SCIENCE NEWS

THE WEEKLY NEWSMAGAZINE OF SCIENCE

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iq gene id'd?

detecting fetal flaws

cosmic rays from galactic cores

yellowstone on the rise

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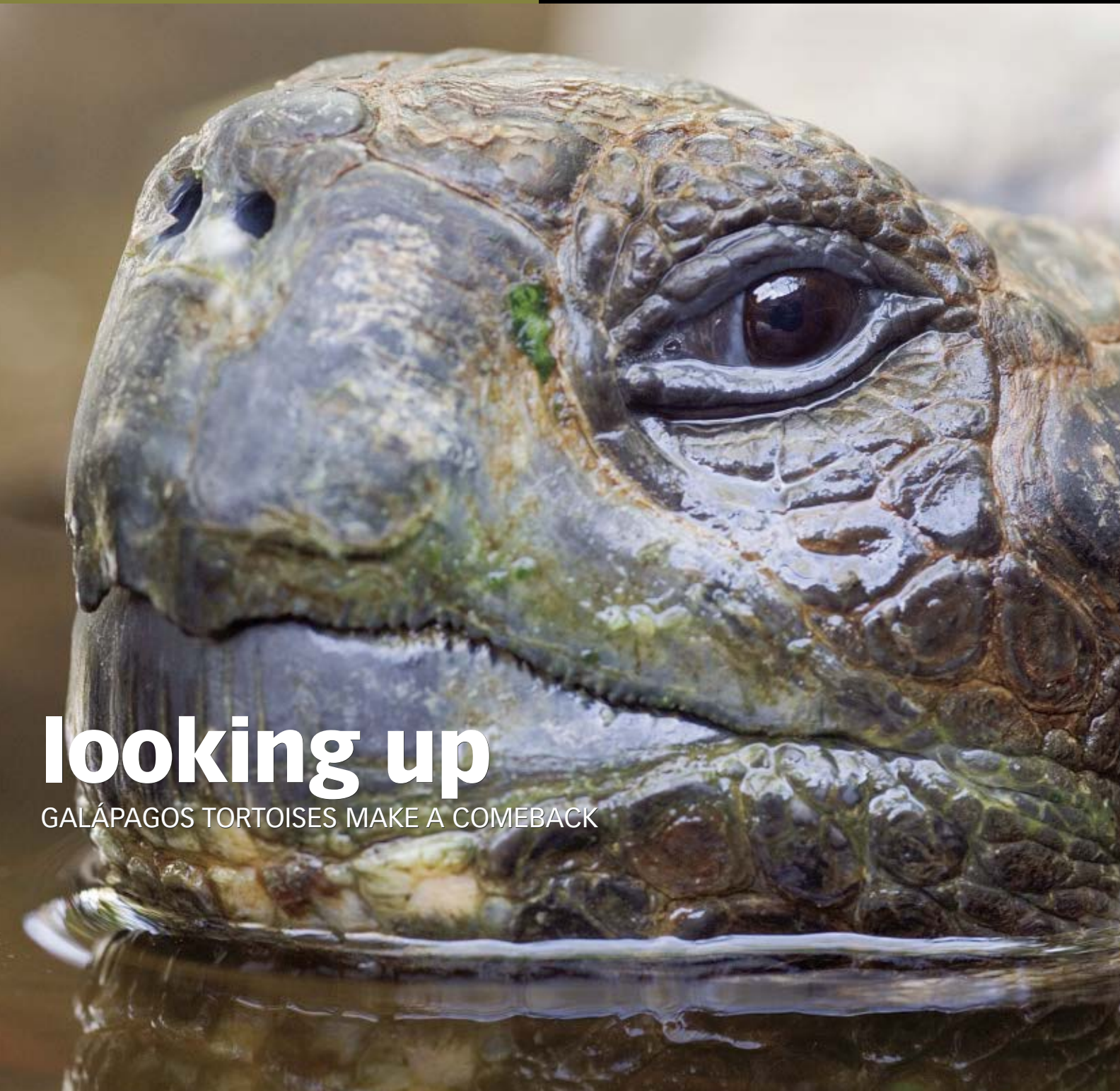

looking up

GALÁPAGOS TORTOISES MAKE A COMEBACK

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Cover This giant tortoise surveys the scene at the Charles Darwin Research Station on the Galápagos Islands. Geneticists and conservation biologists are unraveling the complex history of the numerous species of Galápagos tortoises in order to safeguard their future. (© Paul Souders/Corbis) Page 298

THIS WEEK ONLINE
http://blog.sciencenews.org/

MathTrek Young children who show sophisticated storytelling skills go on to demonstrate greater mathematical ability.

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Smarty Gene

Breast-fed kids show DNA-aided IQ boost

Scientists have achieved a breakthrough in deciphering the genetics of intelligence. Ironically, they did it by accounting for a key environmental factor.

Breast-feeding boosts children's IQs by 6 to 7 points over the IQs of kids who weren't breast-fed, but only if the breast-fed youngsters have inherited a gene variant associated with enhanced chemical processing of mothers' milk, reports a team led by psychologist Avshalom Caspi of King's College London.

QUOTE

"Genes work via specific environmental experiences to shape intellectual development."
AVSHALOM CASPI, King's College London

The new finding supports the controversial hypothesis that fatty acids in breast milk enhance newborn babies' brain development. Moreover, the results demonstrate that intelligence researchers must examine how children's genetic natures interact with the ways in which they're nurtured.

"Genes work via specific environmental experiences to shape intellectual development," Caspi says.

He and his colleagues present their data in an upcoming *Proceedings of the National Academy of Sciences*.

Two groups of children participated in the study: 1,037 boys and girls born 34 to 35 years ago in New Zealand, who are still living there; and 2,232 boys and girls born 12 to 13 years ago who are growing up in England.

In DNA isolated from blood samples, the researchers probed the gene *fatty acid desaturase 2*, or *FADS2*. This gene assists in breaking down fatty acids present in human milk. *FADS2* comes in two forms, one of which enables the body to process fatty acids more efficiently than the other does.

Only breast-fed children who carried one or two copies of the more efficient gene displayed an IQ advantage.

In the two groups of children, 90 percent of youngsters possessed the critical *FADS2* gene variant. Roughly half of all participants were breast-fed regularly during infancy, according to reports collected from the mothers when their children were 1 to 3 years old. The formula-fed infants typically received no fatty acids in their diets.

The New Zealand children completed standard IQ tests at ages 7, 9, 11, and 13. The British children took an IQ test at age 5.

The scientists ruled out several alternative explanations of the findings. For instance, normal- and low-birth-weight babies carrying the critical *FADS2* gene displayed equal IQ hikes when breast-fed. The same held for children from wealthy and poor families, and for kids with high-IQ and low-IQ mothers.

Also, no evidence indicated that mothers carrying the more efficient *FADS2* gene produced better-quality milk or breast-fed more often than mothers carrying the other gene variant did.

Until now, researchers have largely failed in attempts to find genes that affect intelligence independently of environmental factors, Caspi says. However, a new genomewide analysis of more than 10,000 7-year-olds tagged six regions as weakly but significantly associated with IQ, including one on *FADS3*, another fatty acid gene. That study, directed by King's College psychologist Lee M. Butcher, appears online Nov. 2 in *Genes, Brain and Behavior*.

"Both of these new findings suggest an important role for the regulatory mechanism of dietary fatty acids and its possible interaction with environmental factors in intelligence," remarks biological psychologist Danielle Posthuma of the Free University of Amsterdam.

Adds psychologist Jeremy R. Gray of Yale University, "An IQ advantage of 6 to 7 points is unquestionably large enough to have a real-world impact on individuals." —B. BOWER

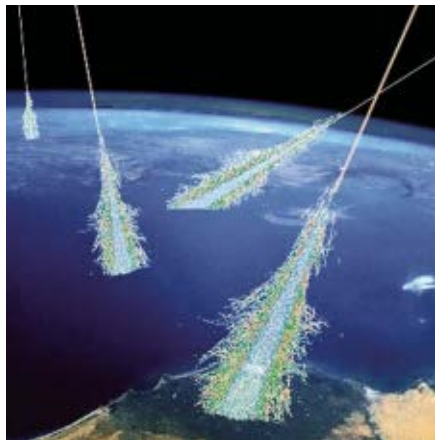
Ray Tracing

Energetic cosmic rays linked to giant black holes

Imagine a single proton smashing into Earth's atmosphere with as much punch as a fast-pitch baseball. For decades, scientists have suspected that protons and other particles with such huge energies, known as ultra-high-energy cosmic rays, arise in the tumultuous surroundings of giant black holes at the centers of galaxies.

Now, the sprawling Pierre Auger Observatory in Argentina has found the first solid evidence linking these exceedingly rare cosmic rays—the most energetic particles in the universe—to supermassive black holes in nearby galaxies.

"This is a landmark finding for both cosmic-ray physics and astrophysics," says Auger researcher Paul Mantsch of the Fermi National Accelerator Laboratory in Batavia, Ill. Mantsch and a vast team of researchers from 17 countries describe the findings in the Nov. 9 *Science*.



CATCHING SOME RAYS Illustration shows air showers generated by the highest-energy cosmic rays, the most energetic particles known in the universe.

S. SWORDY/UNIV. CHICAGO, NASA

In the study, the observatory recorded 27 cosmic rays with energies greater than 57 billion billion electron volts (eV). The team found that 20 of these 27 ultra-energetic rays came from points in the sky coinciding with the known locations of active galactic nuclei (AGN). Mantsch and his colleagues say there is less than a 1 percent chance that the alignment between the cosmic rays and the AGN is merely random. The AGN all lie relatively close to the Milky Way, no more than 326 million light-years away.

In AGN, black holes feed on swirling disks of gas and dust. As this material spirals into the abyss, it heats up and emits energetic radiation. In addition, AGN typically shoot out enormous jets of high-speed gas. The jets might blast some cosmic rays to energies millions of times as great as those created in the most powerful accelerators on Earth. But "we don't really know" the acceleration mechanism, cautions Mantsch.

The Auger facility, which opened in 2004, detects the cascade of secondary particles generated when cosmic rays smack into Earth's upper atmosphere. This cascade, known as an air shower, can spread over an area as large as 40 square kilometers by the time it reaches Earth's surface. The observatory includes some 1,600 water tanks—particle detectors that scintillate when particles from the shower pass

through—spread over 3,000 km², along with 24 ultraviolet telescopes that record the faint light generated by the shower as it speeds toward Earth's surface.

Lower-energy cosmic rays may arise from supernova remnants within the Milky Way, but such particles are so easily deflected by magnetic fields that their direction of arrival provides almost no information on where they originate. In contrast, the ultra-high-energy cosmic rays travel nearly in straight lines from their source. The most energetic cosmic rays are also the rarest. They strike a square kilometer of Earth about once per century and require an observatory as vast as Auger to find them.

The close coincidence between the cosmic-ray directions and AGN locations suggests that most of the energetic cosmic rays are protons that have traveled through relatively weak intergalactic magnetic fields en route to Earth, says Mantsch.

Cosmic-ray astronomer Trevor Weekes of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., says that the study is significant but adds that he's a bit surprised that more of the energetic rays aren't associated with the most powerful nearby AGN. The new findings, he says, make a compelling case for building a second Auger observatory in the Northern Hemisphere. —R. COWEN

Silencing Pests

Altered plants make RNA that keeps insects at bay

Two teams of researchers have modified plants to produce genetic material that disables critical genes in insects that eat the plants. The technique could provide a new strategy for agricultural-pest control.

Looking for a new way to protect corn plants, James Roberts with Monsanto in Chesterfield, Mo., and his colleagues turned to a mechanism known as RNA interference, in which segments of the genetic molecule RNA block the translation of information from a target gene (*SN: 7/2/05, p. 7*). The researchers found RNA sequences that would target critical genes in the western corn rootworm and two other related pests, and then modified corn so that it would generate those sequences.

In rootworms that fed on the modified corn, RNA from the plants shut down the target gene, stunting or killing the insects' larvae, the researchers report. Modified corn plants infested with corn-rootworm eggs suffered less root damage than did normal corn.

In the other study, Xiao-Ya Chen and his colleagues at the Chinese Academy of Sciences in Shanghai used a similar trick to increase the cotton bollworm's sensitivity to gossypol, a defense chemical produced by the cotton plant.

Although large doses of gossypol stunt the growth of bollworm caterpillars, the pests can tolerate the chemical at low concentrations. Chen and his colleagues found the insect gene responsible for this tolerance, and then modified *Arabidopsis*, a widely used lab plant, to produce silencing RNA for that gene. Insects that feasted on the modified lab plants ingested the RNA, and stopped growing when fed gossypol. The researchers are trying to reproduce their results with cotton plants.

TAMING PESTS **Roots of a modified corn plant (top) resist damage by corn rootworms better than normal corn roots (below).**

Both teams report their findings online and in the November *Nature Biotechnology*.

Previous research had shown that RNA injected into insects could shut down specific genes. The critical innovation of the new work is oral delivery of the silencing RNA from plant to insect, Roberts says.

The Monsanto researchers "have produced protection of a crop plant against a real pest," says Peter Waterhouse of CSIRO Plant Industry in Canberra, Australia. Although the Shanghai-based study uses a model plant to deliver the RNA, it "has a very ingenious strategy ... to kill off the insects' counterdefense against a defense chemical," he adds.

For many years, farmers have planted crops engineered to possess a bacterial gene that produces an insect poison known as Bt. But this poison doesn't work on all insects, and scientists worry that pests could eventually evolve resistance to it (*see www.sciencenews.org/articles/20030830/food.asp*).

With the appropriate choice of target gene, "in principle, the [RNA] strategy is applicable to any herbivorous insect," Chen says. And the ability to carefully design the RNA sequence could allow researchers to evade insect-resistance strategies, Waterhouse says.

More research will be needed to show how applicable the technique is to other pests and how well it would work in the field, he adds. Government regulatory bodies would have to grant permission before such crops could be grown in open fields. But Waterhouse says the initial results suggest that RNA interference could be a powerful strategy for controlling insect pests. —S. WEBB

Not Like Clockwork

High-fat diet disrupts daily routines of mice

Most mice sleep, eat, and exercise on a predictable 24-hour cycle, thanks to their precise internal clocks. But mice fed a fatty diet have trouble sticking to their schedule, new research shows. Genetic activity, not just behavior, drives the changes.

Understanding this novel link between daily cycles, known as circadian rhythms, and the metabolic system could help reveal the mechanism behind some cases of obesity and diabetes in humans, scientists say.

Earlier research had shown a connection between circadian rhythm and eating behavior. For example, mutations in mouse genes that help maintain the internal clock's rhythm cause the animals to overeat and gain weight. Studies have also shown that people who work night shifts are at higher risk for obesity than their day-shift counterparts, and that getting too little sleep can raise a child's risk of developing diabetes later in life.

Joseph Bass of Northwestern University in Evanston, Ill., suspected that diet and sleep/wake cycles are linked in other ways. So he and his colleagues fed male mice diets high in fat and recorded their daily behavior.

"We don't normally wake up during the middle of the night hungry," says Bass. "Our clock controls that." But after only 2 weeks on the fatty diet, the usually nocturnal mice began waking up and eating during the day.

To get at the root of how diet was changing the mice's circadian rhythms, the researchers tracked hormone and gene expression in different tissues of the animals for 24 hours. A high-fat diet altered the daily activity cycle of genes involved in appetite and metabolism, the team reports in the November *Cell Metabolism*.

The diet dampened the usual cyclic variation in the activity of some genes. For other genes, the frequency or amplitude of the cycle changed. The changes varied among

MONSANTO

tissues, says Bass, because circadian rhythms play different roles in different organs. Mice that got only 16 percent of their calories from fat, instead of 45 percent, did not exhibit the changes.

Bass says that understanding the two-way link between circadian rhythms and diet in mice could help explain sleep and metabolic disorders in people. "Maybe a very common perturbation, high-fat feeding, is one of the factors that disrupts the circadian rhythm," he says. "And disrupting the circadian rhythm, in turn, affects appetite. It's a vicious cycle."

Tamas Horvath of Yale University calls the new findings "incredibly novel" and says that the next step is to probe exactly how, at a molecular level, a high-fat diet alters the expression of "clock genes." Future studies could test how different diets change circadian rhythms, Horvath says.

"Is it the combination of fat and carbohydrate that matters?" ponders Horvath. "What if you put the mice on an Atkinslike diet with more protein? Would that be different?" —S. WILLIAMS

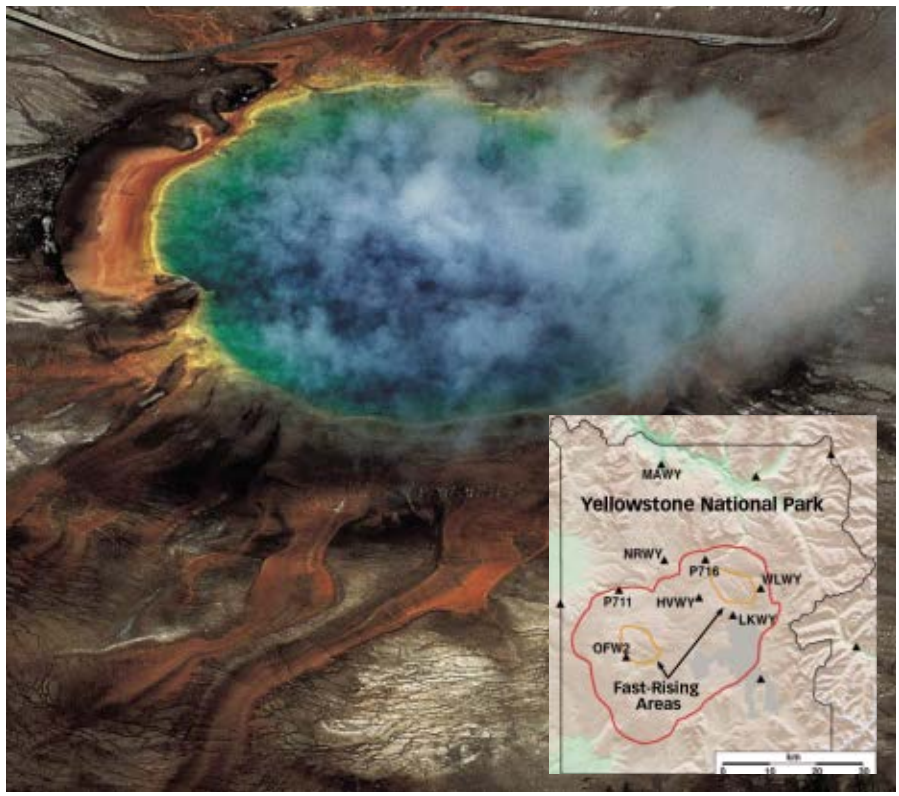
Yellowstone Rising

Magma floods into chamber beneath park

From mid-2004 through 2006, parts of the terrain in Yellowstone National Park rose as much as 7 centimeters per year, a rate about three times that previously measured. Analyses suggest that the rapid uplift results from the flow of molten rock into a Los Angeles–size zone of strata beneath the park.

Yellowstone, one of the most active hydrothermal regions on Earth, contains more than 10,000 geysers, hot springs, and steaming volcanic vents (*SN: 6/5/04, p. 357*). The heat fueling this activity comes from molten rock welling up beneath the park's central basin from deep within Earth, says Wu-Lung Chang, a geophysicist at the University of Utah in Salt Lake City. The main plume of hot, buoyant material rises to depths of about 50 kilometers, he notes, but occasionally blobs of molten rock break off the plume, rise, and replenish a broad magma chamber nearer the surface.

Detailed surveys of Yellowstone began in 1923. In recent years, scientists have supplemented those efforts with data from ground-based Global Positioning System equipment and satellite-based radar, says Chang. The rise and fall of the park's landscape reflect the immense and often complex subterranean motions of molten rock and groundwater, he notes.



ON THE RISE **Yellowstone's Grand Prismatic Spring is one of the geothermal features heated by a reservoir of molten rock beneath the park's central basin (inset map, red). Magma flow into that reservoir from mid-2004 through 2006 triggered rapid uplift in several areas, including those in yellow. Triangles denote locations of Global Positioning System equipment.**

H. HOLDSWORTH; (INSET) CHANG ET AL.

Between 1923 and 1984, some spots in the basin rose, on average, about 1.4 cm/yr. Then, from 1985 to 1995, the terrain subsided almost 1 cm/yr, says Chang. In the following 6 years, the basin floor began to rise again, and an area northwest of the basin rose even faster. From 2000 to 2003, the northwestern area rose about 3.6 cm while the basin floor sank about 2.8 cm.

The Yellowstone basin's record-setting inflation, about 7 cm/yr in some locales, began in July 2004, Chang and his colleagues report in the Nov. 9 *Science*. Around 3 months later, terrain to the northwest began to subside, a trend that the researchers attribute to a flow of groundwater from that area.

The team's analyses suggest that the magma chamber beneath Yellowstone is about 10 km below the surface and underlies an area of roughly 1,200 square kilometers. From mid-2004 through 2006, about 0.1 cubic kilometers of molten rock flowed into that reservoir, says Chang.

The Yellowstone region has seen numerous volcanic eruptions, including a massive eruption about 640,000 years ago that spewed about 1,000 times the volume of lava that Mount St. Helens ejected in 1980, says Jake Lowenstern, a geophysicist with the U.S. Geological Survey in Menlo Park, Calif. The new findings, however, don't suggest that an eruption is imminent. "The last 3 years have been seismically quiet," he notes. Moreover, there have been no significant changes in geyser activity.

The new findings are "very fascinating" and offer scientists insights into how the park's subterranean plumbing works, says Hank Heasler, a National Park Service geologist at Yellowstone's headquarters in Mammoth, Wyo.

Although recent uplift in Yellowstone has been rapid, detailed observations have been available only for a few decades, notes Heasler. "Statistically, that's not a very long time," he says. —S. PERKINS

Ladies First

Genes skew sex ratios in evolutionary struggle

Competition among genes within an indi-vidual male fruit fly can cause its sperm to produce a high proportion of female offspring. Now, scientists have identified a gene responsible for this well-known phenomenon as well as the gene that later evolved to restore gender balance.

In essence, the two fruit fly genes engage in a tug-of-war in which each succeeds evolutionarily if it can spread widely among future generations.

The imbalance favoring females happens because the sex-skewing gene, called

Distorter on the X (*Dox*), is located on the X chromosome. Females each have two X chromosomes, and pass on only Xs to their offspring. Males each have an X and a Y. They pass an X chromosome to their female offspring and a Y to their male offspring.

To ensure its evolutionary success, *Dox* somehow sabotages the maturation of sperm carrying Y chromosomes. As a result, a male fruit fly carrying *Dox* would produce a generation of offspring that is more than 90 percent *Dox*-carrying females.

This imbalance allows *Dox* to spread widely among a population in only a few generations, but the gene's success sets the stage for its own defeat.

Once the population is dominated by females, a male will have many chances to reproduce and pass on its genes. This fact changes the game, because it means that a gene can now spread rapidly if it's carried by a male.

The preponderance of females "creates a strong pressure for the evolution of a [*Dox*] suppressor gene," says Yun Tao of Emory University in Atlanta, who led the research. If a gene on one of the nonsex chromosomes could shut down *Dox*, its odds of being passed on to male offspring would increase. Since the population is mostly female, males that inherit the suppressor gene would have many opportunities to breed and pass on the gene.

Indeed, Tao's team found a gene that suppresses *Dox*. They dubbed it *Not much yang* (*Nmy*). Tao says that *Nmy* could have arisen in fruit flies only a few dozen generations after *Dox* first appeared. The researchers found that the two genes share regions of identical genetic code, which suggests that *Nmy* is simply an altered copy of *Dox*. That finding could explain how *Nmy* could have evolved so quickly, the researchers report in the November *PLoS Biology*.

The fact that the genes share genetic code could also explain how *Nmy* silences *Dox*. Molecular transcripts of *Nmy* would match up with and bind to transcripts of *Dox*, which would block the *Dox* transcripts from crippling a male's Y-bearing sperm.

"It's absolutely remarkable," comments John Jaenike of the University of Rochester in New York, who has performed related research on skewed sex ratios. "This is the first report ever that identifies the suppressor and proposes an extremely elegant model of suppression."

Fruit flies have at least two other pairs of sex-skewing and suppressing genes, but no one has managed to identify them. Tao says that it's uncertain whether people carry similar gene pairs. —P. BARRY

Mr. Not Wrong

Not my species? Not a problem

Female toads that flirt with a male of another species may have their own best interests at heart.

The plains spadefoot toad spawns offspring that grow up faster if dad is a different species called the Mexican spadefoot, says Karin Pfennig of the University of North Carolina at Chapel Hill. The toads begin life as tadpoles in pools of water that can dry up quickly, so a little hop forward in speed of maturation can mean the difference between life and death. Pfennig has now found that in tough times, females tend to prefer Mexican males to plains males.

Spadefoot toads take their name from foot flaps that help the toads dig into mud. In winter, adult toads shovel their way underground and hibernate inside a mud cocoon. But tadpoles can't survive without a pond, so each mating season becomes a race between the next drought and the growing tadpoles.

In earlier research, Marie Simovich of the University of San Diego reported that the plains spadefoot toad (*Spea bombifrons*) hybridized with the Mexican spadefoot (*Spea multiplicata*) when they happened to share shallow pools. The hybrids were more likely to turn up in the shallowest pools that would dry out earliest.

Evidence suggested that the mixed offspring don't reproduce as readily as the purebred young of each species. Purebred Mexican tadpoles mature fast, however, and the hybrids beat by a day or two the 4 weeks that the plains tadpoles typically take to mature. "In a drying pond, that can be huge," says Pfennig.

She wondered whether females in desperate straits might actually be choosing the Mexican males. Simovich was skeptical, because the hybrids could just be an accident of numbers or crowding.

So Pfennig brought female toads into the lab and set them in tubs of water mimicking either shallow pools about to dry out or safely deep water. She then serenaded the females from speakers propped up in the tub. One broadcast a quacklike mating call of the plains males, and the other played the Mexican males' advertisements, which sound like someone running a finger over a comb. Females expressed a preference by hopping into the water and swimming toward one of the speakers.

Toads collected from ponds inhabited by both species strongly preferred their own males' calls—as long as the water was deep. In shallow water, the preference disappeared. Analyzing the data in more detail, Pfennig found that females with poor body condition were more likely to switch preferences to the Mexican toads. Eggs laid by skinny, stressed females are more likely to develop slowly, so the switch to hybrid young makes the most sense for such females, says Pfennig in the Nov. 9 *Science*.

The idea sounds plausible to Glenn-Peter Sætre of the University of Oslo. He has studied flycatcher nestlings that survive better at the end of the breeding season when dad's species differs from mom's. —S. MILIUS

HEY BABY A male of the plains spadefoot toad species calls to possible mates. In dry conditions, females of his species pay greater attention to the calls of Mexican spadefoot males.

SCIENCE

MOTHER KNOWS ALL

Next generation of prenatal tests finds clues to baby's health in mother's blood

BY SARAH C. WILLIAMS

For 9 months, doctors can at best make educated guesses about a growing fetus' future as a healthy human. Those first fuzzy black-and-white ultrasound images can provide a mother with the peace of mind that she has a boy or girl with a beating heart, but many developmental maladies leave no visible fingerprint. Doctors can diagnose some genetic disorders by means of amniocentesis, in which they examine whole fetal cells extracted from the amniotic fluid that surrounds a baby in the womb. But the risk of complications from this invasive procedure, which requires inserting a long needle into a woman's abdomen, can exceed the risk that something was wrong to begin with.

In the late 1990s, Dennis Lo, now at the Chinese University of Hong Kong, was studying a possible low-risk way to get a window on a fetus' health by searching for fetal cells in pregnant women's blood. But "the problem with fetal cells is that they are incredibly rare," says Lo. One milliliter of a pregnant woman's blood, which contains 6 million cells, might yield only one or two fetal cells.

But then a series of papers caught Lo's eye. The papers examined how cancer tumors shed not just whole cells but also pieces of loose DNA, called cellfree DNA (cfDNA), into a patient's blood. He wondered whether a developing fetus—which, like a tumor, is parasitic on its host and consists of rapidly dividing cells—might also shed bits of DNA. He ultimately proved that it did by showing that he could detect fragments of Y chromosomes, which only males have, in the blood of pregnant women carrying boys.

That research and the work that followed led to the rapid development, by independent companies, of tests that use cfDNA to determine a fetus' sex and blood type. Doctors now use these tests, which give accurate results weeks before an ultrasound exam can, to screen fetal sex in women carrying fetuses at risk of sex-linked genetic disorders such as hemophilia and muscular dystrophy (*SN: 7/22/06, p. 56*). Scientists have even discovered that, by measuring levels of cfDNA in a mother's blood, they can predict premature births and preeclampsia, a disease that causes dangerously high blood pressure in pregnant women. But the tests have limits: Scientists can be sure they are detecting a fetal gene only if they know that the mother doesn't possess the same gene herself.

Researchers seeking to refine and expand this kind of testing have now turned their attention to another part of a fetus' genetic material, called messenger RNA (mRNA). By looking for bits of mRNA in a mother's blood, scientists are learning what genes a fetus expresses as it grows and develops. This new perspective, they hope, will lead to prenatal tests for a plethora of developmental disorders.

THE 9-MONTH PICTURE In the first few days of pregnancy, some cells split from the fetus to form the placenta, the interface between mother and fetus. But cells constantly detach from the fringes of the placenta, says Lo. As they break apart, these cells release DNA, which can pass through to the mother's side of the placenta and enter her bloodstream. The cfDNA lasts about 15 minutes before it degrades.

The biggest limitation to prenatal testing based on fetal cfDNA is the difficulty of distinguishing fetal cfDNA from maternal cfDNA. Scientists can focus on genes that the fetus inherited from its father, which wouldn't normally be found in the mother, or they can look for the so-called silenced forms of specific genes that are expressed in a normal adult but not in a fetus.

> "You could take a mother's blood and see how a fetus' brain is developing, or its eyes."
>
> — JILL MARON, TUFTS–NEW ENGLAND MEDICAL CENTER

For these reasons, fetal-cfDNA tests can diagnose only a few genetic diseases. For example, a fetus will develop cystic fibrosis if it inherits from both parents mutations in a single gene. The parental mutations need not be the same, however. If mother and father carry different mutations, finding the father's version of the gene in the mother's blood means that the fetus has inherited the paternal flaw. But for most genetic diseases, a fetus needs to inherit identical mutations from both parents. This means that the mother would already have that mutated DNA circulating in her blood, making it hard to distinguish the fetal DNA from her own.

"For most genes, you've got a problem because the mother has the gene and the baby has that same gene too," says Lo. One way around this difficulty is to look for mRNA instead of DNA. Cells generate mRNA from a gene's DNA only when they need to manufacture proteins. Many genes involved in fetal development aren't functional in an adult, so mRNA from them would never show up in adult cells. For example, most genes that tell the brain how to grow are needed only as a fetus develops, so mRNA for these developmental genes wouldn't be circulating in an adult.

In the October *Journal of Clinical Investigation*, Diana Bianchi and Jill Maron of the Tufts–New England Medical Center in Boston describe experiments in which they identified more than 100 fragments of fetal mRNA in maternal blood. The researchers looked for mRNA in blood samples taken from nine women before and after they gave birth in order to pinpoint genes that were in the mothers' blood before delivery but not

afterward. To be sure of the origin of the mRNA, they then checked what they found against mRNA in blood taken from the newborn babies.

Many of the genes expressed just before birth, Bianchi and Maron found, mediate development of the neural system. Others allow vision and other senses to function. For a baby about to open its eyes to light for the first time, this makes sense, says Maron. At the point of being born, a baby needs to ramp up the production of everything that will help it face the world.

The researchers hope to pursue this strategy by creating a timeline of normal fetal-gene expression through the whole 9 months of pregnancy. They would then compare this benchmark with fetal-gene expression throughout the pregnancies of women whose babies are known to have certain genetic disorders.

"Since we've started to develop what a normal fetus looks like at term, what if we look at an abnormal fetus at term? Or what if we move back to the second term?" says Maron. "We want to see what's possible. You could take a mother's blood and see how a fetus' brain is developing, or its eyes."

If doctors can track the activity of genes during fetal growth, Bianchi says, they will know, simply by testing a drop of blood, as soon as something abnormal shows up, rather than having to wait until they can see something physically wrong on an ultrasound scan.

"The ultimate goal, of course, is fetal treatment," says Bianchi.

Michael Katz, senior vice president of research and global programs at the March of Dimes in White Plains, N.Y., says that this technique holds immeasurable promise for prenatal diagnosis of rare conditions that doctors currently have no tests for. But Katz cautions against using new tests clinically before they are as accurate as amniocentesis.

"They need a vertical expansion of their technique—more precision—and they need horizontal expansion—more things it can test for," says Katz. "It's long term. I don't mean 20 years, but maybe 5."

COUNTING CHROMOSOMES Scientists have long been stymied in their attempts to find a noninvasive way to detect Down syndrome, which affects about 1 in 700 live births worldwide. Caused by the presence of a third copy of chromosome 21—a phenomenon known as trisomy—Down syndrome can be definitively diagnosed only by amniocentesis or by the slightly riskier chorionic villus sampling, which involves examining cells taken from the placenta.

"Initially, many people suggested we could never crack trisomy with cellfree DNA," says Lo. "Diagnosing a trisomy, by definition, is counting the number of chromosomes in a cell. And if we don't have the entire cell, just DNA swimming outside it, then how can you do that?"

Lo found a solution, however, when he turned to cellfree mRNA instead of DNA. His results appeared in the February *Nature Medicine*.

If a fetus has three copies of chromosome 21, Lo figured, it must have acquired two copies from one parent and one from the other. Lo identified a gene, *PLAC4*, on chromosome 21 that is expressed only in placental tissue—so that any *PLAC4* mRNA in a pregnant woman's blood must come from the placenta. What gives *PLAC4* its diagnostic value, however, is that it comes in two distinct forms that differ at just one location on the gene. About half the population has one form of *PLAC4*, half the other.

Putting together these ingredients, Lo devised a way to test for Down syndrome by examining fetal mRNA in maternal blood. If a mother and father have different versions of *PLAC4*, then finding equal amounts of the corresponding mRNAs in the mother's blood would mean that the fetus is normal, with two copies of chromosome 21. But if there is twice as much of one version as of the other, the fetus must have three copies of chromosome 21—and Down syndrome.

Lo tried this test on 67 pregnant women. Ten of the women carried fetuses already known to have Down syndrome while the rest were known to have healthy fetuses. The blood-based test detected 9 of the 10 cases of Down syndrome.

Although this test doesn't work for fetuses whose parents both have the same version of *PLAC4*, future adaptations of the test could take advantage of other single-spot variations in the *PLAC4* gene. Lo says that a test combining many such hot spots would not only extend its applicability to a larger proportion of the population but would also more accurately diagnose Down syndrome and other syndromes related to extra chromosomes.

Katz, however, worries that such a test still would miss out on disorders that arise when a fetus has an extra part of a chromosome but not the whole chromosome. Six percent of Down syndrome cases, for example, are caused by chromosome 21 being duplicated only in part.

"There's a whole range of other rare birth defects caused by chromosomal abnormalities that depend on repeats," Katz says. Lo responds that as long as a gene like *PLAC4* can be found on the part of chromosome 21 that's duplicated, the test would still work.

The hope, then, is that as the science advances, increasingly sophisticated blood-based tests will allow doctors to peer through the veil of mystery that obscures the developing fetus. While some bits of information may ruin surprises for moms and dads, other tests will confirm the health of their unborn baby—something every parent wants. ■

Questioning the Value of Knowledge

New tests raise ethical concerns

The idea that a finger prick can reveal whether a fetus is a boy or a girl and whether it is afflicted with any genetic maladies doesn't appeal just to parents and doctors. A number of companies see a commercial future in simple, at-home genetic tests.

Ethicists, though, worry that these companies tend to stretch the limits of science. Baby Gender Mentor, marketed by Acu-Gen Biolab, Inc. in Lowell, Mass., is a blood-based cfDNA test to determine fetal sex. The company calls its product "the most accurate DNA gender test" and boasts 99.9% accuracy. But more than 100 women are involved in a class action lawsuit, stemming from false results, that accuses Acu-Gen Biolab of fraud and misrepresentations.

Bioethicist Gail H. Javitt of the Johns Hopkins University says that the problem with products such as this is that they're not regulated enough. Baby Gender Mentor and other tests like it aren't subject to regulation by the U.S. Food and Drug Administration, so it's possible that these types of tests can be developed into commercial products before the science behind them has been fully validated.

"There's so much information coming out of the human genome with the potential to be beneficial to disease treatment and prevention," Javitt says. "But the regulatory system just hasn't kept up with this dramatic change."

As scientists explore the wide horizon of mRNA-based prenatal tests, these regulatory challenges are bound to emerge repeatedly. Although the new generation of tests offers the possibility of accurately diagnosing a broad range of developmental disorders, it also brings forth challenging ethical questions on what information expectant parents ought to know—and what to do with this information. —S.C.W.

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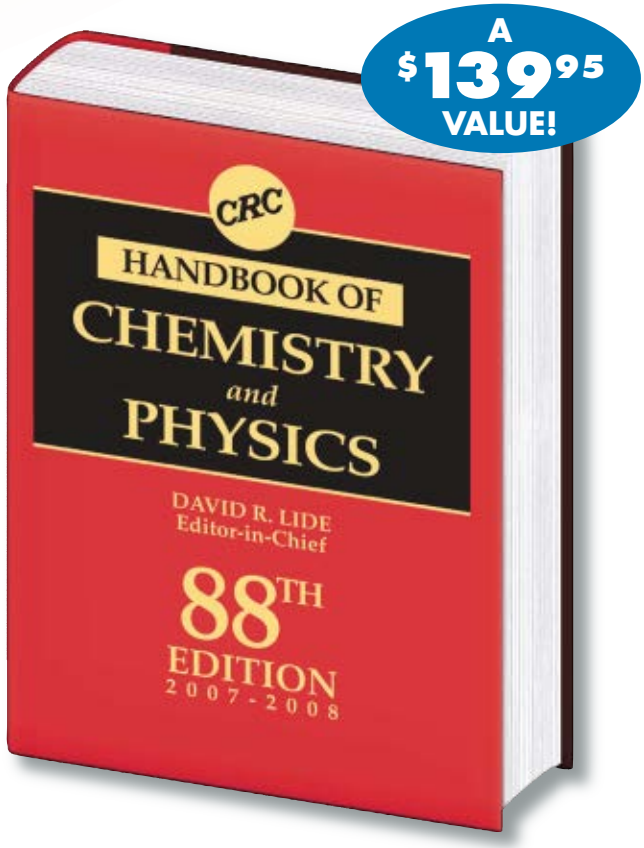

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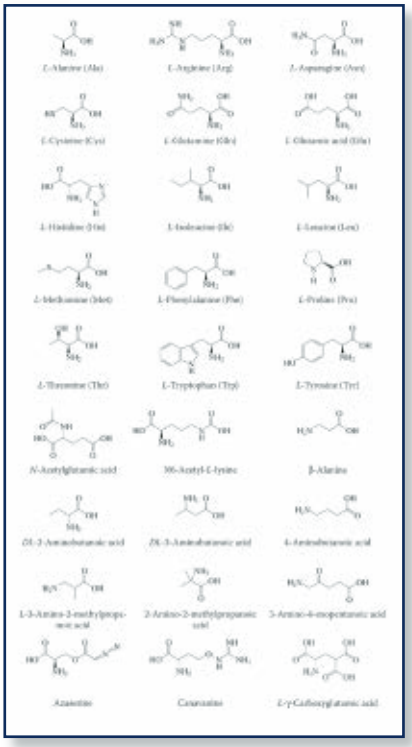

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TORTOISE GENES AND ISLAND BEINGS

Giant Galápagos reptiles on slow road to recovery

BY BRYN NELSON

Not far from where the Galápagos Islands' most famous loner spends his days, tourists disembark by the inflatable boatload at a modern dock. A path takes them past marine iguanas sneezing brine from their salt-caked nostrils and striated herons roosting in the red mangroves to the Charles Darwin Research Station in Puerto Ayora on Santa Cruz Island. Within the station, another walkway leads to a natural enclosure sheltering a misanthropic Galápagos tortoise named Lonesome George.

The confirmed bachelor has been a potent icon of conservation ever since he was spotted on remote Pinta Island in 1971 and captured the next year by a group of goat hunters. Now in his 60s, 70s, or beyond—no one really knows—George may have lived more than half his life in exile. He is quite likely the world's last pure-bred Pinta tortoise, one of the dozen or so closely related species that still lumber around the Galápagos, an archipelago of 19 islands and dozens of islets about 600 miles west of mainland Ecuador.

Last April, however, the surprise discovery that Lonesome George has a genetic cousin on another island cast doubt, in a hopeful way, on George's one-of-a-kind status. The revelation is just one illustration of how genetics and conservation biology are intermingling to rewrite an oversize reptile's evolutionary past and to reshape plans to safeguard the remaining tortoise species well into the future.

ON THEIR WAY — Tortoise hatchlings only a few years old beat the heat at the Charles Darwin Research Station in the Galápagos Islands. These hatchlings, bred in captivity, will be released into the wild at 5 years of age.

REVIVAL SIGNS Estimates of how many giant tortoises remain in the Galápagos vary widely, from less than 10,000 to more than 30,000. Nearly everyone agrees that their prospects are improving, however. "If you look at tortoises today compared to 50 years ago, they are so far ahead of where they used to be," says Linda Cayot, Lonesome George's former keeper and a scientific adviser to the Falls Church, Va.–based Galápagos Conservancy.

But tortoise conservation may be a rare bright spot in the struggle to protect the fragile Galápagos ecosystem. The archipelago is so revered for its unique marine and terrestrial life that it was the first World Heritage Site chosen by the United Nations Educational, Scientific and Cultural Organization (UNESCO). In late June, the organization's World Heritage Committee added the caveat "in danger" to the designation to draw attention to mounting threats, including a surge in tourism and rising immigration from Ecuador's mainland. Increased flights and boat traffic have contributed to a 60 percent escalation in introduced species since 2001.

In April, before the UNESCO announcement, Ecuador's President Rafael Correa acknowledged these concerns by declaring the islands' ecosystem a national priority for conservation efforts. Amid the ensuing calls to scale back residency permits and overhaul a broken tourism model, the discovery of Lonesome George's kin sounded a rare hopeful note. Having compared highly variable regions of DNA from cell nuclei, Gisella Caccone and Jeffrey Powell of Yale University and their colleagues reported in the May 1 *Current Biology* that a tortoise on volcano-studded Isabela Island has about half its genes in common with George. The researchers even suggested that George may have full relatives on the same island.

The potential salvation of George's species, the Pinta tortoise, began in 1994. That year, the Yale team collected blood from 27 tortoises living on the slopes of mile-high Volcán Wolf, an active volcano on Isabela Island's northern end. Unlike single-species populations found elsewhere in the Galápagos Islands, the Volcán Wolf tortoises display an unusual combination of carapace shapes. Some are dome shaped, others have Lonesome George's distinctive saddle-back form, and some show characteristics of both types.

By 2002, the researchers had retrieved enough nuclear DNA and maternally inherited mitochondrial DNA from other Galápagos populations to tease out some unexpected links. The Volcán Wolf group seems to include a hodgepodge of lineages arising from multiple colonizations, while Lonesome George appears most evolutionarily related to saddle-backed tortoises on Española and San Cristóbal Islands, more than 180 miles to the southeast. Caccone speculates that some tortoises on the southern islands may have floated on the strong ocean currents that flow northwest to Pinta.

In 2003, a joint expedition by the Galápagos National Park and the Oviedo, Fl.–based Chelonian Research Institute failed to find any signs of tortoise life on Pinta Island but did uncover the skeletons of 15 former male residents. By extracting DNA from those remains and from others stashed away in museum collections, Caccone and her collaborators were able to compile a robust genetic profile of the Pinta species. Later, the researchers found a partial match in the nuclear DNA of a young male tortoise from the previously sampled Volcán Wolf population. The tortoise's mitochon-

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drial DNA indicated that his mother had been born on Isabela. But it was clear that he had a Pinta male for a father, making him a hybrid of the two species.

"We had it all along but didn't know it until we had the new samples from Pinta," Caccone says. Because they have already uncovered one half-match among 27 Volcán Wolf tortoises out of a total estimated population of 2,000 to 8,000, she says, that "the chance of finding another hybrid, or even a pure [Pinta], is pretty high."

Caccone hopes to send three teams back to the steep volcano to collect more tortoise-blood samples next summer. If DNA tests reveal the presence of pure-blood Pintas, researchers could set up a new breeding program.

Discovering more Pinta tortoises would be "thrilling," agrees Johannah Barry, president of the Galápagos Conservancy, "but it would probably not be critical to the restoration of the Pinta Island ecosystem." Beyond the small chance of finding enough individuals to constitute a robust population, she says, back breeding any half-relatives to recover a pure Pinta bloodline could take decades.

How a Pinta father ended up on Isabela remains unclear. A strong current runs the roughly 50-mile route from Pinta to Volcán Wolf, and historical accounts leave open the possibility that tortoises may have washed ashore after being dumped overboard by pirates or whalers.

Genetic studies may allow researchers to reconstruct the history of specific tortoise populations and to determine whether they may have long-lost relatives on other islands. Even so, Caccone warns that genetic patterns are often deceptive within endangered species. Diverse genotypes, normally a hallmark of older populations, can be rapidly depleted through human interference and result in populations with artificially youthful profiles, she says.

POOL PALS — A Galápagos tortoise shares a morning swim with a white-cheeked pintail in a duckweed-covered pool in the Santa Cruz highlands.

TORTOISE TALES Millions of years before Europeans first caught sight of the Galápagos in 1535, ancestors of the islands' tortoises were likely roaming the South American continent. Mitochondrial-DNA comparisons suggest that the small Chaco tortoise found in the southern half of South America is the closest living relative of its much larger Galápagos counterparts, although Caccone believes that their common ancestor was also oversize. A combination of genetic evidence and geological estimates of when the islands were formed suggests that tortoises likely arrived no more than 2 to 3 million years ago, she says.

As for how the animals made the 600-mile ocean voyage, the chilly Humboldt Current that flows north from the tip of South America and then west along the equator could have been a conduit. "It's a great highway," Caccone says. Whether carried along on her own or on a floating mat of vegetation, a single female laden with eggs could have founded the entire Galápagos population.

Apart from their size and buoyancy, Galápagos tortoises can stay alive for 6 to 9 months without food or water, an evolutionary adaptation that became a curse when 17th- and 18th-century buccaneers and subsequent waves of whaling crews discovered that the reptiles would provide a plentiful and long-lasting source of meat. The logbooks of whaling ships record crew members often loading tortoises by the dozens into bilges and cargo holds, including up to 100 Pinta tortoises at a time.

At least two species went extinct. And by the early 1900s, American and British researchers had retrieved only a handful of live tortoises on Pinta, all of which were killed by the collectors or died en route to distant museums. The fate of the Pinta population remained murky until 1971, when a snail expert conducting research on the island saw a single tortoise and took a few pictures, unaware that his sighting was anything unusual. Peter Pritchard, director of Chelonian Research Institute, recalls that the researcher casually mentioned his sighting when the two were dining together. "Well, I was flabbergasted," Pritchard says.

Eventually, word reached the Charles Darwin Foundation, which receives its funding from a range of nonprofit organizations, countries, and individual donors and advises the Ecuadorean government on conservation issues. The foundation's research station launched an expedition in early 1972. Pritchard, who was studying marine turtles at the time, joined the trip to look for turtle nesting sites. By the time his boat arrived, he says, a resourceful student had already found the Pinta tortoise, and the expedition's goat hunters had tethered it to a cactus so that it wouldn't disappear.

MOVING FORWARD In the 35 years since then, Lonesome George has been living at the research station on Santa Cruz, spurning two female tortoises from Isabela, ignoring frisky males who have provided sex-ed lessons, and spurring a barrage of speculation over what ails him in the reproduction department.

George's keepers have looked into diet, erectile dysfunction, and other bodily functions but have yet to find an answer. Nor do researchers know enough about reptile physiology to try cloning him. In 1994, Cayot learned a sperm-retrieval technique from a German zoo veterinarian and taught a Swiss volunteer how to fondle a rather ticklish George. "She could get the other tortoises to ejaculate in 15 minutes," Cayot recalls. "We worked with George for months and got nothing."

Pritchard says that he has a videotape of George "energetically chasing a female, mounting her, and getting pushed off as she goes under a low branch"—testament to his intact, if unrefined, libido. But if the bachelor tortoise can't, or won't, keep the bloodline going, Caccone hopes that her hunt for a living relative with a stronger inclination will let George off the hook.

In the meantime, conservationists have assisted the wild tortoises on Isabela and other islands with a massive effort to eradicate one of their worst enemies: feral goats. Introduced as a food source by whalers, fishermen, and settlers, goats can chew plants down to the nub. Large herds can tear up the landscape, leading to severe erosion and even ecological collapse. On Isabela Island, goats didn't arrive until the 1970s. Less than 3 decades later, their ranks had swollen to an estimated 75,000 to 125,000.

Project Isabela, run jointly by the Charles Darwin Foundation and the Galápagos National Park, employed helicopters, hunters, and trained dogs to track down the island's unwelcome interlopers. The collaborators also fitted female "Judas goats" with radio collars to betray the locations of male admirers. Last year, researchers announced that the northern part of Isabela was goat free, adding to earlier successes on Pinta, Santiago, and Española Islands.

On Española, a tourist favorite in the archipelago's southern reaches, goats were removed by the thousands in the 1970s, but by then the island's resident tortoise population had dwindled to 12 females and 2 males. Volunteers evacuated the survivors to the Charles Darwin Research Station and set up an emergency breeding program. A third male from Española was later located at the San Diego Zoo and called into service. Remarkably, they and their repatriated descendants now number more than 1,400.

Tortoise-breeding centers are operating on the islands of Santa

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Cruz, Isabela, and San Cristóbal. At 5 years of age, most tortoises are too large to be threatened by invasive black rats and Norway rats, and can be resettled on their native islands with about an 80 percent survival rate. The Galápagos Conservancy's Barry says that the impressive track record should be a model for other resource-limited locations. "I think that restoring what we had a hand in removing is a fairly nice spin of the cosmic wheel," she says.

Success in culling the goats has not only made Pinta Island safe for tortoises again but has also intensified calls for their return. Without a major herbivore to break up the vegetation and regulate access to sunlight, botanists fear a loss of diversity among native plants and habitats and have lobbied for a full-scale tortoise reintroduction.

Officially, the Charles Darwin Foundation is neutral on the proposal, though Bryan Milstead, its head of vertebrate research, is leaning in favor of it. Tortoises are the major herbivores in the Galápagos and vital regulators of the ecosystem, he says. If Lonesome George cannot sire a new generation of tortoises for Pinta Island, Caccone's genetic research has shown that "the next best thing would be to bring an Española tortoise there."

Caccone hopes to repopulate Pinta Island with its native species, whether by George or a relative, though she concedes that much will depend on what her team finds on Isabela. She has a precedent, though, for believing that her DNA comparisons may turn up the unexpected. Two years ago, Caccone's team discovered the genetic signatures of three separate lineages on Santa Cruz instead of its presumed single species. Among their finds, the researchers determined that an isolated dome-shelled group of about 100 tortoises known by their geographic location, Cerro Fatal, should be considered a new species and added to the radar of conservationists.

STANDING PROUD — A Galápagos tortoise with a distinctive saddle-backed carapace poses at the Charles Darwin Research Station.

As one species comes into being, taxonomically speaking, conservationists are struggling to keep hundreds of other types of native plants and animals from disappearing. Critically endangered mangrove finches are being terrorized by rats. Any importation of the West Nile virus could decimate the Galápagos penguin population. And guavas are among the hundreds of introduced plants that now far outnumber native ones.

Park officials are still grappling with tortoise poaching in some remote areas of Isabela, and they are reviewing a plan to kill invasive rats that eat native-born hatchlings on Pinzón and other islands. Even so, the evolutionary icons that so intrigued Charles Darwin are adapting better than many other Galápagos species. Although the guava is overtaking native plants, its fruit is fast becoming a favorite among the tortoises. "They're tough beasts, as long as people don't roll them over and chop them open," Pritchard says.

Like Pinta's potentially lost population, tortoises on San Cristóbal were once given up for dead. But on a trip to San Cristóbal in June, Pritchard's group counted 128 tortoises in 4 hours. "Give them a chance," he says, "and they will recover." ■

NELSON

OF NOTE

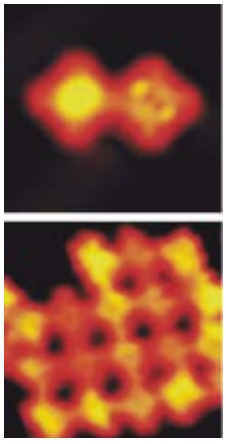
NANOTECHNOLOGY

Hooking up

Researchers have created molecules that spontaneously form sturdy networks on a surface, a step that could bring molecular-scale electronic circuits closer to reality.

Leonhard Grill of the Free University of Berlin and his colleagues synthesized flat, square molecules with arms extending from all four sides. The team engineered the molecules to be either reactive or inert at the tip of each arm, and then temporarily capped the reactive tips with bromine atoms.

Deposited onto a gold surface, the molecules slid around, nudged by random thermal jittering. Heating the surface to 270°C for about 15 minutes drove off the bromine caps. As the molecules continued to wander, their reactive ends began to find one another and form stable, covalent bonds.



LINKED IN Self-assembling molecules connect to form a pair (top) or a grid (bottom) in these electron microscope images.

Versions of the molecules with just one reactive arm got together in pairs, while those with two or four reactive tips got together in chains and grids, respectively, the researchers report in the November *Nature Nanotechnology*.

Grill says that the method is more promising than other techniques for self-assembling molecules that rely on weaker chemical bonds. Other molecules could be engineered to form more-complex structures such as circuits, he adds. —D.C.

BIOMEDICINE

Earache microbe shows resistance

A microbe that causes middle ear infections has developed resistance to a wide range of antibiotics, a new study finds.

The offender is a strain of *Streptococcus pneumoniae*, a bacterium best known for causing pneumonia. However, the microbe can also trigger middle ear infections, meningitis, sinus infections, and various respiratory ailments.

Immunization with a vaccine called pneumococcal 7-valent conjugate prevents infection by the seven most common strains of *S. pneumoniae*. But in recent years, physicians have encountered strains of *S. pneumoniae* not covered by the vaccine that have become impervious to some antibiotics.

Janet R. Casey and Michael E. Pichichero of the University of Rochester (N.Y.) now report that one strain that has cropped up in at least nine children since 2003 is resistant to all 18 drugs currently approved for use against middle ear infections in children. They report the finding in the Oct. 17 *Journal of the American Medical Association*.

The researchers used an adult antibiotic called levofloxacin (Levaquin) to kill the resistant microbe. But this drug isn't approved for use in children.

Between 2001 and 2006, health officials in Massachusetts recorded 94 invasive pneumococcal infections caused by this same *S. pneumoniae* strain. By 2004, signs of drug resistance showed up in many of these infections, which typically manifest as pneumonia, bacteremia, or meningitis.

Stephen I. Pelton, a pediatrician at the Boston University Medical Center, says that the troublesome strain accounted for only 5 percent of *S. pneumoniae* infections in 2000 in Massachusetts but more than 40 percent by 2004. Pelton and his colleagues report the data in the Oct. 19 *Morbidity and Mortality Weekly Report*.

The *S. pneumoniae* strain, dubbed 19A, has also shown up in Canada, France, Spain, and Israel. "It's a disturbing trend," Pelton says. —N.S.

EARTH SCIENCE

Groundwater use adds CO_2 to the air

Using groundwater for crop irrigation or industrial purposes adds more planet-warming carbon dioxide to the atmosphere than volcanoes do, a new study suggests.

As water soaks through soil, it picks up carbon dioxide that's generated when organic matter in the soil decomposes. On average, groundwater holds from 10 to 100 times as much carbon dioxide as the water in lakes and rivers, says Gwen L. Macpherson, a hydrogeologist at the University of Kansas in Lawrence. When groundwater is pumped to Earth's surface, carbon dioxide escapes to the atmosphere, where it acts as a greenhouse gas.

In recent years, people have been pumping about 740 cubic kilometers of water from the ground each year, Macpherson said last week in Denver at a meeting of the Geological Society of America. Chemical analyses of groundwater suggest that this water use adds about 300 million metric tons of carbon dioxide to the atmosphere annually.

The volume of groundwater-generated carbon dioxide is, at most, a small percentage of the emissions produced by burning fossil fuels, says Macpherson. Nevertheless, her analysis suggests that the amount of carbon dioxide fizzing from groundwater is about three times that spewed skyward by volcanoes, a major natural source of atmospheric carbon dioxide. —S.P.

FOOD SCIENCE

Salmonella seeks sweets

Salmonella enterica, a major food-poisoning germ, can enter the tissues of fresh lettuce where no amount of surface washing will evict it. The scientists who reported that finding earlier this year now think that they've gotten to the root of the issue.

To model salmonella soil contamination from livestock wastes, the researchers seeded sterile manure with one of three toxic strains of *S. enterica*. They then planted lettuce seeds in nearly 300 pots of soil fertilized with either clean or treated manure. Six weeks later, 18 to 25 percent of the young plants grown with infected manure hosted surface salmonella contamination.

The scientists ground up half of the 300 plants to look for internal infections—and found only three instances. They also grew plants in a sterile solution to which various *S. enterica* strains had been added. Internal infections turned up in 59 to 93 percent of these plants. Michel M. Klerks of Wageningen University in the Netherlands and his colleagues report their findings in the November *ISME Journal*.

Lettuce roots emit a sugarlike secretion. Its presence, Klerks' team found, turns on genes in salmonella bacteria that produce sugar sensors. When germs detect the lettuce secretions, they make a beeline into the plant's roots. En route, they become more infective by turning on genes that help them glom on to plant cells (*SN: 10/20/07, p. 250*).

Klerks now suspects that relatively few manure-treated plants developed internal infections because the many harmless bacteria in soil offered the salmonella tough competition for food and root access. —J.R.

NATURE NANOTECHNOLOGY

MEETINGS

American Society of Human Genetics
San Diego, Calif.
Oct. 23–27

SCIENCE & SOCIETY

Burdens of knowledge

The gold rush of human genetics is well under way. Now that tools for profiling genome activity are widely available, scientists have found more than 80 disease-related variations in human DNA, many of them in the past year.

As a result, ethical arguments about how to handle people's genetic information—and related disease-risk information—are no longer academic. Much of the discussion this year among human-genetics researchers has revolved around the sticky issues posed by the windfall of new knowledge. For example, how can scientists keep genetic data from research studies truly anonymous when DNA is, by its nature, the ultimate fingerprint?

Furthermore, studies looking for genetic variations involved in a disease often include thousands of people as research subjects. Scientists use microchiplike wafers to test each person for hundreds of thousands of DNA variants simultaneously, usually under agreements of confidentially and nondisclosure.

What, then, should researchers do if they stumble across genetic variations in a study participant that put the person at increased risk for a life-threatening disease unrelated to the condition being studied? If the disease is treatable, saying nothing could be unethical, but telling the subject about the risk would require breaking the anonymity of the data and violating the study's nondisclosure agreement.

"If the original agreement was that you wouldn't tell them, then you shouldn't tell them," said Francis S. Collins, director of the National Human Genome Research Institute in Bethesda, Md., in an interview with reporters. "The triumph of discovering these variants should not be taken as evidence that we should necessarily offer this [information] to patients."

After all, while a gene variant might double a person's risk for a deadly disease, often the actual risk would remain low—8 percent rather than 4 percent, for example. "Would that change your behavior?" Collins asked. Knowledge of this greater risk could cause the person distress without being usefully "actionable," he said.

The debate is far from settled, but Collins suggested that disclosure should be reserved for cases that meet certain criteria. The risk of disease would have to be high, and the disease would have to be preventable or curable. Such a case would raise important questions, Collins noted: "Is this the kind of scenario [the subject] imagined when they said they didn't want to be notified? And if not, is it the benevolent thing to do to notify them anyway?"

Collins gave the example of a mutation in a gene called *MutL homolog 1, colon cancer, nonpolyposis type 2* (*MLH1*) that's known to increase a person's lifetime risk of developing colon cancer from an average of about 6 percent to around 60 percent. The disease is often fatal, but frequent screenings starting before age 30 and removal of benign polyps can save a person's life.

This would be a case in which Collins suggests that informing the person would be the correct ethical decision. —P.B.

INFECTIOUS DISEASES

Nongene DNA boosts AIDS risk

A newly discovered genetic variation raises some people's vulnerability to infection by HIV, the virus that causes AIDS.

People who have this difference in a single letter of their genetic code would have about a 15 percent greater risk that exposure to the virus will lead to infection than would individuals without the genetic variation. That conclusion comes from laboratory studies of human cells by Samuel Deutsch of the University of Geneva and his colleagues there and at the University of Lausanne in Switzerland.

Once infected, furthermore, those with the variation fare worse than other HIV-positive people. In a comparison of 805 HIV patients not yet receiving antiviral drugs, the 56 people with the mutation showed a more rapid decline of the immune system than did those without the mutation.

Yet this variation occurs in a region of DNA far from any protein-encoding genes, in an area of so-called junk DNA that scientists once presumed to have no function.

"This is in the middle of nowhere," Deutsch says. The discovery is another example of scientists finding that some DNA formerly considered junk actually regulates the activity of genes. Because of this regulatory role, mutations in these regions can influence cell behavior and sometimes contribute to disease (*SN: 9/8/07, p. 154*).

The HIV-related mutation appears to regulate a cluster of about six genes with unknown functions, though further research is needed to confirm this, Deutsch says.

Whatever the regulated genes turn out to be, the proteins they produce are likely to be part of the mechanism by which HIV infects human cells. The proteins would therefore provide targets for developing drugs that combat infection. —P.B.

EVOLUTION

Doing the DNA shuffle

During the 6 million years since humans' ancestral lineage diverged from the ancestors of the other great apes, DNA near the ends of human chromosomes has evolved more rapidly than scientists had previously realized.

A new comparison of macaque, orangutan, ape, chimpanzee, and human genomes shows a surprising amount of DNA reshuffling in these chromosome regions, called subtelomeres. These volatile areas of roughly 150,000 to 500,000 genetic units lie between the main body of a chromosome, which varies little among related species, and the caps on each end of the chromosome, called telomeres.

Previous studies had underestimated how much swapping and reordering of DNA segments has occurred within subtelomeres because scientists had looked only for segments in the apes' genomes that matched parts of the human genome, says Katie Rudd of Emory University in Atlanta. That approach overlooked segments in the apes' genomes that had been lost in the human genome.

To get a fuller picture of subtelomere evolution, Rudd and her colleagues closely examined the ends of chromosomes 14 and 15 in all the species studied. They found that chunks of subtelomere DNA had been thoroughly shuffled over evolutionary time. The chromosomes had occasionally lost or picked up chunks of DNA, often by swapping bits with the subtelomeres of neighboring chromosomes.

"This is crazy. This is off-the-charts different," Rudd says. "No one knew that the subtelomeres [of related ape species] have totally different structures." The cause of this high rate of rearrangement is still unclear, Rudd says.

The subtelomeres studied by Rudd's team contained genes for smell receptors. A gene involved in a form of human muscular dystrophy is also found on a subtelomere. Scientists don't yet know what other genes lie in these rapidly evolving areas, Rudd says. —P.B.

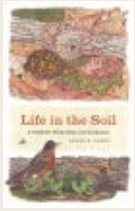
Books

A selection of new and notable books of scientific interest

LIFE IN THE SOIL: A Guide for Naturalists and Gardeners

JAMES B. NARDI

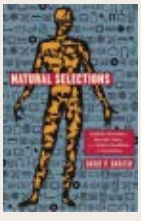
Soil is more than just a planting medium. The ground beneath our feet is teeming with life, forming an ecosystem that provides the elements necessary for plant growth. Soil's underappreciated biological niche gets an in-depth look in this informative illustrated guide. Biologist Nardi profiles the organisms living in the soil, ranging from bacteria to 10,000-year-old fungi to predatory beetles, and describes how each organism contributes to soil chemistry. Part one of the book explains how soil is formed and describes the relationship between specific plant roots and their bacterial and animal partners. Part two describes each organism in a soil ecosystem. Each entry includes the organism's scientific name, place in the food web, impact on gardens, size, and number of species. Nardi ends with suggestions on how people can work as partners with creatures in the soil. ***Univ. Chicago, 2007, 293 p., color plates and b&w illus., paperback, $25.00.***



NATURAL SELECTIONS: Selfish Altruists, Honest Liars, and Other Realities of Evolution

DAVID P. BARASH

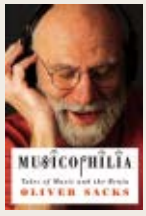
As the debate between proponents of intelligent design and of evolution rages on, Barash demonstrates how natural selection can explain various aspects of the human experience. He tackles some of the harder questions, such as whether or not we have free will. He debunks the notion that humans are exceptions in the process of evolution. He takes on common arguments against evolution, such as the assertion that it's only a theory and that natural selection cannot create anything new. He addresses the fallacy behind the belief that all natural phenomena are good. Many attributes of human behavior, such as infanticide and racism, he proposes, may be wired into our genes. He muses on the relationship between culture and biology, considering, for example, why people keep pets. ***BLP, 2007, 192 p., hardcover, $25.00.***



MUSICOPHILIA: Tales of Music and the Brain

OLIVER SACKS

A piece of music can compel us to move our bodies or move us to tears. How can a series of notes, strung together, have such a profound effect? And what can that experience tell us about the human brain? Author and physician Sacks explores the experience of music from the point of view of musicians, everyday people, and patients struggling with strange, music-related maladies. For instance, he describes a man who developed an overwhelming desire to compose music after being struck by lightning. Another patient developed a fear of music after certain pieces triggered seizures. A third patient lost memory for everything except music. Sacks describes a condition known as Williams' syndrome, whose sufferers are often endowed with extraordinary musical talent. In addition to focusing on pathologies, he explores everyday phenomena, such as music's ability to conjure up images and the annoying tendency for a musical phrase to sometimes stick in one's mind. ***Knopf, 2007, 381 p., hardcover, $26.00.***



HOW TO BUILD AN IGLOO AND OTHER SNOW SHELTERS

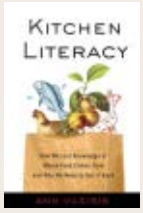
NORBERT E. YANKIELUN

Forget snowmen. To really impress the neighbors after the next snowstorm, build an igloo. Igloos are marvels of engineering—warm, solid structures composed of a cold, soft material. In this whimsical illustrated guide, Yankielun explains how to construct an igloo and other snow shelters step-by-step. After covering safety concerns such as dealing with windchill and frostbite and space constraints, Yankielun launches into the details of construction. He offers advice for selecting the appropriate site and snow, sizing the igloo, and cutting and arranging snow blocks. Igloos can be customized by adding windows, and adjoining igloos can be connected with snow tunnels, he notes. Yankielun explains how to construct lesser-known snow structures such as *quinzees*, hollowed-out, dome-shaped mounds of snow; and spruce traps, shelters formed under the base of evergreen trees. Finally, he offers instructions for forming emergency snow shelters and surviving under harsh cold conditions. ***W.W. Norton, 2007, 148 p., b&w illus., paperback, $17.95.***

KITCHEN LITERACY: How We Lost Knowledge of Where Food Comes From and Why We Need to Get It Back

ANN VILEISIS

Most people today are far removed from their foods' natural sources: They have no idea of how food gets to supermarket shelves. Historian Vileisis describes how centuries' worth of food knowledge has been lost as we increasingly rely on advertising and nutritional guidelines as a basis for choosing our daily menus. Two centuries ago, many of the vegetables eaten in a typical household were grown in the family garden. As food-manufacturing technology advanced, less and less of the work associated with agriculture and food processing was done at home. Vileisis describes how some people eventually began to regard factory-produced foods as unnatural and traces marketers' subsequent attempts to connect their products with natural goodness. She describes the resurgence of local and organic food and touts the advantages of this trend. ***Island Press, 2007, 332 p., b&w photos, hardcover, $26.95.***



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LETTERS

Thinking it through

Bjorn Merker says that "the tacit consensus concerning the cerebral cortex as the 'organ of consciousness' ... may in fact be seriously in error" ("Consciousness in the Raw," *SN: 9/15/07, p. 170*). But the real tacit consensus is that the cerebral cortex is the organ of conceptual consciousness, of thinking and reasoning, and that is not challenged by studies that identify the brain stem as orchestrating the basis of awareness. Awareness per se isn't the same thing as conceptual awareness.

TIBOR R. MACHAN, SILVERADO, CALIF.

Merker argues that basic forms of thinking and reasoning occur in primary consciousness. —B. BOWER

Pride and privilege

Other than people with HIV or AIDS, the prime model for a group overrepresented among those taking the option of physician-assisted suicide ("No Slippery Slope," *SN: 10/6/07, p. 212*) would appear to be educated, insured, financially comfortable, psychologically fit, nondisabled white males between the ages of 21 and 80. Perhaps the research simply demonstrates that we are loath to yield control, even in death.

WILLIAM MOCK, CHICAGO, ILL.

Unclear advice?

A researcher cited in "Exhaust fumes might threaten people's hearts" (*SN: 9/29/07, p. 205*) recommends that people at risk of heart attack should avoid exercising outdoors on highly polluted days. What an odd conclusion, on two counts: First, that avoidance, instead of elimination of the poison from the air we breathe, is the recommended course of action; and second, that only "at risk" people need take any special action regarding diesel pollution.

GEORGE CAMMAROTA, SAN JOSE, CALIF.

No, the other one

My cat has been doing for years what scientists at the University of St. Andrews reported of orangutans: motioning for healthy portions of their favorite foods ("Orangutans hand it to researchers," *SN: 9/8/07, p. 158*). Except that four tins of cat food later, my cat is still motioning "Not that kind, wrong flavor."

SALLY YOUNG, NEWPORT NEWS, VA.

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