Asteroid Travel Guide | Tallying Tuna's Toxicity | 174 Yoctos of Force

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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ MAY 22, 2010

Fatal Friends

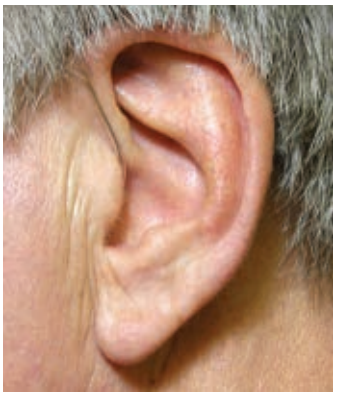
How the body fights back when fungi turn deadly

Metal Motes May Foul Young Minds

Chimps in Mourning

Quantum Trick Spins Off New Form of Matter

Pioneering audiologist invents "reading glasses" for your ears.



Neutronic Ear is the easy, virtually invisible and affordable way to turn up the sound on the world around you.

You don't have to pay through the nose to get Personal Sound Amplification Technology.

It's amazing how technology has changed the way we live. Since the end of the Second World War, more products have been invented than in all of recorded history. After WWII came the invention of the microwave oven, the pocket calculator, and the first wearable hearing aid. While the first two have gotten smaller and more affordable, hearing aids haven't changed much. Now there's an alternative… Neutronic Ear.

First of all, Neutronic Ear is not a hearing aid; it is a PSAP, or Personal Sound Amplification Product. Until PSAPs, everyone was required to see the doctor, have hearing tests, have fitting appointments (numerous visits) and then pay for the instruments without any insurance coverage. These devices can cost up to $5000 each! The high cost and inconvenience drove an innovative scientist to develop the Neutronic Ear PSAP.

Neutronic Ear has been designed with the finest micro-digital electronic components available to offer superb performance and years of use. Many years of engineering and development have created a product that's ready to use right out of the box. The patented case design and unique clear tube make it practical and easy to use. The entire unit weighs only 1/10th of an ounce, and it hides comfortably behind either ear. The tube is designed to deliver clear crisp sound while leaving the ear canal open. The electronic components are safe from moisture and wax buildup, and you won't feel like you have a circus peanut jammed in your ear. Thanks to a state-of-the-art manufacturing process and superior design, we can make Neutronic Ear affordable and pass the savings on to you.

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The Evolution of Hearing Products

Invention	Date	Easy to Use?	Invisible?	Affordable?
The Ear Horn	17th Century	No	Hardly	Maybe
Wearable Hearing Aid	1935	weighed 2.5 pounds	No	No
Digital Hearing Aid	1984	No	No	Not for most people
Neutronic Ear	2010	Yes	Yes	Yes

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MAY 22, 2010 • VOL. 177 • NO. 11


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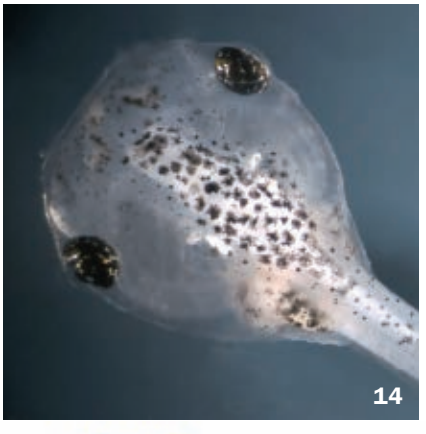
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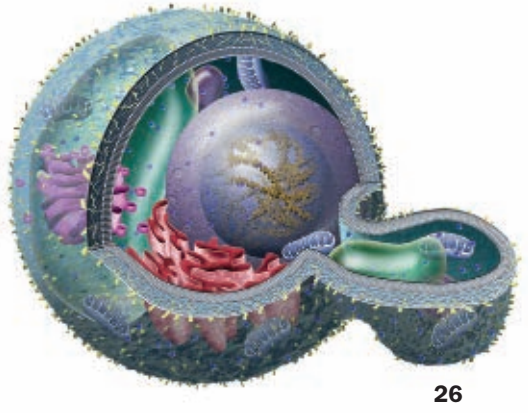
26

ScienceNews

In The News

Features

Departments

COVER Though it may look friendly, the green mold *Aspergillus ustus* can cause inflammation in the lining of the human heart, as well as skin and ear infections. *Dennis Kunkel/ Photolibrary.com*

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FROM THE EDITOR

Science News goes public: available on newsstands

With this issue, we embark on a new strategy for bringing news from the world of science to the public. For the first time in decades, *Science News* will be available for purchase on newsstands.

In the era of digital delivery via smart phones, pods and pads, selling a paper publication in a brick-and-mortar building might seem quaint. But if you've visited a Borders or Barnes & Noble lately, you might have noticed that numerous magazines are still on display, several devoted in some way to science. Hardly any, though, offer the thorough presentation of news from across the spectrum of scientific disciplines that has long been available to subscribers of *Science News*.

As those subscribers know, *Science News* is one of the world's best-kept secrets. Many readers interested in keeping up with science have been unaware of its existence. A presence on newsstands will make it known to a much broader audience.

Newcomers will find that *Science News* is no compilation of news releases or aggregation of others' work, but is rather a journalistic package of articles by experienced specialists who venture into the nooks and crannies of science where few bloggers dare to traipse. In this issue, for example, Alexandra Witze reports on one of the hottest new fields in physics — the study of curious materials called topological insulators (Page 22), substances that exploit quantum phenomena to do things that no other material has done before. On Page 16, Senior Editor Janet Raloff explores growing concern that tiny airborne pollutants exert damaging effects not only in the lungs, but also in the brain. And correspondent Laura Beil offers an engaging foray into the world of fungi (Page 26), focusing on how the body's infection-fighting mechanisms cope (or not) with the peculiarities of fungal biology.

Preceding those features are short accounts of news from multiple disciplinary frontiers. From the latest meeting of cancer researchers (Page 15) and the newest scientific view of the sun (Page 13), to the genome of an amphibian (Page 14) and chimps' reaction to death (Page 9), you'll find reports from all realms of science, from conferences and journals both prominent and obscure, from wherever news of science can be found. For those of you holding *Science News* in your hands for the first time, welcome. For our loyal long-time readers, we're sorry to have revealed your secret pleasure to the world. But we think more copies of *Science News* in circulation will make the world a better place. —*Tom Siegfried, Editor in Chief*

Explore the Mysteries of the Mind

Consciousness. It's as essential to human existence as water is to a fish, yet every night we surrender it gracefully. We recognize that we have it, but we can never be sure anyone else does. It has been the subject of philosophical and scientific debate for millennia, but we've yet to understand its true essence and purpose.

Join distinguished philosopher, psychologist, and award-winning Professor Daniel N. Robinson and explore fascinating questions about this unique mental state in **Consciousness and Its Implications**. Over the course of 12 thought-provoking lectures, probe the depths of this mysterious subject from the perspective of the philosopher, the psychologist, the scientist, and the doctor. A master storyteller, Professor Robinson brings this riveting topic to life with real-world examples and striking anecdotes about smart computers, sleepwalkers, comatose patients, zombies, and more. An in-depth guide to a fascinating subject we're learning more about every day, this course will change how you think about your own mind.

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Scientific Observations

"Evolutionary biology is in large part about reconstructing the past. One of the things I am excited about is our increasing ability to do just that with true genetic precision. For example, by identifying mutations that affect phenotypes, we can start to reconstruct traits of extinct species.... Using ancient DNA, we now know some Neanderthals were red-headed, and some mammoths were blond. Now imagine reconstructing or sequencing whole genomes and being able to predict the morphology, physiology or even behavior of ancient creatures. To what extent this will be possible is still unclear, but if we have reliable data, we can properly understand the direction and nature of evolutionary change in an unprecedented way." —**HOPI HOEKSTRA, BIOLOGIST AND MAMMALS CURATOR AT HARVARD UNIVERSITY'S MUSEUM OF COMPARATIVE ZOOLOGY, IN THE APRIL 13 *CURRENT BIOLOGY***

Science Past | FROM THE ISSUE OF MAY 21, 1960

PARENTS AND DELINQUENCY — A study of 400 juvenile delinquents in a mental hospital showed with "regular frequency" that the parents unconsciously fostered the delinquent behavior in their own children.... The parents show an addiction to the child's delinquency that is much like drug addiction. They even suffer acute "withdrawal symptoms" when psychiatric treatment results in the child's abandoning his delinquent behavior. Then the parent is likely, unconsciously, to find excuses to interrupt the treatment or place obstacles in the way of its progress. This unconscious interference by parents is one of the major difficulties in treating the child and his parents ... psychiatrists reported.

Science Future

June 3
2010 Kavli Prize winners in neuroscience, astrophysics and nanoscience are announced. See www.kavliprize.no

June 25–29
Evolutionary scientists host a joint meeting in Portland, Ore. See www.evolutionsociety.org

July 12–15
Computer scientists and engineers meet in Las Vegas for 22 joint conferences. See www.world-academy-of-science.org/worldcomp10

For Daily Use

Contrary to widespread belief, battling excess bulge may be a good thing in older patients, a small new study shows. Doctors worry about unintentional weight loss in the elderly because it can signify conditions such as diabetes or cancer and cause disease complications. But a study of 316 obese older patients found that those assigned to a diet or diet-and-exercise program were less likely to die in the eight years after the study than patients who did not diet. On average, dieters lost 10 pounds during the 18-month-study, compared with a three-pound loss in nondieters, researchers report in the May *Journal of Gerontology: Medical Sciences*.

SN Online

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EARTH

A Caribbean lake of asphalt replete with microbes could serve as a model for studying extraterrestrial life. See "Life in the sticky lane."

GENES & CELLS

Pea aphids (shown) are the first animals known to make their own carotenoids, nutrients usually found in plants. See "Aphids make their own bright colors."

Science Stats

MORE AND LESS FRIENDLY FISH

Mercury pollutants concentrate more in certain species of seafood, making those riskier eats than others. That's true even for different species of tuna.

Mercury levels in selected fish and shellfish

VERY LOW
Shrimp
Salmon

BELOW AVERAGE
Crabs
Catfish

ABOVE AVERAGE
Cod
Canned light tuna

MODERATELY HIGH
Halibut
American lobster

HIGH
Grouper
Canned/Albacore tuna

VERY HIGH
Swordfish
Tuna sushi/Bluefin tuna

SOURCE: E. GROTH/*ENVIRONMENTAL RESEARCH* 2010

CLOCKWISE FROM TOP LEFT: BEAR CIERI; CHARLES HEDGCOCK, RBP

“This particular image can arguably be called ‘Eagle Nebula on steroids.’” —MARIO LIVIO, PAGE 13

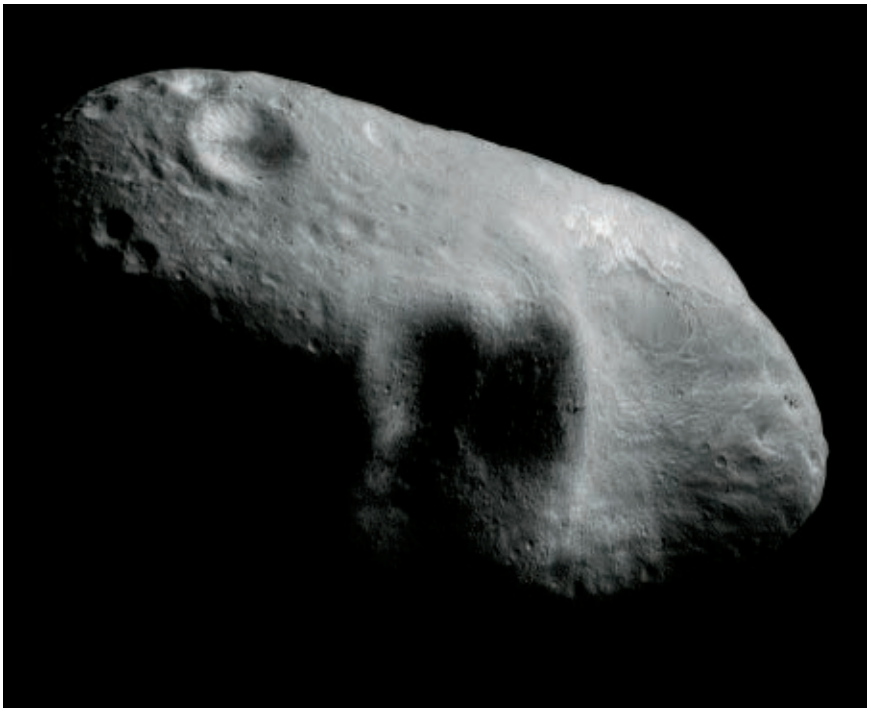
In the News

STORY ONE

Asteroid-bound: Scientists look for a worthy rock

Proximity and slow spin rate are desirable for exploration

By Laura Sanders



NASA-JPL, APL/JHU

Scientists have begun working out what makes an asteroid, Eros shown here, suitable for a crewed mission. Size, shape and composition are a few considerations.

The Little Prince, who stood tall on his fictional house-sized asteroid B612, may soon have company. Since President Obama announced last month that NASA plans to send people to an asteroid by 2025 (*SN: 5/8/10, p. 10*), scientists have been scrambling to fill in the details. Before astronauts can embark on such a journey, they need to choose a destination.

Already, researchers have begun culling the list of potential candidates. Martin Elvis of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., proposed criteria for identifying “potentially visitable objects” on April 28 in Brookline, Mass., at a meeting of the American Astronomical Society’s Division on Dynamical Astronomy.

Asteroids come in a menagerie of sizes, shapes and trajectories. Some are little more than giant loose rubble piles, while others are densely packed. Though Obama’s proposal didn’t point to any specific destinations, Elvis says that a worthy asteroid ought to have a few key features, including a slow spin rate, no problematic satellites and a solar orbit that allows for a long and recurring launch window.

“Are they spinning rapidly? Are they elongated? Is there strange, irregular gravity?” Elvis asks. If the asteroid is “lumpy and nasty, that’s not good.”

The most important consideration, though, is that the asteroid is easy to get to. While the majority of asteroids reside in a belt between the orbits of Mars and Jupiter, some come close to Earth. A relatively nearby asteroid that circles the sun at a speed similar to the Earth’s would be ideal, Elvis reported. So far, six of 6,699 known near-Earth asteroids seem to have amenable orbits.

For many researchers, the visit will be a mini–Mars-mission — a chance to test strategies and equipment before traveling to the Red Planet. A round-trip journey to a nearby asteroid might take about half a year. A mission to Mars would take more than twice as long.

“If you want to climb Mount Everest, you don’t climb K2 first,” says astronaut and astronomer John Grunsfeld of the Space Telescope Science Institute in Baltimore. Practicing deep space maneuvers on a nearby asteroid would be like climbing Washington’s Mount Rainier before tackling the Himalayas.

To find their Mount Rainier, astronomers first need to map all the asteroids. Scientists have pinpointed many of those big enough to destroy the Earth, but a lot of the rocks smaller than a kilometer in diameter haven’t been identified, says planetary scientist Bill Bottke of the Southwest Research Institute in Boulder, Colo. Bottke recently coauthored a

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SN Today at **www.sciencenews.org**

National Research Council report outlining possible approaches to cataloging all asteroids near Earth.

Once the asteroids are tallied, selection criteria such as those proposed by Elvis can be considered. (Regardless of choice, it is unlikely that the asteroid will have enough gravity to allow a landing. Rather, astronauts would probably tether their spacecraft to the asteroid and move as it moves, possibly zipping to the rock in a smaller vehicle.)

Planetary scientist Paul Abell of NASA's Johnson Space Center in Houston says an asteroid's composition might also affect its desirability. Visiting an asteroid that holds water-ice, for example, might help astronauts figure out how to extract water for drinking and for fuel, a technique that could come in handy during pit stops on a long trip to Mars.

Of course, a crewed mission to an asteroid would garner rich scientific rewards in its own right. Visiting an asteroid "tells you about what existed back when planets were forming," Bottke says. Asteroids may host carbon-containing molecules, which could hold clues to the beginning of life on Earth. So far, scientists have gleaned much of their information about the early solar system from meteorites that have landed on Earth, but these samples lose a lot of material as they flame through the atmosphere, he says.

Though robots have successfully landed on two asteroids so far — Eros and Itokawa — people could accomplish experiments that robots couldn't. "Having humans in the mix gives you a lot of flexibility," Abell says. A human with a hammer could pick up a rock and then choose to discard it in favor of a more intriguing rock somewhere else.

But having "non-artificial intelligence," as planetary astronomer Andrew Rivkin of Johns Hopkins University's Applied Physics Laboratory in Laurel, Md., puts it, doesn't mean a thing unless the astronauts survive the trip. Keeping them safe on a long flight to an asteroid, as well as to Mars, will pose new challenges.

The NEAR spacecraft (artist's illustration shown) landed on Eros in 2001.

"Going to an asteroid is a new idea, but I don't think all of the complications have been thought through," Bottke says. "I think everyone's being a little cavalier about jumping on the bandwagon."

For instance, researchers will need to quantify the doses of radiation that astronauts will experience on the journey. An inopportune solar flare could be deadly, and the requisite protective shielding could be too heavy to carry.

If researchers can identify a flight plan that will keep astronauts healthy and safe, Earth's homebodies may be protected as well. Another hope — and another reason to probe a nearby asteroid — is that such a mission could uncover new ways to deflect or destroy a life-threatening rock careening toward Earth.

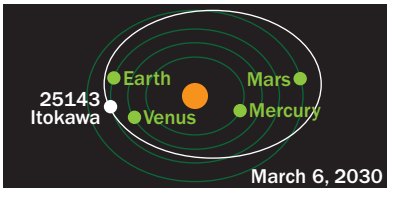
"A lot of the objects that we'll be able to get to as human beings are the ones that represent the greatest threat," Abell says.

The difficulties of the proposed visit are great, but the morale boost from accomplishing the mission is "powerful, if not tangible," Rivkin says.

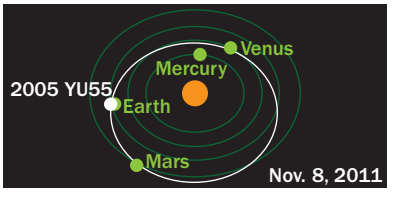
Adds Grunsfeld, who has been on five space flights: "This is about the bigger picture. It's the start of humans going out and exploring the solar system." ■

Back Story | ASTEROIDS TO WATCH

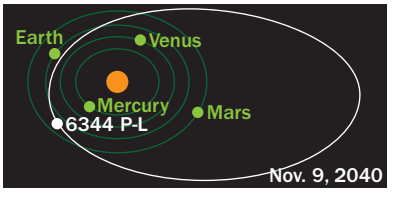
More than 6,500 asteroids are known to enter Earth's neighborhood. Of these, 1,100-plus are classified as "potentially hazardous" — meaning they can approach Earth relatively closely and have diameters larger than 150 meters. The orbits of a few of these asteroids are shown below.



25143 Itokawa grabbed public attention when it became the target of the Japanese Hayabusa mission, which launched in 2003, imaged the asteroid and attempted to collect soil samples. (The recovery capsule is expected to land in Australia in June.) The asteroid's next close approach will be in March 2030, when it will pass within 56.3 million kilometers of Earth.



Recent observations suggest asteroid **2005 YU55** is 400 meters long, twice as large as previously thought. The measurements were taken in April as the Arecibo telescope in Puerto Rico tracked the asteroid passing within 2.3 million kilometers of Earth. On its next approach, in November 2011, the body is expected to get much closer — a mere 325,000 kilometers away.



6344 P-L was first discovered in 1960, but then researchers lost track of it. The asteroid was rediscovered in 2007 and given the name 2007 RR9 before it was recognized. The asteroid has a highly elongated orbit that takes 4.7 years to traverse, and its next close approach to Earth will be in November 2040, when it will pass within 11 million kilometers.

FROM TOP: SPL/PHOTO RESEARCHERS, INC.; BACK STORY: NEO.JPL.NASA.GOV/NEO/PHA.HTML

What's the fastest way to learn a language?

ACT LIKE A BABY.

It's not the advice you'd expect. Learning a new language seems formidable, as we recall from years of combat with grammar and translations in school. Yet infants begin at birth. They communicate at eighteen months and speak the language fluently before they go to school. And they never battle translations or grammar explanations along the way. Born into a veritable language jamboree, children figure out language purely from the sounds, objects and interactions around them. Their senses fire up neural circuits that send the stimuli to different language areas in the brain. Meanings fuse to words. Words string into structures. And language erupts.

Three characteristics of the child's language-learning process are crucial for success:

First, and most importantly, a child's natural language-learning ability emerges only in a speech-soaked, immersion environment free of translations and explanations of grammar. Second, a child's language learning is dramatically accelerated by constant feedback from family and friends. Positive correction and persistent reinforcement nurture the child's language and language skills into full communicative expression. Third, children learn through play, whether it's the arm-waving balancing act that announces their first step or the spluttering preamble to their first words. All the conversational chatter skittering through young children's play with parents and playmates — "…what's this…" "…clap, clap your hands…" "…my ball…"— helps children develop language skills that connect them to the world.

Adults possess this same powerful language-learning ability that orchestrated our language success as children.

Sadly, our clashes with vocabulary drills and grammar explanations force us to conclude it's hopeless. We simply don't have "the language-learning gene." At Rosetta Stone, we know otherwise. You can recover your native language-learning ability as an adult by prompting your brain to learn language the way it's wired to learn language: by complete immersion. Our award-winning, computer-based method does just that. Dynamic Immersion® unlocks the innate language-learning ability you acquired before birth and mastered as a child. By recreating the immersion context in which you learned your first language, you understand, speak, read and write your new language with confidence and accuracy from the beginning — without translations and explanations. At every step and in every skill, you receive instant, actionable feedback, including speech recognition and analysis technologies that prepare you for everyday conversations. And Adaptive Recall® brings back material just when you need it to reinforce and perfect your learning.

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The northeast Pacific transient killer whale may be a distinct species.

One ocean, four (or more) species

New genetic analysis splits killer whales into multiple taxa

By Tina Hesman Saey

Determining whether animals belong to the same species is not as black and white as you might think.

Take killer whales. Scientists have long debated whether the ocean-dwelling mammals all belong in one species. Now, DNA evidence suggests that killer whales should be classified in at least four species, and maybe more.

Scientists once thought killer whales all belonged to the species *Orcinus orca*. But as researchers began observing more closely, they discovered that the whales seem to belong to different groups, called ecotypes, with distinct feeding habits and appearances. Killer whales from different ecotypes don't seem to breed with each other — one criterion for being classified as separate species. So some scientists proposed that killer whales should be grouped into different species.

Early genetic analyses didn't support that idea. Studies that looked at pieces of mitochondrial DNA, a type of genetic material that can be used as a molecular clock to measure the time since two genetic lineages split, concluded that the various killer whale groups are similar enough to fall into a single species.

But recently, researchers have come to realize that not all molecular clocks keep the same time. The mitochondrial DNA of Adélie penguins, for example, evolves faster than previously thought (*SN Online: 11/17/09*). Killer whales and other cetaceans, on the other hand, have molecular clocks that tick more slowly than other species' clocks do, says Phillip Morin, a marine mammal geneticist at the Southwest Fisheries Science Center in La Jolla, Calif.

Morin and colleagues analyzed the mitochondrial genomes of 139 killer whales from around the globe and found that the animals fall into several genetically distinct groups.

"The genetic data show that they are each independently evolving lineages," Morin says.

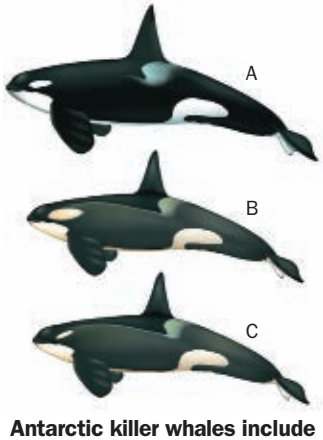
There is enough evidence to split off three new killer whale species, Morin and his colleagues propose in a study published online April 22 in *Genome Research*.

Two of the proposed new species live in the Antarctic. One eats only marine mammals, frequently knocking seals off pack ice to catch its prey. The other pursues fish underneath the pack ice.

A third proposed species lives in the northeastern Pacific and eats a variety of other marine mammals, including seals, sea lions, whales and dolphins. Morin's team estimates that this group diverged from other killer whales as a separate species about 700,000 years ago. The other groups split off more recently.

"I suspect there's going to be another four or five species," says Robin Baird, a cetacean biologist at the Cascadia Research Collective in Olympia, Wash. Killer whales seem to be evolving into new species based on their eating habits, he says, a scenario that is common among birds but extremely rare for mammals.

Not everyone is convinced, however. Mitochondrial DNA is passed from mothers to offspring, so it reflects only one part of a species's genetic heritage, points out A. Rus Hoelzel of Durham University in England. Going yet another step by analyzing the full DNA makeup of killer whales might reveal genetic exchange between killer whale groups that is not detectable looking exclusively at the maternal lineage. Exchange of genetic information between the groups would indicate that they aren't separate species.



Antarctic killer whales include the classic *Orcinus orca* (A) and two types that may be separate species: pack ice whales (B) and Ross Sea whales (C).

Morin agrees that a fuller genetic picture of killer whales is called for; he thinks the additional data will validate the finding from mitochondrial DNA that orcas should be classified into a handful of distinct species.

"My gut feeling is that these really are separate species, but the careful scientist in me wants to get a little bit more data ... to really nail it down," he says.

FROM TOP: DAVE ELLIFRIT/NOAA ALASKA FISHERIES SCIENCE CENTER; UKO GORTER

45 years | Estimated upper end of chimp life span in the wild

60 years | Estimated upper end of chimp life span in captivity

Chimps may sense others' deaths

Apes can react in intriguing ways when their companions die

By Bruce Bower

Pansy the chimpanzee died surrounded by friends and family who cared for her as best they could and reacted to her demise with silent somberness. Pansy's story, along with stories of two mothers unable to let go of their deceased infants, raises the possibility that chimpanzees know when a companion has died and realize that it will never return, two studies report in the April 27 *Current Biology*.

"Chimpanzees may have greater awareness of the finality of death than has previously been believed," says James Anderson, a psychologist at the University of Stirling in Scotland who studied Pansy's death.

Pansy's case gives a first glimpse of how chimps respond to the natural deaths of companions, he says. Video cameras in an indoor enclosure at a safari park recorded activity before and after Pansy died.

In the days before Pansy's demise three adult chimps, including her daughter, groomed her regularly. Grooming increased as Pansy's breathing became labored in the 10 minutes before death. After she died, a male pulled at Pansy's arm and tried to open her mouth. He charged in an aggressive display, pounded on Pansy's body and then ran off.

The next day, the three chimps watched silently as keepers removed the body. The chimps avoided sleeping on Pansy's deathbed for five days and, for a few weeks, were less active and ate less than usual.

"These incidents strengthen the inference that apes have some sort of conception of death," says William McGrew of the University of Cambridge in England.

Anderson says it may be more humane to allow elderly apes to die among companions in research facilities and zoos, rather than isolating them for treatment or euthanasia.

In the wild, chimps' reactions to a death vary greatly from one individual to another, much as in people, says Elizabeth Lonsdorf of Chicago's Lincoln Park Zoo. Old and sick chimps often find sheltered spots to die alone or are taken by predators, with the group resuming its daily foraging. "We don't know yet if chimps can grieve for the loss of a group member," Lonsdorf says.

> "We don't know yet if chimps can grieve for the loss of a group member."
> ELIZABETH LONSDORF

Uncertainty also surrounds the intentions of two female chimps that literally wouldn't let go of infants that died of infections in 2003, as described in another study. These chimps inhabited forests surrounding Bossou, Guinea. One, the mother of several other chimps, carried her 1-year-old on her back while foraging for 68 days. The other, a first-time mother, carried her 2-year-old in the same way for 19 days. Then the bodies were abandoned.

In both instances, tropical weather dried and preserved the corpses in a natural mummification process. Mothers groomed the dead infants' bodies. Over time, mothers increasingly let other group members handle and play with the bodies.

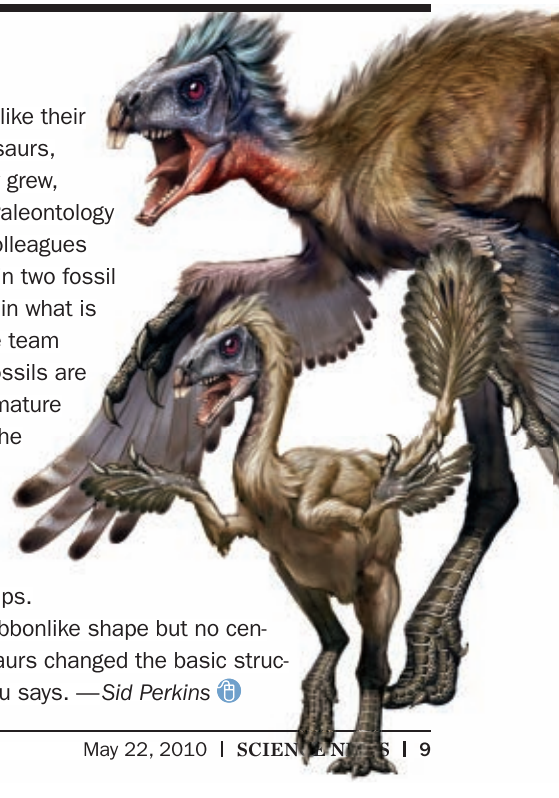
Dora Biro of the University of Oxford in England, who led the study, is cautious about interpreting the behavior as reflecting an awareness of death. "These mothers understood that there was something unusual about their infants," Biro says. "But whether for them that indicated that the infants would never come back to life remains a fascinating open question."

Ape mothers' refusal to let go of dead infants makes evolutionary sense, says primatologist Frans de Waal of Emory University in Atlanta. Close emotional ties to one's youngster prevent chimp mothers from prematurely abandoning sick and near-dead infants, in his view.

"Chimpanzees may know something of someone else's mortality, but we have no way of knowing whether they understand their own mortality," de Waal remarks.

Dino feather shift

Some young dinos sported a look totally unlike their elders, a new study shows. Feathered dinosaurs, like modern birds, may have molted as they grew, says Xing Xu of the Institute of Vertebrate Paleontology and Paleoanthropology in Beijing. Xu and colleagues discovered an age-related shift in plumage in two fossil specimens of *Similicaudipteryx*, which lived in what is now China about 125 million years ago, the team reports in the April 29 *Nature*. While both fossils are juveniles, the larger and presumably more mature of the two—a dinosaur with a body about the size of a goose—had long feathers on the forelimbs and tail that look like modern bird feathers. But in the smaller pigeon-sized creature, the feathers on the forelimbs and tail look modern only near their tips. Closer to the body, those feathers have a ribbonlike shape but no central shaft. Unlike today's birds, these dinosaurs changed the basic structure of their feathers during adolescence, Xu says. —*Sid Perkins*



XING LIDA, SONG QIJIN

Environment

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Reeling in tuna with lower mercury

Level of toxic metal depends on species, two studies find

By Janet Raloff

The way it boosts neural development in babies and protects the hearts and minds of adults, tuna could be considered health food. Yet mercury found in the fish can erase these advantages and even trigger profound mental and cardiovascular harm. Two recent papers now suggest how diners with enough information about the fish they're buying might be able to negotiate the mercury minefield and tap tuna's dietary benefits.

Mercury content can vary dramatically between tuna species, depending on size. Bigger fish tend to accumulate more of the toxic metal. Yet groceries and restaurants in the United States do not typically identify fresh tuna by species.

Jacob Lowenstein of Columbia University and his colleagues used genetic analyses to identify the species of fish in 100 samples of sushi tuna from 54 restaurants and 15 supermarkets in New York, New Jersey and Colorado, tracing the samples to bigeye, bluefin and yellowfin tunas. As the researchers had anticipated, mercury tended to increase with fish size from yellowfin to bluefin.

A couple of samples from supermarkets and roughly 15 from restaurants exceeded the 1 part per million level that gives the U.S. Food and Drug Administration legal authority to remove a product from the market, Lowenstein's group reported online April 21 in *Biology Letters*. Owing to the high mercury content in some samples, the team recommended that health agencies consider adding bigeye and bluefin tuna to lists of fish that should be avoided by especially vulnerable groups such as young children and pregnant or nursing women.

Shawn Gerstenberger and his colleagues at the University of Nevada, Las Vegas also went shopping for tuna, but they sought out a more prosaic form of this fish: canned. The toxicologist and his team bought 155 cans of solid-white, chunk-white and chunk-light tuna from three of the most popular national brands.

The team found that average mercury concentrations in all three brands exceeded 0.5 ppm, and the average for one brand was more than 0.7 ppm. Depending on the brand, 4 to 7 percent of samples surpassed FDA's 1 ppm action level.

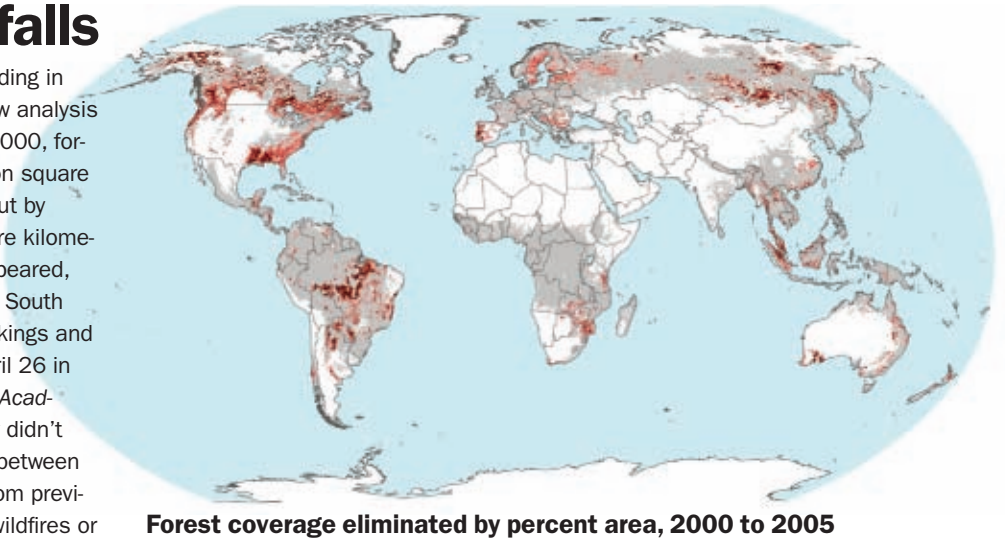
Mercury levels in some tuna could be high enough to erase its health benefits.

In the February *Environmental Toxicology and Chemistry*, the team reported that light tuna offers the better choice for mercury-conscious diners. Its contaminant level averaged 0.28 ppm mercury, versus 0.5 ppm in chunk-white tunas. White tuna consists of albacore only; light is mostly skipjack.

At the 0.5 ppm level, a 25-kilogram (55-pound) child can safely eat only one serving of white tuna every two weeks, the researchers calculated. The data suggest that "stricter regulation of the mercury in canned tuna is necessary," Gerstenberger says.

When a tree falls

About 3 percent of forests standing in 2000 were gone by 2005, a new analysis of satellite images reveals. In 2000, forests blanketed almost 33 million square kilometers of land worldwide. But by 2005 more than 1 million square kilometers of those forests had disappeared, geographer Matthew Hansen of South Dakota State University in Brookings and his colleagues report online April 26 in the *Proceedings of the National Academy of Sciences*. The new study didn't measure the spread of forests between 2000 and 2005, so recovery from previous natural disasters such as wildfires or storms wasn't included. —*Sid Perkins*



FROM TOP: RASIMON/ISTOCKPHOTO; HANSEN *ET AL.*/*PNAS*

Matter & Energy

1 yoctometer — One-millionth the diameter of a quark

23 yoctoseconds — Half-life of a hydrogen-7 atom

1.7 yoctograms — Resting mass of a proton

Measuring the teensiest push

Supercold beryllium atoms feel 174-yoctonewton force

By Laura Sanders

A blob of cold beryllium ions has measured the smallest force yet. The charged atoms registered the minuscule tug of an electric field to be 174 yoctonewtons. That's about equal to the force of Earth's gravity on a 70-kilogram person divided by a million, then by a billion, then by a billion again, then by four.

"The forces measured are astonishingly small," says quantum physicist Dick Slusher of the Georgia Institute of Technology in Atlanta. Slusher, who was not affiliated with the study, calls the research "wonderful work."

The measurement technique, developed by researchers at the National Institute of Standards and Technology in Boulder, Colo., may lead to more precise ways of analyzing the fine-grained surface properties of materials.

The team first trapped about 60 beryllium ions with electric and magnetic fields and then used lasers to cool the atoms to about half a millikelvin. At such cold temperatures, the ions are very sensitive to the slightest perturbations and form the cores of "exquisitely sensitive force detectors," the researchers led by Michael Biercuk, now at the University of Sydney, report online at arXiv.org.

Next, the researchers hit the vulnerable ions with a force, delivered in the form of an electric field. The field bumped into the ions, changing their movement. As the ions moved, laser light that the team bounced off the ions changed wavelength. This telltale shift was detected using a technique called Doppler velocimetry, allowing the tiny force to be calculated. (Cops' radar guns use the same basic principle to measure car speeds.)

Adopted in 1991 as the smallest prefix in the International System of Units, known as the SI system, "yocto" means one part in a million billion billion, or 10^{-24}.

Derived from the base "octo" (for the 8th power of 10^3), yocto got its "y" to prevent its abbreviation from being "o," and thus confusable with the number zero. Yocto, and its gargantuan partner yotta, for 10^{24}, are currently the smallest and largest recognized SI unit prefixes, although a group led by University of California, Davis students is pushing for formal recognition of "hella" to mean 10^{27}.

Many researchers are no strangers to such small forces, says Dietrich Leibfried, also of NIST in Boulder. But until now, "people never really cared to write down how small such forces are," he says.

Humans

To hear how one speaker shortens the word *yesterday*, visit **www.sciencenews.org/yeshee**

Practice. Dream. Improve. Repeat.

Nap-time reveries may be the brain training itself to do better

By Bruce Bower

People who have nap-time dreams about a task they've just practiced get a memory boost on the task upon awakening, Harvard researchers report online April 22 in *Current Biology*.

Memory-fortifying brain processes during sleep actually cause dreams, the paper's authors propose, as a structure called the hippocampus integrates recently learned information.

That's a "tempting speculation," says physiological psychologist Jan Born of the University of Lübeck in Germany.

In the study, 99 college students age 18 to 30 spent 45 minutes navigating a virtual 3-D maze on a computer. They were instructed to remember the location of a particular tree in the maze, then given a five-hour break. For the first 90 minutes of the break, students were assigned either to take a nap or to engage in quiet activities such as watching videos.

In a second try at the maze, nappers who reported dreaming about the maze task — four out of 50 — found the tree much faster than they had in initial trials. These individuals described dreams such as seeing people at particular locations in a maze or hearing music that had played in the lab during testing.

A 90-minute snooze mostly involves non–rapid eye movement, or NREM, sleep. Previous studies have found links between brain activity during NREM sleep and better learning by rats and people. Neural activity sparked by recent learning has not been observed during rapid eye movement, or REM, sleep, which often includes especially vivid and bizarre dreams. The researchers now plan to examine whether people who have REM dreams about a maze task during a full night's slumber navigate that maze better the next day.

Wha'yuh mean, wha'm I sayin'?

Phonetic shortcuts in daily speech often go unnoticed

By Rachel Ehrenberg

BALTIMORE — Talk may be cheap, but that doesn't keep people from budgeting their speech.

When casually conversing, people routinely streamline their utterances by dropping segments, syllables and even whole words, researchers reported April 20 at the spring meeting of the Acoustical Society of America. Insights into these conversational shortcuts could improve foreign language instruction and help scientists design better speech recognition programs, which are typically attuned to carefully enunciated words rather than everyday talk.

"Most of the speech we communicate with is not careful speech at all," said Natasha Warner, director of the Douglass Phonetics Lab at the University of Arizona in Tucson. "You try and give this stuff to a speech recognition program, and it totally goes to pieces."

To parse the spontaneous speech of everyday life, Warner and her colleagues had 13 undergraduate students sit in a sound booth, each with a recording microphone on one ear and a telephone on the other. The researchers recorded the students having 10-minute conversations with friends or family members. The same students were also recorded while reading a list of words and while reading a story in which particular words were embedded.

The researchers then analyzed several aspects of the students' speech, such as how long consonants lasted, the presence or absence of "bursts" (when the lips come apart, as in "apple") and the duration of vocal cord vibration.

The standout findings were not what was said during casual conversation, but what wasn't.

The standout findings were not what was said during casual conversation, but what wasn't. "Reduction is the norm, not the exception," said Warner. "It's massive — syllables are gone."

For example, when a speaker uttered the phrase "We were supposed to see it yesterday, but I felt really bad," the word "yesterday" shrank considerably. When said carefully, the word is three distinct syllables. But within a spoken sentence it is often reduced to a two-syllable, barely identifiable "yesh-ee."

The research pertains not just to understanding regular speech, but to creating it as well. Speech-generating devices and therapies for people who can't speak because of conditions such as amyotrophic lateral sclerosis might also be aided by the research, said Sandra Combs of the University of Cincinnati, who studies speech disorders.

Warner added that this natural speech isn't lazy, it's just more efficient — as long as a listener can still understand. And while the study focused on undergraduates, this efficient speech isn't just for the young. Warner's research was prompted in part when she noticed herself dropping sounds while asking her son if he wanted a peanut butter and jelly sandwich (the "t" sound in butter tends to disappear). "I thought, if I'm doing that," Warner said, "how often does it happen, and what does the listener do?"

Atom & Cosmos

"The idea is to observe all of the sun all of the time." —PHILIP SCHERRER

Satellite spots some sun secrets

First images released from Solar Dynamics Observatory

By Ron Cowen

There's plenty new on the sun, both inside and out, a recently launched spacecraft has discovered.

After staring at the sun for only a few weeks, NASA's Solar Dynamics Observatory has already recorded the interplay between a small sunspot on the surface and disturbances high in the sun's outer atmosphere. The Earth-orbiting observatory has also made the first high-definition recording of a solar eruption over a broad range of ultraviolet wavelengths.

Unlike several other orbiting solar observatories, Solar Dynamics looks at the sun's entire disk in high resolution, taking images about once every second over a wide spectrum of wavelengths.

"The idea is to observe all of the sun all of the time, so you can see what happened before an important event occurs," says Philip Scherrer of Stanford University, lead scientist on the observatory's helioseismic and magnetic imager. "We haven't been able to do that before."

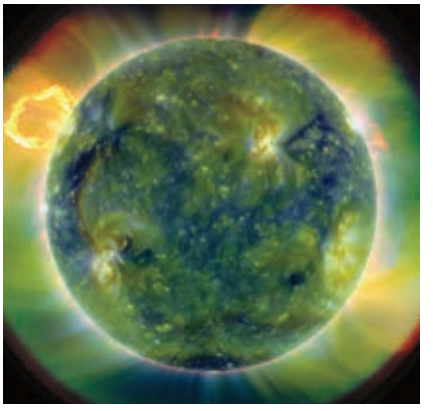
On April 21 NASA released some of the first images from the craft, launched on February 11. It is expected to collect data for five years, including pictures tracking the tangle of magnetic fields that drive solar activity and the roiling gases revealing patterns of acoustic waves reverberating beneath the solar surface.

An ultimate goal of the observatory is to better predict and understand the origin of giant eruptions, called coronal mass ejections, in the sun's outer atmosphere. When directed at Earth, these billion-ton parcels of magnetized gas can disrupt electric power grids and satellites.

On March 30 the observatory captured one of those eruptions as it lifted off the sun. Images reveal that the disturbance's magnetic field contains a twist, a feature that some solar physicists believe is necessary to initiate a solar eruption.

Observations of the sun's magnetic field before the eruption should settle the ongoing debate about whether the twist is necessary to start the eruption, says solar physicist Spiro Antiochos of NASA's Goddard Space Flight Center in Greenbelt, Md. A key advance, he adds, is the observatory's ability to simultaneously observe both the magnetic field at the sun's visible surface and the detailed structure of its outer atmosphere, or corona, at several temperatures.

Images taken April 8 show disturbances in the corona around the time that a small, roughly Earth-sized sunspot disappeared. It's likely that the magnetic field from the sunspot drove the activity in the corona, scientists say.



The Solar Dynamics Observatory captured this ultraviolet image (shown in false color), revealing a magnetized loop of gas lifting off the sun's surface.

FROM TOP: NASA; NASA, ESA, M. LIVIO AND THE HUBBLE 20TH ANNIVERSARY TEAM (STSCI)

Hubble's instant classic

With a smorgasbord of celestial targets to choose from, it wasn't easy picking a portrait to celebrate the Hubble Space Telescope's birthday. But after a debate that began last year, Hubble astronomers finally settled on taking a new, close-up portrait of part of the Carina nebula, a star-forming region that Hubble first captured in 2007 with a less sophisticated camera. "We wanted to have an image that will be at least as spectacular as the iconic 'Pillars of Creation,' " says Mario Livio of the Space Telescope Science Institute in Baltimore, referring to a 1995 Hubble image of the Eagle Nebula. "This particular image can arguably be called 'Eagle Nebula on steroids,' " says Livio, who led the new observations. NASA released the image two days before the 20th anniversary of Hubble's launch on April 24, 1990 (*SN: 4/10/10, p. 16*). The image, which shows starlight eroding pillars of gas and dust, highlights the newly installed Wide Field Camera 3's capabilities, Livio says. — *Ron Cowen*

Genes & Cells

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Genes don't tell whole story of MS

Twin study points to a still unknown environmental trigger

By Tina Hesman Saey

A new study has come up empty-handed in the pursuit of a genetic explanation for why one identical twin develops multiple sclerosis while the other does not.

Researchers transcribed the complete genetic blueprints for a pair of identical twins, looking for differences that might explain why one has multiple sclerosis and the other is healthy. No trace of what caused the discrepancy appeared in the twins' DNA. And comparisons of gene activity levels in the sick and healthy twin, and in two other sets of twins, found no smoking gun either, the team reports in the April 29 *Nature*.

"We looked under a lot of rocks, and we found no differences that we could replicate," says Stephen Kingsmore, a geneticist at the National Center for Genome Resources in Santa Fe, N.M., and leader of the new study. The finding "points to some novel environmental trigger that must be very important to the disease. We don't know what it is."

But the new study is small; it examines only three pairs of twins and one type of T cell, an immune cell known to be involved in multiple sclerosis. A telling difference between sickness and health might be found in other types of cells, such as immune cells called B cells or in oligodendrocytes, which make the nerve cell insulation called myelin, says Esteban Ballestar of the Bellvitge Biomedical Research Institute in Barcelona. "They are closing a door here, but I think, perhaps, the door should be open," he says.

In multiple sclerosis, the immune system attacks and damages the myelin sheath that helps speed electrical communication between nerves, the equivalent of scraping the coating away from an electrical wire. The damage results in pain and symptoms such as loss of coordination and vision.

In the new study, Kingsmore and his colleagues determined the entire genetic makeup of immune cells called T cells from a pair of female twins. One of the women developed multiple sclerosis at age 30 while her twin remained healthy. The twins are now old enough that the healthy one is not likely to develop the disease.

Identical twins share the same genetic makeup, and the researchers confirmed that both women carried variants of genes already known to increase the risk of getting multiple sclerosis. Scientists had thought that maybe the sick twin had developed an additional mutation in her DNA that finally triggered the disease. But the team found no such mutations.

Another way to rev up the immune system and induce it to attack the body is to increase the activity of certain genes. Upping gene activity doesn't necessarily involve changing the genes themselves, but can be done by altering chemical tags on the DNA. In the three pairs of twins, the team examined about 2 million DNA locations that had been tagged with a common label, known as a methyl group, that keeps gene activity in check.

In a previous study, Ballestar's group found lower levels of methylated DNA when comparing people with lupus *(SN: 1/16/10, p. 13)*, also an autoimmune disease, to their healthy identical twins. In the new work, Kingsmore and his colleagues found no such differences that could account for one twin getting MS.

The team also measured overall gene activity in three sets of identical twins, including the sisters who had their genomes sequenced. The researchers did find some minor differences, but none could explain why one twin got sick and the other didn't. ■

SCIENCE/AAAS

Scientists bag frog genome

Frogs have hopped onto the list of organisms that have had their genetic coding unraveled. A study in the April 30 *Science* lays out the genetic blueprint of the Western clawed frog, *Xenopus tropicalis* (tadpole of the species shown). A larger cousin of *X. tropicalis*, called *X. laevis*, is a popular lab organism for studying development. But with a genome about half the size of *X. laevis*', the Western clawed frog has easier DNA to decipher, says Uffe Hellsten of the Department of Energy Joint Genome Institute in Walnut Creek, Calif. Analysis of the Western clawed frog's genome shows that versions of 80 percent of genes that have been linked to disease in humans also turn up in these frogs. Researchers hope that the genome sequence will help reveal the molecular mechanisms behind amphibians' high sensitivity to hormones and other toxins, as well as offering clues to what's driving the worldwide decline of the animals. —*Tina Hesman Saey* ■

Body & Brain

96 percent Five-year survival after breast cancer diagnosis with tumor 13 to 17 millimeters diameter

66 percent Five-year survival after breast cancer diagnosis with tumor 48 to 52 millimeters diameter

Marker protein could help doctors spot breast cancer in early stages

In study, EGFR appeared up to 17 months before disease

By Nathan Seppa

High concentrations of a protein already implicated in several cancers may be an early warning sign of undiagnosed breast cancer, a new study suggests. The study found women who eventually developed breast cancer had elevated levels of the compound compared with those who didn't develop the disease, scientists reported April 20.

Although the marker isn't a sure indication of incipient breast cancer, it might eventually help doctors diagnose the disease early, said study coauthor Christopher Li, a physician and epidemiologist at the Fred Hutchinson Cancer Research Center in Seattle who presented the findings. "There is considerable investment in trying to discover this disease while it is most treatable," he said.

> "There is considerable investment in trying to discover this disease while it is most treatable."
> CHRISTOPHER LI

Li and his colleagues analyzed blood samples from 688 women who developed breast cancer while participating in a large medical trial and from 688 others in the trial who remained healthy. The two groups were matched for race, ethnicity and age.

The blood samples had been drawn up to 17 months before the cancer patients' diagnoses. The researchers focused on EGFR, or epidermal growth factor receptor, a protein on a cell's surface that can trigger pro-growth activity in cells, including proliferation, survival and migration.

In a subgroup of 396 women, those with the highest levels of EGFR were nearly three times as likely to develop breast cancer as women with the lowest levels. Among women who were taking estrogen and progesterone for menopausal hormone therapy at the time blood was drawn, those with the highest EGFR levels were nine times as likely to develop breast cancer.

The new work shows that subtle changes in the protein population in the blood may indicate the presence or increased risk of otherwise hidden tumors, Li said. But the test for elevated EGFR only modestly distinguishes between people with breast cancer and those without. "We don't believe EGFR will be a stand-alone marker," said Li. "Our goal would be to compile a panel of such markers."

Because EGFR has already been linked to several cancers, doctors sometimes order testing for the marker when treating people who have already been diagnosed with cancer of the breast, lung, colon, pancreas or other tissues. Those tests can help determine whether specific drugs that are based on antibodies to EGFR are likely to be effective.

Using biomarkers like EGFR as predictors of cancer remains a possibility, but the science needs to be further refined, said David Solit, a medical oncologist at Memorial Sloan-Kettering Cancer Center in New York City. "As a diagnostic," he said, "this is in a very early stage."

As more biomarkers show value, doctors might check them in high-risk populations, such as women with a family history of breast cancer. High EGFR, in combination with other predictors, might push a doctor to look harder, order a biopsy or repeat biomarker tests at regular intervals to see if the scores change.

MEETING NOTES

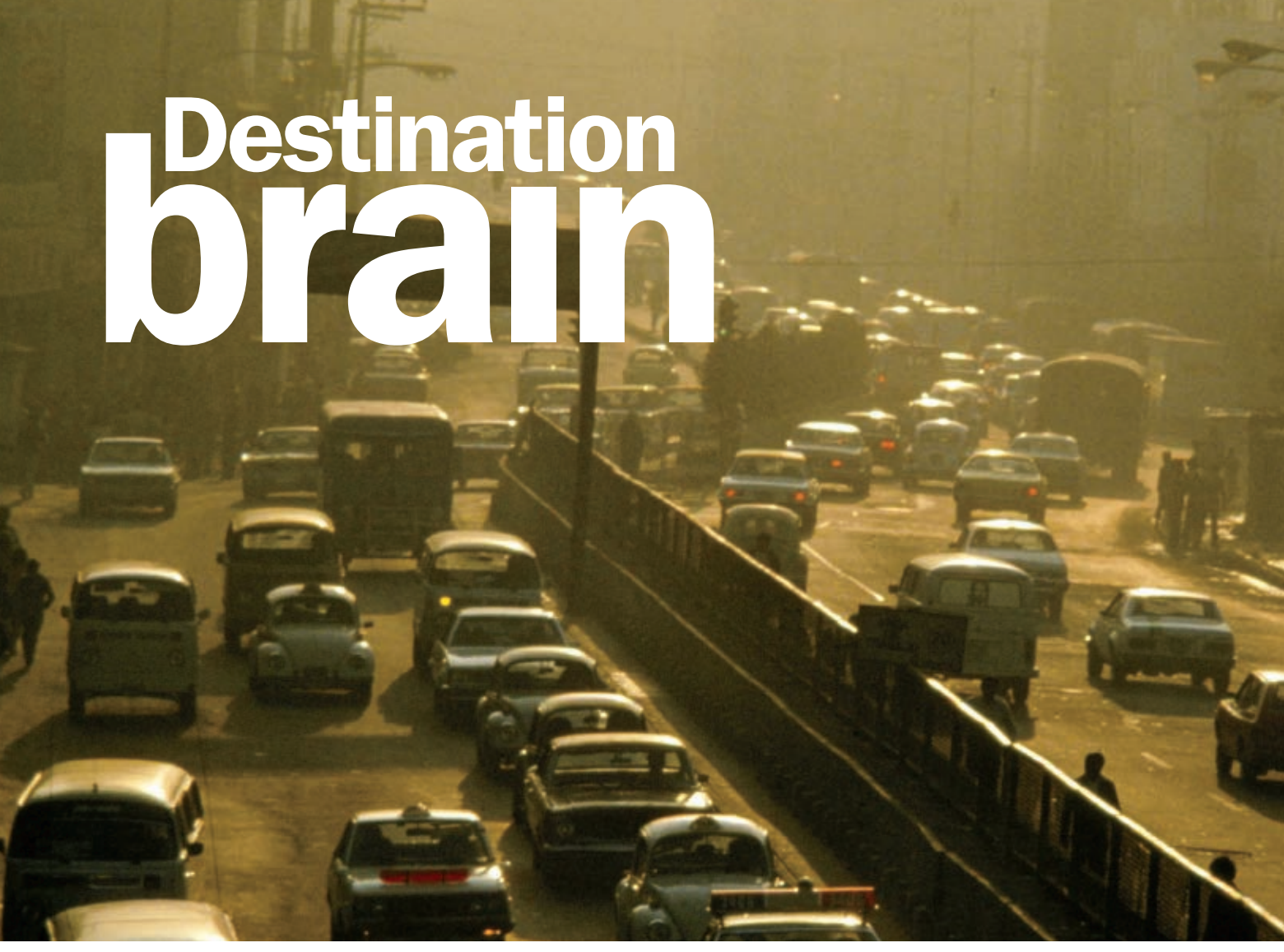
Colorectal cancer risk linked to bacterial infection, inflammation

Chronic stomach infection or inflammation in the digestive tract may raise a person's risk of colon cancer and might even serve as an early warning sign of the disease, according to two studies presented April 19. One study, conducted by researchers at Howard University in Washington, D.C., found that blacks over age 40 who carried stomach infections of the ulcer-causing bacterium *Helicobacter pylori* were more likely to have colorectal polyps, which can be precancerous. In the other study, researchers at Vanderbilt University in Nashville, the National Cancer Institute in Bethesda, Md., and the Shanghai Cancer Institute found that Chinese women with high levels of C-reactive protein, an indicator of inflammation, were more likely to have colon cancer than those with lower levels. Past research has linked chronic infection and inflammation to risk for other cancers, so the new studies suggest that "colon cancer is the next one knocking on the door," said medical oncologist William Nelson of the Johns Hopkins Kimmel Cancer Center in Baltimore. —*Nathan Seppa*

BATTLE trial finds benefits in custom lung cancer treatment

A new study suggests that treatments tailored to a patient's particular type of lung cancer may boost survival. Phase two of the BATTLE trial evaluated four chemotherapy treatments in 300 patients whose tumors had been biopsied and tested for genetic mutations and other molecular changes. Each drug's comparative success depended on the specific type of changes. —*Tina Hesman Saey*

Destination brain



Inhaled pollutants may inflame more than the lungs

By Janet Raloff

When Lilian Calderón-Garcidueñas recruited children for a study probing the effects of air pollution, Ana was just 7. The trim girl with an above-average IQ of 113 "was bright, very beautiful and clinically healthy," the physician and toxicologist recalls.

But now Ana (not her real name) is 11. And after putting her and 54 other children from a middle-class area of Mexico City through a new battery of medical and cognitive tests, Calderón-Garcidueñas found that something has been ravaging the youngsters' lungs, hearts — and, especially troubling, their minds.

Brain scans and screening for chemical biomarkers in the blood pointed to inflammation affecting all parts of the brain, says Calderón-Garcidueñas, of the National Institute of Pediatrics in Mexico City and the University of Montana in Missoula. On MRI scans, white spots showed up in the prefrontal cortex. In the elderly, she says, such brain lesions tend to denote reduced blood flow and often show up in people who are developing dementias, including Alzheimer's disease.

In autopsies of seemingly healthy Mexico City children who had died in auto accidents or other traumatic events, Calderón-Garcidueñas uncovered brain deposits of amyloid-beta and alpha-synuclein, proteins that serve as hallmarks of Alzheimer's and Parkinson's diseases. Several years earlier, she had found similar abnormalities in homeless Mexico City dogs and exaggerated versions of the abnormalities in local 20- to 50-year-olds.

She has published studies linking the insidious changes to the metro region's air quality. The area's 20-plus million inhabitants make it one of the world's largest megacities, a roughly 7,000-square-kilometer region choking with smog and particles containing carbon, metals and more (*SN: 9/8/07, p. 152*). Most are nanoparticles — too small to see but just the right size to migrate into tissues throughout the body. Further clouding the air are solvents and other reactive gases — as well as toxins contributed by livestock feces.

Scientists have known that air pollution can impair airways and blood

STEPHANIE MAZE/GETTY IMAGES

Mexico City's air is choked with smog. But scientists are finding that another menace—particles too small to see—may pose a serious health threat.

vessels. The emerging surprise is what it might do to the brain. Increasingly, studies have been highlighting inflammation-provoking nanopollutants as a potential source of nerve cell damage.

Calderón-Garcidueñas has been correlating Mexico City's stew of air pollutants with a suite of symptoms in people of all ages. In March in Salt Lake City at the annual meeting of the Society of Toxicology, Calderón-Garcidueñas unveiled some of her latest data. At age 11, Ana shows persistent, growing brain lesions, the toxicologist reported. As do the other Mexico City children surveyed. They also exhibit cognitive impairments in memory, problem solving and judgment and deficiencies in their sense of smell compared with age-matched children from a cleaner city 120 kilometers away.

Other toxicologists at the meeting presented data, largely from animal studies, tracking the movement of billionth-of-a-meter–scale particles into the brain, where they triggered inflammation and abnormalities characteristic of Alzheimer's or Parkinson's.

Until recently, most air pollution toxicology has focused on impacts to the lungs and heart, observes James Antonini of the National Institute for Occupational Safety and Health's lab in Morgantown, W. Va. The challenge now, he says, is to identify which pollutants are harming the nervous systems of Ana and others who live in areas with particularly dirty air.

Fuzzy thinking

Mexico City is not the only source of real-world pollution that has been linked to mental impairments.

Ulrich Ranft and colleagues at the Environmental Health Research Institute at Heinrich Heine University in Düsseldorf, Germany, studied 400 or so highly functioning local women in their mid- to late 70s. Elderly women who lived within 50 meters of very busy streets exhibited poorer memory skills than did women of the same age whose homes were well removed from highly trafficked roadways, the team reported in the November 2009 *Environmental Research*.

The study turned up no similar link between cognitive scores and average levels of particles in the women's communities. That makes sense, Ranft says, because the levels of ultrafine motes emitted by traffic can be quite high along streets, "but drop off very fast, falling to almost background levels when you get just 100 meters away from the road."

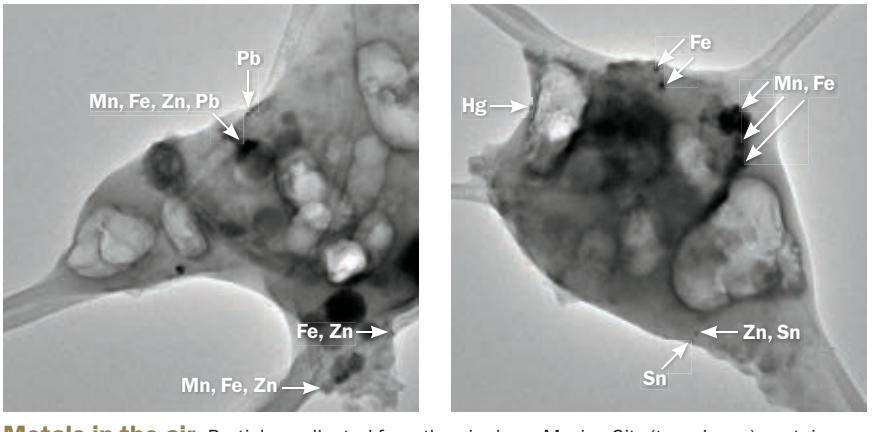
Young children's minds may be especially sensitive to tiny airborne particles spewed by traffic, according to Shakira Franco Suglia of the Harvard School of Public Health in Boston and her colleagues. In studies of roughly 200 Boston 10-year-olds, the researchers found that those living in areas with the highest average airborne concentrations of soot, a pollutant primarily associated with traffic, had lower IQs and lower scores on memory tests.

The team divided the kids by exposure levels into four groups. The average IQ drop between one group and the next averaged about three points—comparable to that seen in kids whose mothers had smoked during pregnancy, Franco Suglia's group reported in 2008 in the *American Journal of Epidemiology*.

Taking note of non-scents

A few studies, including the recent one by Ranft's group, have also observed a somewhat impaired sense of smell among people living in polluted regions.

At the toxicology meeting, Calderón-Garcidueñas reported that kids and young



Metals in the air Particles collected from the air above Mexico City (two shown) contain metals including manganese, iron, zinc, tin, lead and mercury (labeled above). When inhaled, these tiny pollutants can travel into the lungs and other parts of the body. New research suggests that the particles can end up in the brain, where they may cause inflammation.

KOUJI ADACHI AND PETER R. BUSECK/*ENVIRONMENTAL SCI. TECHNOL.* 2010

adults in Mexico City have a somewhat worse sense of smell than those living in cleaner cities. Roberto Lucchini of the University of Brescia in Italy reported much the same for adolescents living in communities around now-closed iron-alloy manufacturing plants. Both groups' data also turned up signs the youngsters have been experiencing at least subtle nerve damage.

The findings, the researchers say, are especially worrisome since a number of studies have shown that a sense of scents wanes in people developing Alzheimer's and Parkinson's.

Though metal pollution hasn't been confirmed as a cause of these diseases, Lucchini was able to link pollution in Brescia to reduced smelling abilities and to motor impairments.

Until 2001, alloy plants in northern areas of the province spewed a number of metals into the air. Manganese remains a substantial pollutant in the air, soil and house dust in this part of Italy. Work by Lucchini's team uncovered unusually high rates of symptoms including tremors, slowed movement and rigidity among adults living near the now-defunct plants. The local prevalence of these and other Parkinson's-like symptoms is about 400 per 100,000 inhabitants. That's two and a half times the usual rate in Italy.

Lucchini's team, which had already planned to examine 300 middle schoolers for neural effects of local pollution, included a smell assay in the tests. To measure exposures, the researchers collected blood and urine from the 11- to 13-year-olds. A third of the kids also carried a backpack fitted with an air-sampling device and a GPS to pair up readings and precise locations. Some children lived near the former alloy plants, others at Garda Lake, a relatively clean comparison region in the province.

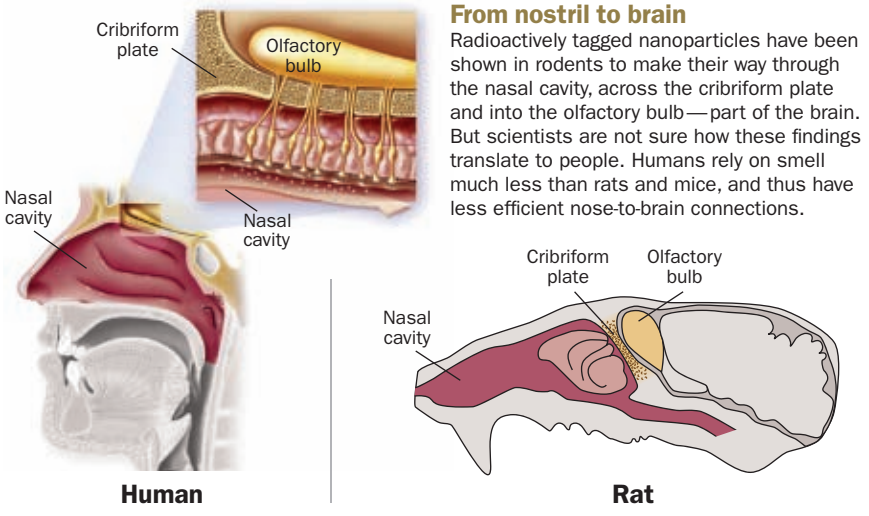
At the toxicology meeting, Lucchini reported that among kids living near the alloy plants, "Odor identification was clearly impaired compared to children living in the [Garda Lake] region." The smell-threshold reduction was "preclinical," he explains, meaning the children wouldn't notice the change but it could be picked up with testing.

His team also linked exposures to manganese-rich dust particles with motor impairments — such as a reduction in the speed at which children could clench their hands or sequentially touch the fingers of each hand to the thumb. Though it's too early to speculate about whether the symptoms will evolve into something resembling Parkinson's disease, Lucchini says, these are the first data to link such motor impairments to inhaled manganese.

Nosing out the problem

While these data are just coming in, a growing body of evidence suggests that nerves in the nose can provide a highway along which some inflammatory pollutants, such as metals, motor directly from the outside world to the brain.

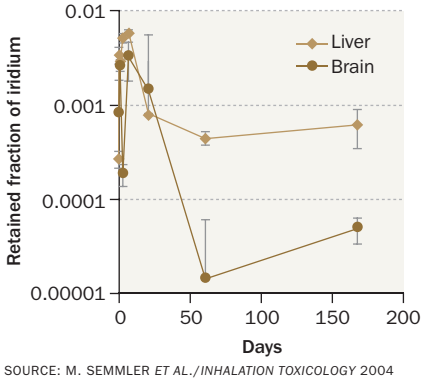
How efficient the conduit is varies by pollutant particle, according to new experiments by Wolfgang Kreyling of the Helmholtz Center and the German Research Center for Environmental Health in Munich. In rats, 20-nanometer–diameter agglomerations of at least 100 radioactively labeled iridium



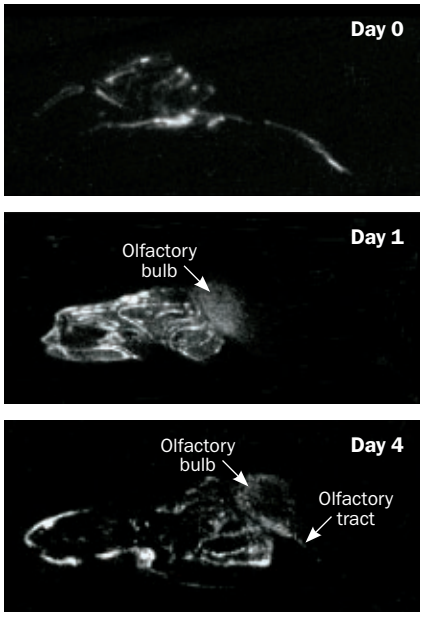
From nostril to brain Radioactively tagged nanoparticles have been shown in rodents to make their way through the nasal cavity, across the cribriform plate and into the olfactory bulb — part of the brain. But scientists are not sure how these findings translate to people. Humans rely on smell much less than rats and mice, and thus have less efficient nose-to-brain connections.

Traces of nanoparticles A growing body of research is documenting the type and amount of nanoparticles that can end up in a rodent brain. The images at right show that manganese particles can travel into a rat's olfactory bulb and olfactory tract by four days after inhalation. The chart below reveals that inhaled iridium particles can motor to the liver and the brain, where the particles remain detectable even after a few months.

Inhaled particles retained in rat organs



SOURCE: M. SEMMLER *ET AL./INHALATION TOXICOLOGY* 2004



SOURCE: D. DORMAN ET AL./*JOURNAL OF TOXICOLOGY AND ENVIRONMENTAL HEALTH* 2002

TOP: HUMAN: BSIP/PHOTO RESEARCHERS, INC.; RODENT: T. DUBÉ

particles entered the brain whether inhaled through the nose or pumped directly into the lungs.

By comparing what has been deposited after one-hour exposures via the two routes, Kreyling's team showed that for such small particles, two-thirds of what ends up in the rat brain comes directly from the nose, the rest via a more circuitous route that starts in the lungs, moves into the bloodstream and then goes to the brain.

Well under 1 percent of inhaled particles made it to the brain via either route, Kreyling reported at the toxicology meeting. However, he added, once those insoluble particles arrive in the brain, "we do not see much clearance." So continuous exposure over time could leave substantial amounts of inflammatory particles in the brain, he speculates.

Change the 20-nanometer iridium to same-sized soot particles and the uptake rate falls by 75 percent. Expose animals to 20-nanometer particles made from titanium dioxide or to 80-nanometer particles of iridium, and the rate of brain uptake drops by about 90 percent.

But no one's sure how well such studies model what happens in people, points out David Dorman of North Carolina State University in Raleigh. Long-snouted rodents depend far more than humans do on the sense of smell and have evolved a much bigger and more efficient system linking the outside environment to the brain. For instance, Dorman notes that half of the nasal cavity of a rat is lined with olfactory-system cells. In humans, this receptive area is much smaller, he says — "only about 3 to 5 percent."

His team has shown that even for particles that begin moving up the olfactory highway, some stop partway. One type that does seem to go the distance: manganese. When Dorman's group exposed rats to manganese, the same metal that taints the dust Lucchini has been studying in Italy, nearly all of the pollutant particles entering the nasal tissue migrated at least as far as the olfactory bulb, a structure in the brain.

Regardless of what percentage of

particles make it all the way, such data suggest that inhaled airborne motes can enter the brain, where they would be expected to foster inflammation, a primary underlying trigger of tissue damage and neurodegenerative disease.

Moreover, Calderón-Garcidueñas has linked the pollutants with a breakdown in the lining of the nose, which could facilitate particles' access to olfactory highways serving the brain.

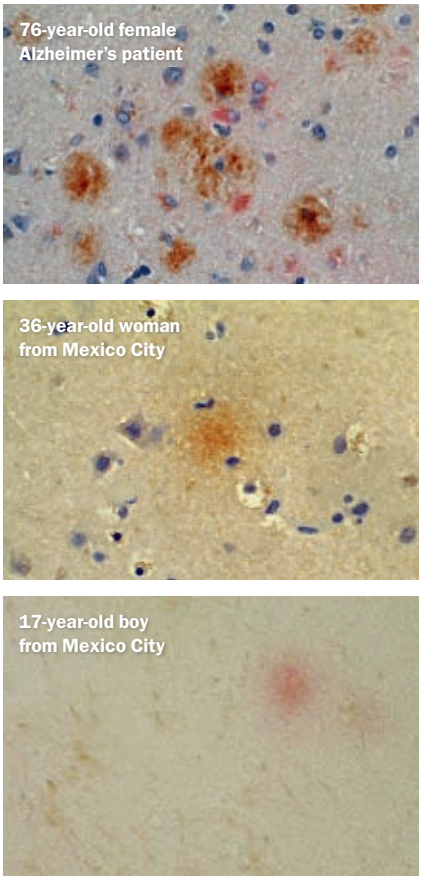
A burning issue

Although the source and chemistry of air pollutants affecting the brain differ, all seem to share the same toxic modus operandi: inflammation. Some pollutants turn on genes that release inflammatory chemicals, others call out immune cells that quash invaders and clean up trash using inflammatory mechanisms. Still more induce biologically destructive electron-stripping chemical reactions that won't quiet down without a copious release of antioxidants.

Krishnan Sriram, a neurotoxicologist with NIOSH, reported at the toxicology meeting that following 10-day and 28-week exposures to manganese welding-fume particles, rodents developed brain changes resembling many of those in Parkinson's patients — nerve-cell inflammation, tissue damage from oxidation and loss of nerve cells from a region of the brain that makes dopamine.

In addition, his team looked at some of the family of *Park* genes; mutations in these genes are associated with an elevated risk of developing Parkinson's disease. In rodents exposed to manganese, the researchers saw a reduction in the genes' production of proteins that normally help rid the body of misshapen nerves and that quash the oxidation responsible for excessive inflammation.

Bellina Veronesi of the U.S. Environmental Protection Agency and Lung-Chi Chen of the New York University School of Medicine laboratory in Tuxedo reported data at the toxicology meeting from mice exposed to dense concentrations of pollutants collected from outdoor air. Animals without functioning apolipoprotein genes, which normally help control the production and activity of certain fats in the bloodstream, experienced runaway brain inflammation and nerve damage. This finding suggests that properly working apolipoproteins may be essential for coping with tiny inhaled particles.



Unnerving signs Diffuse amyloid plaques (brown blotches), like those seen in Alzheimer's patients (top), have shown up in adults (middle) and children (bottom) from Mexico City. Though its unclear whether the damage is caused by air pollution, the findings worry some scientists who report that such plaques are not normally seen in young people living in clean cities.

People born with a particular apolipoprotein gene variant — known as *APOE-4* — face a greatly elevated risk of developing late-onset Alzheimer's disease and more general cognitive declines. In North America, Calderón-Garcidueñas says, roughly one-fifth of people carry this variant. And in Mexico City, she has found that children and young adults with the variant exhibit the most inflammation, the greatest cognitive declines and the most rapid deposition of amyloid-beta.

But Calderón-Garcidueñas has yet to prove that deposition in the brain of air particles primarily explains the brain inflammation she's measured, says Dorman. One has to wonder, he says, whether the "widespread nasal damage" that she depicted was a major contributor to inflammatory brain damage or independent of it.

Calderón-Garcidueñas is aware of the issue. She notes that work by others has shown that inflammation-provoking cells or chemicals have the potential to migrate from distant sites into the brain, triggering fallout damage there.

But whatever the source of inflammation in the brain, Calderón-Garcidueñas would like to see people who may face an elevated risk for pollution-triggered neural damage identified and counseled about lifestyle changes that could reduce that risk. For instance, people with the *APOE-4* gene variant might give up cigarettes, take low-dose anti-inflammatory drugs or find jobs that won't expose them to inflammatory agents, such as the endotoxin in chicken manure.

These people might also look to change their diet, eating foods rich in inflammation-limiting antioxidants, like brightly colored fruits and vegetables, or dark chocolate. She recently began feeding commercially available chocolate rich in polyphenols, a class of natural antioxidants, to treat inflammation ravaging the hearts and minds of mice.

The data are still preliminary, cautions Calderón-Garcidueñas. But from all appearances, she chuckles, the chocolate "works wonders!" ■

Explore more

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L. CALDERÓN-GARCIDUEÑAS

Wife Discovers Secret Office Romance... And She's Thrilled

Keep Your Secret Until After Dark When You Present the 14K gold-fused Midnight Sapphire Drop Necklace.

I should have seen it coming. The luxury business is full of temptation. After months of international travel and long, lonely nights in some of the world's most romantic cities... I was setting myself up for the fall. And I fell hard. When our designer showed me the ***12-Carat Midnight Sapphire Drop Necklace,*** it was love at first sight. After he told me we could offer it at ***only $79***, I knew this was more than just a fling.

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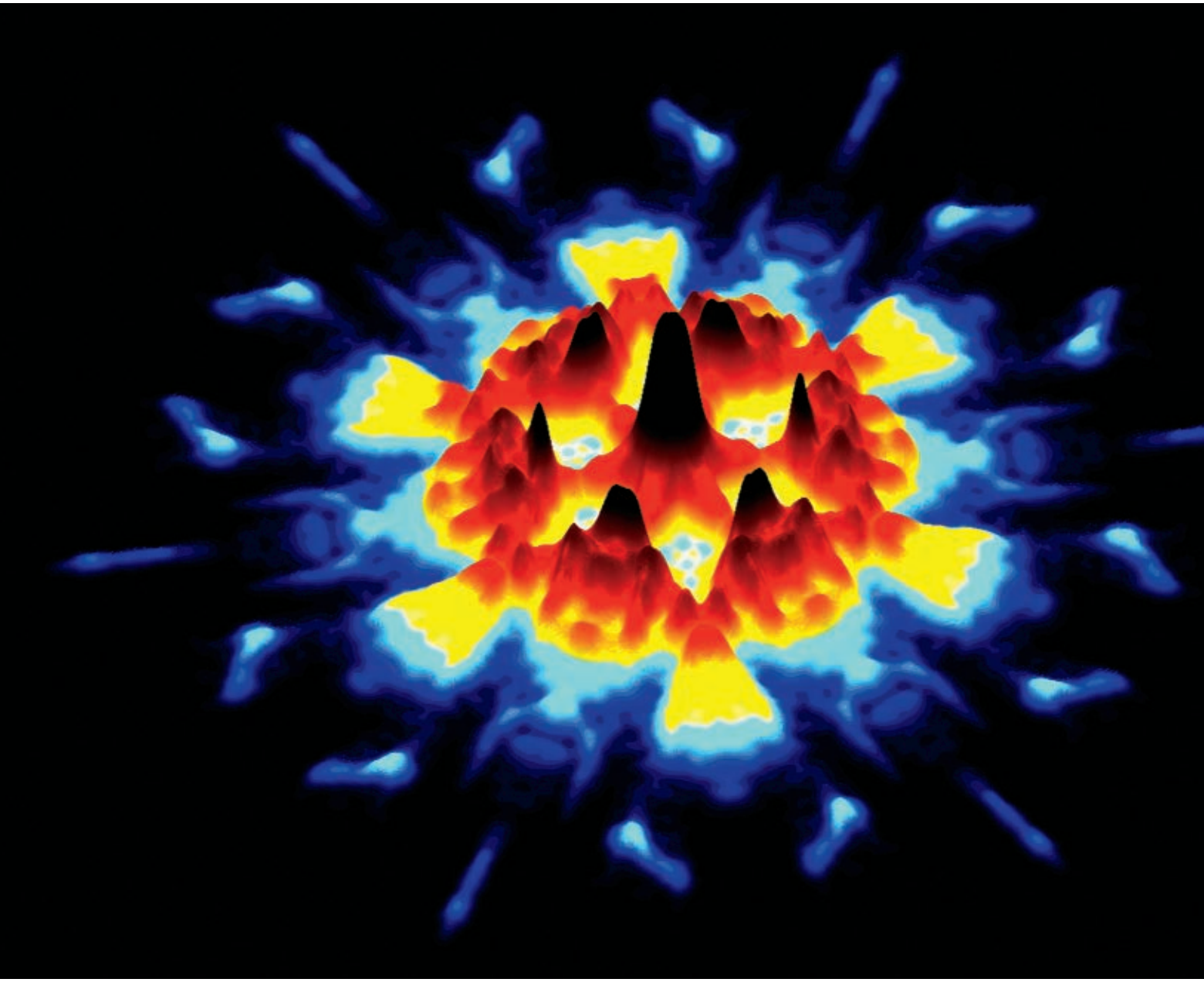


For the hottest thing in condensed-matter physics, check out the local liquor store. Hidden inside a device for chilling wine is the unusual compound called bismuth telluride.

For physicists, bismuth telluride does more than keep champagne frosty. Under the right laboratory conditions, this crystal can start behaving in weird and wonderful ways. Over the past couple of years, researchers have made several toast-worthy new discoveries involving bismuth telluride and other related materials, known as topological insulators.

These materials exhibit a split personality when it comes to conducting electrons. The bulk of the material is an insulator — in other words, it blocks the flow of electric current. But sometimes the surface can act as a conductor, shuttling electrons merrily along their way.

Just a few years ago, no one thought that materials could both insulate and conduct at the same time in this way. "This is a new state of matter — in condensed-matter physics this is the highest goal," says Shoucheng Zhang, a theoretical physicist at Stanford University. "It is such a beautiful thing."

Beyond their theoretical beauty, topological insulators might also one day prove practical in the electronics industry: Already researchers at Princeton University have made a topological insulator behave as a superconductor, transporting electrons without any resistance. And topological insulators might serve as a laboratory for creating and studying new types of particles never before seen in nature.

YAZDANI LAB/PRINCETON UNIV.

Physics on the edge

Electrons get moving along the surfaces of a new class of materials

By Alexandra Witze

Scientists are exploring new types of crystals, called topological insulators, that conduct electricity at their surface but not in their interior. The image at left reveals how electrons scatter on the surface of a bismuth-based material.

Topological insulators are "a new way of thinking of the states of matter," says Zahid Hasan, an experimental physicist at Princeton. "They're teaching us totally new physics."

From the top

The story of topological insulators begins with another one of physics's trendiest materials: graphene, a sheet of carbon atoms in a honeycomb pattern just one layer thick. Researchers have been keen to discover what unusual properties result from this atomic arrangement — a search that led to the discovery of a behavior called the quantum spin Hall effect.

Electrons moving through a material come in two flavors, called "spin up" and "spin down." These flavors refer to the electrons spinning like tiny tops in opposite directions, meaning that they have opposite angular momentum. In materials that exhibit the quantum spin Hall effect, electrons don't move in a crystal's interior but instead flow along its edges; electrons with spin up move in one direction, while electrons with spin down flow the other way. Such an orderly flow of electrons excited physicists, who thought they might be able to take advantage of the quantum spin Hall effect to build new kinds of electronic devices.

By 2005, theoretical physicist Charles Kane of the University of Pennsylvania in Philadelphia and a colleague had proposed that graphene could exhibit the quantum spin Hall effect, and they began pondering what other materials might do so. One crucial issue in this theoretical scenario was that graphene's spin-up and spin-down electrons were traveling right on top of one another — meaning the electrons might smash into each other.

Kane realized, however, that a mysterious feature of quantum physics can prevent such crashes by making the spin-up and spin-down electrons behave as if they move in separate, well-defined traffic lanes. "The lightbulb went off in my head," he says. "That's when I realized there was something really new about this."

His calculations showed that certain crystals would display these unusual edge effects with divided highways of flowing electrons. The electronic structures of these materials were closely related, but not identical, to graphene. Coaxing graphene to behave in this way required difficult-to-reach low temperatures and crystal purity. But new materials — those now known as topological insulators — provided a more feasible alternative.

Kane's initial paper, published with his Pennsylvania colleague Eugene Mele in *Physical Review Letters* in 2005, was the first recognition that topological insulators could exist. By 2007, a team led by Laurens Molenkamp of the University of Würzburg in Germany had observed mercury telluride behaving as a topological insulator in two dimensions in the laboratory. And

the following year, a group led by Hasan at Princeton found crystals of bismuth antimony operating as topological insulators in three dimensions. All of the materials behaved as insulators in the bulk, but as metals — conducting electricity — at their surfaces.

Soon papers were following fast and furious, laying out predictions for what materials might act as topological insulators and then reporting observations of that behavior. "Most of us were shocked by the rapidity of the experimental developments," says Joel Moore, a physicist at the University of California, Berkeley who helped coin the term topological insulators.

Into the lab

In part, scientists say, the field took off because topological insulators don't require complicated laboratory setups. Unlike other areas of quantum physics, which demand specialized cooling devices, powerful lasers or other expensive equipment, the study of topological insulators can be conducted at room temperature using materials found in many labs around the world.

Like bismuth telluride. That material is used in wine chillers for its thermoelectric properties, meaning it can convert a temperature difference to an electric current, and vice versa. Because of these properties, scientists had already been studying it in the lab. Along with its relatives bismuth selenide and bismuth antimony, bismuth telluride also turns out to be easy to work with. "Here is a bulk crystal you can hold in your hand, and it has this exotic state," says Robert Cava, a chemist at Princeton.

Bismuth telluride (representation of electrons' states shown) has become a popular material to study because it has unusual thermoelectric properties and is easy to use in the laboratory.

But researchers need to find just the right kind of crystal to observe topological insulator behavior. "Any old crystal will not do," Cava says. "We have to make them free from chemical and physical defects to make them work."

Any defects could interfere with measurements of the topological insulator properties — for instance, by causing electrons to move around within the crystal's interior, where it is supposed to be an insulator. That, in turn, makes it harder to detect the electrons moving on the surface.

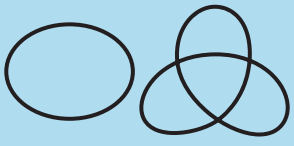
Cava and his Princeton colleagues have continued to work with bismuth-related topological insulators, and in February of this year reported a new feat with them in *Physical Review Letters*. By doping bismuth selenide with a bit of copper, the researchers were able to make it superconduct — a first for a topological insulator. Making topological insulators into superconductors could have big implications for various fields of physics, such as in efforts to build a quantum computer.

Beyond the bismuth compounds, other groups are hunting among various materials for signs of topological insulator behavior. In Germany, Molenkamp's group continues to work with mercury telluride, which is often used in devices that detect far-infrared radiation.

A different kind of knot

A branch of mathematics known as topology can shed light on which materials might function as topological insulators — that is, conduct electricity at the surface but insulate in the interior.

Objects are considered topologically identical if they can be transformed from one into another without cutting or pasting; the classic example is a doughnut and a coffee mug, which are topologically identical because one shape can be stretched into the other. In contrast, a simple loop and a trefoil knot (inset) are topologically distinct from each other, because one cannot be smoothly shape-shifted into the other.



But topology is more than just a way of describing the shapes of objects; it can also be used to describe how certain physical properties can't change smoothly. In the case of topological insulators, one such property is the "wave function," the equation that describes the behavior of every electron in a material. If the wave function of a free electron is viewed as a simple loop, then the wave function of a topological insulator is knotted, like the trefoil. Changing the electrons' wave function could turn a regular insulator into a topological insulator. — *Alexandra Witze*

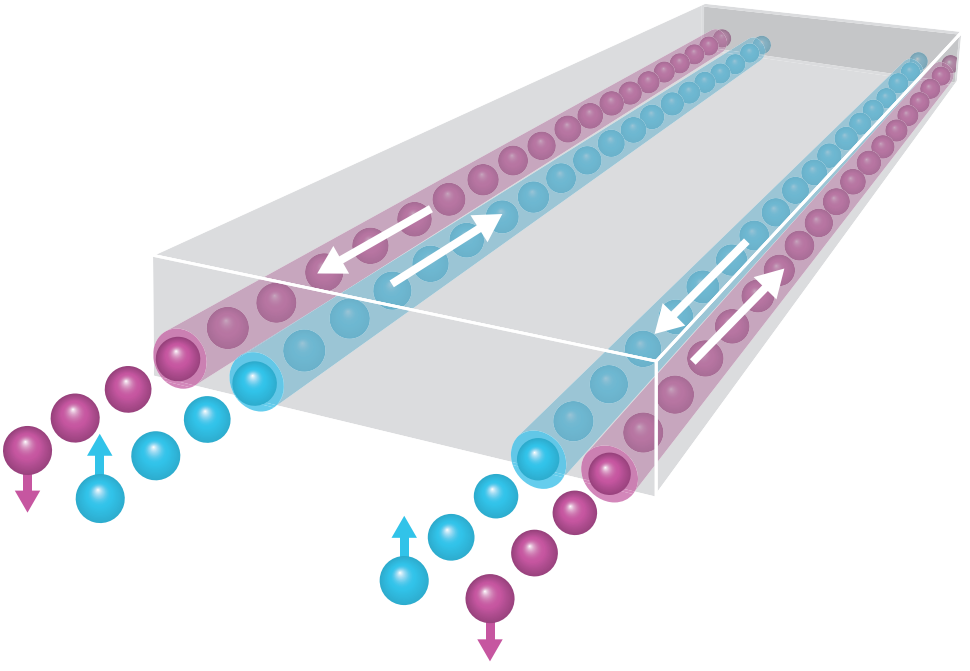
FROM TOP: COURTESY OF YULIN CHEN, Z.X. SHEN; DIANE39/ISTOCKPHOTO

Mercury telluride was the first topological insulator demonstrated in two dimensions and now, in preliminary work, Molenkamp thinks he sees a three-dimensional chunk of the material behaving the same way in the lab.

Theoretical papers have proposed many other classes of topological insulators, and at a recent meeting of the American Physical Society in Portland, Ore., Molenkamp and other researchers ran a tutorial for an overflow crowd on how researchers could enter the field and start finding topological insulators of their own.

As some researchers look for new kinds of topological insulators, others are playing around with slicing and dicing the existing ones to see how that changes their electrical behavior. At Stanford, for instance, Yi Cui and colleagues have made tiny "nanoribbons" out of bismuth selenide. The long thin strands have a much higher surface-to-bulk ratio than a chunk of bismuth selenide, so Cui and his colleagues have a lot more of the conducting edge states to watch than they otherwise would.

At the physics meeting in Portland, the researchers reported that they can observe electrons zipping around and around the nanoribbons. Similarly, a team led by Qi-Kun Xue of Tsinghua University in Beijing has been laying down thin sheets of topological insulators atop one another to see how that changes their behavior.



Spinning in line Electrons at the surface of a topological insulator travel in well-defined traffic lanes depending on a property called spin. Spin-down electrons (purple) and spin-up electrons (blue) move in opposite directions in their designated lanes, permitting charge to flow at the surface of the material even though the interior of the material does not conduct electricity.

Where the wild things happen

Within the next few years, physicists expect a raft of new discoveries — not only toward making practical devices, but also toward probing some fundamental and exotic physics.

For instance, putting a topological insulator right next to a superconductor could create an unusual state at the interface in which unseen particles could pop into existence and be observed. These include the long-sought Majorana fermion, a particle that can act as its own antiparticle and has never been observed directly.

The proximity of the topological insulator and the superconductor would set up a kind of vortex — a quick-and-dirty way to get a single Majorana fermion, researchers say. "It would be the RadioShack way to make Majorana fermions," says Moore of UC Berkeley. Among other things, these fermions could potentially be used to store information in a quantum computer, bypassing some of the challenges that bedevil other proposals for storing quantum information.

Other researchers think that they might be able to use topological insulators to spot a different, never-before-seen particle called the axion. The axion is one of the candidate particles that could constitute dark matter, the unseen stuff that makes up 85 percent of matter in the universe. Astronomers can't see dark matter but know it must exist because of the gravitational effect it exerts on other matter, and they have been hunting for more direct signs of its existence.

In a paper published online March 7 in *Nature Physics*, Zhang's group at Stanford, with colleagues at Tsinghua University, argue that the math describing how topological insulators behave in an electromagnetic field is the same as the math that describes how axions move in that same field. So topological insulators could create conditions for elusive particles like axions to pop into existence, says Zhang. The insulators could serve as a sort of "baby universe" on a tabletop, he speculates, allowing the creation, manipulation and study of particles that physicists wouldn't otherwise have access to.

As exotic as these scenarios sound, they may not be that far in the future, scientists say. And with the pace of research into topological insulators picking up dramatically, additional unexpected findings may be just over the horizon. No matter what the next few years in the field brings, says Cava, "an exciting period is ahead." ■

Explore more

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T. DUBÉ

I, Mold

Conquering the rising tide of infection is hindered by the many similarities between humans and fungi

By Laura Beil

Common in house dust, *Aspergillus fumigatus* is harmless to healthy people. But for those with weakened immune systems or respiratory problems, inhaling the fungus' spores can lead to infection. The tiny spores (blue) cover the fruiting bodies, shown in this false-color scanning electron micrograph.

In the germ world, fungi usually lack the flair of viruses or bacteria. To people with normal, healthy immune systems, a fungus will rarely show itself — even though you carry around a microscopic film of fungus on your hair and skin, and take in invisible clouds of fungal spores with each breath. While many other microbes prefer to make a living through disease and death, a fungus is often content to wait for its host to die of something else.

In fact, throughout the history of civilization fungi have mostly been humans' friends, providing the bounty of bread and beer, recycling trash and enabling plants to extract nutrients from the soil. Scientists estimate that roughly 1.5 million species of fungus inhabit the Earth, but only a handful are capable of causing human disease.

Problem is, when they do, fungi can be remarkably lethal: For example, about half the patients who develop serious infections from the fungus *Aspergillus* will not survive. The mortality rate for the most common fungal infection in hospitals, candidiasis, has been reported to be just as high — and though numbers are hard to come by, reports suggest overall fungal infection rates have been on the rise. Doctors have also recently become concerned about a once-rare infection from the *Cryptococcus* fungus spreading in the Pacific Northwest (*SN Online: 4/22/10*).

Understanding what transforms a fungus from pal to pathogen has occupied researchers for more than a century. Yet scientists have only recently discovered some key principles that govern how fungi operate, and that allow a normally peaceful fungus to turn against people. In trying to decode the molecular conversation between microbe and human host, fungus explorers have also found some surprising secrets about the human immune system.

"We're now starting to see studies that rival anything else done before them," says William E. Goldman, a microbiologist at the University of North Carolina at Chapel Hill. Since 2004, when the complete genetic blueprint of *Candida albicans* was published, researchers have cataloged the genomes of about a dozen species of fungus that cause disease. Studies of these genomes may soon reveal how fungi survive in people's bodies — and suggest new ways to extinguish the germs. Scientists have also recently discovered families of molecules on human immune cells that alert the body to the presence of fungi and other invaders, as well as mechanisms that allow a fungus to evade those cells.

Taken together, these findings may soon solve one of the most challenging aspects of treating fungal infection: how to get rid of the germ that is your closest relative.

In old biology classes, fungi were lumped with plants, presumably because both forms of life could sprout from dirt. Today, fungi are recognized as their own kingdom, a diverse group of organisms that live in the inky depths of the ocean, the sub-zero snows of Antarctica and the forgotten apple in the refrigerator.

Fungi include mold, yeast, mushrooms and other growths that don't make energy from chlorophyll and light. To reproduce, many fungi shed microscopic spores, each one capable of propagating. Even if you don't see fungi, you live with them daily. A 2005 study found that about a million spores are nestled in your pillow alone.

Fungi are usually the vultures of the ecosystem, preferring food that is almost or already dead. (*Aspergillus*, for example, usually hangs around rotting leaves and compost piles, feasting on decaying matter.) But sometimes, when conditions are right, a fungus starts to germinate while its host is still among the living. In people, this generally leads to troublesome, but not fatal, infections of the skin and nails.

Fungal attack Cases of serious fungal infections are not tracked nationally, but a variety of studies and hospital-based reports suggest that infections have increased over the past 30 years and that they often prove fatal.

U.S. estimated prevalence and mortality

Fungus	Cases per 100,000 people	Mortality rate
Candida	10–24	40% to >50%
Aspergillus	5	45% to >80%

SOURCE: J. PERLROTH *ET AL./MEDICAL MYCOLOGY* 2007

"Most fungal pathogens are pretty wimpy," Goldman says. "They are not very good at causing disease in normal hosts with normal immune systems."

But a growing population of people have not-so normal immune systems. Fungal infections are so deadly in part because most patients who become seriously ill are already weakened by AIDS, cancer, transplants or medications that handicap the body's ability to mount a strong defense. More and more of these patients have taken high doses of antibiotics to prevent other infections, fundamentally changing the body's ecology and allowing unnatural fungal growths to take over. More patients are also undergoing medical procedures that breach normal immune barriers with catheters and other devices.

While relatively rare a generation ago, candidiasis — a blood infection from the fungal genus *Candida*, which normally lives on the skin — has become the fourth most common infection acquired in hospitals. Although infections from *Aspergillus* are not carefully tracked, studies suggest that the number of deaths quadrupled during the 1980s and '90s.

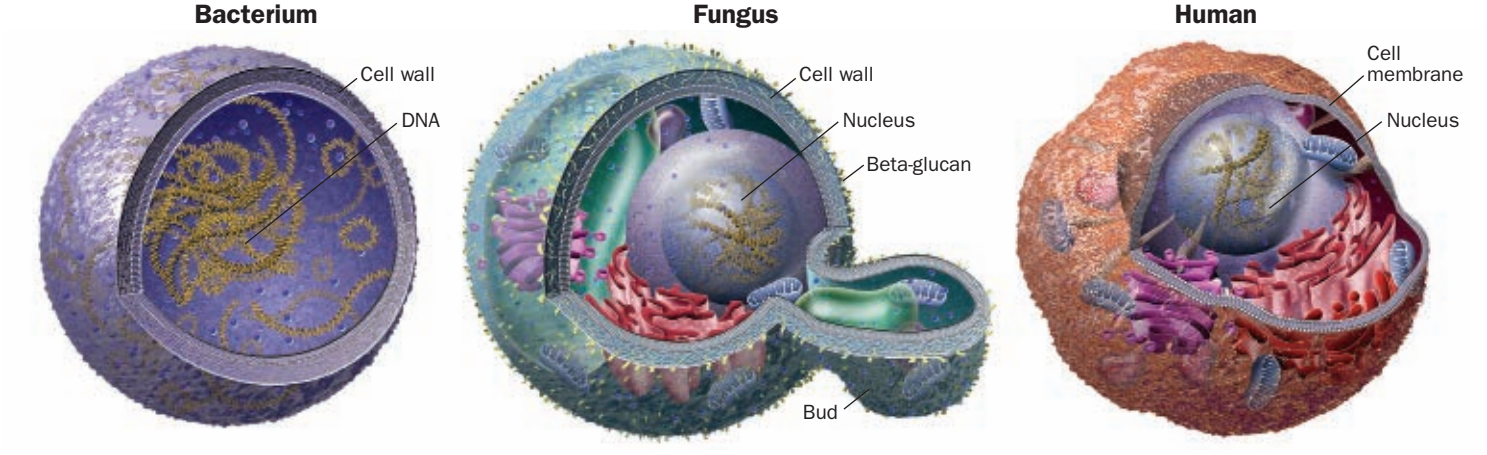
Drugs to treat fungal infections have been difficult to develop because fungi share many properties with people; from an evolutionary standpoint, fungi are closer to the animal kingdom than any other form of life. If you put mushrooms on your pizza, the mushrooms have more in common with you than with the tomato sauce. Fungi are also much more closely related to humans than are viruses and bacteria, which makes attacking fungal infection a tricky business.

Unlike other kinds of germs, both people and fungi are eukaryotes — among other commonalities, their cells have a nucleus, and the nucleus has its own membrane. In fact, fungal cells are so much like animal cells that much about the basics of human life has been gleaned from studies of baker's yeast.

To fight infection, antimicrobial drugs often exploit some molecular difference between an invading organism's cells and human cells. But with fungal treatment, human tissue is more likely

JUERGEN BERGER/PHOTO RESEARCHERS, INC.

Cell-by-cell comparison On a cellular level, fungi have more in common with humans than they do with bacteria. The yeast *Candida albicans* shares as many as half of its protein-coding genes with humans. A thick cell wall and beta-glucan molecules are two features that fungal cells (a yeast cell, reproducing by budding, is shown) possess that human cells don't. These differences could be exploited in new treatment strategies.



to find itself in the line of fire. Although more modern antifungal drugs are less harsh than their predecessors, one of the first widely used antifungals — amphotericin B — had a reputation for being highly toxic.

"When we are evolutionarily so similar, it's hard to get drugs that target fungi alone," says Bruce Klein of the University of Wisconsin–Madison. Drugs that treat bacterial infections often aim for molecules in the bacterial membrane. However, if drugs attack fungal membranes, the treatments often hit human cells too.

Fungal targets

There are, however, distinctions between human and mold. Most notably, fungal cells enclose themselves in a tough outer wall that shields them from abrupt changes in moisture and temperature.

"The fungal cell wall is the major difference between us and them," says Stuart Levitz of the University of Massachusetts Medical School in Worcester. "But it can be their Achilles' heel. It's what protects them in the environment, but also what flags them as being different."

The most important building blocks of this wall, at least to the immune system, are large glucose-based molecules called beta-glucans. In recent years, researchers have begun to compile a laundry list of receptor molecules on the surfaces of human immune cells that recognize and interact with the beta-glucan molecules from fungi. Receptors act as gatekeepers, linking the outside of a cell to its internal workings. By examining these receptors, researchers can eavesdrop on the molecular crosstalk between fungi and people.

Studies have found that just after fungi enter the body, defense relies mostly on "innate immunity" — a general, shotgun-like immune response that enlists certain types of white blood cells to find and destroy invaders. The other type of immunity, "adaptive immunity," takes longer to kick in, involving specialized infection-fighting white blood cells known as T cells and the production of antibodies that confer long-lasting protection against a specific target.

While humans produce plenty of antifungal antibodies, innate immunity is thought to be the first responder against a fungus. This basic defense mechanism is found throughout the animal kingdom; even horseshoe crabs protect themselves from fungi using innate immunity.

Scientists speculate that one reason fungi don't cause as much human disease as other microbes is because "our innate immunity has evolved very, very well so we're able to recognize and respond to fungi by a variety of different mechanisms," says Levitz. "Possibly as a consequence of that, the fungi have not evolved to become significant human pathogens to the extent that bacteria, parasites and viruses have." (Plants have not been so fortunate; despite plants and fungi's long and close interaction, fungi are significant plant pathogens that spoil about 10 percent of the world's harvests each year.)

Among the most important type of proteins that recognize fungi are the toll-like receptors, so named because they resemble a similar fruit fly molecule called toll. As receptors, they switch on when they encounter proteins from fungi and bacteria, setting off other reactions inside the cell. A team of French researchers reported in 1996 in the journal *Cell* that flies with mutations in the gene for a toll receptor were unusually vulnerable to infection with *Aspergillus*. In human white blood cells, two toll-like receptors in particular — TLR2 and TLR4 — appear to be involved in the body's fungus-fighting ability.

In 2008, scientists from the Fred Hutchinson Cancer Research Center in Seattle helped demonstrate the fungus-fighting role of TLR4 in a study in the *New England Journal of Medicine*. The researchers examined patients who had received bone marrow transplants and later developed *Aspergillus* infections. In general, about 10 to 15 percent of transplant patients will develop the life-threatening condition aspergillosis, but it's not clear why the other 85 to 90 percent of patients escape unscathed.

The researchers discovered an inherited gene that causes a malfunctioning

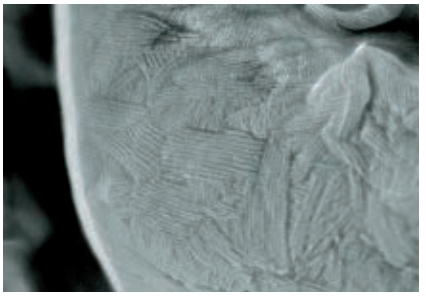
NICOLLE RAGER FULLER

TLR4 in the patients who had become ill. Without a normal TLR4, the scientists proposed, these patients' immune responses may have been weakened. Genetic testing for this mutation among blood stem cell donors may one day identify patients who need special care or attention following a transplant, the authors pointed out.

Two other reports in the *New England Journal of Medicine* last year described genetic flaws that caused increased susceptibility to fungal disease, confirming the role of other receptors in fungal protection. One involved dectin-1, a receptor first recognized as key to fungal defense in 2001. Dectin-1 partners with the TLR receptors to produce substances that both attack fungi and deploy other white blood cells to help fight infection.

Last October, an international team of researchers described genetic studies of one family in which otherwise healthy women seemed particularly prone to chronic *Candida* ("yeast") infections of the vagina, fingers and toes. The researchers found an inherited genetic alteration that led to a defect in dectin-1.

A second team investigated another family whose members were prone to recurrent, and sometimes fatal, infections of *Candida*. A team led by University College London researchers found a different inherited mutation that made a person vulnerable to fungal infection. When dectin-1 detects the fungus, it sets off a chain reaction that gets immune cells in battle mode. A mutation can interfere with one link in that chain, a molecule called CARD9, the researchers found. In this case, dectin-1 was triggered correctly, but the mechanism jammed farther down the line.



SOPHIE PARIS *ET AL./APPLIED AND ENVIRONMENTAL MICROBIOLOGY* 2003

Rodlet fibers form a pattern on an *Aspergillus* spore — and conceal the fungus from the immune system.

While these and other discoveries have brought new understanding to how immunity works, much about how the human body handles its relationship with fungi remains unclear.

"The thing that's most on my mind is how these organisms can manage to survive and proliferate in such a close relationship with host cells without triggering alarms," Goldman says.

Stealth spores

Goldman's work focuses largely on *Histoplasma*, which can cause lung infections. These fungal spores grow inside white blood cells called macrophages — innate immunity cells assigned the job of destroying invading organisms like fungi. "Here is an organism that gets inside the very cell that's supposed to be destroying them," he says.

While the stealth tactics deployed by *Histoplasma* remain largely a mystery, scientists recently reported that *Aspergillus* may dodge the immune system by borrowing a tool from Harry Potter: a cloak of invisibility.

Though each cubic meter of inhaled air may contain a thousand or more *Aspergillus* spores, the immune system doesn't seem to notice. Scientists had been unclear why. Then, writing in August 2009 in *Nature*, researchers from the Pasteur Institute and elsewhere offered an explanation: The body's immune system can't react to the spores because the immune system doesn't know they are there. Normally, the spores are coated with a thatching of small fibers called the "rodlet layer." In experiments with mice, the researchers found that the fibers alone do not excite the immune system. However, when researchers stripped the rodlets from the outside of the cells, the exposed spores invoked a robust immune response.

It appears that the rodlet layer may allow the fungal spores to hide in the body, waiting until conditions are favorable to germinate (such as death). The researchers also noted that when *Aspergillus* spores start to grow, the outer coating disintegrates and the immune system kicks in.

Scientists have recently discovered other deceptive feats. In the March 18 *Nature*, researchers revealed that members of a fungus genus that attacks plants are capable of passing off genes to one another — a lateral handoff thought to occur almost exclusively in bacteria. The discovery means fungi that develop genetic resistance to a drug treatment could theoretically share that secret with neighboring organisms.

The shocking thing, says study coauthor Michael Freitag of Oregon State University in Corvallis, was the ease with which fungi traded genes. Freitag and his colleagues simply put genetically distinct samples of the fungi side by side on a petri dish, incubated them together and tracked genetic movement. "It's not that far removed from what would occur in natural conditions," Freitag says. "I was surprised it would work that well."

While no one knows whether other species of fungus are capable of sharing genes so readily, the findings reinforce the idea that nothing about fungi should be underestimated. To succeed in conquering infections, the next generation of treatment will need to hit several targets at once.

Freitag likens current antifungal treatments to the cancer treatments of the '60s and '70s, most of which were designed to target cells that grew rapidly, and not cancer cells specifically. Today, doctors have a number of drugs that can zero in on the specific defects of a malignant cell, and physicians prescribe drug cocktails that try to disable several mechanisms simultaneously. Fungal infections appear to have similar complexity, including sharing properties with nontargeted cells, and will require treatment just as sophisticated, Freitag says. "Just like now we can attack very specific targets in cancer," he says, "we are going to have to do that with fungi." ■

Explore more

- For more information about fungi and disease, visit www.doctorfungus.org

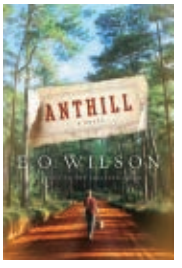
Anthill: A Novel

E.O. Wilson

Connoisseurs of fine fiction had a shock in January when the *New Yorker* published a short story in which all the characters were ants. Now the greatly anticipated source of that excerpt has appeared as a full-length novel, with humans as characters too.

Mostly the book follows Raphael Semmes Cody, who grows up in the fictional Nokobee County, a place inspired by real longleaf pine forests near Mobile, Ala. Wilson, a renowned Harvard ant expert and Pulitzer Prize–winning author, roamed those parts as a boy, and his deft details render the setting so vivid that the pine ecosystem becomes a character in its own right. The novel turns on the clash between a love for the last remnants of the South's once-great forests and the region's long-sought economic development.

When the tale digs into the ants' world, Wilson highlights the same themes of conflict and environmental challenge. He speculates about how ants view humans, but otherwise research-based reality provides drama enough — with ant tournaments, cemetery protocols and extreme soldiering. These ants don't sport cutesy names or break laws of biology by turning up as male workers. But, as Wilson reveals in the acknowledgments, the insect characters are a composite, allowing him to describe behaviors of several real species as a rich counterpoint to human society.



The novel quickly turns back to tales of humans, but the sections dovetail in tone. Wilson approaches humans too with a naturalist's eye, elucidating the behavior of species he knows well, including *Homo alabamensis* and *H. harvardii*. And in the end, the humans do match the ants for drama. — *Susan Milius*

W.W. Norton & Co., 2010, 378 p., $24.95.

The Private Lives of Birds: A Scientist Reveals the Intricacies of Avian Social Life

Bridget Stutchbury

As the outdoor reading season opens, Stutchbury's new, informal work on bird behavior just begs to be read under a backyard tree. The book could serve as beach reading too; marine birds such as the albatross and rhinoceros auklet put in appearances. But Stutchbury, a biologist at York University in Toronto, has done much of her research on songbirds, and tales of their behavior form the heart of the book.

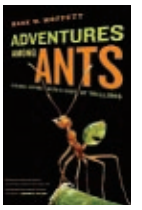
Stutchbury examines big issues in the family life of any species — courtship, kids, infidelity and so on — and describes relevant research projects. Some examples come from her own work with her husband, evolutionary biologist Gene Morton, and some from other scientists.

Behind-the-scenes details set the book apart from typical wildlife guides. In one vignette, Stutchbury recalls conveying nestlings to and from weighing sessions by climbing ladders while clenching paper bags of baby birds in her teeth. The book takes a conversational approach to research, yet Stutchbury packs in a good number of intriguing findings while presenting the science clearly.

Throughout, she discusses how human activity is changing the way birds manage life's challenges. At sewage-treatment plants in England, for example, earthworms reveling in sludge pick up traces of estrogen-mimicking chemicals that are passed along to starlings. In a surprise twist, the hormones give males unusually fine singing powers, possibly misleading females into selecting hormone-laden males as prime mates.

While outlining these problems, Stutchbury reminds readers that nature has remarkable resilience, if given a chance to recover. — *Susan Milius*

Walker & Co., 2010, 272 p., $25.



Adventures Among Ants: A Global Safari With a Cast of Trillions

Mark W. Moffett

A biologist and photographer (who earned his Ph.D. under E.O. Wilson) captures the hidden worlds of ants. *University of California Press, 2010, 280 p., $29.95.*

Saturday Is For Funerals

Unity Dow and Max Essex

Stories from Botswana bring to life the challenges of solving the AIDS crisis in Africa. *Harvard University Press, 2010, 218 p., $19.95.*

Blindspots: The Many Ways We Cannot See

Bruno G. Breitmeyer

A vision expert explores how the eyes, brain and thoughts work together to form mental images. *Oxford University Press, 2010, 266 p., $39.95.*

The Tuning of Place: Sociable Spaces and Pervasive Digital Media

Richard Coyne

Smartphones and iPods are changing the way people use public spaces, both real and virtual. *MIT Press, 2010, 330 p., $35.*

The Intimate Ape: Orangutans and the Secret Life of a Vanishing Species

Shawn Thompson

A journalist travels to Sumatra and Borneo to study the apes and interview scientists. *Citadel Press, 2010, 292 p., $14.95.*

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Making morphine

The article "Chemists pin down poppy's tricks for producing narcotic painkiller" (*SN: 4/10/10, p. 5*) may presage geopolitical changes in Afghanistan, regardless of whether there are engineered virus attacks or alternative crop programs. A technological advance like this one will eventually be used in the United States and Europe. Even if governments continue to treat morphine as a controlled substance, producing it domestically will trump the costs and difficulties of smuggling.
Terry Franklin, Amherst, Mass.

Gut sense

I've been vindicated ("Stomach's sweet tooth," *SN: 3/27/10, p. 22*). Ever since I first read about the links between diabetes and diet soda consumption, I have thought: "Artificial sweeteners were designed to be noncaloric and to taste sweet. They were not designed (nor tested) for glycemic responses in the digestive system, so perhaps there is good reason to expect them to affect insulin responses just as sugars do." As one who abhors the aftertaste from artificial sweeteners, I have no trouble suggesting that they should be avoided or reduced in diets just as much as natural sugars should. Our bodies are not evolved to regularly digest pure sugar nor to be challenged by sugar mimics.
David Adams, Garnet Valley, Pa.

If the stomach and other organs can taste sweetness, how valid are tests using sugar pills as an "inert" placebo?
William Davis, Seattle, Wash.

Feathery escape for dinos

Sid Perkins' article "Feathered dinosaurs, bold and in living color" (*SN: 2/27/10, p. 9*), in which he discusses why dinosaurs developed feathers, gives reasons such as "sending visual signals" to "startle an attacking predator" or to signal "come here, cutie."

Watching predators such as foxes and dogs try to catch chickens, it's easy to see a reason why feathers may have developed: When the bird escapes, it leaves a mouthful or paw full of feathers behind. My theory is that feathers are scales that became detachable. Fluffy features make it more difficult for an attacking animal to grab its prey. This is probably something the world has already thought of, but if not, could you pass the idea along?
Glynn Willett, Potomac, Md.

Missing nukes

After reading Secretary of Energy Steven Chu's comment in Scientific Observations (*SN: 1/2/10, p. 4*), I concluded that he has never heard of nuclear power.
Terrell Perry, Los Alamos, N.M.

Send communications to: Editor, Science News, 1719 N Street, NW, Washington, D.C. 20036 or **editors@sciencenews.org**. Letters subject to editing.

S. James Gates Jr.

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Confronting a third crisis in U.S. science education

Is science education broken in the United States? And if so, how should the country fix it? A working group of the President's Council of Advisors on Science and Technology (PCAST) has been investigating these long-standing questions and is expected to issue a report on its policy recommendations this month. Science News *Contributing Editor Alexandra Witze spoke with the working group's cochair, physicist S. James Gates Jr. of the University of Maryland in College Park. Gates also serves on the Board of Trustees of Society for Science & the Public, the parent organization of* Science News.

What is the outlook for U.S. science education?

If you look at U.S. performance on various international metrics, depending on which one you use, we come out something like 24th or 25th in the world. A lot of people might argue: "Well, who cares? It's just science." The only problem with that theory is we're moving into a time in the development of the world economy when innovation and the formation of novel approaches will clearly come from countries best situated to create a population that can innovate in science and technology.

We're not doing this because we want to make more scientists. The reason we are doing this with urgency is because it's connected to our country's future economy.

The Obama administration has announced a number of science education initiatives. Will they do enough?

I think the true test is yet to come. Does one put one's money where one's mouth is? To some substantial degree, this administration has stepped up to the plate with its increased support of science. On the other hand, we have heard concerns about sustainability of this commitment in light of current economic constraints.

How is PCAST approaching its deliberations about science education?

We're trying to be mindful of the tremendous number of efforts that have gone before; there are at least 40 to 50 such reports that one could list. We have found discussions in the literature all the way back to the '60s where people were raising issues of science and technology education.

When I look at the country's current crisis with regard to STEM [science, technology, engineering and mathematics] education, this is in fact the third such crisis.

The first one was World War II in my opinion. If you look at the way this nation prosecuted the war successfully, it was because the United States innovated at a level far beyond its competition.

Crisis two in my opinion was the launch of Sputnik. Once again there was a public resolution. You create NASA, our space program, and we successfully get men on the moon by 1969.

In this third STEM crisis, what we really need to do is start thinking in light of our previous experience. What might be policy structures that could bring to bear the kind of transformational and long-term vision to allow our nation to progress to higher levels of performance?

How do we do that?

My [PCAST working group] cochair [Eric Lander of the Broad Institute of MIT and Harvard in Cambridge, Mass.,] says the problem is that we in this nation do not have the structures that have allowed us to get inside the innovation cycle in education in the way that we have in scientific research.

One way to look at this problem is to look at education as a system to be engineered and to ask how one might do this. What I've been looking for is maps between how research works in this country and how education works in this country. In particular I have been struck by the fact that there is nothing like DARPA [the Defense Advanced Research Projects Agency] for education. You need something like that in the system to drive innovation like we haven't seen.

The reason we are doing this with urgency is because it's connected to our country's future economy.

Previous education has been mostly about the delivery of facts; you wanted people to remember and manipulate facts. But one thing that's different now is in a world that has a Web [and] access to information at your fingertips, the memory of facts won't be that important. What's going to be important is the capability of people to marshal those facts to solve the kinds of problems they're engaged in.

Are you optimistic about the country's future in science education?

I'm optimistic in the long term. There's lots of evidence that this country solves difficult problems, especially if you give us enough time. In the short term, I'm afraid it's going to be very painful. I fear that we may not be able to impress upon the larger society quickly enough that the issues around science, technology, engineering and mathematics are not tied to just those fields — that in fact this is the basis for our wealth formation. If we can't get that message out quickly enough, the nation's not going to respond quickly enough. ■

JOHN CONSOLI/UNIVERSITY OF MARYLAND

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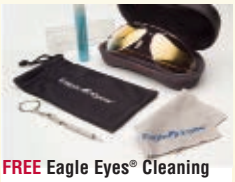

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