's, or sicca, syndrome (in which inflammation results in dry eyes and a dry mouth) and in the livers of patients with primary biliary cirrhosis (although, complicating matters, it was also frequently found in the livers of patients with other non-autoimmune diseases). Furthermore, two recent papers on myositis, a degenerative muscle condition, reported finding more maternal cells in children with this

## Women are uniquely subject to a kind of reverse inheritance from their children.

disease than in those without. Our scleroderma team has detected maternal cells in adult children, some of whom were scleroderma sufferers and others not. Other researchers have found they could induce lupus in laboratory mice by injecting parental cells into offspring, suggesting that maternal cells could be involved in this disease as well.

If foreign cells are indeed involved in autoimmune disease, why is there often such a long lag between the time of cellular transfer and the onset of illness? The most likely explanation is that these cells may, under the right genetic circumstances, predispose a person to an illness, but that some kind of triggering event—exposure to an infectious agent, for example, or an environmental toxin—must also occur in order for the disease to develop.

At this stage, we don't yet know *how* one person's cells cause malfunctions in the body of another. We think it less likely that damage results from a direct attack on host tissues than that the foreign cells operate like a computer virus, disrupting the sensitive network of communication between cells responsible for immune regulation in the body of the host.

The study of microchimerism in human health and disease is a new frontier. My research has convinced me that the movement of cells from one individual to another will prove to be an important factor in at least some diseases. There are, however, other interesting (and not mutually exclusive) possibilities. Persistent foreign cells—whether fetal or maternal—may sometimes be neither detrimental nor neutral, but beneficial. Some studies, for instance, have sug-

gested that once a woman has given birth, she may in fact have a reduced risk of developing rheumatoid arthritis, and we are currently investi-

gating the possibility that reverse inheritance may be providing some protection here. And we have found that when fetal and maternal cells are genetically mismatched in a particular way, a woman who already has rheumatoid arthritis may sometimes enjoy remission from the disease during her pregnancy.

Future research will undoubtedly reveal much more about the cellular traffic between mother and fetus, including whether this form of microchimerism is simply an unavoidable side effect of pregnancy or whether—if beneficial effects should prove to outweigh the negative ones—it might actually have been the target of natural selection. Obstetricians may never feel the need to hand out pamphlets entitled "Warning: The genes you don't inherit may be harmful to your health." But one thing is certain: microchimerism research has shed new light on the age-old question "Who am I?"

*J. Lee Nelson is an associate member in the Program of Human Immunogenetics at the Fred Hutchinson Cancer Research Center and an associate professor in the Division of Rheumatology of the University of Washington, Seattle.*

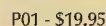


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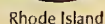
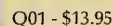
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## IN SUM

**SNAKY FAKERY** According to a theory of mimicry promulgated by English Victorian naturalist Henry Walter Bates, some harmless creatures warn away predators by evolving a strong resemblance to poisonous ones. Thus, several kinds of king snakes mimic the venomous coral snake's distinctive pattern of alternating red, black, and yellow or white bands. Now an experimental field study has demonstrated that the king snakes can get away with the charade only if coral snakes inhabit the same locality.



Sonoran mountain king snake

Western coral snake

David W. Pfennig, of the University of North Carolina at Chapel Hill, and colleagues predicted that the protective effect of looking like a coral snake would break down in areas where the genuine article was absent. The researchers placed a total of 1,200 plasticine models of snakes at forty different woodland and desert locales in two wilderness areas, one in Arizona and another in North and South Carolina. About half the models in each area were placed inside coral snake territory, and half were placed outside it. When raccoons, coatis, foxes, coyotes, skunks, or bears bit the models, they left tooth marks in the plasticine.

Since there were far fewer attacks on king snake mimics within the ranges of real coral snakes, Pfennig believes that predators in these areas have developed an innate avoidance of snakes with bright-colored band patterns. How these mammals acquire their instinctive fear remains a question, as a single coral snake bite might well remove the "learner" from the gene pool. ("Frequency-

Dependent Batesian Mimicry," *Nature* 410, 2001)—Richard Milner

**EAU DE GENES** Scientists and lovers alike have long known that fragrance plays a role in sexual communication. Now, research done by Manfred Milinski and Claus Wedekind while they were at the Universität Bern in Switzerland suggests some evolutionary explanations for odor preferences.

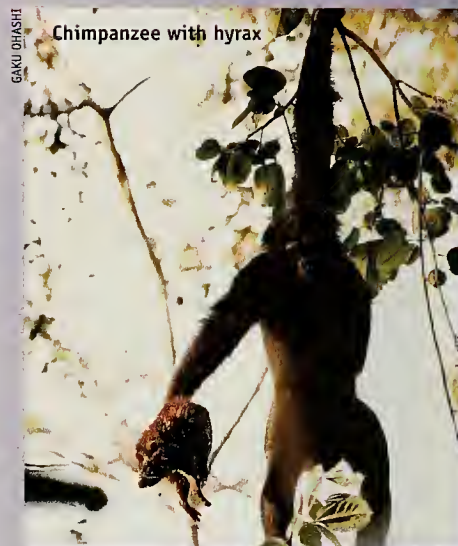
A set of genes involved in both scent recognition and conferring immunity to infection—known as the major histocompatibility complex (MHC)—is widespread in vertebrates. Both mice and humans, for example, have been shown to prefer the body odor of partners that possess MHC genotypes different from their own.

Milinski and Wedekind asked both women and men to assess various scents, indicating whether they would "like to smell like that" themselves or whether they would like to smell them on a partner. There was a positive correlation between the subjects' own MHC genes and the scents they chose for themselves—but a negative correlation for fragrances they selected for potential partners.

The researchers believe that people prefer fragrances that amplify—rather than mask—their natural body odor. By using perfumes that broadcast their genetic makeup to potential mates, and by selecting partners whose MHC genes are dissimilar to their own, individuals may reduce inbreeding as well as susceptibility to certain diseases. ("Evidence for MHC-Related Perfume Preferences in Humans," *Behavioral Ecology* 12, 2001)—Kirsten L. Weir

**PLAY OR PREY** If there's one fact emerging from all the field studies of chimpanzees, it is that chimp behavior and customs ("cultures") differ widely from place to place. Communities vary in their greeting behavior, use of tools, and food preferences. Because chimps are so closely related to us, primatologists carefully document any unusual behavior.

One recurrent question concerns the extent of ape carnivory. Observers have verified that chimp populations at Mahale Mountains Wildlife Research Centre in Tanzania kill ten differ-



Chimpanzee with hyrax

ent species of mammals and eat them, while other chimp groups at Bossou, in Guinea's southeastern corner, hunt much less frequently and prey only on tree pangolins. Still other communities are almost entirely vegetarian.

Satoshi Hirata, Gen Yamakoshi, and colleagues from Kyoto University in Japan have reported incidents in which chimps at Bossou captured and killed western tree hyraxes but did not eat them. At Bossou, they recently observed a mixed-sex group, including the alpha male, scream excitedly and then attack a hyrax that fell out of a tall tree.

In another incident, an adolescent male was seen moving through a tree swinging a live hyrax, then slamming it against the tree's branches. An eight-year-old female soon got hold of the motionless carcass, swung it in the air, and carried it. She finally settled into a tree nest, where she slept with the dead hyrax all night; the next morning, she groomed its fur, carried it around, and eventually let it drop to the ground.

Although chimps at Mahale do consume hyraxes, those at Bossou either let them go or kill them without eating them. Bossou chimps, which only rarely interact with hyraxes, apparently do not consider them prey. For further information, go to [www.pri.kyoto-u.ac.jp/chimp/Bossou/Bossou.html](http://www.pri.kyoto-u.ac.jp/chimp/Bossou/Bossou.html). ("Capturing and Toying With Hyraxes (*Dendrohyrax dorsalis*) by Wild Chimpanzees (*Pan troglodytes*) at Bossou, Guinea," *American Journal of Primatology* 53, 2001)—Richard Milner



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## CELESTIAL EVENTS



Ancient Peruvians reacting to a solar eclipse, nineteenth-century illustration

LEONARDO DE SELVA/CORBIS

your lifetime, chances are you are going to have to go to it.

Which helps explain why eclipse cruises and land expeditions have become popular—and the best such tours not only get you there but also provide lectures and briefings on what to expect when totality arrives. That's not to say that the eclipse cruise I took in 1999 didn't have a casino or an onboard band featuring "The Girl From Ipanema" in its repertoire. Nor was every passenger "chasing totality," as eclipse veterans like to say: on the morning of the main event, fifteen passengers passed up the final and most extensive preparatory briefing in favor of a napkin-folding tutorial. Still, those of us who did go to the middle of the Black Sea for a good look at what some

tour organizers were billing as "the last total solar eclipse of the millennium" found ourselves trying to get the most out of our two minutes and twenty-one seconds.

Among the phenomena any observer of a total solar eclipse can anticipate are

**Weather.** After the edge of the Moon meets the edge of the Sun, the temperature may begin to drop noticeably. Aboard our ship, idling in the August heat of the Black Sea, it fell 21° F altogether, to 83° at totality.

**Shadow.** Just before the beginning of totality, the shadow of the Moon will visibly race across the landscape—in our case, the calm surface of the sea—from the west.

**Baily's beads.** When the last rays of the Sun poke through the valleys

# There Goes the Sun

Witnessing an eclipse today may not be the mystical experience it once was, but it's no less impressive.

By Richard Panek

What you'll see," one of the cruise ship's official guides was saying, "you won't be able to describe to your family. People cry. People scream. People babble." An unofficial guide—the director of a planetarium back in the States and one of our fellow passengers—had a more frankly romantic interpretation of what we were about to experience: "I equate it with love."

Can any natural event (even love) possibly live up to such advance billing? The passengers aboard a special cruise on the Black Sea in August 1999 were about to find out—as will people taking similar sea cruises or land expeditions in Africa this month, when the moon once again totally eclipses the Sun. Such an event isn't particularly rare. Unlike spectacular

comets, which tend to streak into view only once or twice a decade, total solar eclipses occur on average about once every other year. What makes them *seem* so rare, however, is their inaccessibility.

You can witness the Moon precisely superimposing itself upon the Sun only if you happen to be in the right place at the right time. The right place—the path of the Moon's umbral shadow—is 170 miles across at its widest, and the right time—totality itself—can be seven minutes, thirty-one seconds at the longest and usually lasts several minutes less. A total solar eclipse will be visible at a given place on Earth only once about every 375 years on average, so if you want to see any of the three dozen or so such events that are going to occur during



along the perimeter of the Moon's disk, they create an effect that nineteenth-century British amateur astronomer Francis Baily described as "a string of bright beads."

**Diamond-ring effect.** The final Baily's bead appears together with the visible band formed by the solar corona.

**Wildlife.** Birds and beasts will be responding, perhaps starting to bed down for the "night" as the sunlight dims. But the response of human beings will be no less notable. As my fellow cruise passenger Robert J. Bonadurer, director of the Minneapolis Planetarium, said, "You can tell yourself that you don't believe the world is going to come to an end—but you *do*. And then you understand people shooting arrows at the Moon."

To be sure, a total eclipse of the

Sun is not without its scientific applications, whether it's Arthur Eddington using photographic images of the 1919 eclipse to help validate Einstein's general theory of relativity or today's astronomers monitoring the event to view the corona, the highly ionized gases surrounding the Sun. But the psychological impact of seeing the perfect fit between Moon and Sun—the only such coincidence visible from the surface of a planet in our solar system—is what casual observers remember.

They'll be watching this month (see "The Sky in June," below, for details). And if you need any evidence that the impact of a solar eclipse is more psychological than scientific, consider what happened on our cruise immediately after totality. As the sight of the Moon creeping across the

Sun—the spectacle that minutes earlier had awed everyone—continued to play itself out, only in reverse, hardly anyone paid attention. The band resumed playing; the totality chasers drank champagne and danced. Off to one side, Anthony F. Aveni, professor of astronomy and anthropology at Colgate University and one of the official onboard experts, noticed that our ship as well as several others in our immediate vicinity were already barreling back across the Black Sea. "We're all heading for the Bosphorus," Aveni laughed. "We've abandoned the midline. What a bunch of eclipse hypocrites we are!"

*Richard Panek is the author of Seeing and Believing: How the Telescope Opened Our Eyes and Minds to the Heavens (Penguin, 1999).*

## THE SKY IN JUNE

By Joe Rao

**Mercury** swings between Earth and the Sun this month, reaching inferior conjunction on June 16. This is not the best month for Mercury hunters, as their target will be cloaked by the blinding solar glare.

**Venus**, by far the brightest of the planets, rises every morning just before dawn. At twilight it is unmistakable as it ascends in the east. The planet attains its greatest elongation as early as June 8. On the 17th the Moon appears well below and to the right of Venus; on the 18th the Moon is a similar distance below and to its left.

**Mars** dominates the night skies this month, reaching opposition on June 13, when it shines at an eye-popping magnitude of -2.4. On June 21 it is only 41.8 million miles away, its closest approach to Earth since October 19, 1988. Mars retrogrades among the stars of Ophiuchus all month. At 40° north latitude, it rises about an hour after

sunset at the beginning of June and appears well to the right of an almost full Moon, rising together with it on the 6th. By the 13th the fiery planet rises as the Sun sets, and by month's end, it is already well above the east-southeastern horizon at dusk.

**Jupiter's** solar conjunction occurs on the 14th, and the planet is invisible for most of June. At month's end, it might be glimpsed just above the east-northeastern horizon about forty-five minutes before sunrise. This marks the beginning of a yearlong apparition, when the giant planet will blaze within the stars of Gemini.

**Saturn** will be too close to the Sun during the first two weeks of June to be seen, but after midmonth, it begins to emerge low in the east-northeastern sky about two hours before sunrise. On the morning of the 19th the yellowish planet will be below and to the left of the Moon's crescent.

**The Moon** is full on June 5 at 9:39 P.M. Last quarter comes on June 13 at 11:28 P.M. The new Moon falls on the 21st at 7:58 A.M., and first quarter is on the 27th at 11:19 P.M.

**A total solar eclipse**, the first of the century, will get under way at 6:37 A.M. on June 21 and can be viewed along a narrow swath starting in the South Atlantic, crossing Angola, Zambia, Zimbabwe, and Madagascar, and ending in the Indian Ocean. The Moon's umbral shadow cone will first touch Earth far off the coast of Uruguay. The open waters of the South Atlantic will experience the longest totality: four minutes and fifty-seven seconds.

**Summer solstice** occurs on June 21 at 3:38 A.M. in the Northern Hemisphere.

*Unless otherwise noted, all times are given in Eastern Daylight Time.*



## THIS LAND

# How the West Was Swum

At Nevada's Berlin-Ichthyosaur State Park, fossils of giant marine predators point to the region's watery past.

By Richard L. Orndorff, Robert W. Wieder, and Harry F. Filkorn

Berlin-Ichthyosaur State Park sits on the western flank of Nevada's Shoshone Mountains, about 7,000 feet above sea level. Today a seemingly endless expanse of sagebrush covers the relatively featureless landscape of the valleys, while the rocky slopes of the mountains support scattered sagebrush and stands of juniper and piñon trees. This sparsely populated part of Nevada has changed little since the first settlers arrived more than one hundred years ago. But it has changed much since the largest marine predators of the Triassic Period lived here, more than 200 million years ago.

Extinct reptiles that plied ancient oceans, ichthyosaurs had highly streamlined bodies resembling those of some of today's fastest fish, such as swordfish, marlin, and tuna. Despite their fishlike exteriors, ichthyosaurs

Adapted from *Geology Underfoot in Central Nevada*, by R. L. Orndorff, R. W. Wieder, and H. F. Filkorn. © 2001 by the authors. Reprinted by permission of Mountain Press Publishing Company, Missoula, Montana.



*Shonisaurus* of Triassic Nevada

had to surface to breathe air and they gave birth to live young. Their elongate mouth and strong jaws held rows of pointed conical teeth, similar in shape to those of modern toothed whales. A circular set of overlapping bony plates internally reinforced the disproportionately large eyes of some ichthyosaurs and compensated for changes in water pressure when the animals dived or surfaced. This feature enabled them to consistently maintain their highly developed sense of vision at all swimming depths.

Fossil ichthyosaurs are common in

the sedimentary rocks that make up Nevada's Luning Formation. About 230 million years ago, when these sediments were deposited, North America was part of the northern supercontinent known as Pangaea, and the Panthalassa Ocean covered much of what is now the western United States. Outcrops, or protruding layers of rock, of the Luning Formation are scattered throughout the mountain ranges of central Nevada, and paleontologists have found ichthyosaur remains in the West Humboldt Range and the New Pass Range, as well as in



Union Canyon in the Shoshone Mountains, the site of Berlin-Ichthyosaur State Park.

The first ichthyosaur bones discovered in Union Canyon were excavated by gold prospectors from the mining town of Berlin (now a well-preserved ghost town and part of the state park). While the miners saw the bones as novelties and sometimes used them to decorate their cabins, the fossils did not become known to the scientific community and recognized as the remains of ancient reptiles until 1928. The first field expedition to Union Canyon was launched by Berkeley paleontologist Charles L. Camp and his colleague Samuel P. Welles in 1954. Today a stroll through Union Canyon takes visitors by Camp's cabin, where he and other scientists worked diligently for years to reconstruct the ichthyosaurs' skeletons and solve the puzzle of their presence here. Union Canyon has yielded at least thirty-seven mostly complete ichthyosaur skeletons. In 1966 an A-frame shelter was built over the main quarry to protect some of the exposed fossils and allow visitors to see them. The skeletons of nine individuals, their bones still embedded in rock, are on view. While Camp considered the Union Canyon fossils to be of three different species of ichthyosaurs, all the specimens are now thought to be the remains of a single species, *Shonisaurus popularis*, named for the surrounding mountain range.

Thanks to the abundance of *Shonisaurus* specimens collected in the region, scientists know more about the skeleton of this species than about any other Late Triassic ichthyosaur. Fifty or more feet long and weighing an estimated forty tons, *Shonisaurus* was one of the largest creatures of its time—about the size of a modern sperm whale and twice the size of a killer whale. Larger individuals had six-foot-long front fins, twenty-five-foot-long tails, and ten-foot-long

skulls with elongate jaws, filled with conical teeth. In contrast, the well-preserved ichthyosaurs found in shale quarries in Holzmaden, Germany, which date from the Jurassic Period, were only the size of today's dolphins.

While fossil bones tell us about the body plans of extinct creatures, the composition of the surrounding rock can give clues to the environments they inhabited and help answer such questions as how so many large ocean-going predators ended up in the Shoshone Mountains. At the time the Union Canyon ichthyosaurs lived, the region was a tropical sea, situated along the west coast of what is now North America. The bedrock in the area indicates that the ichthyosaur bones were deposited in a deep ocean shelf environment. One of the most convincing pieces of evidence is the fine-grained sedimentary rock that encloses the fossils. As rivers carrying sediment spill into the ocean, freshwater mixes with standing marine water, and the momentum of the flow decreases; as a result, coarse sediment drops to the bottom near the shore. Fine sediment remains in suspension much longer and travels far out to sea, where it settles slowly to the bottom. The sediment layers in the Union Canyon rocks suggest they accumulated in the deeper areas of the continental shelf.

Other fossils found with the ichthyosaur remains also support the idea that this was a deepwater environment. Most of these are from swimming organisms, such as ammonoids and nautiloid mollusks that lived just above the deep seafloor.

The general scarcity of fossils of scavenging marine animals also fits well with this interpretation, because scavengers tend to be less numerous in deep marine waters. In contrast, other rock layers of the Luning Formation contain an abundant and diverse assemblage of mollusks, corals, echinoderms, and sponges that typically lived in shallower waters.

Most of the *Shonisaurus* skeletons are articulated; that is, the bones are still in the correct anatomical position relative to one another. Strong ocean currents would have moved at least some of the carcasses during their decomposition, and the bones would have been scattered during their transport. Thus currents must have been fairly weak, as they typically are in deeper water. The corroded and pitted surfaces of some of the bones, and the presence of brachiopod shells on them, suggest that the bones lay exposed on the seafloor

for a time, but the relative completeness of the skeletons indicates that they were buried on the seafloor soon after the flesh had decomposed.

One major unresolved problem concerns the explanation for exactly how so many ichthyosaur skeletons came to be preserved so close to one another. Two plausible explanations exist. The ichthyosaurs may

have died singly over an extended period; according to this scenario, their carcasses sank to the bottom, and weak currents naturally concentrated them into a depression, or submarine valley, on the seafloor. Alternatively, this deposit of multiple ichthyosaur skeletons could represent a massive die-off.



**Ichthyosaur bas-relief and trail of vertebrae**

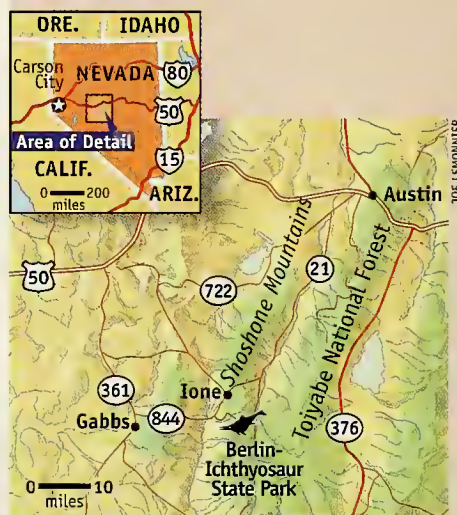


Based mainly upon studies of modern marine vertebrates, various theories, of varying plausibility, have been proposed to account for the sudden-mass-mortality scenario. Changes in the physical or chemical conditions of the seawater have been proposed as a cause of death, but any change drastic enough to kill ichthyosaurs would also have killed other marine organisms. However, no other fossil evidence exists along with the ichthyosaur bones to indicate any such additional die-off. Volcanic eruptions could have killed many ichthyosaurs, but then we should see some evidence, such as a volcanic ash layer, preserved in the sediments. None is present in the rock record. Similarly, the stratigraphic record lacks evidence of a severe storm, for example, a coarser-grained layer of sediment. The notion that some ichthyosaurian behavior—such as spawning, stranding, or coastal foraging—brought these *Shonisaurus* together is improbable. As dramatically evidenced in fossils from other sites, ichthyosaurs bore live young, so they did not spawn en masse as do many fish. And while mass mortality by stranding was previously supported as a cause for the concentrations of skeletons here, researchers now consider that scenario unlikely, given the evidence for an ocean shelf, rather than a coastal, setting.

One intriguing possibility is that the ichthyosaurs ate fish or shellfish

## Park Ranging

The state of Nevada, which has adopted *Shonisaurus popularis* as its state fossil, first officially recognized the importance of Union Canyon in 1955, when it designated the site Ichthyosaur Paleontological State Monument. In 1970 the boundaries were expanded to include the mining ghost town of Berlin, and the



tainted with a neurotoxin that paralyzed them. (Paralytic shellfish poisoning in humans also results from a neurotoxin.) Such a poison may have originated at the base of the marine food chain, in oceanic plankton, and then become concentrated in the tissues of the animals that ate the plankton. The *Shonisaurus*, top predators and consumers in the food

chain, may have eaten these plankton-feeding organisms and perished. This type of poisoning mechanism has triggered some of the mass kills of modern whales along the coast of New England.

Over the years, scientists have revised

site was renamed Berlin-Ichthyosaur State Park.

Located about twenty-three miles east of the town of Gabbs, the park offers guided tours of the Fossil Shelter, the ghost town of Berlin, and the Diana Mine rock tunnel used by the Berlin prospectors. Camping is available, and an extensive system of hiking trails can help visitors enjoy the juniper and piñon woodland, examine the local bedrock, or catch sight of wildlife. Eagles, rattlesnakes, deer, pronghorn antelope, foxes, coyotes, and mountain lions roam the area.

For visitor information on seasonal access, tour schedules, and rules and regulations, contact:

Berlin-Ichthyosaur State Park  
HC 61 Box 61200

Austin, NV 89310

Tel: (775) 964-2440

Fax: (775) 964-2012

[www.state.nv.us/stparks/bi.htm](http://www.state.nv.us/stparks/bi.htm)

and refined their views of how *Shonisaurus popularis* lived as an active predator and how it came to rest on the seafloor. Each new generation of paleontologists builds on the work of those who came before, providing some answers but invariably providing many more questions. A visit to Berlin-Ichthyosaur State Park is an opportunity to ponder the giant predators and their ancient environment. As you investigate the *Shonisaurus* fossils, you might want to consider some of these questions. And ask some of your own.

*Richard L. Orndorff is assistant professor of geology at the University of Nevada, Las Vegas; Robert W. Wieder is a biologist at the California Department of Agriculture; and Harry F. Filkorn is a paleontologist at Kent State University in Ohio.*



*Shonisaurus* skull and other Triassic marine fossils

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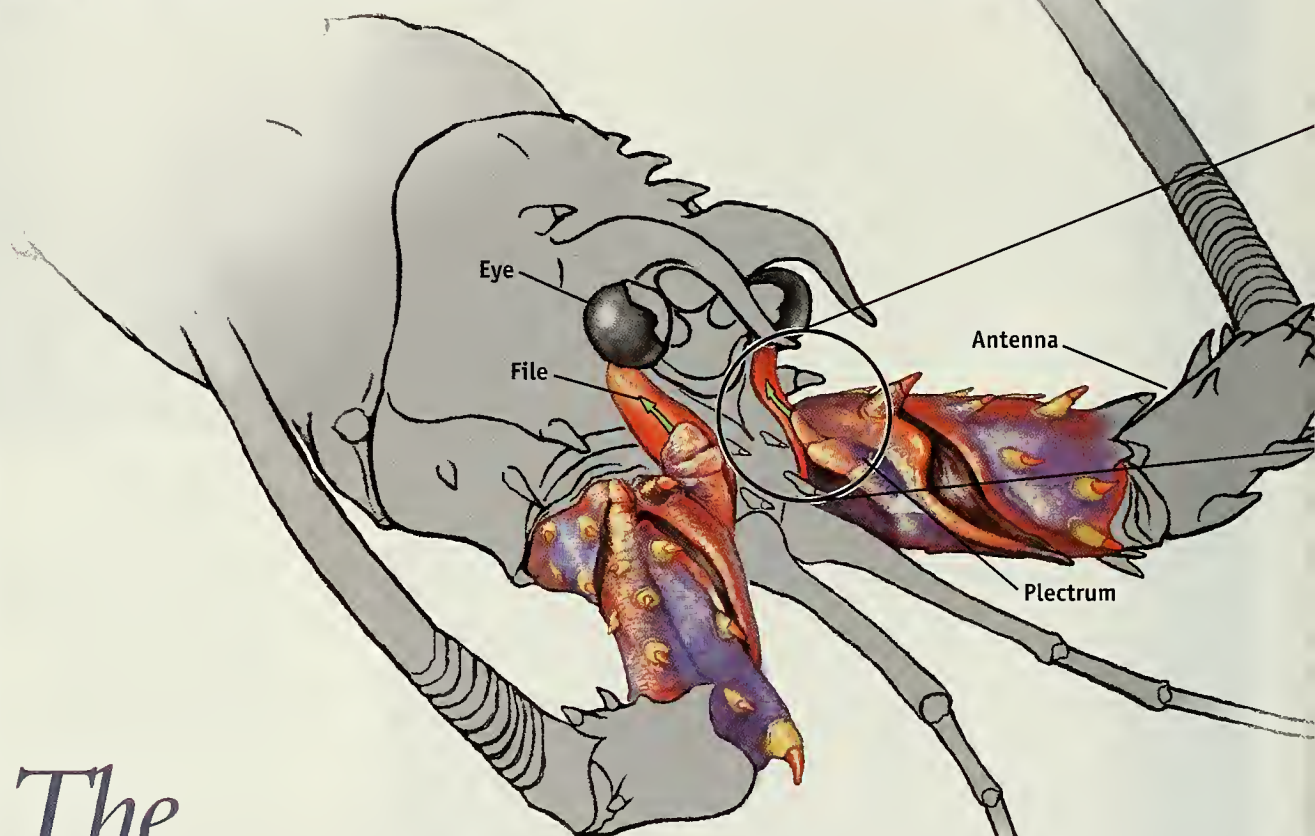
## BIOMECHANICS

Some lobsters and many eight-year-old violinists have a knack for making unpleasant noises; amazingly, crustaceans and humans use much the same mechanism to produce these awful sounds. The lobsters in question are not Maine's clawed variety but members of a family known as the Palinuridae, or spiny lobsters. These clawless marine invertebrates, found worldwide, often appear on menus as rock lobster or New Zealand lobster tail. Instead of a showy pair of claws,

two long antennae are their most striking feature. The base of each antenna (where it joins the head) is thick and spiny—the reason for the lobster's common name.

Many invertebrates, such as crickets and cicadas, make noise by “plucking” a series of spikes or ridges (usually on their legs or wings)—much like a person drawing a thumbnail across a comb or a pick across guitar strings. But Sheila Patek, of Duke University, has discovered that spiny lobsters produce sound in a very different way:

by drawing a bow across a vibrating surface. In this case the “bow,” called the plectrum, is a flattened protuberance (actually a series of soft ridges) emerging from the basal segment of each antenna. (Earlier researchers thought the lobster's plectrum functioned like a pick; hence the confusing mix of terms.) The analogue of the violin string is the file—an oblong lump, or pad, one on either side of the lobster's head. By wagging an antenna, the lobster draws the plectrum across the file;



# The Lobster's Violin

It's enough to give a predator pause.

Story by Adam Summers ~ Illustration by Sally J. Bensusen



the result is a surprisingly loud, rasping buzz. (One striking difference between lobster and violinist, of course, is that no amount of practice will turn this buzz into music.)

This type of mechanism is known as stick-and-slip motion. Imagine a box of rocks sitting on a conveyor belt, but instead of being able to move freely along the belt, the box is secured to a wall by a spring. As the belt moves, the box rides with it, stretching the spring. At some point, the tension of the spring becomes greater than the

frictional force (the amount of resistance to movement that occurs between two moving objects in physical contact) between box and belt, and the box skitters along the belt toward the wall. This backward movement shortens (and thus reduces the tension of) the spring, permitting the box once again to ride the belt. Each time the box skips back across the belt, it makes an audible rumble; when the box rides smoothly, there is silence. The key to stick-and-slip sound production is friction. If the conveyor belt was greased, the box would move forward until the spring was stretched taut. Then the box would ride in place, with the belt sliding smoothly and soundlessly underneath it. No friction, no sound.

Violinists enhance the friction between the horsehair bow and the nylon or gut strings of their instrument by rubbing rosin on the bow. For lobsters, the friction comes from microscopic shingles on the otherwise smooth files. Each time the lobster's plectrum skids on the file, it produces a pulse of sound. As it travels the length of the file, the plectrum generates between two and twenty-four of these pulses, creating the characteristic raspy squeak. The duration of the sounds depends on the length of the file, which varies considerably from genus to genus. In fact, seven of the nine genera of spiny lobsters can be identified by the shape of their files and plectra. (The other two do not have files and thus make no noise at all.)

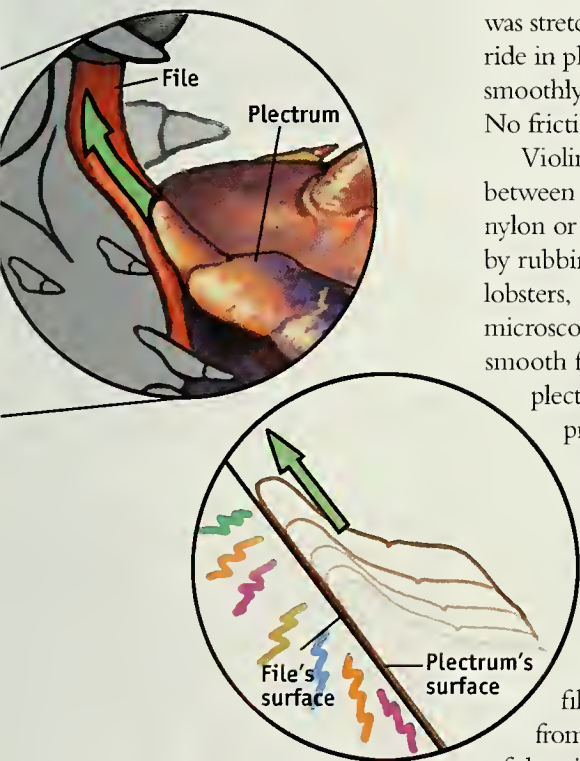
Patek believes that lobsters make these raucous sounds to deter predators. Think how you would react if a hot dog let out a loud squeak when you picked it up. However, the sound may do more than just startle potential predators. Spiny lobsters can do considerable damage with their

stout antennal bases, which may be several inches long. In captivity they wield these spiky clubs aggressively and even catch the occasional fish dinner by slamming their antennae together. In the wild, lobsters may use sound to warn a predator that it is about to get clunked. Or the noise may simply inform a shady character that the element of surprise has been lost—the lobster version of "I've got my eye on you."

In any case, there is a very important biological reason a lobster would prefer a violin to a guitar. Lobsters, like all animals with exoskeletons, periodically shed their armor as they grow. As anyone who has appreciated soft-shell crabs can attest, naked crustaceans are both tasty and easy to eat. If a spiny lobster had to produce sound the way guitarists so often do—by plucking a hard plectrum across a series of hard ridges—the animal would be obliged to fall silent just when it would benefit most from an antipredator noisemaker: during the vulnerable few days it takes for the carapace to harden following a molt. The great advantage of the stick-and-slip approach is that a soft structure rubbing against another soft structure works just as well right after a molt as it did beforehand.

Many animals produce sounds to communicate with their own species—to issue warnings or invitations or to affirm their presence. Spiny lobsters appear to have developed this communication system solely to talk to other species. Their predators can certainly hear sounds in the range produced by the plectrum and file, but as far as we know, the lobsters themselves are completely deaf to their own playing.

*Adam Summers is an assistant professor at the University of California, Irvine.*



**To generate its loud, raspy buzz, the spiny lobster waggles one or both of its antennae, causing a flattened projection (the plectrum) on each antenna's spiky base to skid across an oblong lump (the file) located on either side of the animal's head, near the eye. Microscopic shingles on the file create friction, which is essential to sound production.**



## ESSAY

# Are Genes Real?

Our understanding of heredity has been propelled by the oscillations of a conceptual pendulum, arcing between the gene as a real entity and the gene as an abstraction.

*By Nathaniel C. Comfort*

At the entrance to the California Academy of Sciences in San Francisco, a 235-pound brass pendulum bob swings on a thirty-foot cable fixed to the cathedral ceiling. It is known as a Foucault's pendulum, after Jean-Bernard-Léon Foucault, a French physicist of the nineteenth century. Each arc of the pendulum cuts a diameter across a ring of metal pegs set on the floor. As the earth rotates, each peg in turn moves into the path of the giant bob and is knocked over. Although the pendulum swings in a straight line, it never returns to the same spot twice in succession.

Science often behaves more like a Foucault's pendulum than like the for-

ward march of progress depicted in textbooks and newspapers. An idea may oscillate between two extremes, yet as the world of science shifts beneath its path, each swing results in a different incarnation of the idea. One

**Gregor Mendel**



James Clerk Maxwell



Charles Darwin



Wilhelm Johannsen

APPH

such idea is whether or not genes are real. If the accumulation of scientific knowledge were linear, the idea of the gene ought to have started out vague and become progressively more sharply delineated with time. In fact, over the past 150 years, our understanding of heredity has been propelled by the oscillations of a conceptual pendulum,



William Bateson

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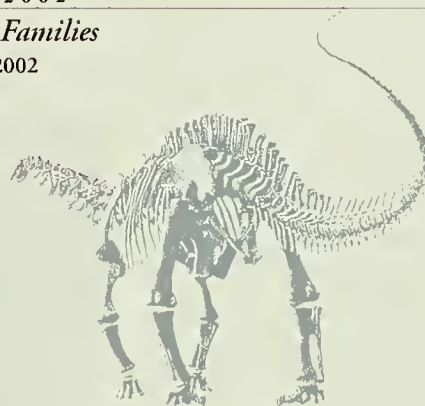
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arc between a real gene and an abstract one.

The word “gene” was coined in 1909 by Wilhelm Johanssen, a well-known Danish botanist of the late nineteenth and early twentieth centuries. During the preceding four decades, Gregor Mendel, Charles Darwin, and many other scientists had proposed theories of how hereditary traits were passed down through the generations. Mendel, the canonical father of genetics, modeled the inheritance patterns of seven carefully chosen “differ-

Gregor Mendel did not distinguish between the traits seen in a plant—such as seed color and texture, pod shape, and stem length—and the hereditary elements that produced them.

entiating characters” (*differierende Merkmale*)—for example, stem length, pod shape, color and texture of the seeds—in garden peas. At the end of a paper published in 1866, Mendel hinted that the *Merkmal* might lie in the cell nucleus, but he did not distinguish between the traits seen in the plant and the hereditary elements that produced them. For Mendel these elements were abstractions, useful in understanding the patterns of inheritance.

The hereditary elements proposed by Darwin were more physical—and therein lay their downfall. In 1868, unaware of Mendel, Darwin put forth his hypothesis of pangenesis, in which trait-

bearing particles that he called gemmules budded off from the body’s tissues and collected in the reproductive cells, where they waited to be passed on to the next generation. Pangenesis won few adherents. The great physicist James Clerk Maxwell even digressed from his 1875 *Encyclopædia Britannica* essay “The Atom” to take a swipe at it. “Some of the exponents of this theory of heredity have attempted to elude the difficulty of placing a whole world of wonders within a body so small and so devoid of visible structure as a germ,” he wrote, referring to Darwin’s gemmules. To Maxwell—an expert on particles—no simple particle could conceivably explain the wonders of heredity and embryology. “To explain differences of function and development of a germ without assuming differences of structure,” he continued, “is to admit that the properties of a germ are not those of a purely material system.” Advocating hereditary particles seemed tantamount to mysticism.

Nevertheless, in the late 1800s it was Darwin’s theory, not Mendel’s, that had the most influence. Mendel died in 1884, and his paper was largely ignored until the turn of the century; it was cited just a handful of times and never as a landmark in the study of heredity. Though Darwin’s theory of pangenesis was soundly rejected, it continued to echo throughout the rest of the century. The major theories of heredity put for-

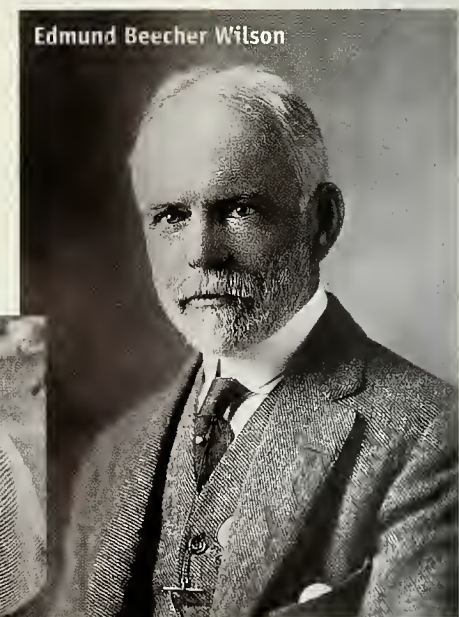
ward in the 1880s and 1890s also assumed the existence of real, physical particles, with such exotic-sounding names as *ids*, *biophors*, and *pangenes*.

When Mendel’s principles, with their mathematical treatment of heredity, were rediscovered in 1900, however, the pendulum swung back toward abstract genes. One of Mendelism’s staunchest



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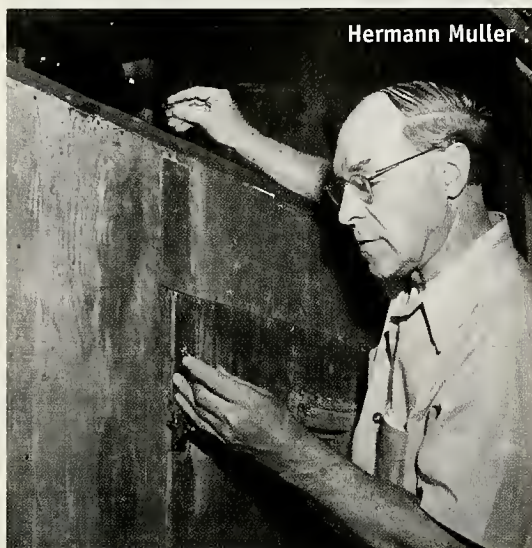


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Hermann Muller

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Edward Tatum

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George Beadle

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Richard Goldschmidt

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partisans was William Bateson, an English biologist who saw in it a weapon for his attacks on the Darwinian idea of continuous variation in nature. Making the units of heredity abstract helped Bateson contradict this view, which was predicated on material particles. Bateson referred to the Mendelian elements as unit characters—a deliberately abstract term—and he passionately denied their material existence.

A young American embryologist named Thomas Hunt Morgan followed this trend. In 1903 Walter Sut-

ton, an American cytologist, offered a cellular explanation of Mendel's principles, suggesting that Mendelian elements lay on the chromosomes. Two years later, geneticist Nettie Stevens (a former student of Morgan's) showed that sex was associated with the mysterious "accessory," or X, chromosome. Morgan, however, was skeptical. Given the limited evidence then available, too many assumptions were required and not enough biology was explained. Advocates of the chromosome theory seemed to him enthusiasts, bandwagon jumpers. In 1905 he wrote that his colleagues at Columbia University, especially the distinguished cytologist Edmund Beecher Wilson, were "wild over chromosomes," making Morgan feel he lived "in an at-

mosphere saturated with chromosomal acid."

In 1910 Morgan abruptly tipped the other way. Among the thousands of fruit flies he was breeding in his laboratory, he found a single male with white eyes, rather than the usual red. Breeding experiments revealed that eye color was inherited together with a "factor" that determined sex. Stevens's results could no longer be ignored. Sex and eye color were linked by association with the X chromosome.

Morgan and his graduate students at Columbia made genes real again. Dur-

To Thomas Hunt Morgan and the other early fruit-fly geneticists who put together the first gene maps, a gene was roughly synonymous with a physical point on a chromosome.

ing the following years, they developed the first gene maps, assigning genes for various traits to different chromosomes and measuring the distance between genes in terms of the likelihood that two traits would be inherited together, much as blond hair often goes with blue eyes. To these early fruit-fly geneticists, a gene was roughly synonymous with a locus, a physical point on a chromosome. In 1922 Hermann Muller, one of Morgan's former students, went further, describing genes as "ultra-microscopic particles." Muller dreamed of one day being "able to grind genes in a mortar and cook them in a beaker."

Practitioners of this "classical" school of genetics mostly ignored the question of what genes were made of. The question of most interest to them was what genes *did*. Working at Stanford University with the bread mold



*Neurospora*, George Beadle, a geneticist, and Edward Tatum, a chemist by training, provided an elegant answer in 1941. They identified genetic mutations that disabled specific steps in the synthesis of a complex molecule. Knowing from biochemistry that each step was catalyzed by a particular enzyme, they concluded that each mutation knocked out one enzyme. In classical genetics, genes had been defined as things that, when mutated, changed a trait: one mutation, one gene. Beadle and Tatum refined the definition by showing that a gene was a thing on a chromosome that specified an enzyme: one gene, one enzyme.

As Beadle and Tatum's work became accepted, more and more scientists thought of genes as real entities. This did not happen overnight, however, and there were critics. One of the most eloquent—and most aggravating to advocates of real genes—was Richard Goldschmidt, a brilliant, cantankerous German who in 1936 fled Nazi Germany and took up a post at the University of California, Berkeley. "There are no genes," Goldschmidt wrote in 1937, and "no gene mutations." In 1951 he continued his attack on the gene by suggesting this analogy: "If the A-string on a violin is stopped an inch from the end, the tone C is produced. Something has been done to a locus in the string, it has been changed in regard to its function. But nobody would conclude that there is a C-body at that point."

Within two years, Goldschmidt's provocative criticism seemed absurd. In April and May of 1953, James Watson and Francis Crick published their two papers describing the double-helical structure of DNA. The genius of their model was that the structure of the molecule and the structure of the gene were one and the same. With the double helix finally came insight not just into how genes worked but also into what they were made of. A gene was merely a particular sequence of nu-

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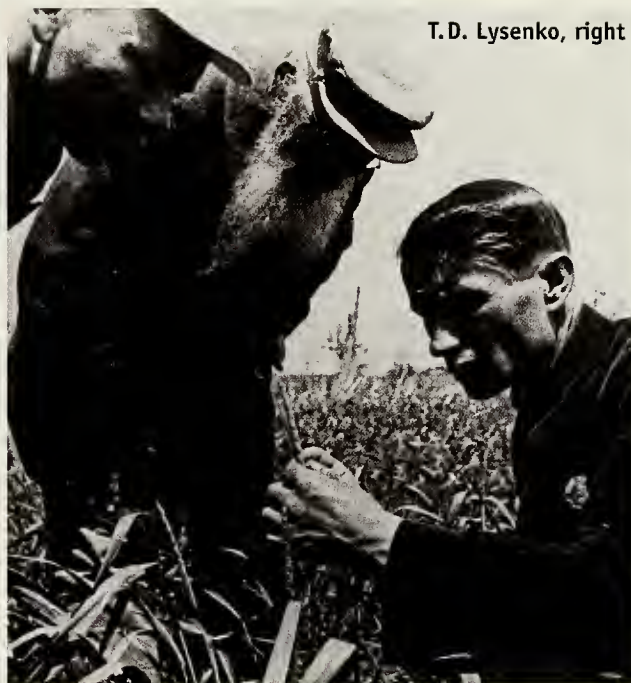
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T.D. Lysenko, right

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James Watson, left, and Francis Crick

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cleotide subunits of a DNA strand—not a pearl on a string but the string itself.

For most geneticists, the discovery of the double helix instantly and unequivocally settled the debate in favor of real genes; from that point on, skeptics provided little more than comic relief. Horace Freeland Judson reports an anecdote told by Russian biochemist Vladimir Englehardt about his meeting in 1961 with Trofim Denisovich Lysenko, the infamous Soviet agronomist who rejected Mendelism and Darwinism as inconsistent with Stalinist ideology. Lysenko scoffed, "All this DNA,

DNA! Everybody speaks about it, but nobody has seen it!" Englehardt explained that in fact plenty of people had seen it, and he sent his secretary to fetch some. When she returned with a vial of powdered DNA, Lysenko retorted, "Ha! You are speaking nonsense! DNA is an acid. Acid is a liquid. And *that's* a powder. That can't be DNA!"

Yet just when it became clear that only cranks could deny the reality of genes, the scientific ground began to shift again. In 1957 Seymour Benzer, a physicist turned viral geneticist at Purdue University, proposed that more than one type of gene existed, and he suggested the term "cistron" for a segment of DNA that encodes a protein. This was, in essence, the gene of Beadle and Tatum expressed in the language of

Watson and Crick. Cistrons caught on; the term is still used today. Reconstructions, two other types of genes proposed by Benzer, soon fell by the wayside, though, because it quickly became clear that they merely amounted to a single unit of DNA. Nevertheless, the very suggestion that biologists should think in terms of several senses of "gene" created fissures in the concept of a monolithic gene.

Meanwhile, a group of French geneticists, led by François Jacob and Jacques Monod, were showing that the gene's boundaries were fuzzier than bi-



ologists had thought. First, genes often act in clusters: Jacob and Monod portrayed "the" gene as a set of structural genes, which encode proteins, and regulatory genes, which switch the structural genes on and off in response to signals from the cell. Furthermore, Jacob and Monod showed that genes are not restricted to chromosomes. They found free-floating genetic elements, called episomes and plasmids, in bacteria; other scientists soon found these elements in higher organisms as well. Mitochondria, the cell's power plants, and chloroplasts in green plant cells were later found to have their own genes, inherited independently from those on the chromosomes.

In 1967 James Shapiro, an American then working at the University of London's Royal Postgraduate Medical School, and Sankhar Adhya, of the University of Wisconsin, discovered another wrinkle: regions of bacterial DNA that can cut themselves out of

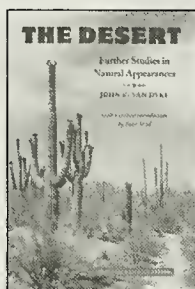
A group of French biologists, led by François Jacob and Jacques Monod, showed that the gene's boundaries are fuzzier than had been thought and that genes are not restricted to chromosomes.

one site on a chromosome and reinsert themselves at another site. They called these regions insertion elements. Twenty years earlier, Barbara McClintock, the great cytogeneticist of maize at the Carnegie Institution of Washington's Department of Genetics, had demonstrated that certain chromosomal elements—she did not believe them to be genes—can move, but

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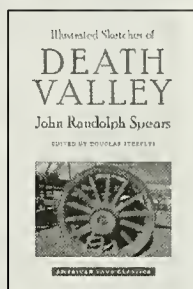
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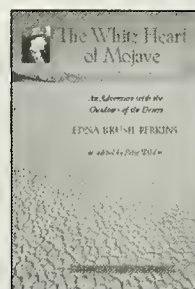


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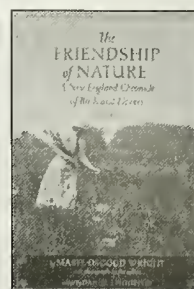
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Jacques Monod, left; André Lwoff, center; and François Jacob

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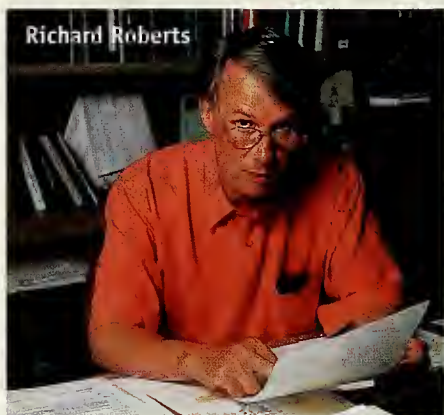
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Shapiro and Adhya were the first to show how this movement takes place. Within ten years, insertion elements were found to be widespread in nature. Bacteria use them to pass along genes that confer resistance to antibiotics—which is one reason that drug-resistant strains of disease-causing germs spread so rapidly. Insertion elements enable retroviruses (HIV, for example) to incorporate their genes into their hosts' chromosomes. By 1980 biologists had accepted that certain genes routinely



Phillip Sharp

COURTESY OF PHILLIP SHARP'S OFFICE



Richard Roberts

KAREN WILSON WONG

move around—within a chromosome, between chromosomes, within a species, between species. Movable genes torpedoed the idea of the gene as a site on a chromosome.

Today a reasonable working definition of a gene might read: one or more segments of DNA that specify a protein. The DNA is transcribed into an intermediary called RNA, which ferries the genetic message out to the ribosomes, where it is translated into a protein chain. This definition is a useful distillation of much that we learned about genes in the last century, but it is really just a starting point for thinking about all that a gene can be.

In 1977 two research groups, one led by Richard Roberts at Cold Spring Harbor Laboratory and the other led by Phillip Sharp at MIT, found that the many DNA segments that constitute a

single gene are sometimes quite distantly separated on the chromosome. In such genes the segments are then spliced together to compose the RNA message. Furthermore, the same segments can be combined in different ways, which means that one gene is capable of specifying a whole family of products: one gene, sometimes several enzymes.

And the story gets even more complicated. Biologists have found examples of genes within genes and even overlapping genes. In some cases, the same DNA sequence specifies one protein when read in the "forward" direction and another when read "in reverse." Muddling things further, the instructions encoded in the DNA do not always reach the ribosome as a literal translation. In a phenomenon known as RNA editing, an enzymatic highwayman intercepts the RNA message en route and alters it, so the resulting protein is not identical to that specified by the DNA.

In a sense, as the reality of the gene has become more and more certain, the gene has again become an ideal, a measuring stick against which scientists compare the exceptions and deviations of real biology. Little wonder, then, that some writers, such as the historian and philosopher of biology Evelyn Fox Keller, have advocated scrapping the term "gene" altogether, in favor of some term or set of terms to better express the dynamism of the chromosomes. DNA is not made of discrete units with fixed boundaries; it comprises great lengths of sequence that are altered, shuffled, and reused. Perhaps Goldschmidt was right.

Yet the pendulum continues to swing. Some of the most exciting research in biology today employs devices known as DNA chips to provide snapshots of the activity of every gene in a cell. The chip, a square inch or less of glass or plastic, is first dotted with DNA from every gene in an organism. The genome—that interactive, responsive, deeply integrative set



of all the genetic material on the chromosomes—is then digitized as an orderly array of DNA microdots: one gene, one dot. Using special tracers, biologists can mark on the chip just those genes that are active at a given instant. Computers scan and analyze the chips, comparing the set of active genes both before and after various experimental manipulations. Thus, biologists can see which genes are activated in response to a specific procedure. It is an immensely powerful technique, one with promise for developmental biology, immunology, cancer biology, and drug discovery.

Recently, biologists have found genes within genes, overlapping genes, and DNA sequences that specify one protein when read “forward” and another when read “in reverse.”

This new research makes genes real once again by imposing physical boundaries between them. And so the pendulum completes another arc. But the world of science continues to revolve. As scientists probe genomes using DNA chips, they will, in all likelihood, find that our current notions about genes are inadequate, and the next swing of the pendulum will undoubtedly bring us to a new understanding.

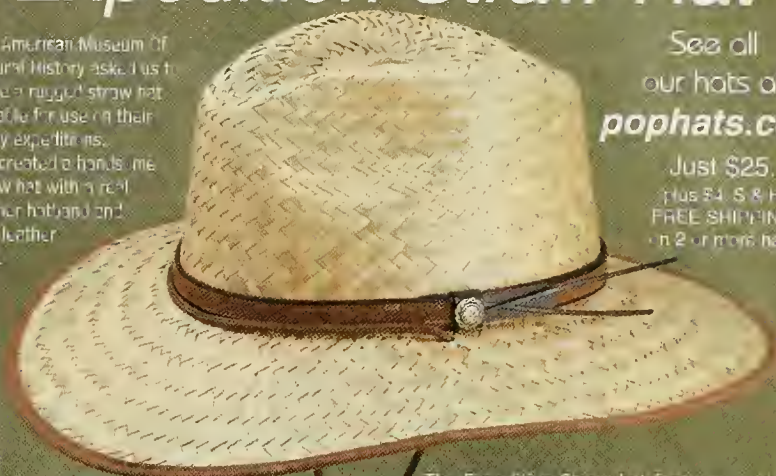
Nathaniel C. Comfort is deputy director of the Center for History of Recent Science and assistant professor of history at The George Washington University in Washington, D.C. He is author of *The Tangled Field: Barbara McClintock's Search for the Patterns of Genetic Control* (Harvard University Press, 2001).

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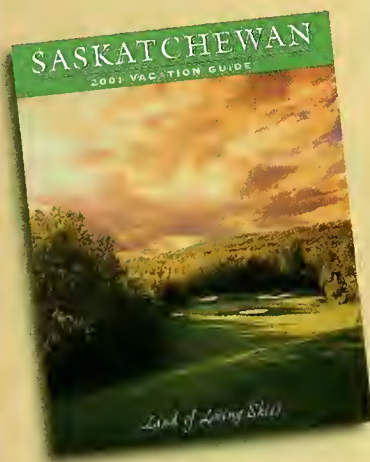
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# THE BEAST WITH FIVE GENOMES

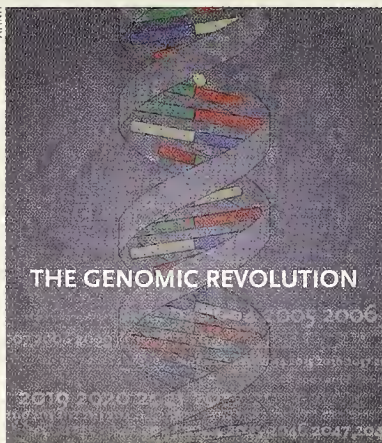
Inside a termite's gut lives *Mixotricha paradoxa*, a microscopic organism comprising hundreds of thousands of smaller life-forms. *M. paradoxa* is an extreme example of how all plants and animals—including ourselves—have evolved to contain multitudes. **By Lynn Margulis and Dorion Sagan**

**T**he hullabaloo over mapping the human genome—the sum of all the genes in an individual—might lead one to think that each species has only a single genome and that the genetic makeup of individual organisms is discrete and unitary. Such is far from the case. Paraphrasing Walt Whitman, we multicellular beings contain multitudes. All animals' cells have at least two interacting genomes. One is the DNA in the cell nucleus; this is the genome that has recently been

“mapped.” The other is that of the DNA in the mitochondria—the cell's multiple oxygen-breathing organelles that are inherited only through the maternal line. For more than a century, some scientists have known that every organism is in fact a multiple being, but until recently these unorthodox researchers were ignored.

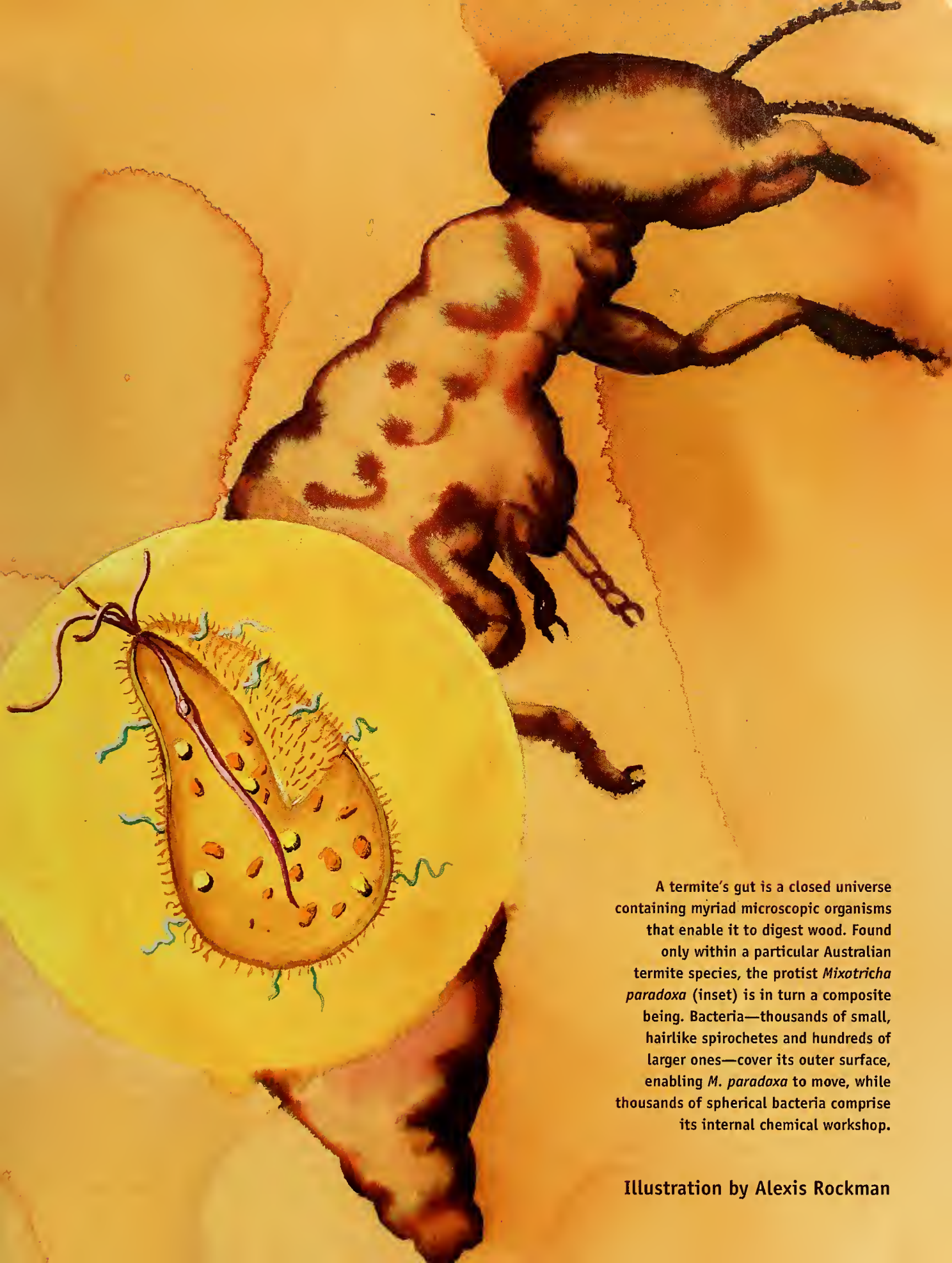
In most of the animals we think we know best (mammals, reptiles, insects), the genomes that determine limbs, eyes, and nervous systems, for example, are very similar to our own. These animals, like us, are doubly genomic. Even some unicellular beings that do not have eyes, limbs, or nervous systems—such as amoebas and paramecia—contain both nuclear and mitochondrial genomes. Plants and algae have these double genomes as well, plus a third genome, of symbiotic origin. During their evolutionary history, they ingested (but did not digest) photosynthetic blue-green bacteria. Therefore, all visible photosynthetic organisms have at least three genomes. But many organisms—such as the protists that inhabit termites—contain within them up to five or more genomes.

The great nineteenth-century naturalist Joseph Leidy, one of the founders of the Academy of Natural Sciences in Philadelphia, was the first to take a close-up look at the contents of a termite's gut. “In



**“THE GENOMIC REVOLUTION”**  
Opening May 26 at the American Museum of Natural History, a landmark exhibition explores the emerging role of genomics in science, technology, and everyday life. The exhibition runs through January 1, 2002.





A termite's gut is a closed universe containing myriad microscopic organisms that enable it to digest wood. Found only within a particular Australian termite species, the protist *Mixotricha paradoxa* (inset) is in turn a composite being. Bacteria—thousands of small, hairlike spirochetes and hundreds of larger ones—cover its outer surface, enabling *M. paradoxa* to move, while thousands of spherical bacteria comprise its internal chemical workshop.

Illustration by Alexis Rockman



watching the Termites from time to time wandering along their passages beneath stones," he wrote, "I have often wondered as to what might be the exact nature of their food." What he saw under his microscope amazed him. If the termite's intestine is ruptured by the experimenter, he wrote, "myriads

**The pioneering biologist  
Konstantin S. Merezhkovsky  
argued in 1909 that the little  
green dots in plant cells,  
which make sugar in the  
presence of sunlight, were  
originally separate  
organisms.**

of the living occupants escape, reminding one of the turning out of a multitude of persons from the door of a crowded meeting-house."

Leidy immediately realized that what he knew as "white ants" were actually composed of dozens of different kinds of tiny life-forms, including bacteria and what we now call protists. (Protists are microbes with nuclei; more complex than bacteria, the group includes amoebas, slime molds, and algae.) We now recognize

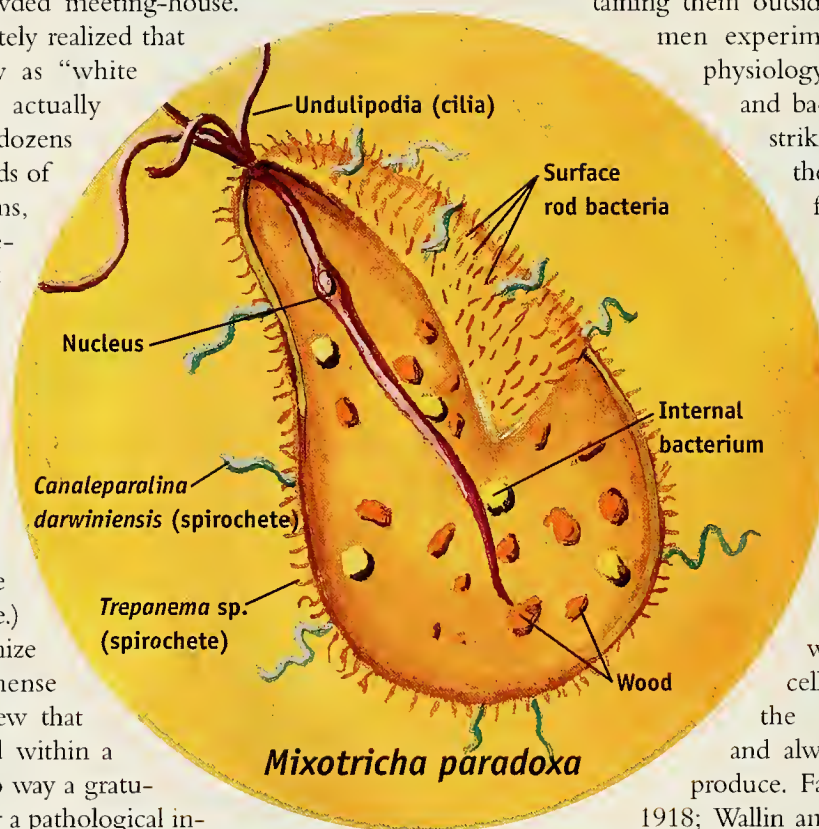
that the immense and motley crew that Leidy observed within a termite is in no way a gratuitous add-on or a pathological infection. Rather, it is a necessary part of the termite's digestive system and is organized as a particular tissue: an aggregate working mechanism that turns the refractory compounds lignin and cellulose (the main constituents of wood) into food. This composite fabric, or living consortium, has

evolved in the nearly oxygen-free closed system of the termite's abdomen for probably 100 million years; without the living, wood-degrading factories that have become their digestive systems, these termites starve.

The pioneering biologist Konstantin S. Merezhkovsky first argued in 1909 that the little green dots (chloroplasts) in plant cells, which synthesize sugars in the presence of sunlight, evolved from symbionts of foreign origin. He proposed that "syntrophogenesis"—a term he coined for the merger of different kinds of life-forms into new species—was a major creative force in the production of new kinds of organisms. A Russian anatomist, Andrey S. Famintsyn, and an American biologist, Ivan E. Wallin, worked independently during the early decades of the twentieth century on similar hypotheses. Wallin further developed his unconventional view that all kinds of symbioses played a crucial role in evolution, and Famintsyn, believing that chloroplasts were symbionts, succeeded in main-

taining them outside the cell. Both men experimented with the physiology of chloroplasts and bacteria and found striking similarities in their structure and function. Chloroplasts, they proposed, originally entered cells as live food—microbes that fought to survive—and were then exploited by their ingestors. They remained within the larger cells down through the ages, protected and always ready to reproduce. Famintsyn died in 1918; Wallin and Merezhkovsky

were ostracized by their fellow biologists, and their work was forgotten. Recent studies have demonstrated, however, that the cell's most important organelles—chloroplasts in plants and mitochondria in plants and animals—are highly integrated and well-organized former bacteria. Using





new methods, scientists have been able to raise and resolve the question of how these bacteria became permanent symbionts.

Like other animals, we harbor in our intestines an assortment of specific microbes that help us digest food, although some are also able to live outside humans. Few of our microbes are organized as layers of tissue, as they must be in termites. Nevertheless, without these hitchhikers to help digest fiber and produce vitamins, we—like termites—weaken and even die. Entirely integral to our bodies, however, are the mitochondria in our nucleated cells. These tiny entities use oxygen to generate the chemical energy needed to sustain life. They reproduce on

**Acceptance of the composite nature of the individual revolutionizes evolutionary biology. Bacteria are exemplary genetic engineers: splicers and dicers and mergers of genomes par excellence.**

their own, independently of the nuclear DNA, and multiply more quickly after short bursts of muscular exercise, leading to stronger, more mitochondria-packed muscles. Because mitochondria are so genetically integrated into each of our cells, no one has yet succeeded in growing them in test tubes.

We believe that Wallin and Merezchkovsky were fundamentally correct when they claimed that all nucleated living things evolved by symbiogenesis, generally because of preexisting bacterial genomes physically associated with other organisms. Reef-building corals, for instance, are now known to have five different genomes of once independent organisms. And *Mixotricha paradoxa*, a compound beauty found in a termite's gut, also has five genomes. Indeed, *M. paradoxa* could well be the "poster animal" for symbiogenesis.

In 1933 Australian biologist J. L. Sutherland first described and named "the paradoxical being with mixed-up hairs" (she mistakenly thought it was the only microbe that swims by simultaneously using both flagella and cilia). Studies done by A. V. Grimstone of Cambridge and the late L. R. Cleveland of Harvard in the 1950s with the electron microscope showed that *M. paradoxa* was a hundred times larger

than its close relatives, that four different kinds of bacteria were part of its body, and that it lacked mitochondria.

For many years, we have studied and photographed this organism. Under low magnification, *M. paradoxa* looks like a single-celled swimming ciliate. With the electron microscope, however, it is seen to consist of five distinct kinds of creatures. Externally, it is most obviously the kind of one-celled organism that is classified as a protist. But inside each nucleated cell, where one would expect to find mitochondria, are many spherical bacteria. On the surface, where cilia should be, are some 250,000 hair-like *Treponema* spirochetes (resembling the type that causes syphilis), as well as a contingent of large rod bacteria that is also 250,000 strong. In addition, we have redescribed 200 spirochetes of a larger type and named them *Canaleparolina darwiniensis*.

Acceptance of the composite nature of individuals, we predict, will soon revolutionize evolutionary biology. Bacteria are exemplary genetic engineers: splicers and dicers and mergers of genomes par excellence. Devoid of immune systems, always reproducing without mate recognition, bacteria are supremely promiscuous beings in which infection and sex—that is, gene flow—are virtually the same thing. The sexual proclivities of bacteria include (when their survival is threatened) rampant exchange of genes—next to which our own species' most bacchanalian orgies look like rather subdued affairs.

Biologists have always puzzled over why there are so many kinds of beetles. Perhaps symbionts beneath the surface, generating variety at the genomic level, account for nature's beetlemania. Insects have integrated bacterial genomes to an extraordinary degree. In many cases, bacteria reside in all the tissues, accumulate in the eggs, and are inherited. Beetles have developed partnerships with an extremely diverse assortment of bacteria; many more kinds live inside their tissues than live in most other groups of animals.

Eventually we may well realize that natural selection operates not so much by acting on random mutations, which are often harmful, but on new kinds of individuals that evolve by symbiogenesis. Scrutinizing any organism at the microscopic level is like moving ever closer to a pointillist painting by Georges Seurat: the seemingly solid figures of humans, dogs, and trees, on close inspection, turn out to be made up of innumerable tiny dots and dashes, each with its own attributes of color, density, and form. □







# SEX, ERRORS, AND THE GENOME

Can human beings keep evolving? Or does the error-ridden process of reproduction prevent us from getting more complex than we already are? **By Mark Ridley**

When extraterrestrial visitors land on Earth in their space saucer, they will be excited to see that ours is one of those rare planets on which complex life has evolved. They will already have found microbes—organisms resembling our viruses and bacteria—on every other life-bearing planet. And they will know that the real fun begins not in trying to understand how life on Earth came to exist at all but in how such complex forms as humans and butterflies and clams and whales and trees came about. And a question they will certainly ask is, How many copying mistakes does earthly life make when it reproduces the hereditary molecules of its DNA code?

When we (and other life-forms) produce offspring, our genome—the sum of all our individual DNA—is copied. But the repeated copying that takes place prior to pregnancy, during numerous divisions of our reproductive cells, can alter the messages in our genome—much as the children's game of Chinese Whispers (called Telephone or Gossip in the United States) distorts a verbal message as it is repeated from one person to the next. By the end of the line in Chinese Whispers, the message is laughably corrupted.

*In sexual reproduction, a male's and female's genomes are reshuffled, increasing the odds that some offspring will be produced without serious DNA copying errors.*

Rajah Bhup Singh of Guler under a quilt with his rani, ca. 1800

Through 3.5 billion years of evolution, life-forms have been able to perpetuate themselves—and become more complex than their ancestors—partly because they evolved ways of dealing with these copying errors. Double-stranded DNA (which appeared quite early in the history of life) and certain enzymes work within the cell nucleus to prevent errors from happening in the first place. Other enzymes correct most of the errors that nonetheless arise: proofreading and repair enzymes correct errors in the code, and developmental troubleshooting enzymes correct the expression of a faulty code without correcting the code itself. But the most important factor in the evolution of complex forms, which contain many genes (and therefore the possibility of making many errors), was the evolution of sex. Because sex takes one set of genes from each parent and recombines them, it shuffles the errors that manage to slip through all the defenses and improves the odds that at least some healthy, error-free offspring will result. This crucial innovation probably arose about 2 billion years ago, around the time that a more complex cell type—called the eukaryotic cell—originated.

Nonetheless, human beings are quite error prone. When we copy our DNA, we make more mistakes than most, if not all, other forms of life on Earth. In fact, every human being is conceived in 200-fold copying error. How many of these 200 mutations are harmful is not known. Most errors seem to be neutral, and a very few may actually help the organism, but even rigorous accounting cannot squeeze the harmful-error rate to below about 2 per conception. A figure of 5 to 10, or even 20, harmful mutations per conception may be quite likely. These high numbers are a consequence of our complexity. A human being contains 30,000 genes, included within a total of some 6.6 billion or

*Based on the forthcoming book The Cooperative Gene, by Mark Ridley. Copyright © 2001 by Mark Ridley. To be published by The Free Press, a division of Simon and Schuster, Inc., N.Y. Adapted with permission.*



## MUTATIONS: MOTHER VERSUS FATHER

As their life spans stretch out, men and women travel different evolutionary roads, and the amount of DNA copying that goes on in their gonads contributes to the error level of their genomes in different ways. Men manufacture sperm throughout their lives. About 40 cell divisions in the reproductive cells have occurred in a human male by the time he reaches puberty. After that, the DNA in his sperm is copied every sixteen days, or 23 times per year. A twenty-year-old man's genome has been copied more than 200 times, and a forty-year-old's more than 600 times. Compare that with the average adult male rat: its DNA has been copied only 58 times in its short life, and the DNA in its spermatozoa is therefore relatively error free.

A female human, on the other hand, already possesses her lifetime supply of eggs—with about 33 cell divisions behind them—by the time she is a late-stage fetus. When a thirty-year-old man breeds with a thirty-year-old woman, his DNA has been copied 430 times against her 33. With about thirteen times as many errata in his DNA, about 185 of the 200 copying mistakes in each human conception may come from the sperm. However, a woman's eggs are more likely to carry serious errors in chromosome numbers, and these errors increase with maternal age. Some disorders, such as Down syndrome, are the result of eggs that deliver the wrong number of chromosomes during conception.

All the DNA messages in a sperm and an egg can be compared with all the text in two sets of encyclopedias. If publishers made errors in book production at the same rate fathers and mothers do in transcribing their DNA, buyers of *Britannica* would receive sets with 200 printing errors on average, and half the time they'd be sent the wrong number of books.

more units of DNA. A bacterium might have on the order of 2,000 genes and 2 million units of DNA. The unit error rate, however, is similar in humans and bacteria. Humans therefore make more mistakes than bacteria do, for much the same reason that a scribe is more likely to make mistakes when copying the Bible—a job that took about a year and a half in the Middle Ages—than when copying a single psalm.

Another, and perhaps even more important, reason we humans are error prone is that we are long lived, with an average of thirty years between one generation and the next. Mutation rates are higher in long-lived animals such as humans because we copy our reproductive DNA a number of times in the interval between when we ourselves are conceived and when we beget our own children. (See "Mutations: Mother Versus Father," above.) And—

complicating matters further—not only do most human embryos contain about 200 copying errors, or "typos," in individual DNA messages, but about 50 percent of these conceptions have a botched number of chromosomes. The length of a typical generation is probably a factor here, too, because the percentage of such errors in rabbits or guinea pigs—with generations measured in weeks or months—is negligible. Even if half of all embryos have chromosomal errors, that still leaves 50 percent that are potentially available to carry the human species forward. And these will of course have, on average, 2 to 20 damaging typos.

Any individual may produce some faulty young, but for humans or any sexually reproducing form of life to persist, the average parent must produce at least one error-free offspring. Luckily, in addition to having enzymes that prevent or correct copying errors, we also have sex, which provides each offspring with a helpful redundancy of genes. In fact, our cells contain four copies of the information for each genetic instruction—a paternal and a maternal double helix. A correct version in one set will usually override a copying mistake in the other, so the average parent has a reasonable chance of producing a baby that will itself survive to reproduce. Unfortunately, sexual reproduction does not always prevent an embryo from picking up a whole extra chromosome or two. In this case, natural selection comes into play after conception: embryos with the wrong number of chromosomes almost always die in the very earliest stages of their intrauterine existence. (Those who have extra chromosomes but do survive, such as people with Down syndrome, often have significant health problems.)

What implications does our high error rate have for human evolution? Can we keep on evolving

## AT CONCEPTION, HUMAN EMBRYOS AVERAGE ABOUT 200 COPYING ERRORS, AND 50 PERCENT OF THE EMBRYOS HAVE A BOTCHED NUMBER OF CHROMOSOMES.

and pick up more genes for more functions? How high can the error rate go if a sexually reproducing life-form is to be indefinitely sustainable? Equations have been written to address this question, but the real answer remains unknown.



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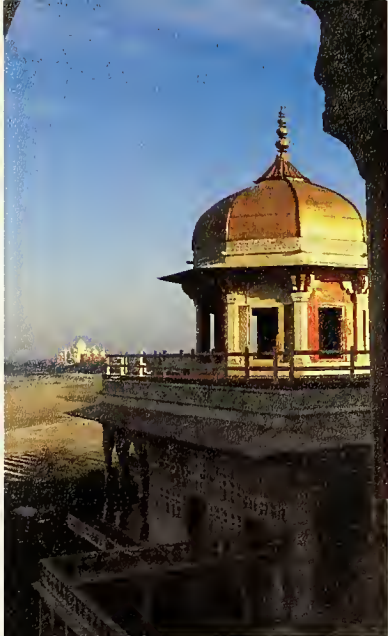
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**Among the highlights** of the program will be time in Delhi and Agra exploring the monuments of Old and New Delhi and visiting the incomparable Taj Mahal. This exquisite monument to love is one of the most impressive examples of landscaping and architectural symmetry in the world.

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## Study Leaders

### ANNAPURNA GARIMELLA

Departure: February 12 - March 2

A native of India, Annapurna Garimella is a specialist in Indian and Islamic art and architecture. A graduate of Columbia University, where she received her Ph.D. in art history, she was curator of the exhibition *Saris of India* at California State University Northridge, and documented paintings and wrote interpretive materials for the 1998 exhibition *Sakki: Friend and Messenger in Rajput Love Paintings* at the Sackler Gallery of Art. She is currently researching modern Indian religious architecture as a Visiting Fellow at the Center for the Study of Culture and Society in Bangalore, India.

### TO BE ANNOUNCED

Departure: February 19 - March 8

### JAMES CLAD

Departure: February 26 - March 16

James Clad holds the Henry R. Luce Foundation Research Professorship of Southeast Asian Studies at Georgetown University. A diplomatic officer posted in India from 1976-77, Professor Clad chaired the "Georgetown India Forum" last year. His views on India and Indonesia are frequently quoted in leading newspapers nationwide, and he regularly appears as guest commentator on Asia issues for National Public Radio, CNN, and CNBC.

### DAMODAR R. SARDESAI

Departure: March 5 - 22

Professor Emeritus of Indian History at the University of California, Los Angeles, Damodar Sardesai has written over a dozen books, including *India Through the Ages*, and more than 200 articles, papers, and book reviews. Among many academic honors, he has received honorary fellowships to the Royal Historical Society in 1979, Father Heras Society of Bombay in 1991, and was elected President of The Asiatic Society of Bombay in 1989.





## Itinerary

**Day 1**  
**USA | DEPARTURE**

**Day 2**  
**EN ROUTE**

**Day 3**  
**DELHI, INDIA**  
Arrive in Delhi and transfer to the *Taj Mahal Hotel*. Afternoon tour of New Delhi.

**Day 4**  
**DELHI**  
In the morning, explore Old Delhi. Tour the Red Fort, built by Mughal Emperor Shah Jahan as his royal residence; the Jami Mosque; and the Raj Ghat, the memorial to Mahatma Gandhi. This afternoon, visit the National Museum and its extensive collection of artifacts.

**Day 5**  
**DELHI | SIKANDRA | AGRA**  
This morning, visit Akbar's Mausoleum, an extraordinary work of architecture blending Hindu, Christian, Islamic, Buddhist, and Jain motifs. Continue to Agra and transfer to the *Jaypee Palace Hotel*. Later in the afternoon, tour the massive Agra Fort.

**Day 6**  
**AGRA | TAJ MAHAL | FATEHPUR SIKRI**  
This morning, visit the incomparable Taj Mahal. This afternoon, tour the abandoned yet perfectly preserved Mughal city of Fatehpur Sikri.

**Day 7**  
**AGRA | BHARATPUR | JAIPUR**  
Drive to renowned Keoladeo National Park this morning. Later in the afternoon, arrive at Jaipur for a two-night stay.

**Day 8**  
**JAIPUR**  
Tour Jaipur, including the Hawa Mahal, or Palace of the Winds. Continue to the Amber Fort. Also visit the Jantar Mantar Observatory and City Palace.

**Day 9**  
**JAIPUR | MUMBAI (BOMBAY)**  
Board a morning flight to Mumbai. In the afternoon tour the Prince of Wales Museum, Victoria Terminus, Marine Drive, Chowpatty Beach, and the Municipal Dhobi Ghats. Accommodations are at the *Hotel Taj Mahal*.

**Day 10**  
**MUMBAI | EMBARKATION**  
Enjoy an excursion by local boat to Elephanta Island, the small island famous for its eighth-century temple caves carved out of rock. This afternoon, transfer to the port to embark *Clelia II*.



*Royal Botanic Gardens, Peradeniya*

**Day 11**  
**GOA**  
After a morning at sea, arrive in Panaji for an excursion to Old Goa.

**Day 12**  
**MANGALORE | MUDABIDRI | KARKALA | MANGALORE**  
From Mangalore an excursion leads to several revered Jain shrines that attract pilgrims from all over India, including Chandranatha Basti, with an imposing entrance gate and two large columned halls, and Chaturmukha Temple, noted for its symmetrical proportions.

**Day 13**

**AT SEA**

**Day 14**

**TUTICORIN | TIRUNELVELI | TIRUCHENDUR | TUTICORIN**

Morning arrival in Tuticorin, once a thriving Portuguese colony. In Tirunelveli visit the 13th-century Nelliayappa Temple. After lunch, continue to Tiruchendur to explore the Subramanya Temple, one of South India's most sacred temples.

**Day 15**

**COLOMBO, SRI LANKA | KANDY**

Call at Colombo for an excursion to Peradeniya to see its beautiful Royal Botanic Gardens. Continue to Kandy and the lake-front Temple of the Tooth, which enshrines what is said to be a tooth of the Buddha. We also tour the Archaeological Museum, with its superb collection of sculptures and other objects.

**Day 16**

**AT SEA | COCHIN**

After a day at sea arrive in Cochin, the oldest European settlement in India.

**Day 17**

**COCHIN | DISEMBARKATION**

Explore via local boats the backwaters of Cochin to observe typical village life. Afternoon at leisure. This evening, attend a performance of the centuries-old Kathakali dance theater. Accommodations are at the *Taj Malabar Hotel*.

**Day 18**

**COCHIN | USA**

In the morning, tour the old districts of Mattancherry and Fort Cochin. After lunch, transfer to the airport for the return flight to Mumbai to connect with the flight to the United States.

**Day 19**

**ARRIVE USA**

## Departures

Depart	Return
February 12, 2002	March 2, 2002
February 19, 2002	March 8, 2002*
February 26, 2002	March 16, 2002
March 5, 2002	March 22, 2002*

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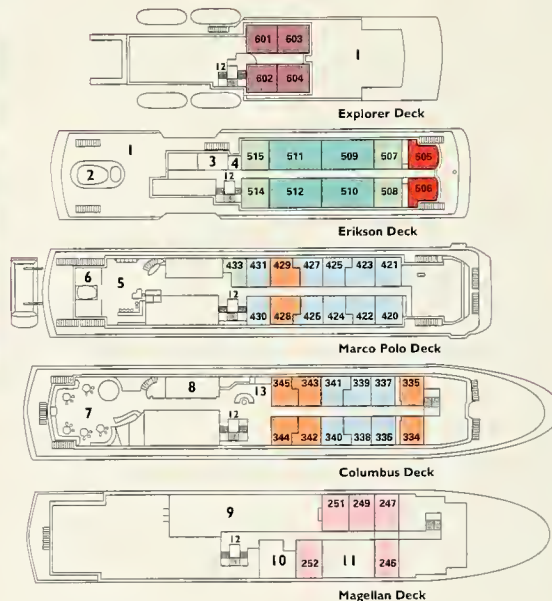


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A complacent conclusion can be drawn if we assume that sexual reproduction—with its ability to compensate for errors—has dramatically raised the upper limit on copying mistakes. If it has done so, then natural selection can easily take care of all the harmful mutations, and *Homo sapiens* is evolutionarily motoring along with more important worries than mutational error.

Another possibility is that the harmful-error rate has already reached an upper limit and we are in a mutational meltdown. This scenario is not only apocalyptic but unlikely. Our ancestors have probably been making 200 copying mistakes per offspring and putting the wrong number of chromosomes in 50 percent of them ever since the span of one hominid generation evolved to the modern figure of thirty years or so. Experts disagree about when this happened. Some argue that chimpanzees and gorillas have a generation time roughly similar to ours (between twenty and thirty years), which would push the origin of that trait back near the origin of great apes, to about 15 million years ago. Others would use a figure nearer 5 million years ago, when the human line branched off from that of the other great apes. Or, if our modern generation length dates back to the origin of the genus

*Homo*, maybe a figure of 2 million years would be better. If we have been just fine for 15 million years, we'd have to conclude that our mutation rate is truly sustainable. There is some evidence to suggest that we are accumulating mutant genes at a higher rate than are other species, but I suspect that our mutation rate is older than we are and is similar to that of chimps and gorillas. We are probably not mutating our way to inevitable extinction.

What does an understanding of genomic error tell us about what we can expect from the new human reproductive and genetic technologies? Such a discussion is futuristic and necessarily uncertain and conditional, but the way we understand evolution, error, and complexity does bear on the answers.

One potential reproductive technology that has aroused tremendous interest is cloning. Although full reproductive cloning—in which an individual

*Perhaps our species would be unharmed if we disposed of sex, but it would be a good idea to find out first.*

**Amorous couple in bed, terracotta, Old Babylonian period, ca. 1750 B.C.**





produces an offspring made from an exact copy of the DNA in his or her sperm or egg cells—might be used by a minority of human beings who have no other reproductive options, I suspect the practice is highly unlikely to become widespread. The reason is simply that cloning has a drawback: it suffers from as many errors as sexual reproduction does but lacks a crucial mechanism (sex itself) for clearing out the errors. If a subset of human beings signed up to use only clonal reproduction in the future, they would also be signing up their progeny for rapid genetic decay. Mutations would accumulate much faster than they could be eliminated. Not many generations would pass before all the clones were so loaded with genetic defects that they could not survive. (See “Why Sex Is Better Than Cloning,” page 49.)

At this stage of our understanding, opting to reproduce by cloning is rather like what volunteering for a heart transplant would have been during the era before the function of the immune system was known. The problem lies in messing with a design feature of our bodies when we do not know the design principles. Whatever factors allowed sexual reproduction to evolve, the advantages they conferred must have been big. Otherwise, the sexual form of life—in which each being is able to pass on only half its genes—would never have evolved in the first place. The lower reproductive rate is probably made up for by a difference in quality: the average sexual offspring is probably twice as good as the equivalent cloned offspring. In other words, sex may have evolved for some reason that we are clueless about, and perhaps our species would be unharmed if we disposed of it. But it would be a good idea to find out first.

Gene therapy, however, may be another story. Gene therapy means medically curing a defective gene by replacing it with a normal version, or by neutralizing it, or by some other technology yet unimagined. The use of such technology will, I expect, prove to be as acceptable as conventional medicine is now. (The idea of gene enhancement—in which an individual's genes are replaced with the aim of improving physical appearance and athletic, mental, or other abilities—will remain controversial.) If one is carrying a gene for a condition such as Tay-Sachs disease, deciding to undergo gene therapy may someday be much easier than the only options currently available: deciding not to have children at all, aborting a fetus that inherits the defect, or giving birth to a baby with an incurable ill-

ness. Such decisions are ghastly, and it therefore seems likely that people will use the new technologies to cure genetic error. Of course, these practices have not yet been shown to be safe for humans. And in any case, we cannot now do much with gene therapy, relative to its potential. In absolute terms, geneticists have identified a large number—

## THE RISK OF HAVING A SCHIZOPHRENIC CHILD IS THREE TIMES GREATER FOR FIFTY-YEAR-OLD FATHERS THAN FOR FATHERS UNDER TWENTY-FIVE.

maybe in the hundreds, maybe in the few thousands—of defective genes, but this is probably only the tip of the iceberg. Every one of a human's 30,000 or so genes will have several mutant, defective versions. Defects also exist in pieces of DNA that do not code for genes.

Whatever the benefits of gene therapy, the future may also bring technologies for preventing copying errors in the first place, thus eliminating the need for repair. Consider the idea of freezing gametes (or preserving them by some other method). In women, the quality of egg cells tends to decline with time. The possibility that a twenty-year-old mother will conceive a baby with an extra chromosome is negligible; the chance that a forty-year-old mother will do so is several percent; and soon after that, the biological clock reaches midnight. In the future, however, we may be able to stay the clock hands. Young women could opt to have some reproductive cells removed and softly embalmed, and then have them revived for pregnancy at a time of their own choosing. One consequence would be a reduction in the mutation rate in individuals (and in species, in proportion to the number of individuals who choose this procedure). Indeed, the mutation rate might be further reduced by harvesting the cells as early as possible—at birth, for example. Of course, the trade-off here would involve particularly knotty ethical problems (the impossibility of getting informed consent being the

*If a life-form is to persist, the average parent must produce at least one error-free offspring.*

Detail from *Offering to Venus*, by Titian, ca. 1518



most obvious). Men, too, could freeze their gametes. An old man's sperm contains so many mutations that the geneticist James F. Crow once joked that the greatest threat to the human genetic future is fertile old men. The broad consequences, if any, of freezing sperm are uncertain, but a recent U.S.

study found that fathers over fifty run three times the risk of having a child that develops schizophrenia than do fathers under twenty-five.

Could we ever evolve to be more complex? It's hard to say, and it depends on whether research proves that sexual reproduction is up to the task of









clearing the errors that would be created by an organism with a longer life, a longer generation time, or a larger genome. Early in the history of microbial life, the evolution of repair enzymes helped reduce the copying-error rate from about 1 in 10,000 to about 1 in 10 billion. If gene therapy by itself could be used to cure a large proportion of human genetic defects, it could become the cultural equivalent of those repair enzymes. The introduction of new gene and reproductive technologies could turn out to be not just a way to prevent individual heart-break but one of the most momentous events in the 2-billion-year history of complex life. It would rate with the handful of evolutionary breakthroughs: reliably replicating molecules, repair enzymes, the Mendelian machinery of inheritance, and the evolution of sex and gender.

Such a breakthrough in reducing error rates might permit the evolution of forms with a whole new level of complexity—in intellect or social organization, for example. But what might such a life-

## GENE THERAPY COULD BECOME ONE OF THE MOST MOMENTOUS DEVELOPMENTS IN THE HISTORY OF COMPLEX LIFE.

form look like? Thirty thousand genes of DNA code give you a complex being such as a human or a mouse, but what would 100,000 give you? Since education in our information-based society uses up a large fraction of the human life span, perhaps we could evolve to live longer. Or we might evolve more efficient learning abilities. Our skill in acquiring language between the first and second year of life is impressive, but genetic programming for early learning probably involves a large number of genes. With extra genes at our disposal, we could acquire other skills the same way, with our brains prompting us in the right direction. Each skill would have its own set of DNA codes. We could then pick up computer programming, for example, or methods of pricing derivatives on the futures market, the way we learn to understand and speak our own languages.

*Unlike sex, cloning has the drawback of creating copying errors that cannot be corrected.*

*Large Triple Elvis, by Andy Warhol, 1963*

Being brainier, however, may not be the best way to become more complex. I have in mind another fanciful idea, inspired by the late evolutionary theorist W.D. Hamilton. If 30,000 genes are needed to code for a human being or a bird, and 20,000 to code for an oak tree or a lobster, 100,000 might code for all four. The resultant organism would not combine the features of all those organ-

## WHY SEX IS BETTER THAN CLONING

All reproduction gives rise to some mutations, or errors, during the copying of DNA messages. Sexual reproduction, which operates according to Mendel's principles of inheritance, has the advantage of redistributing the parents' mutations among the offspring. In effect, a toss of the coin determines whether any particular gene will be "allowed" into each embryo. Meiosis is the fateful cell division in which each gene, whether perfect or mutated, has only a 50-50 chance of making its way into a gamete—a particular sperm or egg. On average, if a male or female with one harmful DNA mutation produces eight gametes, four will have the flaw and four will be free of error. When the sperm and eggs of two parents—both of whom have one harmful mutation—are combined to form eight new organisms, four offspring on average will have one harmful mutation and two will have two, but the remaining two will have no mutations at all. The life-form carries on.

Now consider a clonal life-form. If a parent with one harmful mutation decides to have eight offspring, all eight will inherit the flaw. A life-form that reproduces this way will continue to accumulate errors each time its DNA is copied; over several generations, it becomes unsustainable and will be destroyed by its mutations. Though some life-forms—certain plants, for example—use clonal reproduction, they also have sex from time to time. On the family tree of complex life, only a few odd twigs are exclusively clonal.

Another reason to doubt we could go in for cloning in a big way is that sex is probably a necessary condition for complex life. This conclusion would stem from the theory (put forward by Alex Kondrashov, of the National Center for Biotechnology Information) that sexual reproduction evolved primarily to purge bad genes. Another plausible theory is that sex exists to keep us from being destroyed by parasites and microbial pathogens. Since infectious bacteria, as well as viruses and parasitic protozoans, evolve rapidly as they exploit our bodies as habitats, we need to make genetic shifts in each generation to keep up with them. Even if the parasite-avoidance theory is a better explanation for why sex evolved, cloning would still be a bad idea. Your cloned offspring would be more likely to die of infectious disease rather than genetic disease. Cloning yourself would be like taking your children to a plague-stricken city, where the chance that they will die of plague is doubled.



isms; such a monstrous body would have hopelessly difficult integration tasks. What the extra genes might provide is the opportunity to choose which life-form to become. At some embryonic stage, our large-genomed creature could assess its environment and see where the best opportunities lay. If a niche for oak trees was relatively unoccupied, it could commit to this form and grow up as a tree that produces acorns. If the sea bottom was underexploited, it could grow claws, eight legs, and a spring-action tail—and watch out for lobster pots. Unconstrained by the form of its parents, the embryo would pick the adult form that promised the best reproductive return. The embryo itself would be a complex creature, because it would have to assess all those environments and opportunities. It might start out in larval-assessor form and then undergo a metamorphosis as it switched on all the necessary genes for its preferred adult form. All the unused genes would simply be switched off, perhaps until the next generation.

But there may be many reasons such a flexible form has not evolved. An important one is that natural selection—creating a massive slaughter of mutants in every generation—is needed to ensure that genetic information is not erased by mutational decay. If genes are not expressed, natural selection cannot work on them. Put another way, if a gene is not used, it is lost over evolutionary time.

The destructive force of mutation has prevented earthly life from evolving reserves of occasionally expressed genes. But what if that force were relaxed or resisted? If future technology could accomplish this, or if an otherworldly method of reproduction superior to Mendelian inheritance emerged, life could add to its reserves of DNA. The future would lie with life-forms that, although not necessarily more intelligent than humans, might have genetic subroutines that could be called up as appropriate. After fire and brimstone, such descendants could reinvent themselves as fire-adapted flowers and cover the scorched Earth with fresh foliage. After the deluge, they could grow up as fish and swim safely beneath the waves. □

*If we evolved extra genes, we might acquire computer skills as effortlessly as we pick up our native language.*

*Golconde, by René Magritte, 1953*

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# BACTERIAL REVELATION

**Genomics is providing a wealth of information about some of Earth's littlest, oldest, and most abundant living things.**

Story by Roberta Friedman ~ Illustrations by Robert Grossman

## Pumping Metal

How do certain bacteria subsist amid heavy metals, oils, and rank toxic sludge—substances that kill most other forms of life? Genomics may help researchers answer this question and, as a result, aid in the cleanup of humanity's nastiest messes.

One bacterium that may be useful in mopping-

up operations is *Ralstonia metallidurans*. Through its ability to turn normally poisonous heavy metals into harmless carbonates, this bacterium has the potential to make the environment safe for other forms of life that lack its transformational powers. Since the carbonates accumulate on the surface of *R. metallidurans*, if these microbes are allowed to work for a while and are then removed, the heavy metals can be effectively removed with them.

Last October, as part of its first annual "Microbial Month," the U.S. Department of Energy's (DOE's) Joint Genome Institute in Walnut Creek, California, sequenced the genomes of fifteen bacteria. One was *R. metallidurans*. Although not finalized, the sequences give researchers a good idea of how this microbe survives and even thrives in the most hostile environments. The DOE's draft of this organism's 3,000 genes may in fact reveal the secret of some microbes' ability to pump heavy metals and precipitate them harmlessly.

The genes that confer *R. metallidurans*'s resistance to heavy metals are on a circular bit of DNA called a plasmid. Plasmids are the shuttle buses of the bacterial genetic world, easily transferring genes among microbes—even across species. And according to John Dunn, a biologist at the DOE's Brookhaven National Laboratory, *R. metallidurans*'s plasmids are about ten times the usual size.

Before Microbial Month, only one percent of this bacterium's genome had been known. Now, because of the DOE-led effort, scientists can contemplate, for example, adding genes to *R. metallidurans* that would link its uptake of heavy metals to biolumines-



METAL-MINING MICROBE



NS



cence. Glowing bacteria could then indicate the presence of heavy-metal contaminants. Researchers might also be able to effect the transfer of *R. metallidurans*'s pumping instructions to other bacteria, or to use it as a host for other genes that could improve upon its talents.

The very flexibility of the *R. metallidurans* genome is what allows it to adapt nimbly to changing and challenging environments, says Dunn. Of course, tinkering with such a mobile microbial genome raises concerns, and scientists working on the organism do not propose to release altered genomes without first amassing adequate knowledge of the habits these bugs might adopt. To that end, Dunn expects that within a year, the full genome of *R. metallidurans* will be mounted for study on a DNA microarray chip. Even with microarrays—which will allow scientists to take snapshots of the microbe's genes in action—it will be some time before the story of *R. metallidurans*'s lifestyle is unraveled.

## By the Light of the Sea

Efforts to probe marine ecosystems have always been hampered by the difficulties of exploring the open ocean. But now that the science of genomics is making possible the decoding of complete genetic blueprints, researchers are quickly moving toward a more comprehensive understanding of the marine food chain.

Take the cyanobacterium *Prochlorococcus marinus*. Discovered only fifteen years ago, it is the smallest and most abundant photosynthetic microbe on

Earth: one tablespoon of seawater may contain 10,000 of them. Together, all species of *Prochlorococcus* make up nearly a third of the ocean biomass that uses light to make food. Penny Chisholm, of the Massachusetts Institute of Technology (MIT), points out that, along with other phytoplankton, the carbon dioxide-consuming *P. marinus* plays a key role in the regulation of CO<sub>2</sub> in the atmosphere. "If all the phytoplankton suddenly died," she says, "the CO<sub>2</sub> concentration in the atmosphere would increase two- to three-fold."

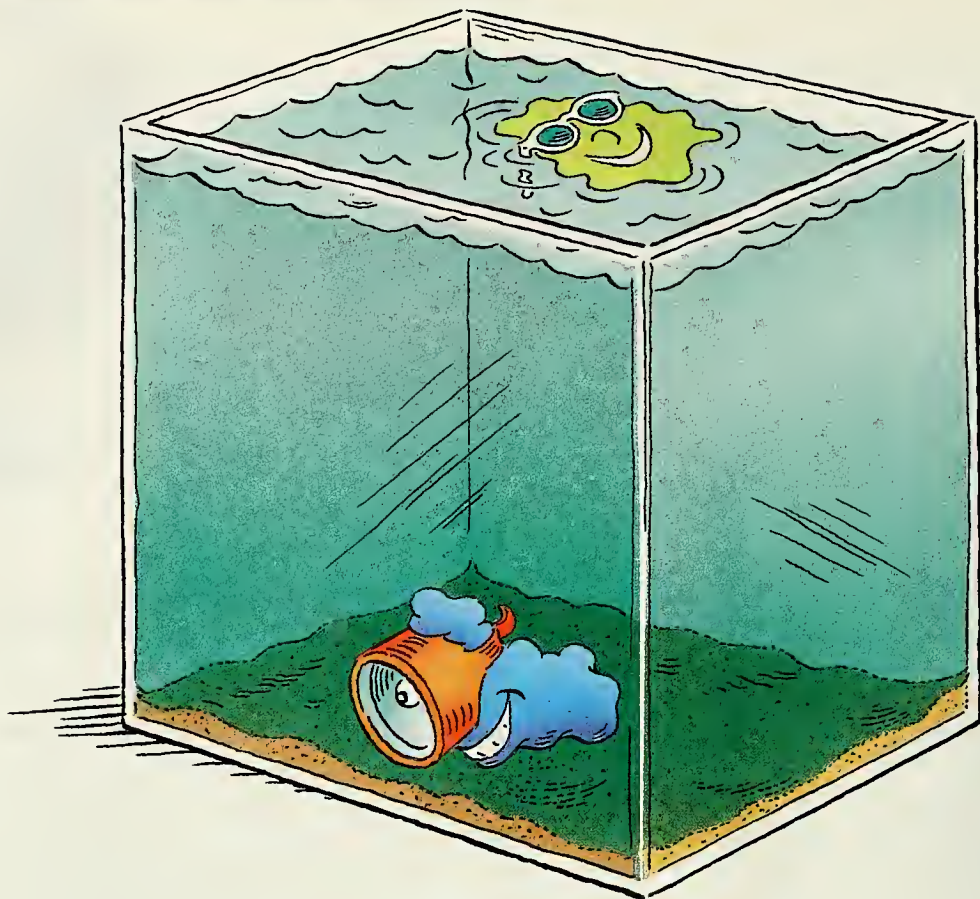
One strain of *P. marinus*, MED4, lives in rather brightly lit surface waters. Another strain, MIT9313, inhabits deeper waters. Researchers at MIT, the Joint Genome Institute in California, and the Oak Ridge National Laboratory in Tennessee have been working to determine the complete genetic instructions of both strains. And it turns out that the two are quite different.

The surface-dwelling cyanobacterium has fewer genes—1,700 compared with the 2,400 of its low-light relative. However, it possesses many more genes that are activated by light. And as befits a surface dweller, it also has an enzyme that repairs damage to its DNA caused by exposure to ultraviolet light.

What does the deepwater specialist have that its shallow-water counterpart doesn't? Apparently, it can make a living off diverse sources of nutrients. For a start, it bears genetic instructions for making enzymes capable of utilizing the nitrite that is present in deeper waters but absent from the surface waters of the open ocean. In addition, the deep-water cyanobacterium carries the codes for several enzymes that handle sugars.

Scientists are also using genomics to examine how certain microbes thrive in novel ways, using the abundant light available in the surface waters of the





## SHALLOW AND DEEPWATER CYANOBACTERIA

ocean. A pigmented molecule called rhodopsin helps many creatures use light; in humans, for example, it is present in the retina. But Edward F. DeLong and Oded Beja, of the Monterey Bay Aquarium Research Institute in California, certainly didn't expect to find rhodopsin in oceanic microbes, because it had never been found in *any* bacteria. But find it they did. They confirmed that these rhodopsins are capable of harvesting biochemical energy from light, thus giving the microbes an energy boost from sunlight. Since rhodopsin-containing microbes are widespread in the sea, the researchers predict that this harvesting is an important oceanic process.

DeLong, Beja, and colleagues also discovered that microorganisms gathered from Monterey Bay and from surface waters of the Pacific Ocean north of Hawaii contain a red-reacting rhodopsin, while those from the Antarctic and the deep waters of the North Pacific have a blue-reacting variant. The difference most likely occurs because blue light reaches the farthest depths of the sea, while red light quickly attenuates with depth. The bacteria appear to have "tuned" their rhodopsins to react

optimally with available light. Without modern genomics, the researchers would never have discovered oceanic rhodopsin or detected the habitat-specific spectral tuning evident in its variants.

## How TB Plays Possum

More than sixty years into the antibiotic era, tuberculosis is still the world's number-one infectious killer. One reason the disease has been so difficult to eradicate is that *Mycobacterium tuberculosis*, the microbe that causes it, has a talent for hiding out in white blood cells—sometimes for decades. A third of the world's human population harbors the bacillus in its dormant state.

*M. tuberculosis* may cause active disease right away, or it may weather the initial attack by the body's defenses and then enter a state of latency, persisting quietly in a kind of equilibrium with the immune system. If a host's defensive line falters (when the immune system is suppressed by AIDS, for example, or weakens with malnutrition or ad-

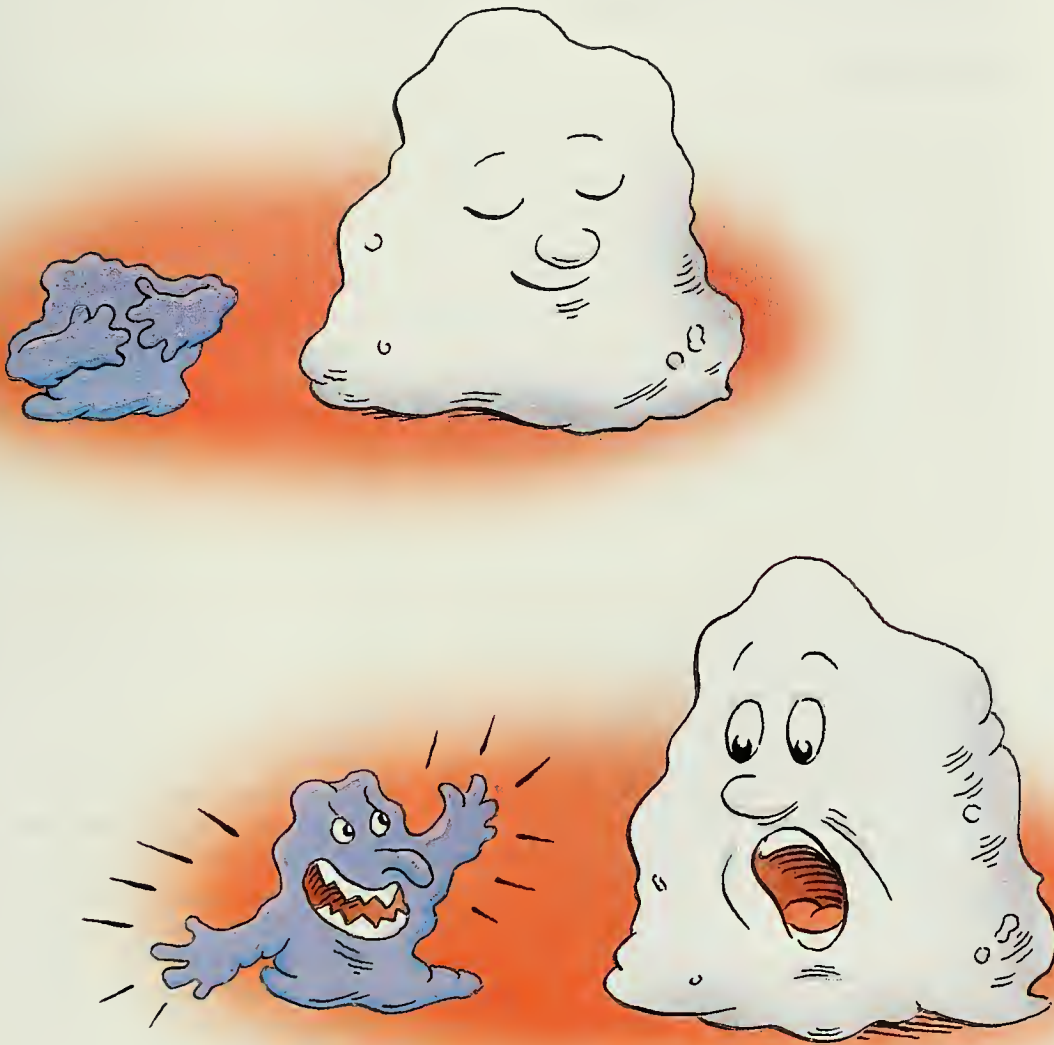


vancing age), active disease ensues. The ability to coexist with its host is the mark of a successfully evolved pathogen, and the tuberculosis bacterium has an extremely effective strategy for doing so, says researcher Gary K. Schoolnik, of Stanford University School of Medicine's Beckman Center.

Schoolnik has been able to show which genes the TB pathogen "turns on" when its hosts—the large white blood cells known as macrophages—start responding to its presence. When the macrophages' immune response is activated, the pathogen undergoes fundamental metabolic alter-

ations to survive in the host cell. One component of this change is a switch to the hosts' fatty acids as a principal source of carbon for energy. The pathogen also responds to nitric oxide, one of the main products of the activated white cell. When nitric oxide levels are high, the TB pathogen seems to slow or stop replicating. When its production declines, replication resumes. The results suggest, but do not prove, that nitric oxide induces the disease's dormant state.

There are many other puzzles for Schoolnik to grapple with as he tries to unravel the secrets of *M.*



TUBERCULOSIS GERM PLAYING PEEKABOO WITH A WHITE BLOOD CELL



*tuberculosis*'s success. For one, the microbe seems to be turning on the genes it uses to acquire iron from its surroundings, even though the nutrient mixture in which he is cultivating the microbe is rich in iron. Perhaps the toxic soup of macrophages and other white blood cells encountered by the microbe lead it to sense that iron is depleted, or perhaps more iron is needed by the bug in this environment.

So far, says Schoolnik, the lesson from the genomic investigation of tuberculosis is that what appear to be genes for ordinary metabolic functioning could in fact be crucial mediators of the infection's virulence.

## Pillars of a Salty World

Sequencing the genomes of the ancient kingdom of microbes called archaea yields evidence that all life shares some basic strategies, despite disparate ways of earning a living. Consider this: the tiny genome of *Halobacterium*, a microbe that thrives in the saltiest, most landlocked bodies of water, shares many similarities with complex (eukaryotic) cells, even those from humans.

Last year, an international team finished sequencing the NRC-1 strain of *Halobacterium*. The vulture

of Earth's salty puddles, NRC-1 grows on the degrading carcasses of less sturdy organisms that die off as salinity mounts due to evaporation. The genome of *Halobacterium* NRC-1 facilitates this process by including instructions for generating such exotica as a putrescine transporter—a molecular version of a waste-disposal truck. The microbe also contains the directions for making proteins that can eject toxic heavy metals such as arsenic and cadmium.

NRC-1 can prosper in water ten times saltier than Earth's oceans and can function happily with hypersalty innards. Unlike many other exotic microbes, it is easily grown in the lab, making it a potential workhorse for future research on the entire group of archaeal microorganisms known as extremophiles.

International efforts have resulted in the discovery that NRC-1 genes are carried on three replicating units, only one of which is as big as a typical chromosome; the other two are "minichromosomes." The genome sequence was used to predict about 2,600 genes, a third of which do not resemble any other known genes. Yet many of NRC-1's genes do carry instructions for familiar proteins—for example, those that facilitate molecular signaling across the cell membrane and those that are used for metabolic (housekeeping) functions. In fact, NRC-1 carries genetic instructions for making many cellular systems similar to those found in plants and animals.

Microbiologist Shiladitya DasSarma, of the University of Massachusetts Amherst, led the international sequencing project. He explains that NRC-1's trick to surviving in ever saltier water is to have proteins on its inside that carry a high negative charge. Bacteria that lack such proteins succumb to salt poisoning.

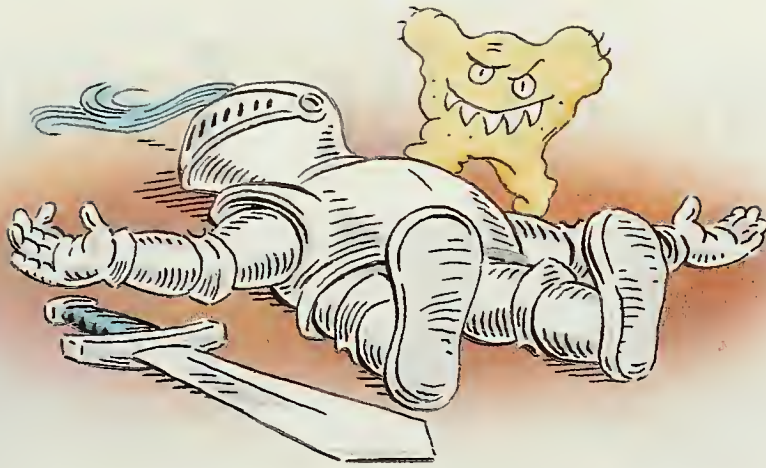
Also found in NRC-1's genes are the machinery for growth in both the presence and absence of oxygen; a primitive photosynthesis system; and a very efficient DNA repair system that protects against damage by harsh sunlight. This hardy strain of *Halobacterium* is also genetically rigged with sensing systems that guide it to the best-lit waters and to the optimal locations where nutrients, temperature, and oxygen can stimulate its growth. NRC-1 even has a protein molecule for a membrane that can be likened to a primitive retina, and a component that in other bacteria (as well as in plants and animals) acts to maintain circadian rhythms.

All in all, the microbe should serve as an impressive genetic ambassador from its ancient kingdom.



HALOPHILIC MICROBE





## TYPHUS – THE KILLER OF SOLDIERS

### Typhus, Cats, and You

Despite having one of the smallest genomes known, *Rickettsia prowazekii*, the typhus-causing pathogen, has played a major infective role throughout history. More soldiers have been killed by typhus (lice spread it) than have been killed in battle. The microbe's genome itself looks like a molecular theater of war, with dead genes strewn among the living. According to Siv G. E. Andersson, of Sweden's Uppsala University, that's because fully a quarter of the microbe's compact genome is made up of a kind of junk DNA—inactivated, ancient genes in their final stages of deterioration.

Andersson, part of the team that sequenced the typhus genome, has also found that the little killer's working genes turn out to be remarkably similar to those that govern some important functions of the mitochondria, the energy-generating packets within all animal and plant cells. Mitochondria have tiny genomes that encode only a few proteins; the rest of the instructions for their task of generating cellular energy reside in the centers, or nuclei, of the cells they inhabit. The similarity between some of the nuclear genes that code for the mitochondria's energy production and the genes of the typhus organism suggests that these instructions were transferred into the nuclear genome from a bacterial ancestor of the mitochondria. According to this scenario, when oxygen levels started to rise in the atmosphere 2 billion years ago, our cellular ancestors had to adapt or die: they responded to the catastrophe by swallowing oxygen-

respiring bacteria and converting them into little energy-producing organelles, the mitochondria. Andersson's sequencing of the typhus genome has yielded remarkable insights into this evolutionary heist.

When a formerly independent organism takes up residence in a host, it begins to depend on the host for its basic needs, and some of its own genetic instructions become redundant. Mutations can occur in the genes of the symbiont without consequences for its survival. Theorizing that the typhus pathogen may have had just such a history, Andersson contrasts its genome with that of *Bartonella henselae*, the microbe responsible for cat-scratch disease. Typhus microbes can survive only within other cells, whereas the cat-scratch pathogen, in the same microbial family, still carries functioning genes that allow for a more independent lifestyle. *B. henselae* microbes are taken into the infected cell, but they don't live directly in the cell's fluids. Rather, they live in a kind of bubble called a phagosome.

*B. henselae* can do even more for itself: it has retained genes instructing it to create the building blocks for proteins and DNA. The typhus pathogen, on the other hand, lacks the genes for making amino acids and nucleosides and depends on its host cell for a steady supply of them. As much as 80 percent of the typhus pathogen's original complement of genes may have been lost or inactivated as it evolved its completely parasitic existence, Andersson speculates.

Meanwhile, evidence accumulating from genomics suggests that the nucleus of the so-called eukaryotes, or nucleated cell organisms, is itself a donation from an ancestor of the microorganisms known as archaea. □



# Born To Be Tame



To attract a mate, a male houbara bustard, above, fans his elongated crown and neck plumes and may perform an animated "display run" for an hour at a time.

*To survive in the desert of Saudi Arabia, captive-bred bustards have to learn to go wild.*

*By Yolanda van Heezik and Philip Seddon*

Imagine you're living on your own for the first time. Away from the comforts of home and in a strange town, you're preoccupied with looking for a place to get a meal and you inadvertently wander onto a dark street. A large man comes toward you. Perhaps he could give you directions. You smile hesitantly. He smiles back and then flourishes a knife. Too late, you realize you've made a fatal mistake.

In the natural world, naïveté is costly, but it is also rare in animals that share their environment with natural enemies. Constantly faced with the danger of becoming someone else's dinner, prey animals are skilled in recognizing and avoiding poten-



tial predators. In some species this ability is largely innate, while in others it seems to be learned from parents or other members of a herd or flock. Animals raised in an artificial environment and without such guidance often lack the predator-detection skills of their wild cousins.

For nine years we worked at a captive breeding center for Asiatic houbara bustards in Saudi Arabia. At the National Wildlife Research Center in Taif, female bustards are artificially inseminated and the chicks are hand-raised and then released. The goal is to reestablish healthy populations of bustards in their Saudi Arabian range. One of the first challenges the program faced was how to prepare naive bustards to survive—to eat and not be eaten—in the desert.

Camouflaged and, in their natural state, wary birds, Asiatic houbara bustards are at home in the undulating steppes and semideserts of the Arabian Peninsula, western and central Asia, and the Indian subcontinent. Superbly adapted to arid environments, wild houbaras do not need to drink water but manage to get all the moisture they need from their food. Indeed, part of the secret of the bustards' survival may be their varied diet. Opportunists at heart, they will try most edible objects they encounter, from juicy berries and young green shoots to crunchy beetles and sunbathing lizards. Though powerful fliers, houbaras prefer to walk and are more difficult to discern when on the ground. When they do take to the air, their size (wingspread is about five feet), their deep wingbeats, and the black patches on their wings and neck make them easy to recognize. Houbaras' strong flight, coupled with a fighting spirit, make them a premier quarry in the ancient sport of falconry. This kind of hunting, in which the falcons are trained to attack much larger birds, is one reason bustards are threatened throughout much of their range.

Houbara bustards are also sensitive to human disturbance. Before Saudi Arabia's oil-fired economic expansion, the wanderings of nomadic herdsman were dictated by the presence of water and green vegetation. Today water is trucked to livestock, and both the herds and the four-wheel-drive vehicles penetrate once pristine landscapes. In

response to the decline of resident houbaras in the Arabian Peninsula, the Saudi government began a conservation program in 1986. This project included captive breeding and the creation of large protected areas. By 1992 the successful program of artificial insemination and incubation had produced a surplus of chicks, enough to begin introducing some of them into the wild. Juvenile birds, from thirty-five to forty-five days old, were at first re-



leased into a predator-free enclosure of about one and a half square miles, where they could learn how to find natural food. In their own good time, the young birds could simply fly out into the wider reserve, a fenced area of 850 square miles that was free of livestock and human predators but had a full complement of natural predators. Some bustards, on first leaving the enclosure, immediately retreated back inside, while others headed off into the desert.

Considering the enormous transition the young birds had to make, many of them fared well as far as food finding was concerned. After growing up on a diet of unlimited food pellets, alfalfa, mealworms, crickets, and water, they were somehow able to rec-

**Tiny and mainly insectivorous, Rüppell's fox, above, rarely preys on newly released bustards, but many of the young birds fell victim to red foxes.**





Houbara bustard chicks, above, are cared for by hand. Right: A young red fox, Sophie, was enlisted to give predator-avoidance lessons.



YOLANDA VAN HEZIK

ognize and collect their natural plant and insect foods. They coped without water in temperatures that soared above 100° F and eventually even figured out how to breed successfully.

The main stumbling block along the road to self-sufficiency was predation. As more and more of the newly free young birds fell victim to deadly attacks, we considered a list of likely suspects. The delicate little Rüppell's fox inhabits the region, but its predominantly insectivorous diet and small size argued against its being the villain. (However, should a hungry Rüppell's fox blunder upon a recently released bustard barely able to fly and searching for its water dish, then the fox is likely to prefer





YOLANDA VAN HEZIK

**Eager to chase bustards, Sophie is kept in hand by author Philip Seddon. Each carefully supervised training bout lasted just three minutes.**

short distances at one month of age. They are taught by their mothers or other bustards how to recognize and avoid foxes and other dangers. The captive-bred houbaras missed out on these lessons, and many paid the price. Navigating a new, seemingly limitless environment, finding appropriate sources of food, and learning to live without water was demanding, yet possible. Having to cope with predators at the same time was too much.

To help get the birds through this critical period, the research center's staff decided to trap foxes living around the predator-free enclosure and move them far away. When the young birds left the enclosure, provided they remained close by, their chances of running into a hungry fox were diminished. Despite these trapping efforts, more than half the bustards released were quickly killed by predators—three-quarters of these by red foxes. Removal of foxes from a buffer zone around the enclosure had only delayed the birds' encounters with predators; the proportion killed was the same.

Our bustards were in danger of becoming "bird nerds"—hand-reared individuals that, if they manage to survive, still have handicaps that even other

the bird over another beetle.) The lovely, wide-faced sand cat could have preyed on some birds, but the habits of this desert species are little known. Later on, we found that eagle owls could become habitual houbara killers once they discovered how easy it was to dispatch naive birds. But our investigation of the tracks and other signs around predator-killed carcasses indicated that the main culprit was the red fox.

Not as common in the region as Rüppell's foxes, red foxes ordinarily pose little problem for houbara bustards. Wild houbara chicks, usually two or three per clutch, stay with their mother for about three months in total, even though they begin to fly



Although primarily ground birds, houbara bustards are strong fliers when they take to the air, right. In the wild, clutches—usually of two or three eggs—are laid directly on the desert surface, below.



XAVIER ESCHNER, NWRC PHOTO LIBRARY



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members of their own species can readily recognize. A study of captive-bred male partridges, for example, has shown that when released, these birds not only lacked some basic survival skills but also were less attractive to females than were wild birds. Hand-reared female partridges were more likely than their wild counterparts to lose their eggs and chicks to predators.

Could our birds be instructed how to recognize predators and behave appropriately around them? At first, we tried exposing the naive birds to simulated attacks, using a stuffed red fox. This method was safe and allowed us to repeat the training trials. But while the young bustards did seem to fear the model, when released they were just as likely to be devoured by a fox as were unschooled birds. We began to think that the birds would benefit by experiencing a more realistic attack. For this we needed a live predator, but we wanted one we had some control over. To this end, one chilly March morning, we raided a fox den in northern Saudi Arabia, carefully extricating a ten-day-old female fox.

We named her Sophie. She spent her first weeks snuggled inside layers of our clothing and waking us up at night with her warbling wails for food. As she grew, her prowess at chewing and climbing was marked by our gradually raising all our possessions onto higher and higher shelves and into cupboards far from fox-reach. Sophie was very playful and liked nothing better than to be entertained by us. Her delightful personality threatened to shift our allegiance from bustards to foxes. We reminded Sophie that she was going to have to work for her keep, and we spent time trying to accustom her to walking on a lead and wearing a muzzle. We failed miserably on both counts. The lead was sometimes ignored, and at other times Sophie went off at a breakneck pace, dragging us behind, until she jerked to a halt to investigate some odoriferous burrow or bone. The muzzle was apparently a grievous insult to a fox and resulted in her complete immobility. We despaired that she would ever be of use in training the bustards.

The day came, however, when young birds were ready to be moved from the captive breeding facility to the enclosure, and Sophie was caught, collared, and taken to a specially designed training cage. The birds had three training sessions, of about three minutes' duration each, over the course of a week. During those three minutes Sophie would tear around the cage, darting both at and away from



the bustards, sending them up and crashing into the cloth walls and roof in a panic. We were treading a fine line between making the sessions frightening enough to teach the birds the lesson and damaging them. Eventually, half the bustards were “trained”; the other half, a control group, did not make Sophie’s acquaintance.

Before being released, all the birds were fitted with radio transmitters that allowed daily monitoring of their movements. We then waited anxiously for news. Past experience had told us that most deaths would occur within about two weeks after the birds left the security of the enclosure. As expected, over the next few weeks, some birds went missing and some were lost due to illness or injury. Even if a bird was killed by a predator, its transmitter usually remained intact. The remains of individuals could be found quickly, and a cause of death could usually be ascribed. Within a few months, we were able to congratulate Sophie on the contribu-

tion she had made to houbara bustard conservation. Her foxiness must have left a lasting impression on the young houbaras, because significantly more birds in the “trained” group survived.

Most of the bustards that fell prey to foxes did so during the first week on their own. None of those that died survived longer than nineteen days, but those that lasted more than nineteen days appeared to be no longer at risk. We believe this is the critical period during which the bustards learn to adapt to a new diet and new environment.

The houbara bustards’ experience with Sophie may have helped them develop some simple rules—the primary one being to react rather than hesitate during a dangerous encounter. Our training technique is now available for use as future releases take place in new sites. We like to think we took a little of the nerd out of the birds and gave them a flying start toward self-sufficiency and a real life in the Arabian Peninsula.

**Stillness and cryptic coloration can be natural defenses in the bustard’s environment.**

**Below: A captive-bred female bustard, equipped with a radio transmitter, is almost invisible as she incubates her eggs.**







The big, bulbous nose of the adult male proboscis monkey consists of erectile tissue that fills with blood when he is excited. This causes the nose to become red and swollen—a display that speaks volumes to the female, whose own nose is petite and upturned.



# A NOSE FOR ALL REASONS

From the size and arrangement of fossil nose bones, paleontologists are certain that many dinosaurs had huge, fleshy noses. The anatomy of some, such as the hadrosaur *Saurolophus*, left, suggests the presence of inflatable nasal sacs. One way to explore this and other possibilities is to study the noses of living animals—the hooded seal, for example.

MARK HALLETT



From courtship to camouflage, sinking to swimming, there's a nose for the job.

By Lawrence M. Witmer

**N**oses are for more than just smelling—or for holding up spectacles, as Pangloss remarks in Voltaire's *Candide*. Olfaction is certainly the nose's ancestral role, and smelling remains important for most animals, but in many disparate groups of vertebrates the organ has been co-opted for a variety of other, quite different functions. The reasons for the nose's evolutionary adaptability are straightforward. For starters, it is well positioned to greet the environment. In addition, it can be modified without compromising essential tasks such as locomotion or chewing. Finally, nasal anatomy makes use of diverse raw materials—bone, cartilage, muscle, blood vessels, connective tissues—on which natural selection can draw.

I was thrust into the arena of nose anatomy by dinosaurs. Along with Scott Sampson, of the University of Utah, I had noticed that certain groups of dinosaurs had enormous and complicated noses, some taking up half the skull. Clearly, something biologically important was going on. Previous researchers had proposed a number of possibilities, based on loose analogies with animals living today. Hoping to unravel the enigma of dinosaur noses, we decided to conduct our own studies of modern analogues. My collaborators (including many students) and I soon discovered just how evolutionarily labile the vertebrate nasal apparatus is.

A variety of mammals, such as tapirs and elephants, have evolved a trunk (nose plus upper lip) capable of manipulating objects. In many species, the nose has been transformed into a display organ, such as the fleshy knob on the snout of the gharial, the inflatable sac of the hooded seal, and the swollen nasal appendage of the proboscis monkey. Often—as in these three species—only the males possess display-worthy noses, which serve to communicate information about their health and status both to females and to rival males. Many birds and some antelopes, notably the dik-dik and the saiga, sport noses that give them an exceptional ability to regulate brain temperature and conserve water.

The list could go on—the nasal snorkel of many aquatic turtles, the nasal leaf of many echolocating bats—but the general point is that all these noses are built from a limited set of anatomical tissues. Evolution “makes do” with what's available. Now our task is to add all this information to the fossil evidence, with the goal of reconstructing the dinosaurs' soft tissues and determining the biological roles of their nasal novelties. Our studies of tapirs, for example, indicate that dinosaurs most likely did not have trunks, as some researchers have proposed. Instead, we think that the nose of *Triceratops* and some other enormous dinosaurs was not a mechanical tool, and we are currently homing in on its precise physiological function. Stay tuned.



MICHAEL AND PATRICIA FOGDEN



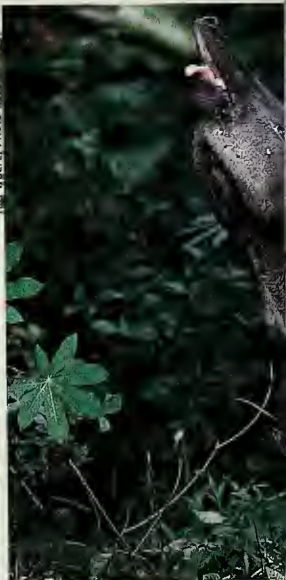
The enigmatic nasal appendage of Madagascar's leaf-nosed snake may enhance camouflage and/or improve the animal's sense of touch. The male's nose ends in a tapering spike and the female's in a flattened "leaf," so the appendage may play a role in social display as well.

MICHAEL AND PATRICIA FOGDEN



The nose of the white bat is shaped like a leaf, an adaptation for echolocation. Leaf-nosed bats vocalize through their noses, not their mouths, and the leaf both modulates and focuses the sound.

ART WOLFE PHOTO RESEARCHERS, INC.



MITSUAKI IWAGO: MINDEN PICTURES







A tapir's trunk works more like an octopus's tentacle than like a typical vertebrate appendage. Made mostly of muscle, fat, and connective tissue, this highly flexible organ is useful for everything from rummaging in debris on the forest floor to "directional smelling."



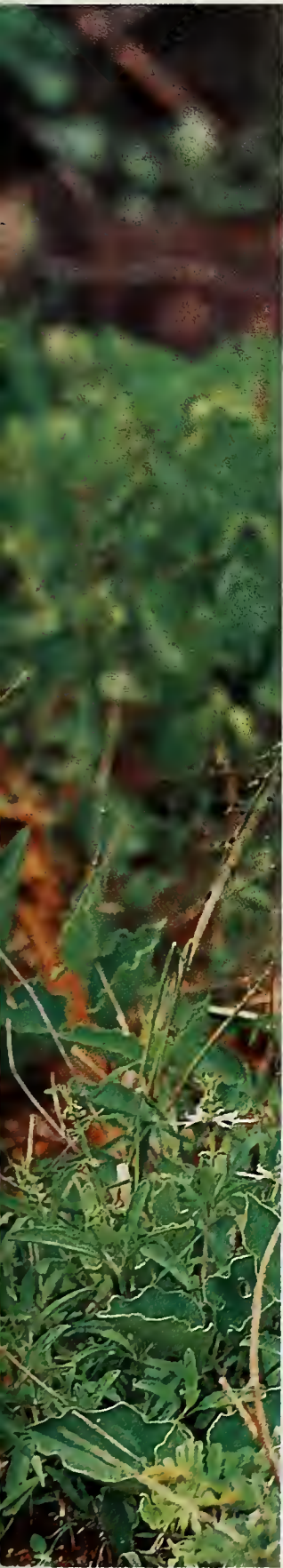
DOUG ALLAN; BBC NATURAL HISTORY UNIT

Male hooded seals have two dramatic displays. The first (inset) involves closing both nostrils and exhaling, thus inflating the highly elastic black skin of the nose. For the second display, the male closes just one nostril and exhales, blowing out the blood-red elastic nasal septum through the other nostril.



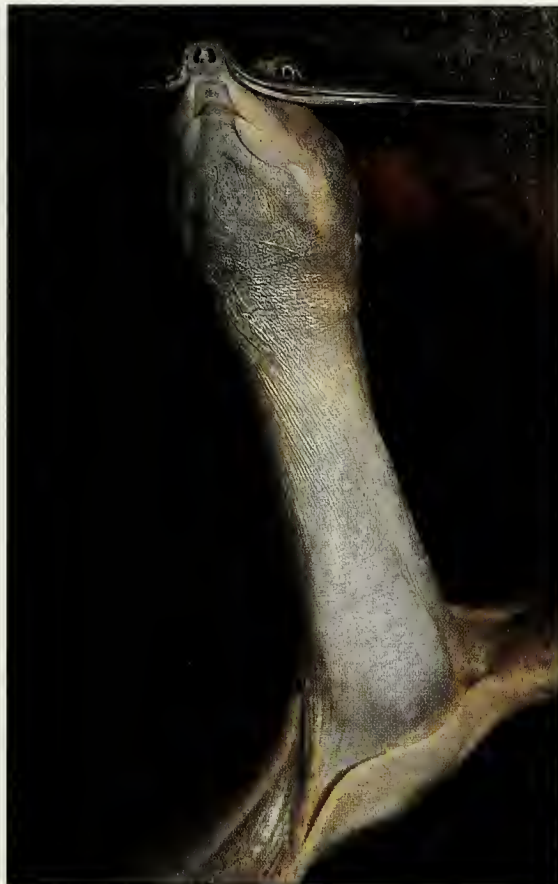






In their hot, arid African habitat, dik-diks may go months without drinking. The anatomy of the proboscis and nasal cavity enables these tiny antelopes to reclaim water otherwise lost during exhalation and to cool blood going to the brain.

ART WOLFE



JOE McDONALD: ANIMALS ANIMALS

In many soft-shelled turtles, the nasal cartilages are greatly elongated, effectively turning the nose into a snorkel. Thus equipped, the turtle can remain submerged, periodically extending its nose above the water's surface to breathe.



MICHAEL QUINTON: MINDEN PICTURES

Moose noses are expanded affairs, with cartilaginous plates and connective tissue pads that make the muzzle both tall and broad and that widely separate the nostrils. Thick nasal pads help seal off the nostrils when the moose submerges to feed on underwater plants.





MICHAEL AND PATRICIA FOGDEN

The snout of the male gharial is graced by a stiff, hollow knob, used in both visual and acoustic displays. Building a nose ornament entailed the evolution of a new means of closing the nostrils: penislike erectile tissue that, when engorged, seals off the airway.



FRANS LANTING; MINDEN PICTURES

Once thought to be airspeed indicators, the nostrils of the southern giant petrel and some other ocean birds help in the excretion of excess salt. Salty fluids drain from glands above the eye, pass through the nostril (a long, horny tube perched atop the beak), and ultimately drip off the bill tip.

FRANS LANTING; MINDEN PICTURES







Formed by thousands upon thousands of small muscles that confer both strength and versatility, the elephant's trunk is perhaps the most remarkable multipurpose vertebrate appendage. It is used for breathing, sniffing, manipulating objects as small as a single blade of grass, lifting an entire tree trunk, taking a shower or dust bath, and snorkeling across a river. In short, it does everything one could ask of a nose—and then some.



## IN THE FIELD



Tundra fire in northwestern Alaska

DANIEL GULLICKSON; BUREAU OF LAND MANAGEMENT

# Arctic Fires

Tundra flowers wait for lightning to strike.

By Peter J. Marchand

For eons, wildfire has played a fundamental role in the evolution of plant traits. Around the world—in grasslands, coniferous forests, chaparral—fire influences the makeup of plant communities, shapes the way many plants grow and reproduce, and regulates the course of natural succession (the orderly replacement of one group of species by another over time). Fire may even play a major role in determining what types of flowers bloom in the cold arctic tundra.

My first experience with tundra fire came in Alaska's Noatak River watershed, where sparse stands of white spruce give way to a seemingly endless expanse of dwarf shrubs and tussock-forming sedges that stretch northward to the farthest horizon. Although the soils in this region are usually cold and wet (permanently frozen ground known as permafrost lies only inches below the surface), plant matter that has accumulated over many seasons often dries out in early summer and becomes

vulnerable, for a brief period, to wildfires triggered by lightning strikes. And the impact of fire here can be far longer lasting than might be expected for a landscape dominated by low-growing shrubs and herbs.

Flying over the confluence of the Noatak and Kuguruk Rivers, where ecologist Charles Racine has established a research site for the study of tundra fires, I could see the extent of a 30,000-acre burn that had once charred the area. Its boundaries were clearly delineated by the fresh green of vigorous new growth, standing out sharply against a charcoal gray backdrop. At the site, Racine and his assistant Kathy Hutchins were monitoring changes in the plant community, quantifying what I had already suspected: the fire had set into motion a complex cycle of events, giving some species all the advantage they needed to gain a foothold. Already, several new colonists—among them bluejoint grass, fireweed, fourpart dwarf gentian,

horsetail, and arctic saxifrage—had invaded the site. It would be many years before the tundra could return to its pre-burn state.

In a place where permafrost is a feature of the environment, the makeup of the plant community depends largely on the depth to which the soil thaws each summer. The depth of the thaw is determined by a tenuous balance between heat lost during the dark winter

months and heat gained during the perpetual days of summer. This balance, in turn, depends greatly on the nature of the ground cover, since this is what absorbs or reflects sunlight and insulates the soil. Adding wildfire to the equation may shift the balance dramatically. A newly fire-blackened surface substantially increases absorption of sunlight, and in the absence of insulating plant cover, the absorbed heat is efficiently conducted downward. By season's end, the depth of thaw in burned areas may be 50 percent greater than in unburned tundra. And it doesn't take plants long to respond. Sedges sprout vigorously, spurred by a flush of nutrients resulting from the accelerated activity of soil microorganisms as well as from the burning of dead plant matter. Other species, unnoticed at the site at the time of the burn, suddenly show up; some are carried in from afar, and some germinate from seed lying dormant in the soil. Even cotton grass, a widespread and dominant tundra species that reproduces year after year by cloning, profits from fire. Its dense, knee-high tussocks are built up through the prolific sprouting of belowground stems, but fire stimulates flowering and seed production, introducing fresh genetic material into a stand and keeping the species adaptable.



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The plants that benefit most from wildfire, though, may well be arctic annuals. Annual species (those that must germinate from seed, grow to maturity, and produce a new crop of seed within a single season) are something of a novelty in this environment, where the likelihood of their success is greatly diminished by compression of the growing season into forty or fifty days. Removal of the established plant cover and exposure of the soil, however, bring out the best in these fast-growing opportunists. Kneeling among a blue profusion of gentian blossoms, Hutchins measured the response of this annual to the fire, counting more than 200 plants and 1,200 flowers per square yard where the soil was blackened and competing vegetation had been destroyed. While she found that the total number of plants at the study site differed little from the number at unburned sites, one flower characteristic stood out: seed maturation at the study site occurred fully one week earlier than it did in plants from unburned areas. For arctic annuals racing to finish seed

production before the first killing frost, this was as good as a three-length lead in the homestretch of the Preakness. Testing the seeds back in the lab, Hutchins found them to be 87 percent viable—as good as any commercial seed grower could hope for. The annuals in burned areas were clearly getting a jump start.

When tundra vegetation remains free from fires and other disruptions, the long-lived sedges form large clumps that are often drier than the surrounding soil and eventually become colonized by lichens, mosses, willow shrubs, and alders. These invaders slowly crowd out their predecessors, changing the environment by shading the ground, which reduces soil temperature and makes it even more difficult for annuals to become established. But fire, if only occasional (it occurs once every three to four years somewhere

in the Noatak River valley), reverses this course of succession just often enough to keep annuals in the game. Like fire-adapted pines, which bank seed for future generations in tightly closed and long-lasting cones, the resourceful arctic annuals invest their future in an underground seed bank. We now know that every square yard of tundra soil holds many hundreds of seeds of diverse species. Biding their time in this cold-storage depository, the seeds await some change to tip the balance of competition in their favor. And in this frozen Eden, there's nothing like a good fire to rouse them from dormancy and spark a summer bloom.

*Peter J. Marchand is currently a visiting scientist at the Carnegie Museum of Natural History's Powdermill Biological Station in the Allegheny Mountains of western Pennsylvania.*



Saxifrage in the Alaskan interior

CHARLIE OTT; NATIONAL AUDUBON SOCIETY/PHOTO RESEARCHERS, INC.

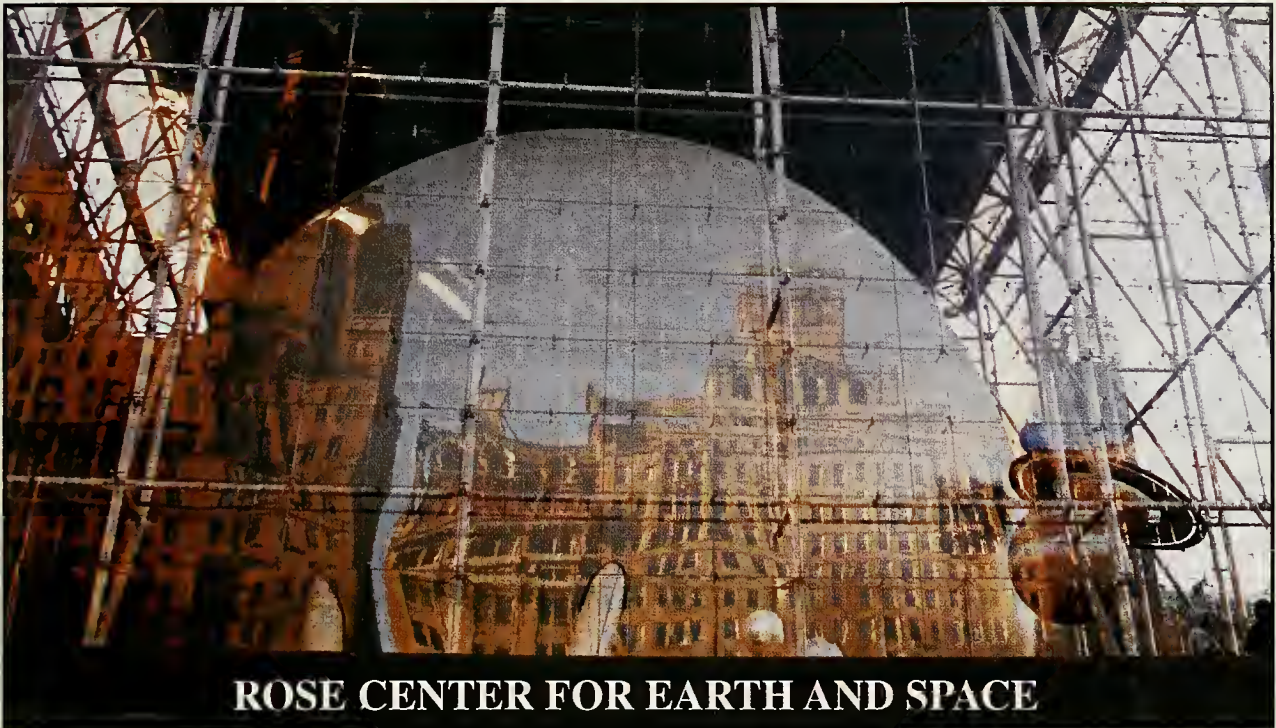


Charred spruce forest near the Koyukuk River

FRED HIRSCHMANN



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Award-winning Jungle Lodge in the Tamshiyacu-Tahuayo Reserve, shown to have the greatest diversity of mammals in all of the Amazon. 1-800-262-9669. [www.perujungle.com](http://www.perujungle.com).

## 5. Amelia Tours

Amelia Tours offers a unique collection of escorted, independent and culinary holidays to Sicily, Malta, Tunisia, Sardinia and Cyprus. 1-800-742-4591.

## 6. American Cruise Lines

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## 7. Belize Tourist Board

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## 8. British Tourist Authority

Wales: Legendary Wales has tempestuous history, timeless beauty and unexpected pleasure - just two hours from London. Visit [www.travelbritain.org/2001](http://www.travelbritain.org/2001) for more information.

## 9. Canadian Tourism Commission

Free copy of Great Canadian Adventures Travel Guide. Hiking, canoeing, cycling, wildlife viewing and more. Detailed tour operator/outfitter directory. Call 1-877-8-CANADA, Op. 407 or visit [www.travelcanada.ca/history](http://www.travelcanada.ca/history).

## 10. Charles County, MD

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## 17. Galapagos Network

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## 19. Irish Tourist Board

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**36. Tourism Authority of Thailand**

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## REVIEW

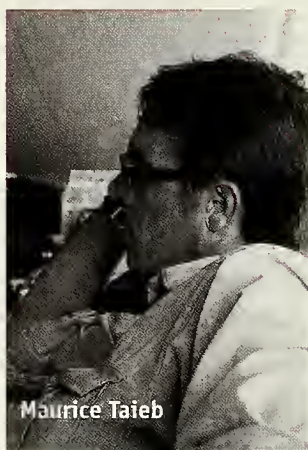
History, they say, is written by the winners. If so, we may have been missing some good books. Jon Kalb's engrossing account of discovery and disappointment in the Afar region of Ethiopia may be one of the best first-person accounts of finding human fossils ever written. In 1971, as a restless and somewhat overmature graduate student in geology, Kalb—galvanized by the fossil-finding successes of well-financed French, American, and Kenyan groups working near the northern tip of Lake Turkana—decided to move his family to Ethiopia and put together his own expedition into the last unexplored segment of the East African Rift System. Alas, once the hominid remains began to turn up, Kalb was the first (but not the only) loser in the appalling academic brawl that ensued, even as Ethiopia was exploding in waves of murderous revolution. Threaded through this vivid story of fieldwork, paleoanthropological politics, and on-the-spot war reportage is Kalb's nervy struggle simply to stay in the game.

In these pages we are backstage for some of the great scenes in human paleontology, a long-running saga of triumphs and jealousies that might have been written by Giuseppe Verdi. Nearly fifty years had passed since Raymond Dart astounded the world with the discovery of the Taung skull in southern Africa, giving anthropologists a new human ancestor to fight over. In the 1970s in South African caves and East African rift valleys, especially Kenya's Turkana basin, discoveries and hard feelings were reaching a crescendo. At first, Kalb and his partner, French geologist Maurice Taieb, had the infernal landscape of Ethiopia's Afar Depression to themselves. It was a jagged wasteland of ovenlike heat, frantic mosquitoes, and unfordable, unsanitary rivers, with a local population that had a history of wiping out exploration parties. Describ-

# Hardball Among the Hominids

Fossil riches in the Horn of Africa have sparked decades of cutthroat competition.

By John Van Couvering

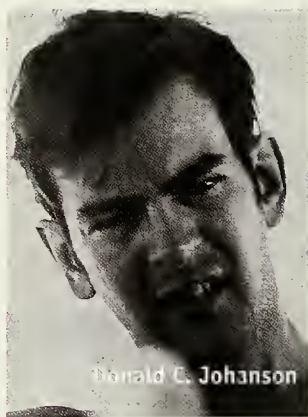


Maurice Taieb



Yves Coppens

PHOTOGRAPHS BY DENNIS PEAK



Donald C. Johanson



Jon Kalb

The prevailing mood at the Afar field camp in 1973

ing how he and Taieb learned to get around in this terrible place (and how they began to find fossil beds wherever they looked), Kalb uses such vivid and compelling imagery that one wishes—almost—to have joined them there.

Some needed no urging. Not long after news that the Afar had fossil beds dating back millions of years reached the who's who of hominid paleontology that was entrenched around Lake Turkana,

Kalb and Taieb were sought out by a young professor, Yves Coppens, representing the French presence in the area, as well as by an ambitious graduate student, Donald C. Johanson from the U.S. team. Louis Leakey, who had been sidelined by illness and politics after organizing the Kenyan contingent, was also eager to know more about the Afar fossils.

When Leakey and his wife, Mary, met Kalb at a congress of prehistorians at Addis Ababa in December 1971, Mary Leakey stressed the importance of Kalb's not trusting

anybody when it came to hominid fossils. As it turned out, she couldn't have been more prescient. From that time onward, the action in Kalb's story quickens inexorably as it dawns on everyone that the Afar is the biggest, the most fossiliferous, and (surely) the most newsworthy of all the fabled locales in human evolution. The Afar Depression, however inaccessible a hellhole, appeared to be the one re-



maining place on earth that had geological potential for a paleoanthropological bonanza. (As Kalb notes, however, there's always the Sudan.)

Various accounts exist of what happened, but the central facts are the same in all of them: In late 1972 Kalb and Taieb signed an agreement to cooperate with their French and American partners and were jointly awarded a permit by the Ethiopian government's Antiquities Administration to study the geology and paleontology of nearly 13,000 square miles of the Afar's Awash River valley. In October 1973 Johanson came across hominid remains—australopithecine leg bones—as the team was exploring at Hadar (in the northern part of the concession). Just eleven months later, Kalb was forced out. Kalb claims that the reason given by the director of the Antiquities Administration was that a rumor of his connection with the CIA had been brought to their attention by Johanson. Within months,

his former partners Taieb and Johanson announced the discoveries of “Lucy” and of the 2.3-million-year-old “First Family,” a cache of 214 fossil bones and teeth of early hominids of both sexes and different ages found in a single locality.

Kalb, meanwhile, managed to retain a corner of the original concession—in the Middle Awash valley, where nobody had yet explored. He assembled another team, and sure enough, in May 1975, they found a vast trove of Acheulean hand axes and cleavers, “the artifacts so dense in places that they [could] be seen from an airplane at 2,000 feet.” In October 1976, in the same place, they found the 600,000-year-old Bodo cranium.

Three proposals for work in the Middle Awash went to the National Science Foundation (NSF) in 1977 from Kalb's eminently qualified associates at Southern Methodist University, New York University, and Harvard—and all

three were rejected. The following year, Kalb was expelled from Ethiopia on six days' notice. The bitterest part of the book is Kalb's dry description of how the area of the Middle Awash was then taken over by J. Desmond Clark and his associates from the University of California, Berkeley, who had previously obtained an overlapping permit.

Denied funds to investigate the Bodo site further, Kalb went to the NSF to see if his supposed CIA involvement was the problem. He was blandly informed that the rejections were strictly on merit—that no such rumor had ever reached their ears. In 1986 Kalb filed a lawsuit against the NSF under the Freedom of Information Act and won a court-stipulated settlement as well as a public apology from the NSF. Their records showed that the CIA rumor was indeed a factor. The information released to Kalb showed that Clark, who moved into the Middle Awash only weeks after Kalb

was out of the way, was one of the referees who had argued privately to reject the Harvard application due to the CIA rumor (the other two applications had vanished from the files).

Now a research fellow at the University of Texas at Austin, Kalb paints an engaging and sympathetic picture of himself as a good guy who got steam-rollered. Skull digging without skull-duggery is hard to imagine, however, and Kalb may just not have been as proficient a hardball player as his competitors. I recall the wry diagnosis of “the hominid game” offered by a paleontologist who was in Kenya studying fossil fish at the time. “It's always a bad combination,” she concluded, “when you get hominid fever on top of testosterone poisoning.”

*John Van Couvering is a geologist and the editor and publisher of the Museum's Micropaleontology Press.*

**nature.net**

# Biology's Giant Leap

*By Robert Anderson*

The preliminary map of the human genome is complete, but as the scientists who worked on the project readily admit, the hard work is just beginning. Researchers will be trying to figure out the role that each gene plays in our complex biochemistry. Even with only 30,000 genes—far fewer than the original estimates—this task could take decades.

For wide-ranging information about the Human Genome Project, try the site at [www.ornl.gov/hgmis/](http://www.ornl.gov/hgmis/). Also, the “Human Genome Special” of the British weekly popular-science magazine *New Scientist* has nice online summaries of what biology's big moment means for all of us ([www.newscientist.com/news/genome.jsp](http://www.newscientist.com/news/genome.jsp)).

For budding student geneticists who want to get in on the action, I recommend starting with the University of Utah's Genetic Science Learning Center site ([gslc.genetics.utah.edu](http://gslc.genetics.utah.edu)). Its “Basic Genetics” section has a primer on how our cells translate DNA's genetic information into the multitude of proteins our bodies need. Science becomes personal in the “Genetic Disorders” section, in which young people suffering from a rare genetic disease, neurofibromatosis, share their experiences. For news about how genetic research is changing the world, click on “Genetics in Society.” Or try the “Students” section, with demonstrations such as “How to extract DNA from anything living”: Place split peas (or onions or chicken liver) in a blender; add water and salt; blend; then add a little detergent, meat tenderizer, and alcohol, in sequence. *Et voilà*, you get strands of sticky DNA, the code of life. Seeing is believing.

*Robert Anderson is a freelance science writer living in Los Angeles.*



## PHOTOGRAPHY



**Primal Forces**, photographs by David Muench and Marc Muench; text by Michelle A. Gilders (Graphic Arts Center Publishing, 2001; \$50)

DAVID MUENCH

## BOOKSHELF

**Brave New Brain: Conquering Mental Illness in the Era of the Genome**, by Nancy C. Andreasen (Oxford University Press, 2001; \$29.95)

Neuroimaging of the thalamus reveals that it is smaller in schizophrenics. According to Andreasen, a neuroscientist, future mapping of the organ holds the promise of finding in "this small haystack . . . the quixotic needle that can be used to slay one of the biggest giants of mental illness."

**The Misunderstood Gene**, by Michel Morange (Harvard University Press, 2001; \$24.95)

"Organisms are algorithms that are incarnated in DNA molecules and in proteins." So writes this French biologist, intent on looking at genes as synthesizers of proteins and on offering precise accounts of how they operate in such fundamental life processes as development, aging, learning, and behavior.

**The Impact of the Gene: From Mendel's Peas to Designer Babies**, by Colin Tudge (Hill and Wang/Farrar, Straus and Giroux, 2001; \$27)

For Tudge, a scientist turned writer, the immense possibilities of biotechnology raise the disturbing question, Is our basic humanity at risk?

**Transducing the Genome: Information, Anarchy, and Revolution in the Biomedical Sciences**, by Gary Zweiger (McGraw Hill, 2001; \$24.95)

Storing and analyzing the genomic data of our species, writes Zweiger, promises to create "a dramatically new understanding of life" but also to impose on us the enormous responsibility of becoming "stewards of our own genome."

**Cracking the Genome: Inside the Race to Unlock Human DNA**, by Kevin Davies (The Free Press, 2001; \$25)

Davies, the founding editor of *Nature*

*Genetics*, gives a lively account of the costly and intensely competitive effort to decipher the full genetic composition of human beings. The Human Genome Project, begun in 1990 and completed this year, "represents an extraordinary technological achievement, and is at best perhaps the defining moment in the evolution of mankind."

**Abraham Lincoln's DNA and Other Adventures in Genetics**, by Philip R. Reilly (Cold Spring Harbor Laboratory Press, 2000; \$25)

Doctor, geneticist, and lawyer Reilly examines the diverse uses of genetic technologies, from the proposal to diagnose a disorder in Lincoln's DNA known as Marfan syndrome to using animal organs in humans.

**Perspectives on Genetics: Anecdotal, Historical, and Critical Commentaries, 1987–1998**, edited by James



*E. Crow and William F. Dove (University of Wisconsin Press, 2000; \$19.95)*

This collection of essays originally appeared in *Genetics*. Written by such contributors to the field as Joshua Lederberg, Richard C. Lewontin, and John Tyler Bonner, they richly document the history of modern genetics research and its continuing evolution.

**Decoding Darkness: The Search for the Genetic Causes of Alzheimer's Disease**, by Rudolph E. Tanzi and Ann B. Parson (Perseus Publishing, 2000; \$26)

Isolating the genes and proteins responsible for this neuron-wasting disorder (now affecting 20 percent of people age seventy-five to eighty-four and 40 percent of those eighty-five and over) has been Tanzi's quest since the early 1980s.

**The Seven Daughters of Eve: The Science That Reveals Our Genetic Ancestry**, by Bryan Sykes (W. W. Norton, 2001; \$25.95)

Modern genetics permits us to journey into the deep past of our species, "way beyond the reach of written records or stone inscriptions," writes geneticist Sykes. "These genes tell a story which begins over a hundred thousand years ago and whose latest chapters are hidden within the cells of every one of us."

**The Way of the Cell: Molecules, Organisms, and the Order of Life**, by Franklin M. Harold (Oxford University Press, 2001; \$27.50)

In 1944 physicist Erwin Schrödinger published his book *What Is Life?* This ageless question is at the heart of Harold's investigation of the ubiquitous "process of living" and the unique capacity of organisms "to reproduce themselves indefinitely, and arise on a millennial time-scale by the interplay of variation and selection that underlies biological evolution."

The books mentioned are usually available in the Museum Shop or via the Museum's Web site, [www.amnh.org](http://www.amnh.org).

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## MUSEUM EVENTS

### JUNE 4

Lecture: "What's the Matter in the Universe?" (Frontiers in Astrophysics series). Astronomer Vera Rubin. 7:30 P.M., Space Theater, Hayden Planetarium.

Lecture: "Dragon Hunter: The Life of Explorer Roy Chapman Andrews." Writer and archaeologist Charles Gallenkamp. 7:00 P.M., Kaufmann Theater. For more information, call (212) 313-7607.

### JUNE 5, 12, AND 19

Three lectures: "Evolution and Genomics," Rob DeSalle, curator of the exhibition "The Genomic Revolution," June 5; "Natural History of the Genome: The Role of Genes in Nature, Extinction, Mutations, and Status," Niles Eldredge, curator, Division of Paleontology, June 12; and "Genetic Diversity and Native American/First Nations Cultural Issues," Linda Burhansstipanov, executive director, Native American Cancer Research Corporation, June 19 (Science of the Genome series). 7:00 P.M., Kaufmann Theater.

### JUNE 6, 9, 13, 20, AND 27

Films and discussions in connection with "The Genomic Revolution": *The Lost Tribes of Israel* (DNA research aids a quest for identity), June 6, 6:30 P.M.; *Gene Hunters* (genetic research and indigenous peoples), June 9, 2:00 P.M.; panel discussion on art and biotechnology, June 13, 7:00 P.M.; *Amrit Beeja* (traditional agriculture and agribusiness in India), June 20, 6:30 P.M.; *After Darwin* (possibilities and ethics of genetic technologies), June 27, 6:30 P.M. Kaufmann Theater (except June 13, Linder

Theater). For additional screenings on May 26 and 30, call (212) 769-5200.

### JUNE 7

Lecture: "Transition From Sail to Steam: Archaeology and the Social History of Ships" (Earthwatch at the Museum series). Anthropologist Richard Gould. 7:00 P.M., Kaufmann Theater.

### DURING JUNE

A fossil of a subadult theropod (125–145 million years old) discovered in northeastern China is on display in the Astor Turret. This dinosaur, covered with well-preserved featherlike structures, probably looked much like a bird.

For information on field trips and workshops for adults and children, inside and outside the Museum, call (212) 769-5315.

Seminars on Science: Six-week online courses for K–12 teachers on sub-

jects including genetics, spiders, fishes, and life in the universe. Continuing education and graduate credits available. For more information, visit [www.amnh.org/learn/pd/sos/](http://www.amnh.org/learn/pd/sos/).

Films at the IMAX Theater: *Lost Worlds: Life in the Balance* (biodiversity and the need for conservation); *Shackleton's Antarctic Adventure* (the dramatic story of the 1914–17 British Imperial Trans-Antarctic Expedition); and *Ocean Oasis* (the biodiversity of the Baja California peninsula).

The American Museum of Natural History is located at Central Park West and 79th Street in New York City. For listings of events, exhibitions, and hours, call (212) 769-5100 or visit the Museum's Web site at [www.amnh.org](http://www.amnh.org). Space Show tickets, retail products, and Museum memberships are also available online.



**June 9: Opening of the Discovery Room, an interactive space for families with children ages five and up. Activities are available in all the Museum's major fields of science and research, from anthropology to astrophysics. Tuesday–Sunday, 10:00 A.M.–5:00 P.M.; Friday 10:00 A.M.–8:45 P.M.**



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THE NATURAL MOMENT



# Woodworks

Photographs by Jim Brandenburg

Minden Pictures





Charles Darwin admired the special adaptations of woodpeckers, citing their stiff tail spines, hard-pointed bills, and shock-absorbent necks. While most of these birds are small and forage by drilling holes in tree bark to extract one grub at a time, the crow-sized pileated woodpecker (*Dryocopus pileatus*) hacks away at wood on a somewhat larger scale—both to excavate nests for its young and to expose whole colonies of ants. During February and March, a male and female may pound on a dead branch or old tree trunk for as long as a month to excavate a nesting cavity in which to raise three or four chicks. Both parents take turns incubating the eggs and bringing food to the chicks. Males sit on the eggs mainly at night. The parent feeding a chick in the photograph (inset) was spotted in a mixed conifer-hardwood forest in northern Michigan, near the Canadian border.

Although their diet includes some acorns and beechnuts in the fall,

pileated woodpeckers eat mostly ants, flying insects, grubs, and some seeds and fruits. Carpenter ants are a special favorite. When the forest floor is blanketed in snow, the birds use their powerful bills to dig out ant nests from tree trunks and tree bases. Jabbing at the wood, they remove chips three to six inches long. According to some observers, these woodpeckers feed on the sap that runs from the trees' wounds and also eat the insects that are lured by the flow of sugary liquid.

Woodpeckers of this species have been known to rescue endangered eggs. In one documented instance, when a dead tree containing a nesting cavity collapsed, the female retrieved each of her three eggs with her bill and flew them to a hollow in another tree. On his return to the original nesting site two hours later, the male began a frantic search of the area and finally managed to locate his family. The pair soon settled in their new home and resumed their breeding efforts.—*Richard Milner*



## ENDPAPER

# Would Darwin Get a Grant Today?

By T. V. Rajan

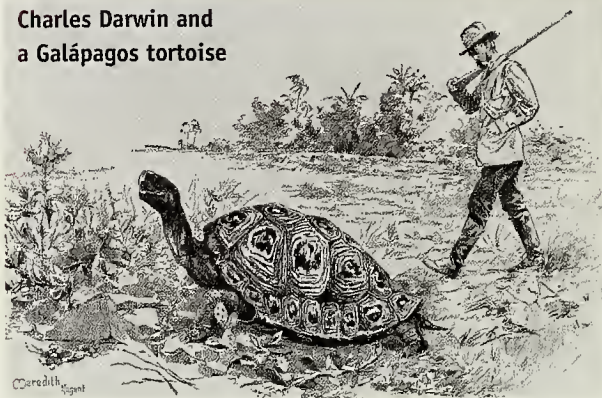
For the first half of the twentieth century, biological research was dominated by scientists who studied whole organisms in their natural settings. In recent decades, this approach to the study of life has increasingly been perceived as a poor cousin to molecular biology and has even, in some scientific circles, been contemptuously dismissed as stamp collecting. The change began as several forces conspired to bring about a reductionist approach. One powerful influence was the 1944 publication of a book entitled *What Is Life?* by Austrian physicist Erwin Schrödinger, whose work was vital to the development of quantum mechanics. In this book, Schrödinger maintained that physics could help explain why genetic traits are so stable—why, for example, a particular malformation of the lips (known as the Hapsburg lip) turned up over several centuries in members of the German royal family.

Physicists who felt that quantum mechanics had exhausted the possibilities offered by inanimate objects were inspired by Schrödinger's words to turn their attention to biology. These scientists brought with them the bias that studying individual molecules was the only plausible approach to understanding systems as complex as living organisms. This approach produced a massive explosion in our understanding of the molecular basis of life, teaching us that organisms derive their enormous complexity from a vast number of relatively simple interactive systems. Unfortunately, I feel, the molecular biology revolution has put the baby at risk of being thrown out with the bathwater.

I am often involved in reviewing grant proposals in my own field—biomedical research—and over the years have witnessed a disturbing trend. Nearly all the worthy molecular studies receive the funding they need, while many promising clinical projects—following the progression of a disease in real human patients, for instance—are turned down. This is regrettable, because both approaches are critical to understanding the many complex factors involved in any disease: the genes that may predispose someone to a particular illness, the environmental or other triggers that may bring on the illness, and the numerous factors (some, but not all of them, genetic) that determine how the illness progresses.

Some scientists believe that genetic information will

Charles Darwin and  
a Galápagos tortoise



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soon render clinical or descriptive research moot. I am skeptical. The precise amino-acid substitution in the hemoglobin of patients with sickle-cell anemia has been known for approximately forty years; so far, however, this knowledge has done little to help manage the disease. The inevitable time lag between a genetic discovery and any alleviation of human suffering that might result from the discovery should be grounds enough to dictate steady financial support of clinical as well as molecular studies.

Furthermore, nature is notoriously frugal with her resources. Most genes play multiple, often little-understood roles in an organism. Altering a gene in order to treat one condition may leave the patient vulnerable to other problems. For example, the early onset of puberty in girls is associated with health risks, including an increase in the chance of developing breast cancer, so why not try to inhibit premature menarche? The enzyme responsible for breaking down the male hormone testosterone (which females also have, though in lesser amounts) may cause premature menarche. Wouldn't it make sense to tinker with the enzyme in order to increase a girl's testosterone level? No, because tinkering with it would adversely affect other areas of her physiology, such as her susceptibility to cardiovascular disease.

My concerns are not limited to medicine. I fear we may be discouraging many young people—the possible intellectual descendants of Darwin—who might otherwise choose to study organisms in the wild. In my view, this is tragic. As much as molecular biology and genomics can teach us, to deeply understand living organisms requires that we also study how their physiology is structured, how they evolved, and how they function in their current ecological settings.

Fortunately, as readers of *Natural History* know, field biology is far from dead, and many field biologists are now incorporating molecular biology into their research programs. As we begin to appreciate the limits of reductionism, I am optimistic that descriptive biology will come back into fashion and once again be recognized as an essential part of our efforts to understand life.

*T. V. Rajan is chairman of the pathology department at the University of Connecticut Health Center in Farmington.*



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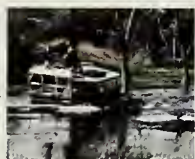
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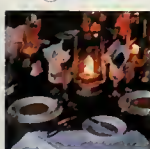
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