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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ JULY 3, 2010

Life Reloaded

In search of origins, biologists try to build a cell

Gulf Oil Spill's Deep Impact

Was Feynman Wrong about Ratchets?

A Bunyanesque Molecule

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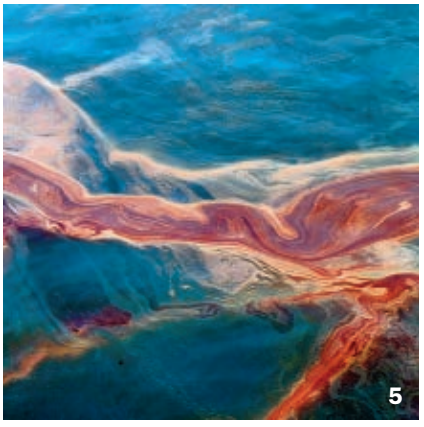

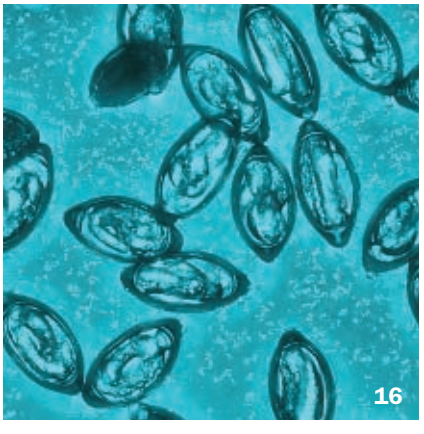

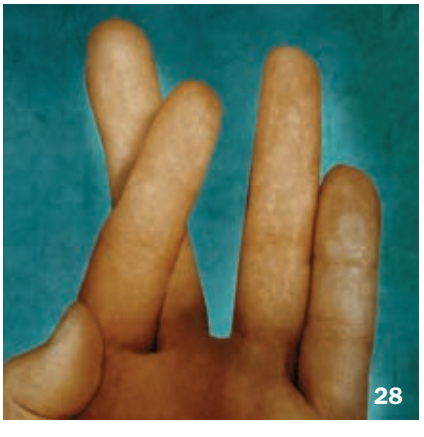

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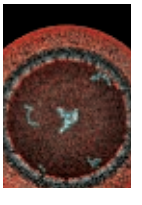

COVER A computer graphic reveals researchers' vision of a protocell, self-replicating molecules enveloped by a simple membrane. *Janet Iwasa*

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FROM THE EDITOR

Creating life in lab depends on power of primitive cells

For many years now, highly evolved complex brains of sophisticated *Homo sapiens* have been trying to figure out how to create the simplest possible form of single-celled life. Some bacteria-like bag of chemicals capable of copying itself is all it would take for scientists to declare victory in the quest to generate a living organism merely by collecting the proper amounts of just the right molecules under conditions conducive to biogenesis.

It doesn't sound like it should be that hard. After all, sometime not quite 4 billion years ago, lifeless molecules gathered somewhere on Earth and self-assembled into an entity that spawned the planet's full repertoire of ancestral life-forms — without help from any fancy laboratory equipment.

Yet despite their modern technological advantages, scientists have not been able to create even a rudimentary biological cell. They are getting closer, though, as contributing correspondent Charles Petit reports in this issue (Page 22). Before too much longer, biochemists may be able to claim that they have found the recipe for replicating the origin of life. That would surely be an accomplishment on par with deciphering the structure of the atom, discovering the expansion of the universe or winning 10 NBA titles in 21 years.

But before celebrating their success, scientists should contemplate the irony of setting their sights so low. Cooking up a cell from scratch might seem special, but bacteria replicate themselves all the time. And when you think about it, primitive cells also begat all the more sophisticated life-forms now populating the planet — it just took a few billion years.

It may even turn out that the real brains are the small RNA molecules that probably got life started. Cells from today's complex bodies host vast numbers of small RNA molecules that pretty much tell the cell what to do, by regulating the activity of various genes. In a 2006 paper in the journal *BioSystems*, physicist Emmanuel Tannenbaum argued that those RNA molecules constitute a "community" directly descended from the primitive RNA that participated in life's origins. In a way, complex organisms like people may merely be vehicles constructed by simple molecules for their own preservation — the molecules offering a repertoire of survival tools in exchange for a comfortable habitat. So when scientists succeed in creating primitive life, it might be appropriate to remember that primitive life succeeded first in creating scientists. —*Tom Siegfried, Editor in Chief*

U.S. Navy photo by Ensign John Gay.

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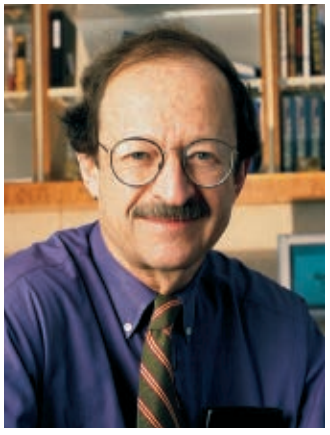

Scientific Observations

"People tend to think of science as a sort of remote activity, carried out behind gates by people who are sort of difficult to understand.... We as a scientific community have made inadequate use of the Internet to tell everybody what we do, to exchange information among ourselves, to bring the results of publicly supported science to the attention of everybody. We forget that although the groups who are doing frontline science may be relatively small, that there are a lot of people who have a big interest … teachers and journalists and people who are in the health service profession and many others who have not just an interest but a need to know what is going on at the front lines of science." **—NOBEL LAUREATE HAROLD VARMUS IN AN INTERVIEW WITH ABC'S ELIZABETH VARGAS AT THE WORLD SCIENCE FESTIVAL IN NEW YORK CITY ON JUNE 3**

Science Past | FROM THE ISSUE OF JULY 2, 1960

HIGH MILK CONTAMINATION FROM NUCLEAR ACCIDENTS — Radioactive contamination of milk is likely to be "the most widespread hazard" resulting from a nuclear accident or explosion depositing fission products on agricultural land, according to recent studies in England reported in a forthcoming issue of *Nature*.... Elements that appeared to cause the greatest contamination are the isotopes of iodine and strontium although barium-140 and cesium-137 also contribute to the peril. These findings resulted from a series of 53 experiments with 44 cows in which the fission products were artificially introduced into the diet of the animals and their milk subsequently monitored for radioactivity.

Science Future

August 8–12
Geoscientists meet in Foz do Iguaçu, Brazil, for an international conference. See www.agu.org/meetings/ja10

August 11–14
The Cognitive Science Society meets in Portland, Ore. Go to cognitivesciencesociety.org/conference2010

September 6
Last day to view the Chicago Field Museum's exhibit on creatures of the Ice Age. See www.fieldmuseum.org/mammoths

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HUMANS
Inhaling someone else's cigarette smoke may harm your emotional health. Read "Secondhand smoke linked to mental distress."

GENES & CELLS
Survival in a tidal environment is in the genes for one species of brown alga (shown). See "Seaweed genome reveals tools for multicellular lifestyle."

SCIENCE & THE PUBLIC BLOG
A chemical that makes chilis hot may affect genes linked with burning fat. Read "Understanding why hot peppers are slimming."

EARTH
Prehistoric waters may have run through Michigan before heading to Colorado. See "Before the Mississippi, minerals show ancient rivers flowed west."

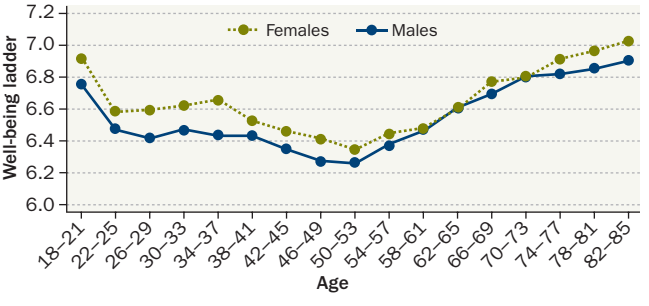
For Daily Use

Fighting food cravings may not be a matter of willpower so much as one of visualization skills. Previous research has shown that people craving a certain food report intense mental images of that food. So researchers in Australia reviewed results from a variety of experiments to see if interfering with those images could reduce cravings. One group of volunteers who had been experiencing food cravings was asked to visualize a common, nonfood image, such as a rainbow. Another group watched an actual pattern of dots on a computer screen. Both groups reported reduced food cravings after these visualization exercises, the scientists note in the April *Current Directions in Psychological Science*.

Science Stats | TRULY GOLDEN YEARS

A U.S. survey of about 340,000 people found that their sense of overall well-being drops in early and mid-adulthood but then rises after age 50.

Mean values for well-being versus age for U.S. men and women



SOURCE: A. STONE *ET AL./PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES* 2010

CLOCKWISE FROM TOP LEFT: MEMORIAL SLOAN-KETTERING CANCER CENTER; YANN FONTANA

“ This was *CSI: Jamestown*. ” —HOWARD SPERO, PAGE 14

In the News

STORY ONE

Surface oil isn't the only threat from Gulf gusher

Scientists try to understand deep-sea hydrocarbon clouds

By Janet Raloff

DAVE MARTIN/AP PHOTO

Oil leaking from the Deepwater Horizon rig can be carried in currents at the surface (above), but teams are now studying the damage that diffuse, deep-sea oil can do.

The catastrophic explosion on April 20 of an offshore oil-exploration platform in the Gulf of Mexico sent oil slicks coasting toward shorelines from Louisiana to Florida. Now research vessels are tracking a more stealthy threat: huge clouds of nearly invisible oil droplets hovering deep below the surface.

New data indicate these invisible plumes form shifting strata that fan out in a host of directions from the gushing wellhead, more than 1.5 kilometers below the water's surface. These clouds could substantially increase estimates of the total amount of oil spilled and could poison deep-dwelling critters that form the base of the marine food web.

BP North America had claimed for weeks that its well was probably spilling no more than 5,000 barrels, or 210,000 gallons, a day. But a new oil-collection system that the company set up on June 3 was hauling in just shy of 16,000 barrels of crude per day by June 10.

Federal officials recently charged several independent research teams with figuring out precisely how much crude oil and natural gas spewed directly into the water in the days following the explosion. As of mid-June, preliminary estimates suggested that more than 1 million barrels of oil could already be sloshing around the Gulf. That means this spill, dubbed the Deepwater Horizon for the name of the platform that sank, released about four times the volume of the next biggest spill in U.S. waters, the 1989 grounding of the *Exxon Valdez*.

Because the oil-collection system now in place over the wellhead can't trap all of the spewing oil, the well will continue to foul Gulf waters until it's shut down in late summer, said Coast Guard Admiral Thad Allen at a June 7 White House briefing.

The big issue is where all of this oil is ending up, because much has still not floated to the surface. An experiment conducted in 2000 off Norway should offer Gulf analysts good clues, notes spill modeler Eric Adams of MIT.

Known as DeepSpill, the Norwegian experiment — sponsored by 23 oil companies and the U.S. Minerals Management Service — released natural gas or a mix of gas and oil into the Atlantic during four tests, none lasting more than two hours. In tests that let off 60 cubic meters of crude oil, “only between 1 and 17 cubic meters (lower and upper bound estimates)” made it to the surface, according to a 2005 analysis by Adams and Scott Socolofsky, now at Texas A&M University in College Station.

Related experiments by teams at MIT and the University of Hawaii help explain the finding. If oil droplets are very small or if enough cold, dense water is mixed in with them, Adams says, “you can get a mixture that could be neutrally buoyant,” meaning it could hover below the surface. It's not clear how oil dispersants currently being used in the Gulf could aggravate such hovering behavior.

Data from DeepSpill and follow-up

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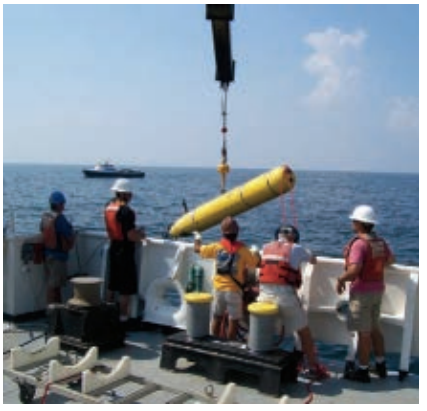
tests may explain the massive clouds of oil reported on June 8 by two independent research cruises plying the Gulf. Oil concentrations were too low to stain the deep-sea water and so couldn't be easily seen, but oil could be detected chemically, acoustically or on filters.

In early June, Samantha Joye of the University of Georgia in Athens and her colleagues found diffuse oil plumes up to roughly 30 kilometers southwest of BP's leaking wellhead. The team measured clouds roughly 3 to 5 kilometers wide. "The part of the water column most impacted was generally 1,100 to 1,300 meters below the surface," Joye notes. "So it's a pretty big patch of water."

The plumes' oil and methane concentrations diminished with distance from the accident site. And when BP slapped a hood on top of the well, a plume these researchers were tracking changed trajectory.

That's "pretty convincing evidence that it was linked to the wellhead and not some natural feature, like a [seabed hydrocarbon] seep," Joye argues.

Deep, diffuse undersea plumes "were totally independent of surface slicks," she notes. Plume distribution is probably influenced by bottom currents, she says, driven by differences in water density (due to temperature and salinity) and by varying seafloor topography.



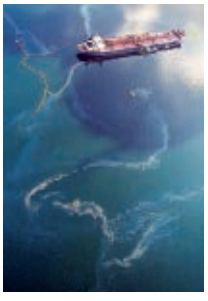
In late May, the Monterey Bay Aquarium Research Institute deployed a robotic submersible to study subsurface oil.

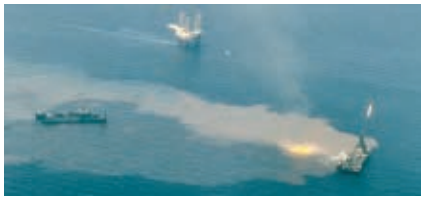
As plumes hit these currents, portions "will shear off and whiz around here, there and yonder," Joye quips, explaining why different research teams may find plumes shooting in different directions at different depths.

Scientists from the University of South Florida in Tampa and the National Oceanic and Atmospheric Administration mapped oil-tainted strata northeast and southeast of the wellhead between May 22 and 28. On June 8, they reported finding oil in several undersea layers more than 70 kilometers from the spill site. Oil concentrations were low, peaking at just 500 parts per billion.

While big fish and porpoises might steer clear of oiled water, small or larval fish probably can't, points out Wes Tunnell of the Harte Research Institute for Gulf of Mexico Studies at Texas A&M's campus in Corpus Christi. Plankton, tiny plants and animals that float with the currents and serve as the base of the marine food web, also can't evade oil. Those that aren't killed outright risk sharing the pollutant with predators that eat them, Tunnell says.

If there is any good news in the Gulf catastrophe, it's that the Gulf's environment often encounters oil from spills and natural seeps, Tunnell says. This history means that there are already plenty of oil-degrading bacteria present, which should help the environment heal over the next several years. ■





Back Story | SPILLS AT SEA

The recent Gulf incident wasn't the first to spew loads of oil into the ocean.

Torrey Canyon
The *Torrey Canyon* (in pieces at far left) was one of the first big oil tankers. She ran aground on Pollard Rock off the coast of England in 1967. More than 700,000 barrels of oil spilled into the sea, polluting the coasts of England and France.

Amoco Cadiz
More than 1.6 million barrels of oil drained into the water when the *Amoco Cadiz* (top right) split in two off the coast of Brittany, France, in 1978. Though the ship put out a distress call, the weather prevented several tugs from stopping the ship from running aground. *Amoco Cadiz*'s sister ship the *M/T Haven* spilled close to a million barrels after exploding off the coast of Italy in 1991.

Atlantic Empress
The *Atlantic Empress* collided with another tanker, the *Aegean Captain*, during a tropical storm off of Trinidad and Tobago in 1979. Though about 2 million barrels of oil leaked into the sea, very little made it to shore.

Ixtoc 1
A Mexican petroleum company was drilling an offshore well, dubbed Ixtoc 1, in 1979 when a blowout occurred (bottom). Before the well could be capped eight or nine months later, about 3.5 million barrels of oil gushed into the Gulf of Mexico.

Gulf War
Iraqi forces dumped oil from several tankers into the Persian Gulf in 1991 to prevent American soldiers from landing there. Estimates vary, but some suggest that up to 13 million barrels were released.

Exxon Valdez
Though not among the largest international spills, with 260,000 or so barrels dumped, the *Exxon Valdez* (middle right) ran aground in 1989 in Alaska's remote Prince William Sound, making response efforts difficult.

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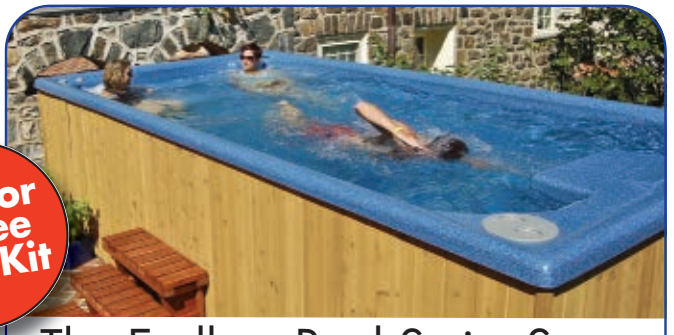

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Molecules

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Designing a giant among molecules

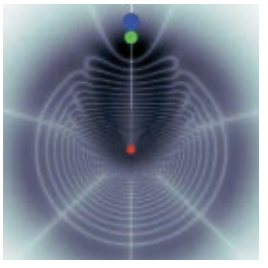
Rydberg atom could form new kind of bond, scientists propose

By Laura Sanders

Physicists have predicted the existence of a new kind of gargantuan molecule, large enough to dwarf a virus, with the potential to be in two configurations at once. Such a molecule might prove useful in storing and transmitting quantum information, the researchers report online June 15 in *Physical Review Letters*.

An atom in an excited state can have an electron that roams very far from its nucleus. These giant "Rydberg atoms" can form molecules more than a thousand times larger than everyday molecules. The newly predicted molecule would be so large that a small virus — itself made of many molecules — could fit inside, says study coauthor Seth Rittenhouse.

In the new study, Rittenhouse and his colleague Hossein Sadeghpour, both of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., predicted what would happen to a giant rubidium atom in the Rydberg state if it were brought near a small molecule, composed of potassium and rubidium, with a dipole moment — a positive electrical charge at one end and a negative charge at the other. This charge separation wouldn't be strong enough to rip the wandering electron away from the giant atom. But the electron would find the dipole irresistible, calculations show. "That extra bit of charge is enough to get the electron to stick near it," Rittenhouse says.



An electron far from its nucleus (red) could bond with a small molecule (blue and green).

In this way, the small molecule and giant atom would form a gigantic Rydberg molecule with a totally new type of chemical bond. "When you talk about chemistry, you talk about bonds," says Rittenhouse. "This type of bond is new."

In 2000, atomic physicist Chris Greene of JILA and the University of Colorado at Boulder and colleagues predicted the existence of a Rydberg molecule made up of an excited atom and a neutral atom. The researchers calculated that the roaming electron of the excited atom would hover around the neutral atom and form an electron cloud that resembled, of all things, an ancient trilobite.

"I think it came as a surprise to a lot of people when we made our original prediction, because diatomic molecules, with just two atoms, were believed by chemists to be completely understood," Greene says. Adding a molecule with a charge takes his prediction a step further: "I view this as a really interesting extension," he says.

The new giant molecule would exhibit a property called superposition. The potassium-rubidium molecule can point in two directions at once, the models predict, with the potassium atom on top and on the bottom of the rubidium atom at the same time. Rittenhouse says that this superposition state might serve as a qubit, a bit of quantum information that could store or transmit a message.

Such a use is "certainly an interesting aspect I hadn't thought of before," Greene says. But he says the life span of the molecules, about 100 microseconds, might be too short for them to be useful.

Turning pigment to poison

One beastie's pigment is another's poison. The marine-dwelling California sea hare *Aplysia californica* (shown) converts pigment from food into a chemical weapon, scientists say in the first report of an animal taking a dietary photosynthetic pigment and turning it into a molecule for deflecting attackers. Some sea hares blast predators, such as crabs, with a defensive spray combining dark purple ink and a whitish substance called opaline. Michiya Kamio of the Tokyo University of Marine Science and Technology analyzed the *A. californica*'s ink and exposed blue crabs to its ingredients. One especially offensive compound was aplysioviolin, Kamio and colleagues report in an upcoming *Animal Behaviour*. The compound's chemical precursor is phycoerythrobilin, a mild-mannered photosynthesis pigment found in red algae. Sea hares graze on the algae, loading up on phycoerythrobilin. Two chemical tweaks by the sea hare convert the precursor into the offensive compound. —*Rachel Ehrenberg*

FROM TOP: S. RITTENHOUSE AND H. SADEGHPOUR/*PHYS. REV. LETTERS*; HOWARD HALL/PHOTOLIBRARY

Body & Brain

73 Concussions reported by 74 Alberta youth hockey teams in one season of games with body checking

20 Concussions reported by 76 Quebec youth hockey teams in one season of games with no body checking

A check on youth hockey injuries

More concussions reported in leagues with body checking

By Nathan Seppa

Children playing ice hockey in leagues that permit body checking have more concussions and other injuries than do youngsters in leagues that prohibit checking, a Canadian study of preteens shows. The work appears in the June 9 *Journal of the American Medical Association*.

Checking in hockey is akin to blocking in football. But in hockey it's a defensive hit, in which a player attempts to stop or limit the progress of an offensive player who has the puck. While a check cannot be delivered with the elbows, knees or a hockey stick, it can involve a violent collision. Some youth leagues disallow checks among preteens, and in the late 1980s the entire province of Quebec banned checking for all players ages 12 and under.

Researchers enlisted 76 youth hockey teams in Quebec and 74 in Alberta, where checking is still allowed, in the new study. Each team had a physical trainer or other adult who recorded injuries to the team's players during an entire season's games and practices. The study included more than 1,000 players in each province. All children were ages 11 or 12, a group referred to in Canada as the Pee Wee League. Nearly all were boys, and all wore helmets and mouth guards.

There were 209 injuries, including 73 concussions, during games involving the Alberta players. The Quebec teams, which don't allow checking, suffered 70 game-related injuries, of which 20 were concussions. Doctors verified the injuries. Among the Alberta players, checking was involved in the majority of injuries.

"The public health implications of body checking in Pee Wee ice hockey are significant," says study coauthor Carolyn Emery, an epidemiologist and physiotherapist at the University of Calgary in Alberta. "In Alberta, we estimate that if Pee Wee ice hockey checking were removed, it would prevent over 1,000 injuries and 400 concussions per year."

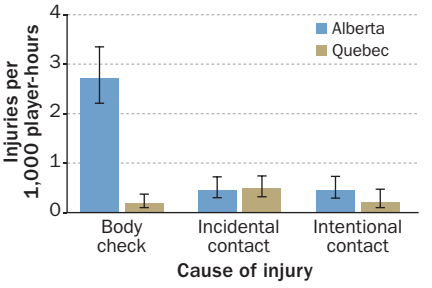
She notes that players below the 25th percentile for weight in these leagues were more prone to injury than heavier players. "It's physics, really," she says.

Injury rates during practices were similar in the two provinces.

The study adds to a growing literature on head injuries in hockey and football. Coaches in these sports have recently taken a more cautious approach to concussions, keeping players out of subsequent games as a precaution. Having a concussion seems to increase the likelihood of getting another one, says Steven Broglio, a kinesiologist at the University of Illinois at Urbana-Champaign.

The definition of a concussion has changed, Broglio says, to include head injuries that cause dizziness, brief memory loss, headache or other symptoms that fall short of a loss of consciousness. "In the past, unless you passed out it was just accepted as part of the game. They'd say you 'had your bell rung,'" he says. "Those are incredibly antiquated terms now. Those were concussions."

Ice hockey injuries in 11- and 12-year-olds



SOURCE: ADAPTED FROM C. EMERY *ET AL./JAMA*

Ice hockey leagues that allow body checks report more injuries in preteens, most related to the defensive move.

Testing a new tool to treat sepsis

Blocking an enzyme active in inflammation shows promise

By Nathan Seppa

By blocking an enzyme that appears to incite inflammation in sepsis, researchers can reverse the deadly condition in mice, a study in the June 4 *Science* shows.

Sepsis is a massive inflammatory state typically triggered by a bacterial infection. Death rates can reach 60 percent.

The enzyme that researchers blocked, sphingosine kinase 1 or SphK1, is known to spur production of inflammatory proteins. But until now, the enzyme hadn't been linked to sepsis, says study coauthor Alirio Melendez, a physician at the University of Glasgow in Scotland.

Melendez and collaborators in Singapore and Europe found that SphK1 is mass-produced by certain immune cells during sepsis. In lab-dish tests, the researchers inhibited SphK1 by silencing a key gene or by adding a compound called 5c. When they injected a bacterial toxin into mice to induce sepsis, mice whose ability to make SphK1 had been blocked avoided sepsis. And mice with full-blown sepsis from intestinal surgery survived longer if they had been pretreated with SphK1 blockers such as 5c.

"This is a long way from getting into a clinical setting, but I think it's promising," says physician Derek Wheeler of Cincinnati Children's Hospital Medical Center.

Timothy Hla, a vascular biologist at the Weill Cornell Medical College in New York City, says the SphK1 inhibitors need to be tested more extensively. "You can give compounds to animals and cure them, but that doesn't tell you the mechanisms at work," he says. "I'm pretty sure this compound [5c] is blocking many things — not just SphK1." Overall, he says the study offers "an interesting finding, but I think it's very preliminary."

Atom & Cosmos

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Crash of '09 suspect named

Asteroid probably to blame for scar on Jupiter

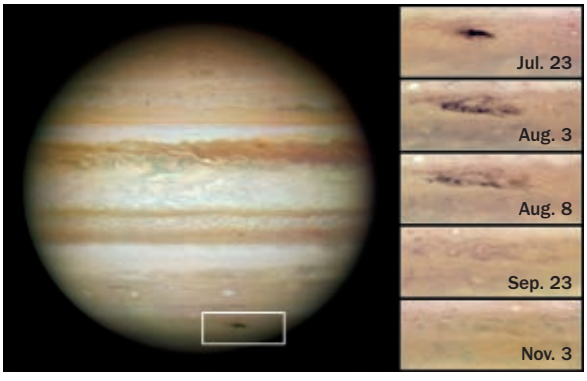
By Ron Cowen

The body that slammed into Jupiter last July almost certainly was an asteroid rather than a comet, and such impacts might happen as often as every 10 to 15 years, new studies suggest.

If researchers are correct, images taken by the Hubble Space Telescope and other instruments may have captured for the first time the immediate aftermath of an asteroid striking a planet.

The evidence for an asteroid comes in part from comparing Hubble images of the 2009 impact site with those Hubble took in 1994, when comet Shoemaker-Levy 9 struck Jupiter.

Before that impact, each fragment of Shoemaker-Levy 9, which had broken apart during an earlier passage by Jupiter, was surrounded by a cloud of dusty debris, called a coma, that clearly identified the fragments' source as a comet. The impact of the tiny particles that made up each coma generated the extended dark halos seen in Hubble's 1994 ultraviolet images, notes Heidi Hammel of the Space Science Institute in Boulder, Colo. In contrast, she says, the single body that smacked into Jupiter in 2009 produced no such halo, which argues for an asteroid as the source.



A blemish (white box) imaged by the Hubble Space Telescope in 2009 marks the spot of an object's impact with Jupiter. Close-ups (right) show the blemish disappearing.

Hammel, Agustín Sánchez-Lavega of Universidad del País Vasco in Bilbao, Spain, and colleagues describe their findings in the June 1 *Astrophysical Journal Letters*. In another article in that issue, the team suggests that an object about 500 meters to 1 kilometer across strikes Jupiter every 10 to 15 years. Previous estimates, based on the 1994 collision, put the rate at about once a century.

Estimates based on tracing the orbit of the 2009 impactor back in time yield about a 50-50 chance that it was an asteroid. Hubble observations, with data from other telescopes, tip the balance in favor of an asteroid, says Carey Lisse of the Johns Hopkins Applied Physics Laboratory in Laurel, Md.

If the culprit is an asteroid, it probably came from the Hilda family of asteroids in the outer part of the main asteroid belt, between the orbits of Mars and Jupiter.

The two new studies indicate that the population of potential impactors in Jupiter's neighborhood is much greater than previously thought and that many are rocky, not icy like comets. In fact, on June 3, amateur astronomers recorded a new impactor hitting Jupiter. The observed fireball didn't leave a bruise.

M.H. WONG/UC BERKELEY, H.B. HAMMEL/SPACE SCIENCE INSTITUTE, I. DE PATER/UC BERKELEY, THE JUPITER IMPACT TEAM, NASA, ESA

Titan chemistry yields hint of life

Other factors could explain lack of acetylene, hydrogen

By Ron Cowen

Studies of chemistry on Saturn's moon Titan raise the possibility that methane-based bacteria might be munching acetylene and hydrogen on its surface.

Two new reports of deficits of acetylene and hydrogen could more readily be explained without invoking biology, says astrobiologist Chris McKay of NASA's Ames Research Center in Moffett Field, Calif. The findings, he says, may nonetheless have implications for the possibility of life on Titan.

One study, posted online March 15 in *Icarus*, focuses on a computer simulation indicating that hydrogen molecules flow downward from Titan's atmosphere but are missing from the surface. Using data from the Cassini spacecraft, Darrell Strobel of Johns Hopkins University in Baltimore found that 10,000 trillion trillion hydrogen molecules fall out of the atmosphere per second. But no corresponding buildup was seen at the surface.

A second paper, posted online April 28 in the *Journal of Geophysical Research*, reports a lack of acetylene. Acetylene and benzene should be produced when sunlight strikes the methane gas in Titan's atmosphere, then fall to the surface. Roger Clark of the U.S. Geological Survey in Denver and colleagues found loads of benzene on the surface but no acetylene.

Taken alone, a lack of acetylene wouldn't mean much, says McKay. But low acetylene plus low hydrogen could equal life. Just as organisms on Earth combine oxygen with organic compounds to get energy, organisms on Titan might react molecular hydrogen with organic materials such as acetylene. It's therefore intriguing that both acetylene and hydrogen are missing on the surface, he says.

When combined with hydrogen, acetylene is a potentially huge source of metabolic energy, "large enough to drive a biosphere," comments planetary scientist David Grinspoon of the Denver Museum of Nature & Science.

460 Approximate number of known extrasolar planets

706 New candidate exoplanets discovered by Kepler mission

Kepler promises planetary bonanza

More than 700 new candidates added to roster of exoplanets

By Ron Cowen

Surveying thousands of stars for telltale twinkles that signal the passage of an orbiting planet, NASA's Kepler spacecraft has discovered a whopping 706 candidate planets beyond the solar system. If confirmed, that mother lode would bring the total number of known extrasolar planets from fewer than 500 to well over 1,000.

The trove includes five stars with signs of full-fledged planetary systems. If verified, they would be the first known multiplanet systems in which each orb creates a minieclipse as it transits, or passes in front of, its star. The amount of dimming and the duration of a transit offer information that cannot be gleaned by less direct methods of detection.

A team including Kepler lead scientist William Borucki of NASA's Ames Research Center in Moffett Field, Calif., posted the findings online June 15 at arXiv.org. The discoveries were made by analyzing Kepler's first few months of data, recorded in the spring of 2009 when it examined 156,000 stars.

"This is a massively historic discovery," says study coauthor Sara Seager of MIT. "This is showing how the Kepler mission will revolutionize exoplanets and change the way we do exoplanet science."

The findings don't include full information on the most interesting 400 of the 706 planets, which orbit the brightest stars Kepler has surveyed and may offer the most promise for finding Earth-mass planets. A complete description of those planets will be made public in February.

Of the five candidate planetary systems, one consists of three orbs, while the other four contain two. The orbiting objects range in size from twice Earth's diameter to slightly larger than that of Jupiter. They are not yet confirmed planets because their masses have not been determined. Astronomers are attempting to measure those masses using ground-based telescopes that can discern the tiny wobble induced in the motion of a parent star due to the tug of orbiting bodies.

Even without confirmation, the candidate systems elevate confidence that such systems are common in the cosmos.

"They show that Kepler will find dozens, and likely over a hundred stars having multiple planets that all transit in front of their host star," says study coauthor Geoffrey Marcy of the University of California, Berkeley. "Apparently, stars commonly house multiple planets."

Genes & Cells

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Some autism cases linked to rare mutations

Extra or missing chunks of DNA offer therapy targets

By Tina Hesman Saey

Each person with autism may have a genetically distinct version of the developmental disorder, a new large-scale study finds.

Rare variations in which part of a person's genetic blueprint is missing or duplicated are responsible for some cases of autism, the study shows. Such missing or duplicated stretches of DNA, known as copy number variants, have been implicated in schizophrenia and other diseases (*SN: 4/25/09, p. 16*).

Published online June 9 in *Nature*, the new study shows that some people with autism may be missing some or all of one or more genes involved in the development and function of the brain. The findings could lead to improved diagnosis of the disorder, perhaps even in infants, and may give new direction to research on drug treatments.

"The exciting thing about ... this study is that it highlights biological pathways that can be targets for therapy," says Geraldine Dawson, chief science officer of Autism Speaks, a national organization that helped to fund the study.

Autism is a group of developmental disorders that impair social interactions and often language development. Researchers don't know the exact causes of autism, but genetic factors are strong suspects. Studies of identical twins show that when one twin has autism, about 90 percent of the time the other twin will too. Autism affects about one in every 100 children in the United States and is more common in boys.

Previous studies have indicated that people with autism may have more copy number variants overall than healthy people do. But the new study found that people with autism had the same number of deletions as people in a healthy control group. People with autism tended to have deletions that removed parts or all of genes, however, while healthy people tended to have deletions affecting stretches of DNA that don't contain genes.

"You and I may have just as many deletions in our genomes, but since they don't hit genes, we don't have autism" or other diseases, says Anthony Wynshaw-Boris, a medical geneticist at the University of California, San Francisco, who was not involved in the new research.

In the study, an international consortium of researchers analyzed the genetic makeup of 996 people with autism and 1,287 people without autism. The researchers found more than 5,000 copy number variants in people with autism, usually places where DNA was missing. Many people with autism had more than one spot in the genome where they were missing large chunks of DNA.

All together, the new study identified 25 places in the genome that may help in diagnosing autism.

Each specific variant was rare on its own. Even the most common appeared in less than 1 percent of the people in the study.

The new work may help settle a scientific debate about whether common diseases and disorders are caused by genetic variations present in many people, or if rare variants contribute more to these diseases. "This definitely suggests a role for rare variations in autism," says Charles Lee, a clinical cytogeneticist at Brigham and Women's Hospital and Harvard Medical School, both in Boston.

Although each person with autism appears to have a distinct set of genetic variations, the genes affected tend to influence similar biological processes. Further studies promise to give researchers a better picture of what causes autism, Wynshaw-Boris says.

Some of the deleted genes had a strong link to autism; missing just a single copy was enough to push a person across the autism threshold, says study coauthor Stephen Scherer of the Hospital for Sick Children in Toronto. Other genes had to be inherited along with more deletions or other genetic factors for autism to develop.

One gene strongly linked to autism in the study, *DDX53-PTCHD1*, is located on the X chromosome. Women who carry a deletion of the gene on one of their X chromosomes will almost always carry a healthy version of the gene on the other X chromosome that will cover for the missing copy. But if a woman passes the X chromosome with the deleted gene on to a son, he will have no healthy copy of the gene (the Y chromosome doesn't carry it) and will get autism.

Researchers also identified several genes involved in forming connections between brain cells that had not been linked to autism before. Genes involved in a cell-to-cell communications system known as the Ras/GTPase pathway were also found to play a role in autism.

All together, the new study identified 25 places in the genome that may help in diagnosing autism. New genetic tests would probably focus on these markers, Lee says. But until scientists understand more about how genetic factors work together to cause disease, no one will be able to make a definitive diagnosis based on genetics, he says.

Even with the new findings, scientists are able to explain genetic causes for only about 10 percent of autism cases, says Steven McCarroll, a geneticist at Harvard Medical School. "What causes autism in the other 90 percent of cases is still on the table," he says. "Every little victory is important, but it's still amazing how little we know."

"People who look genetically like ... fifth cousins aren't necessarily genealogically related."—KARL SKORECKI

Genome maps trace Jewish origins

Roots of far-flung populations reach back to the Levant

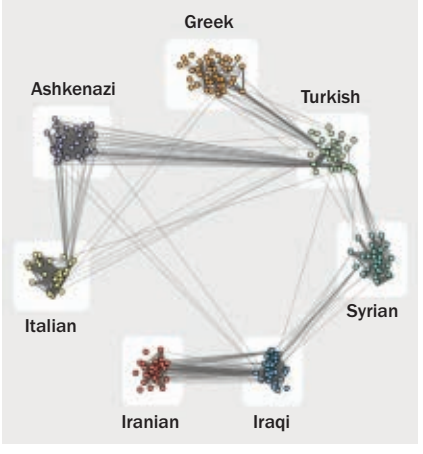
By Tina Hesman Saey

Scientists taking a genome-wide view of ancestry have confirmed what historians, archaeologists and linguists have long known — Jews originated in the part of the Middle East known as the Levant.

Two new studies show that most Jewish groups share large swaths of DNA.

A study of 14 Jewish Diaspora communities and 69 non-Jewish populations, published online June 9 in *Nature*, finds that Jews share genetic heritage with Middle Eastern groups, such as the Druze. The work also indicates that most of the Jewish groups trace their genetic origins to the Levant, which includes present-day Israel, Palestinian territories, Lebanon and other areas. From there, groups of Jews migrated to other parts of the world.

Maps of those migrations were inscribed in the DNA by genetic signatures of local people that Jews interbred with as they moved. Most contemporary Jews carry evidence of a Middle Eastern origin along with genetic heritage from European and North African ancestors.



G. ATZMON *ET AL.*/*AJHG* 2010

Links between geographically distinct Jews (dots) represent genome similarities that are typically seen between distant cousins, a new study finds.

"I like to think of relatedness as a tapestry, and these shared segments [of DNA] are threads in the tapestry," says Harry Ostrer, a geneticist at New York University School of Medicine and the leader of a study published in the June 11 *American Journal of Human Genetics*.

Each of the Jewish groups in the studies has its own genetic signature but is more closely related to the other Jewish groups than to non-Jewish groups. Even though the researchers excluded people known to be directly related, any two Ashkenazi Jewish participants in Ostrer's study shared about as much DNA as fourth or fifth cousins, he says.

But "people who look genetically like they may be fifth cousins aren't necessarily genealogically related," says Karl Skorecki of Technion–Israel Institute of Technology and the Rambam Medical Center in Haifa, a coauthor of the *Nature* study.

That study finds that three major Jewish groups have roots in the Levant: Ashkenazi, Moroccan and Sephardic Jews; Caucasus and Middle Eastern Jews (including Iranian and Iraqi groups); and genetically distinct Yemeni Jews.

In contrast, analyses of DNA from Jews in Ethiopia and India show that those groups share genetic heritage with their neighbors, indicating that these Jews may descend mainly from local converts.

Understanding the genetic heritage of individuals may help researchers zero in on disease genes that are more common in certain ethnic groups, Ostrer says.

The new data also may be used in commercial DNA tests to tell clients how much Jewish ancestry they have. But, says Skorecki, "People get the horribly wrong impression that there's a Jewish version of a gene" defining heritage. That's not the case. "It's more of a genome-wide pattern," he says.

MEETING NOTES

Genetics 2010: Model Organisms to Human Biology, Boston, June 12–15

H1N1 virus lacks killer protein

The H1N1 flu virus just doesn't have what it takes to be a real killer, a new study of the 1918 Spanish flu suggests. The Spanish flu virus, which caused a pandemic that killed 20 million to 40 million people, had a killer combination of three surface proteins, including PB1-F2. That protein prevents the body from making an antiviral compound called interferon, virologist Peter Palese of Mount Sinai School of Medicine in New York City reported June 14. Without interferon to hold it back, the virus can overwhelm the body's defenses within three days after infection. Other vicious pandemic influenza strains also possess PB1-F2. But the 2009 H1N1 "swine" flu virus lacks the protein. "This virus is not as virulent as other pandemic influenza viruses," Palese says. —*Tina Hesman Saey*

Mexican-American and African-American genomes completed

Researchers have compiled the complete genetic instruction books for two people of mixed ethnic ancestry—a Mexican-American and an African-American, Carlos Bustamante of Stanford University School of Medicine reported June 12. The African-American person has genetic roots in West Africa and Europe, while the Mexican-American carries genetic legacies of Native American and European ancestors. Fine-scale DNA analysis shows that Native Americans may have as much genetic diversity as some African populations known to be among the most genetically diverse. —*Tina Hesman Saey*

Humans

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Trash confirms Jamestown drought

Oyster shells discarded in well reveal colonists' water woes

By Sid Perkins

Oyster shells excavated from a well in Jamestown, Va., the first permanent British settlement in North America, bolster the notion that the first colonists there suffered an unusually deep and long-lasting drought.

The shells reveal that water in the James River, where many of those oysters were harvested, was much saltier than along that stretch of the estuary today, says Howard Spero, a geochemist at the University of California, Davis. For the water to have been so brackish, river flow must have been slack compared with today's, a sign that precipitation was dramatically lower when those oysters were growing. Spero and colleagues report the findings in the June 8 *Proceedings of the National Academy of Sciences*.

Oyster shells dumped into an abandoned well by the early settlers of Jamestown, Va., chronicle a lengthy, deep drought that the colonists faced.

Jamestown was established in 1607. Many accounts from its early settlers chronicled the drought, as did trees, Spero says. Studies based on tree rings and documents revealed that the first colonists' arrival coincided with the beginning of a drought that included the driest seven-year interval in almost 800 years.

"It was interesting trying to figure out what was happening in the colony at a time when 70 to 80 percent of the colonists were dying," Spero says. "This was *CSI: Jamestown*."

Now, oysters confirm the tale from trees and historical accounts, comments William M. Kelso, an archaeologist at Preservation Virginia's Jamestown project who was not involved in the study. "We're getting a consistent story from science and the humanities," he notes. "It's pretty fantastic."

The telltale oysters were unearthed from a well that sat within the fort at Jamestown, about 100 yards from the river. Among other material in the well, the shells came from three layers up to 3.5 meters deep. The well's water level originally sat at about 4 meters deep, so Spero and colleagues suggest that the settlers abandoned the well—which either ran dry or was infiltrated by salty groundwater—and converted it into a trash pit.

Historical accounts suggest that settlers dug the well between 1609 and 1616, but items from the well narrow that window. Ratios of oxygen isotopes in the shells suggest that they were harvested before the drought ended in late 1612.

Shoe steps out of Copper Age

Armenian cave search yields oldest known leather footwear

By Bruce Bower

A new find has given archaeologists a rare foothold on Copper Age life. Excavations of an Armenian cave have uncovered the oldest known leather shoe, a slip-on, lace-up model from roughly 5,500 years ago. It's about a modern woman's U.S. size 7.

A team led by archaeologist Ron Pinhasi of University College Cork in Ireland found the shoe in 2008, under a broken jar at the bottom of a pit in the Areni-1 Cave. A deer's shoulder blade, two wild-goat horns, a fish vertebra, scattered reeds and pottery shards rested in the pit as well. A 6,000-year-old human brain was also recovered at Areni-1 in 2008.

The shoe consists of a piece of cowhide that wrapped around the right foot, the scientists report in a paper published online June 9 in *PLoS ONE*. A leather lace running through eyelets pulled the hide together on top of the foot and another connected flaps at the wearer's heel.

Grass was stuffed inside the shoe, probably to maintain its shape during storage. Radiocarbon tests of the shoe and stuffing provided an age estimate.

Excavators discovered this 5,500-year-old cowhide shoe in an Armenian cave.

Cowhide sandals from a Copper Age grave in Israel are thought to be about as old as the Areni-1 shoe but have not been radiocarbon dated.

The shoe found at Areni-1 looks much like shoes worn in modern times on Ireland's Aran Islands.

"This is a shoe in the modern sense, in that the same technology and manufacturing method utilized by the Areni-1 people prevailed until the 1950s in Ireland and other parts of Europe," Pinhasi says.

Researchers know little about when people first started sporting shoes. Stone Age human skeletons from Europe display unusually small toes, suggestive of footwear use by 40,000 years ago.

FROM TOP: PRESERVATION VIRGINIA; BORIS GASPARIAN/INSTITUTE OF ARCHAEOLOGY AND ETHNOGRAPHY OF THE NATL. ACAD. SCIENCES, ARMENIA

Numbers

"Living organisms ... seem to end up obeying some kind of mathematical law." —GANDHIMOHAN VISWANATHAN

Sharks use math to hunt their prey

Marine predators cruise the seas using fractal principles

By Alexandra Witze

The great white shark in *Jaws* knew exactly where it was going — to the closest pair of plump legs around. But where might it head if it didn't have a tasty human snack in its sights?

A new study suggests that some sharks and other marine predators can follow strict mathematical strategies when foraging for dinner. The work, reported in the June 10 *Nature*, is the latest aiming to show whether animals sometimes move in a pattern called a Lévy walk.

Unlike random motion — in which animals take similar-sized steps in any direction, like a drunk stumbling around — Lévy walks are punctuated by rare, long forays in any direction. Draw a Lévy walk on a graph, and its squiggly pattern echoes a fractal, the mathematical phenomenon whose shape remains similar no matter the viewing scale.

"Living organisms, when allowed to make freely willed decisions, seem to end up obeying some kind of mathematical law," says Gandhimohan Viswanathan, a theoretical physicist at the Federal University of Alagoas in Maceió, Brazil, who was not involved in the study.

A team led by David Sims, a researcher at the Marine Biological Association of the United Kingdom in Plymouth, looked at 14 species of open-ocean marine predators, including tuna, swordfish, marlin and sharks. Electronic tags yielded 12 million data points describing how the animals swam over 5,700 days.

Many of the animals displayed Lévy behavior at least some of the time, Sims and his colleagues report — "the strongest evidence yet that these Lévy patterns are exhibited by wild animals," he says. Lévy behavior showed up more often in waters where plankton, fish and other food was scarce. In regions with plentiful food, random motion dominated. This observation, says Viswanathan, fits with earlier suggestions that "animals may use a Lévy flight motion to improve their chances of finding prey."

Not all experts are on board. Simon Benhamou, an ecologist at the National Center for Scientific Research, or CNRS, in Montpellier, France, says that statistical errors can often suggest Lévy behavior where it doesn't exist.

Sims and his team now want to probe the evolutionary history of Lévy behavior — for instance by monitoring the movements of the "living fossil" known as the nautilus.

Life

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Parasites need bacteria to hatch

Study suggests new way to battle an intestinal infection

By Rachel Ehrenberg

The question of which came first, the whipworm or the whipworm egg, leaves out a key player: bacteria.

Eggs of the parasitic whipworm, whose potential hosts include people, won't hatch in their host's intestine until they get the go-ahead from nearby gut bacteria, researchers report June 11 in *Science*.

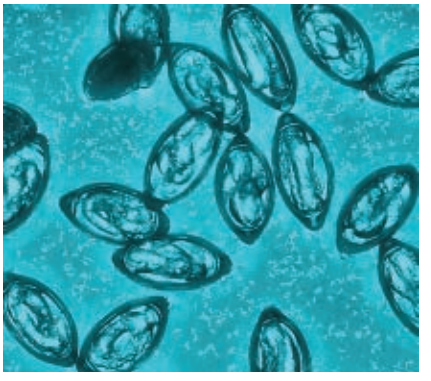
The work reveals how the parasite avoids hatching in the wrong place. It also highlights that parasite-host interactions don't occur in isolation.

"This is a very nice illustration of the interdependence of things," says immunologist Rick Maizels of the University of Edinburgh in Scotland, who was not involved in the work. The finding also suggests a way to stymie whipworm infection, by interrupting the bacterial message that tells whipworm eggs to hatch.

Nearly a billion people are infected by the whipworm species that affects humans. The small eggs can be eaten accidentally with rice or other grains. Once inside, the eggs hatch and the larvae burrow into the intestinal wall, leading to anemia, diarrhea or other problems.

Ian S. Roberts of the University of Manchester in England and colleagues investigated the mouse whipworm, one of about 50 species of the intestinal parasite *Trichuris*. In small sections of mouse intestine, many more whipworm eggs hatched in the presence of live *E. coli* than in the presence of *E. coli* that had been killed by boiling. Some other bacteria, including a staph species, also induced hatching. And giving live mice antibiotics to kill their gut bacteria also reduced their worm load.

Researchers noticed that bacteria clustered at the ends of the whipworm eggs, as though communicating by physical contact with the egg rather than via a chemical messenger. Sure enough, experiments found that thin, rodlike projections from the bacteria were binding to proteins on the surfaces of the whipworm eggs.



Intestinal bacteria (tiny flecks) alert whipworm eggs that it's time to hatch.

Hatching was also influenced by temperature — whipworms emerged only at about 37° Celsius. Taken together, the right temperature and the gut bacteria seem to tell the eggs that they are inside a host and can get hatching.

Possible snake shortage looming

Researchers alarmed by widespread population declines

By Susan Milius

The world might be facing a new kind of sssilent ssspring if researchers are correct that snake numbers are declining.

Out of 17 snake populations monitored in Europe, Australia and Africa, 11 plummeted about 10 years ago and have not bounced back, says herpetologist Chris Reading of the Centre for Ecology & Hydrology near Oxford, England. He and nine other biologists sound an alarm online June 9 in *Biology Letters*.

Losing a lot of snakes can upset the way ecosystems work, Reading says. Snakes often rank as top predators, and even ophidiophobes may appreciate the job that snakes do in controlling rats and mice.

The new finding strikes herpetologist Harry Greene of Cornell University as deeply troubling. Checking for trends in other populations will be difficult, he says, because snakes are notoriously hard to count. "Being secretive is a very snaky thing."

The smooth snake, a protected species, has dwindled in numbers in an English forest.

No data were given on U.S. snakes, but Greene notes worrisome signs from eastern king snakes in Florida and southern hognosed snakes. "Of course, some snakes seem to be doing fine, but overall the trend is alarming," Greene says.

Declines of wild creatures have led to recurring expressions of concern since Rachel Carson's 1962 book *Silent Spring* warned of pesticides threatening birds.

Just what caused the declines between 1998 and 2002 is not yet clear. And of the 17 snake populations studied, five hung in as stable and one increased slightly. Tiger snakes with reliable food sources on an island off Western Australia seem to be doing OK, for example, says study coauthor David Pearson of the Western Australia Department of Environment and Conservation near Perth.

"The jury is still out on whether or not there is a general crisis here," says ecologist Rick Shine of the University of Sydney, "but the reports are alarming."

FROM TOP: KELLY HAYES/UNIV. OF MANCHESTER; GEORGE MCCARTHY/NATURE PICTURE LIBRARY

Matter & Energy

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Bouncing beads outwit Feynman

A machine based on a thought experiment performs work

By Laura Sanders

Researchers have built a machine that harnesses energy from the random motion of bouncing beads. The device, modified from a system dreamt up nearly a century ago, dances around physicist Richard Feynman's dictum that work can't be extracted from such a system.

In 1912, Polish physicist Marian Smoluchowski proposed a thought experiment in which tiny moving particles spin a windmill-type paddle, which then spins a toothed wheel. A pawl prevents the wheel from slipping backward, forcing the wheel to move in one direction only. But Feynman later pointed out, in his famous lectures on physics, that the original calculations missed something. If everything in the system was the same temperature, the pawl would occasionally slip off the wheel, resulting in no net movement, he showed.

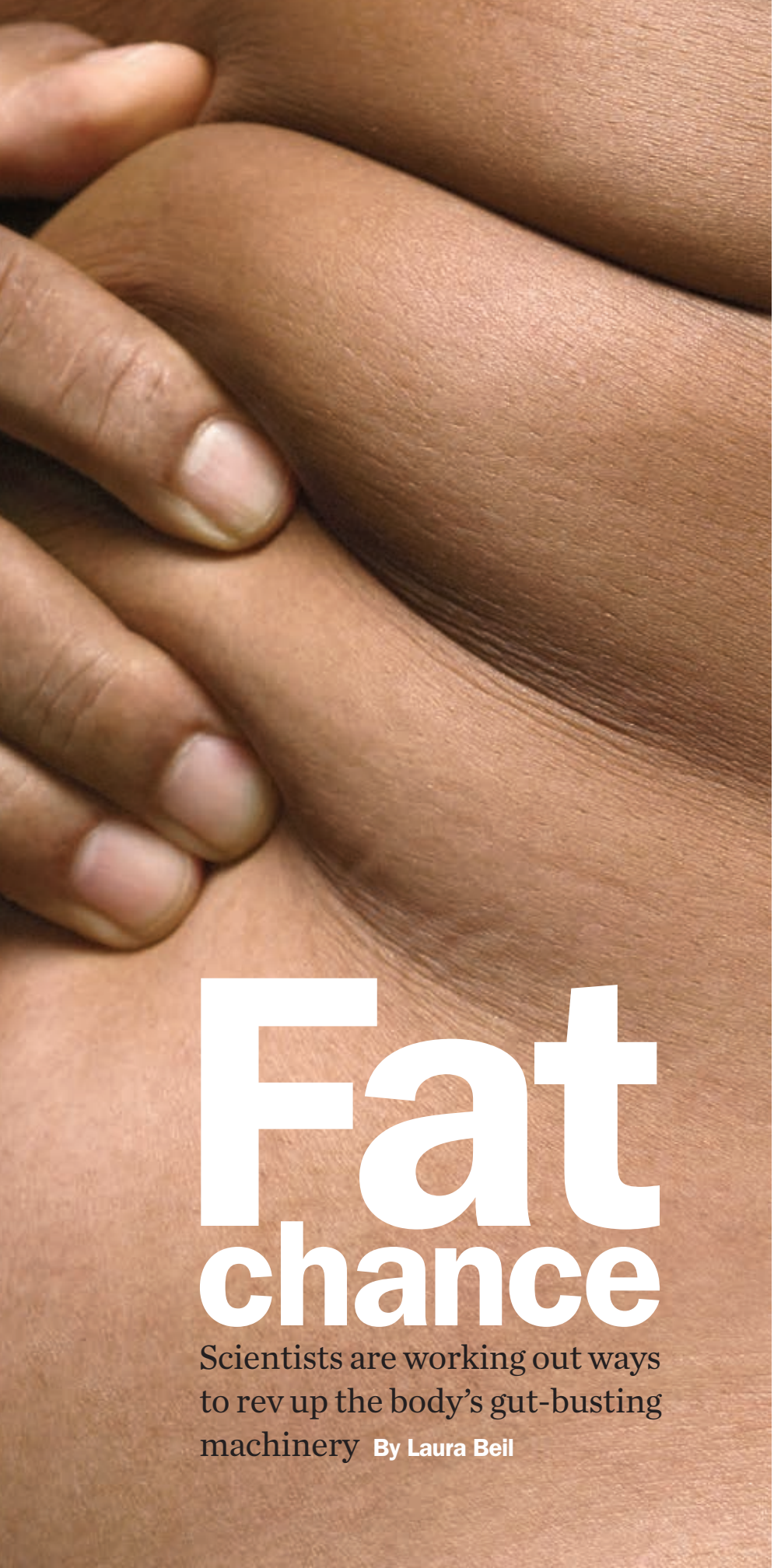
By skirting some of the original rules, the new machine, described in a paper to appear in *Physical Review Letters*, keeps the wheel spinning in one direction. "It's an amusing play on a classical problem," says physicist Bob Behringer of Duke University in Durham, N.C. "By changing an assumption you can actually make this work."

In the new study, Devaraj van der Meer of the University of Twente in the Netherlands and colleagues designed a vigorously shaking platform that sends glass beads flying up like popcorn dancing off a popper. The beads smash into windmill-like vanes, which start turning a rod, which rotates a sensor. If this spinning is directional, it can be put to good use.

If the paddles had the same kind of surface on each side, there was no net rotation — the machine swung back and forth evenly. After the scientists coated one side of each paddle with duct tape, though, the beads lost more energy when they hit the softer taped side of the vanes, causing the system to rotate in one direction.

As the vanes turned, they created a roiling pattern in the beads. This reciprocal give-and-take — beads moving the vanes and vanes moving the beads — could also happen for tiny molecular ratchets, such as those in the body, van der Meer says.

The machine requires energy for shaking and loses most of that energy through heat and sound. "It's an extremely inefficient device," van der Meer says. "In terms of the second law of thermodynamics, there's no problem whatsoever."



Fat chance

Scientists are working out ways to rev up the body's gut-busting machinery **By Laura Beil**

SIRI STAFFORD/GETTY

Throughout the leaner epochs of human history, when food supplies were unreliable, the species would not have survived without a way to hoard calories for later use. That is, without fat. Once a meal has supplied the body's immediate energy needs, any unused fuel gets converted into long molecules called triglycerides, which are dispatched to fatty tissue where they wait for a signal that the body needs them.

But in an era of high-calorie smorgasbords and 24/7 convenience, unused energy can just pile on year after year, a major reason why one-third of the U.S. adult population is struggling with obesity. Laws of physics — the ones about conservation of matter and energy — dictate that schemes for burning off all that fat are pretty much limited to two options: Diet to lower the amount of energy consumed, or exercise to increase the amount of energy the body needs.

Most current antiobesity drugs work on the diet half of the equation, helping people limit calories by dampening appetite or by interfering with the digestion of food. Approaches that knock down cravings are based largely on research in the 1990s that worked out some of the biological underpinnings of hunger.

More recently, though, experiments have deepened scientists' understanding of the way fat locks up and releases surplus calories — providing hope that future therapies may offer a kind of virtual exercise. While there's still no getting around the laws of thermodynamics, scientists are getting closer to finding ways to trick fat cells into releasing their stockpiled fuel.

One day — maybe not soon, but eventually — medical science might even offer pills that activate the body's fat-burning machinery without a trip to the gym (in ways that today's promoters of "fat-burning" products can only dream about).

"I see great opportunities," says vascular biologist Yihai Cao of the Karolinska Institute in Stockholm. Cao studies how fat tissue remodels the circulatory

system as it expands, with the idea of inhibiting blood vessel growth to arrest fat expansion. Recent findings have revealed other secrets about fat as well — among them, the discovery of molecules that help inform fat cells when a person is exercising, and insight into the mysterious role that calorie-burning brown fat plays in weight control. These and other findings could lead to new ways to dissipate fat long after it entrenches itself in the body.

No approach is far enough along to predict how well it might work in people, or what the side effects might be. Despite the popular perception of fat — that it sits idly on the belly and thighs, doing nothing until it is used — fatty tissue is a dynamic, complex and necessary component of life. Any fat-fighting drug has to overcome daunting safety and logistical hurdles, especially since it would have the potential to be used, or misused, by millions. A therapy that attacks fat too broadly could get rid of one health problem only to create a new one.

A body needs fat "not only for energy storage, but also for the hormones it makes," says Joel Elmquist of the Taskforce for Obesity Research at the University of Texas Southwestern Medical Center at Dallas. Fat cells play a role in metabolism and even in the ebb and flow of immune cells. "They are key in regulating almost every system in your body," Elmquist says.

By weight, fat is also one of the most abundant types of tissue in the body. Scientists know that fat grows when existing cells enlarge and when new cells get created. And a bit of bad news here: The number of fat cells can go up, but not down. During weight loss, existing cells shrink. Scientists once thought that fat cells never went away, but a study published in *Nature* in 2008 demonstrated that a small percentage of them do eventually die. The problem is, the body quickly replenishes them with new ones.

And a bit of bad news here: The number of fat cells can go up, but not down.

Bring on the brown

Other recent findings have also challenged traditional thinking about fat. Perhaps most surprising has been the discovery that adults have a kind of fat that actually burns calories as well as storing them. Adipose tissue, which is mostly a conglomeration of fat cells, comes in two types — white and brown. White fat is the main energy-storage depot, and is the kind of adipose that makes your belt too tight. Overweight mammals — humans are not the only ones — have an abundance of white fat. Brown fat is found in infants (though they also have those adorable rolls of white fat) and has the ability to burn energy — primarily, researchers think, to generate body heat. "That's why you have relatively more brown fat in small rodents and newborn babies," says Stephan Herzig, head of molecular metabolic research at the German Cancer Research Center in Heidelberg. "Those little creatures use the brown fat to maintain temperature."

Brown fat gradually erodes with age. Scientists once thought that adults had only rudimentary, inactive deposits between their shoulder blades, if any at all. But in April 2009, three research papers in the *New England Journal of Medicine* (followed by a fourth in July in the journal *Diabetes*) confirmed that adults can indeed possess metabolically active brown fat (*SN: 5/9/09, p. 10*). Taken together, the reports make the case that brown fat helps control body weight, raising the tantalizing possibility of fighting fat with fat.

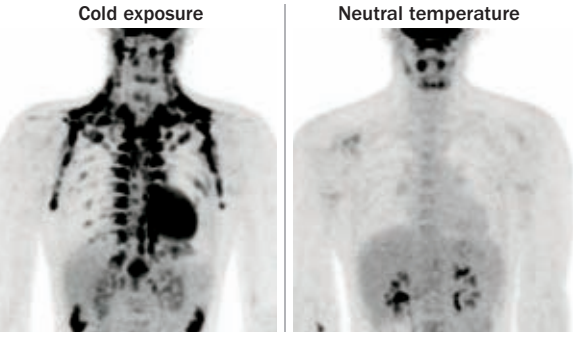
Though the true role of brown fat in adults is still a subject of investigation, its presence might help explain why some people gain weight more easily than others, or why a propensity for obesity tends to increase as people age. In general, the older you are, the less brown fat you have. "If we were able to increase either the mass or the activity of brown fat in adults, in particular in obese adults, that might be a safe and effective way of using the fat stores in the white fat deposits," Herzig says.

One of the *New England Journal* papers described a way to do this — with cold. Dutch researchers studied 24 healthy men, 14 of whom were overweight or obese, and exposed each to two hours of mild chill (about 16° Celsius). Using PET scans to measure the activity of brown fat in the men's bodies, the scientists found energy radiating from the fat, presumably to help hold body temperature.

What's more, the brown fat in lean volunteers used more energy in the cold than did the brown fat in obese men. "Brown adipose tissue may be metabolically important in men, and the fact that it is reduced yet present in most overweight or obese subjects may make it a target for the treatment of obesity," the researchers wrote.

Other than joining a polar bear club, there's no obvious way to boost your brown fat activity. In May, in the journal *Science*, Herzig and his colleagues reported that the enzyme COX-2, which is involved in many body processes, plays a role in turning white fat brown. He and his colleagues described experiments in which they rebooted white fat in mice, turning it brown, after increasing

Brown fat heats up Calorie-burning brown fat shows up (left, black areas) in a PET-CT scan of a man after two hours in a chilly 16° Celsius room but is not apparent in a scan at 22° C, or room temperature. Scientists hope to boost brown fat for weight loss.



W.D. VAN MARKEN LICHTENBELT *ET AL./NEJM* 2009

the animals' exposure to COX-2 and mimicking the physiological changes caused by cold. Even more important, mice with new deposits of brown fat lost weight.

These and other experiments have generated intense interest in the creation of brown fat. Still, "It's naïve and wrong to say we're going to make your white fat disappear," says cell biologist Bruce Spiegelman of the Dana-Farber Cancer Institute in Boston.

Spiegelman's laboratory has revealed much about the biology of brown fat (*SN: 8/29/09, p. 9*), including the discovery that it shares a cellular origin with muscle cells, a finding that helps explain brown fat's energy-burning qualities (muscle being an energy-hungry tissue). He and others are now homing in on the exact genetic switches that prompt the body to make brown fat, with the hope that drugs might one day trigger those genes.

One of the genes of interest is called *PRDM16*. In 2008, Spiegelman and his colleagues reported in *Nature* that activating the gene in myoblasts — immature cells that have not yet committed to becoming muscle or fat — spurred the cells to turn into brown fat. Similar results have been found in other precursor cells for fat. "Certainly natural or synthetic compounds that can induce *PRDM16* in white fat precursors or in myoblastic cells could have great value in human metabolic disease," the researchers wrote in *Nature* in 2009.

Treadmill pills

Brown fat is not the only potential scheme for improved thermodynamics. In one instance, scientists at the Salk Institute in La Jolla, Calif., startled even themselves with experiments in 2008 on an enzyme, called AMP kinase, that is triggered during exercise. To the researchers' surprise, their studies suggested a way to make lazy cells burn energy as if they were exercising.

One of AMP kinase's main jobs is to send out a signal to adipose tissue to release its cargo for muscles to use. A few years ago, Salk molecular biologist Ron Evans and his collaborators began to ask themselves if they could mimic the action of AMP kinase without exercise, in the hopes of tricking fat cells into thinking the body needed energy.

Turns out, there are at least two ways. Experimental drugs known as AICAR and GW1516 can reprogram inactive muscle cells to behave as if they were exercising. Chemically, AICAR looks a lot like AMP kinase. It is similar enough that after mice are given AICAR, they are able to run on a treadmill an average of 44 percent farther than untreated mice, just as if their bodies had undergone the conditioning of repeated exercise. "The fact that a drug could do that was pretty remarkable," says Evans, who reported his results in 2008 in the journal *Cell*. "I was pretty stunned."

The second drug, GW1516, activates a genetic switch in a cell's nucleus that's also triggered during exercise. When that drug was given to mice that exercised regularly — and therefore also had their AMP kinase on board — endurance rose 68 percent. It appears the activation of AMP kinase by AICAR fooled the body into believing it had exercised or, in the case of GW1516,

Burn, baby, burn The natural fat-burning process is complex, but ultimately it's all about turning stored fat into energy that's used when the body exercises or goes without food. Muscles, the liver and even fatty tissue itself are all involved in this process in some way, described below. A few of the ways that scientists are looking to boost fat-burning are also shown (tan boxes).

Muscles

During exercise, cells produce AMP kinase, an enzyme that tells fatty tissue to release its stores for muscles to burn.

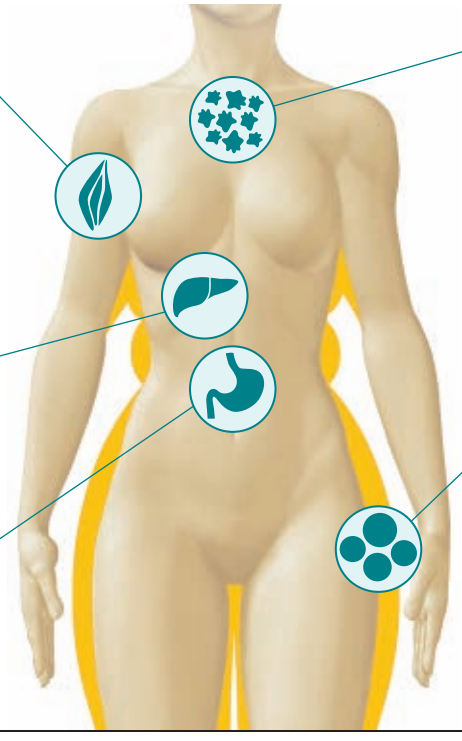
Fake exercise Scientists hope to mimic the action of AMP kinase and other molecules the body releases during exercise to trick the body into releasing energy stored in fat.

Liver

The liver stores glucose from food as glycogen and releases it into the bloodstream when energy is needed. Once glucose runs out, the body starts to burn fat.

Stomach and intestines

After eating, the presence of food in the gut stimulates the pancreas to make insulin. Insulin triggers cells to take up glucose, which is burned for energy, and to store excess fuel.



Brown fat

Brown fat cells are loaded with mitochondria, energy powerhouses that can generate heat. When the body is cold, fat molecules in brown fat are broken down in mitochondria and their energy is released as heat.

Enhance brown fat Researchers are studying genes that tell precursor cells whether to become muscle or brown fat and are also studying the role of the COX-2 enzyme in turning white fat to brown.

Fatty tissue

An enzyme called lipase, made in the blood vessels of fat tissue, helps break up white fat and release it into the bloodstream as both glycerol, which is harvested for energy in the liver, and fatty acids, which are used by muscle.

Starve fat One day, drugs may inhibit angiogenesis, the formation of blood vessels that nourish fat tissue, to arrest fat expansion.

FRANK GEISLER/ALAMY; ICONS: T. DUBÉ

exercised more than it actually had.

Drugs that could activate the fat-burning machinery triggered by exercise might have few harmful side effects, Evans says. Exercise already has a global effect on many body systems, almost always for the better. "Most drugs are designed to inhibit something, to block something in a cell from occurring," he says. Drugs that throw up cellular roadblocks to a biological reaction cause side effects when that same process has a day job in some other tissue. But, he says, a drug that promotes the effects of exercise would flip on chemical pathways that the body already uses regularly. "There's a benefit to activating a natural pathway," he says.

Starving fat

There may also be a benefit to inhibiting the mechanisms that adipose tissue uses to expand itself. For example, some scientists have become interested in the idea of arresting angiogenesis, the growth of new blood vessels into developing tissue. The idea of slowing angiogenesis has gotten attention in cancer research with the introduction of drugs that impair the ability of tumors to develop a new blood supply, in the hopes of simply starving the malignancy to death. And now it has become clear that adipose tissue, like cancer, feeds itself oxygen with new blood vessels.

In 2002, cardiovascular researcher Maria Rupnick of Brigham and Women's Hospital in Boston — who had done some of her graduate training in cancer angiogenesis — began to explore whether fat growth would be a good way to study angiogenesis in healthy tissue. "Fat is a normal organ that expands or shrinks depending on the body's needs," she says. She began to test the effects of cancer drugs that inhibit angiogenesis on obese mice. "Anything we had on the shelf, we tested," she says. "All the animals lost weight."

Fat tissue, like cancer, has a voracious need for oxygen. "It can't expand without expanding its blood vessels, just like a city can't expand without expanding its roads," Rupnick says.

Today's antiobesity drugs There are two ways to lose fat without surgery: exercise or diet. While scientists are looking for ways to spur fat-burning processes, today's prescription diet pills (top sellers listed) generally suppress appetite to reduce the formation of fat in the first place.

	How it works	U.S. sales, 2009
Phentermine	Appetite suppressant; increases levels of the brain chemical norepinephrine, which is involved in the flight-or-fight response and reduces hunger.	$36.3 million
Sibutramine (Meridia)	Enhances the availability of the brain chemicals serotonin, dopamine and norepinephrine in the brain, reducing appetite.	$35.8 million
Phendimetrazine	As a sympathomimetic amine, works similarly to an amphetamine, stimulating the central nervous system to increase heart rate and blood pressure and reduce appetite.	$7.5 million
Diethylpropion (Tenuate)	Also works as a sympathomimetic amine.	$6.5 million

SOURCE: IMS HEALTH

Research in the years since has led to experimentation with almost a dozen antiangiogenesis drugs, many of which are already approved for cancer treatment. In the February issue of *Nature Reviews Drug Discovery*, Cao reports that all but one have led to weight loss or slowed weight gain in mice. However, he concedes that research is still preliminary, and the relationship between adipose tissue and blood vessel growth is highly complex. For example, while angiogenesis may assist the growth of white fat, it promotes energy expenditure in brown fat — so interfering with angiogenesis in brown fat might impair weight loss. "We are learning," he says.

To market

Encouraged by recent findings — and the scent of a huge market — drug companies are already beginning to test potential fat-fighting drugs in people. The experiments highlight just how much remains to be understood about fat. Zafgen, a biotech firm based in Cambridge, Mass., sought to develop an antiangiogenesis obesity drug, only to discover that its candidate drug might work in an entirely different way. At high doses, the drug, called ZGN-433, indeed inhibits angiogenesis. But the company now says it is testing the compound at a fraction of the amount needed to inhibit angiogenesis, and at those levels rodents appear to lose weight and eat less even though the blood supply in the adipose tissue looks unaffected.

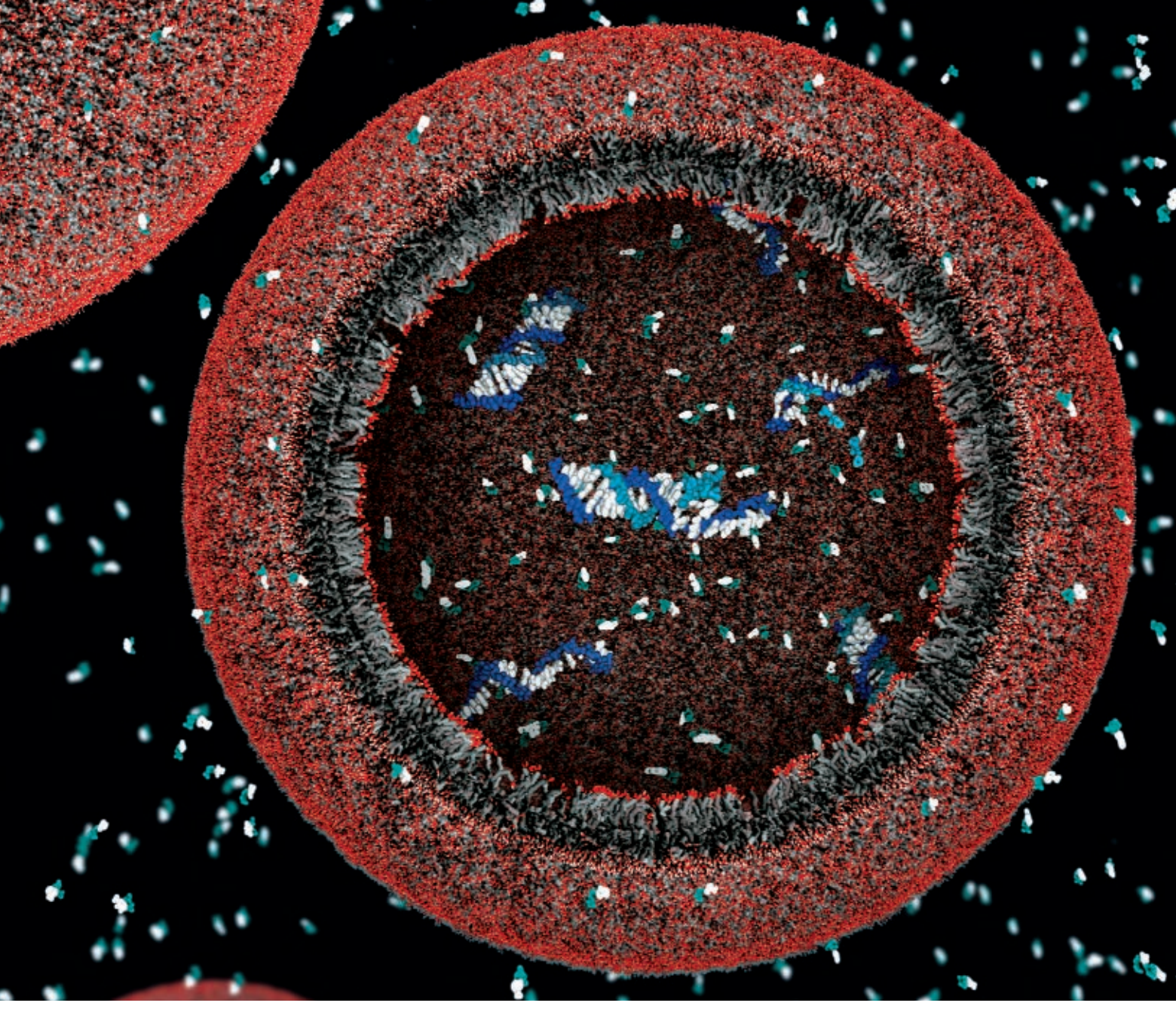
Zafgen is investigating how the drug might work — it appears to affect metabolic machinery in the liver — and is sponsoring a test in people. One day, says CEO Tom Hughes, such a drug could be distributed through the kind of centers that specialize in surgical weight loss, or bariatrics. "If this drug works the way it does in animals, we see it as being a pharmacological alternative to bariatric surgery," he says. He expects to have the first data from human studies by the end of this year.

Researchers know well that antiobesity drugs have had a long and often disappointing history. Many a drug has launched with fanfare only to later shipwreck on its own safety concerns. In January, for example, European regulators said doctors should stop prescribing the drug Meridia, which helps control appetite, because of evidence that it raises risk of heart attacks and strokes in people with cardiovascular disease. Then in May, federal officials changed labels on the antiobesity drugs Xenical and Alli to warn consumers about a rare risk of severe liver injury.

Setbacks in the war against obesity should not be too surprising. For at least a couple hundred thousand years, human biology has been perfecting a means to hold onto fat. It will not easily let go. ■

Explore more

■ E. Ravussin and L.P. Kozak. "Have we entered the brown adipose tissue renaissance?" *Obesity Reviews*. May 2009.



Life from scratch

Relaunching biology from the beginning

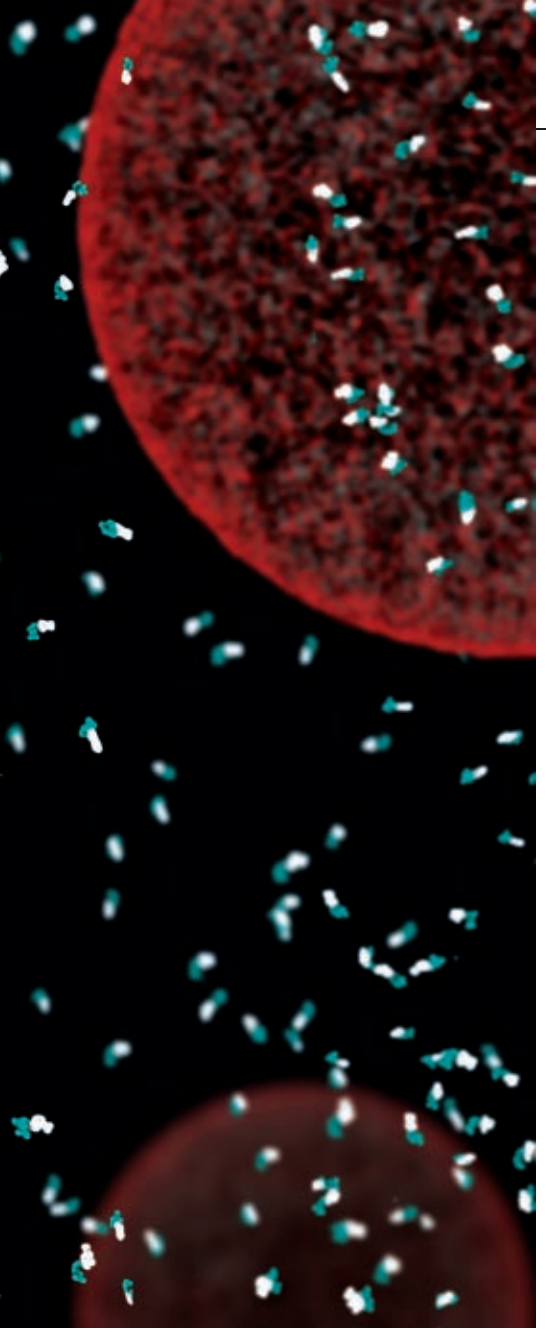
By Charles Petit

A short stroll from Boston's Charles River, behind a sheath of blue glass on the seventh floor of a Harvard Medical School research building, Jack Szostak is getting set to replay the greatest event on Earth.

He and his 15-member team of graduate students and young postdoctoral research fellows are well on their way to starting biology from scratch — more than 3.5 billion years after it first emerged.

The feat would qualify as creation of life in a test tube if it weren't for one thing: Szostak's lab does not rely much on test tubes. "I know exactly where it will happen," said postdoc Alonso Ricardo, from Cali, Colombia. It will most likely be in a 1.5-milliliter tapered plastic centrifuge tube "smaller than my little finger." And unlike the first time — when life formed on its own — the second time it will get a boost from human ingenuity and the lab's elaborate organic chemistry equipment.

JANET IWASA



By creating vesicles containing protogenetic material (depicted in this computer graphic), researchers are trying to watch life pop into existence.

Szostak's endeavor is very different from another artificial life project led by biologist and entrepreneur J. Craig Venter. Venter's team is using chemical sequencing machines to make a panoply of genes for the highly evolved parts of a modern microbe. Recently he and his colleagues announced that they had inserted an entire genetic blueprint, modeled on a known microbe but built from scratch, into a microbe of another species where the synthesized DNA took over (*SN: 6/19/10, p. 5*). Venter's ultimate aim is to build designer organisms with novel and fully contemporary genomes.

Szostak has a far more fundamental aim: to show how unguided natural events might have led to life on Earth in the first place, and to show how the scenario might also play out in myriad other places in the universe. Like bookends on a long row of volumes, the two exercises would frame the story of evolution so far.

In his neat corner office outside the rows of lab benches and work bays, the 57-year-old biochemist leaned forward and explained a deep motivation: "What we'd like to see is, from initial chaos and randomness, how something useful emerges. What we are trying to do, to understand, is how Darwinian evolution can emerge from chemistry.... If we can get a self-propagating chemical system that can evolve, yeah, I'd call that life."

A place to start

Szostak brings a lot of tools to the project. He has already made his mark on biology throughout a career puzzling over and exploring the workings of DNA and its cousin, RNA. He was a winner last October of the Nobel Prize in physiology or medicine, along with Elizabeth Blackburn of the University of California, San Francisco and Carol Greider of Johns Hopkins University School of Medicine in Baltimore. In the 1980s they showed how telomeres, distinctive caps on the ends of chromosomes, protect a living cell's DNA and genes from degradation.

Szostak, a U.S. citizen now, was born in London, where his father was stationed with the Royal Canadian Air Force. After returning home, Szostak enrolled in McGill University in Montreal at age 16. At 19 he took his degree in cell biology to graduate school at Cornell University. He dove into genetics. *Nature* published an extract from his biochemistry Ph.D. thesis — on synthetic RNA. At 26 he joined Harvard's faculty, where he is now a professor of genetics and a Howard Hughes Medical Institute investigator.

By the mid-1980s, electrifying word swept the field of DNA and RNA research. Tom Cech of the University of Colorado at Boulder and Sidney Altman of Yale University independently discovered that RNA — believed to be a mere messenger, carrying genetic blueprints from DNA-based genes to cellular machinery for making proteins — had another trick. It could fold into complex shapes, forming an enzyme that vastly speeds up the natural rate of some reactions (*SN: 11/27/82, p. 342*). Until then, the only known enzymes were specialized proteins. How life had first made proteins without enzymes, which presumably had to be proteins themselves, had been a chicken-and-egg conundrum.

But the discovery that RNA can be an enzyme, dubbed a ribozyme, potentially able to boost its own replication, led to fresh ideas about how life might have started. Cech imagines an early "RNA world," where life could have taken its first steps while ignoring all the amino acids floating in the prebiotic soup. Only later, with ribozymes as tools, need life have developed a genetic code for stringing amino acids into proteins — gradually turning metabolic duties over to those proteins.

Ten years ago Szostak and Pier Luigi Luisi, then at the Swiss Federal Institute of Technology in Zurich, settled an argument by writing a paper together. They had been debating the order of important steps in the origin of life. Luigi Luisi favored the membrane first: It would hold a primitive "protocell" together and keep its vital chemicals concentrated. Szostak figured that genetic machinery able to copy itself and assure that its features get passed from generation to generation was paramount. The team's paper, "Synthesizing Life," written with David P. Bartel of MIT's Whitehead Institute for Biomedical Research, came out in *Nature* in early 2001. It split the difference, arguing that life would need both at the start: a whole cell. Its second paragraph asked "How simple can a cell be and still be considered as living?"

The researchers conceptually stripped it to almost nothing. On the outside, a uniform membrane of simple

and ubiquitous lipid, or fat, molecules. Inside, a few strands of nucleotides, some possibly folded into ribozymes.

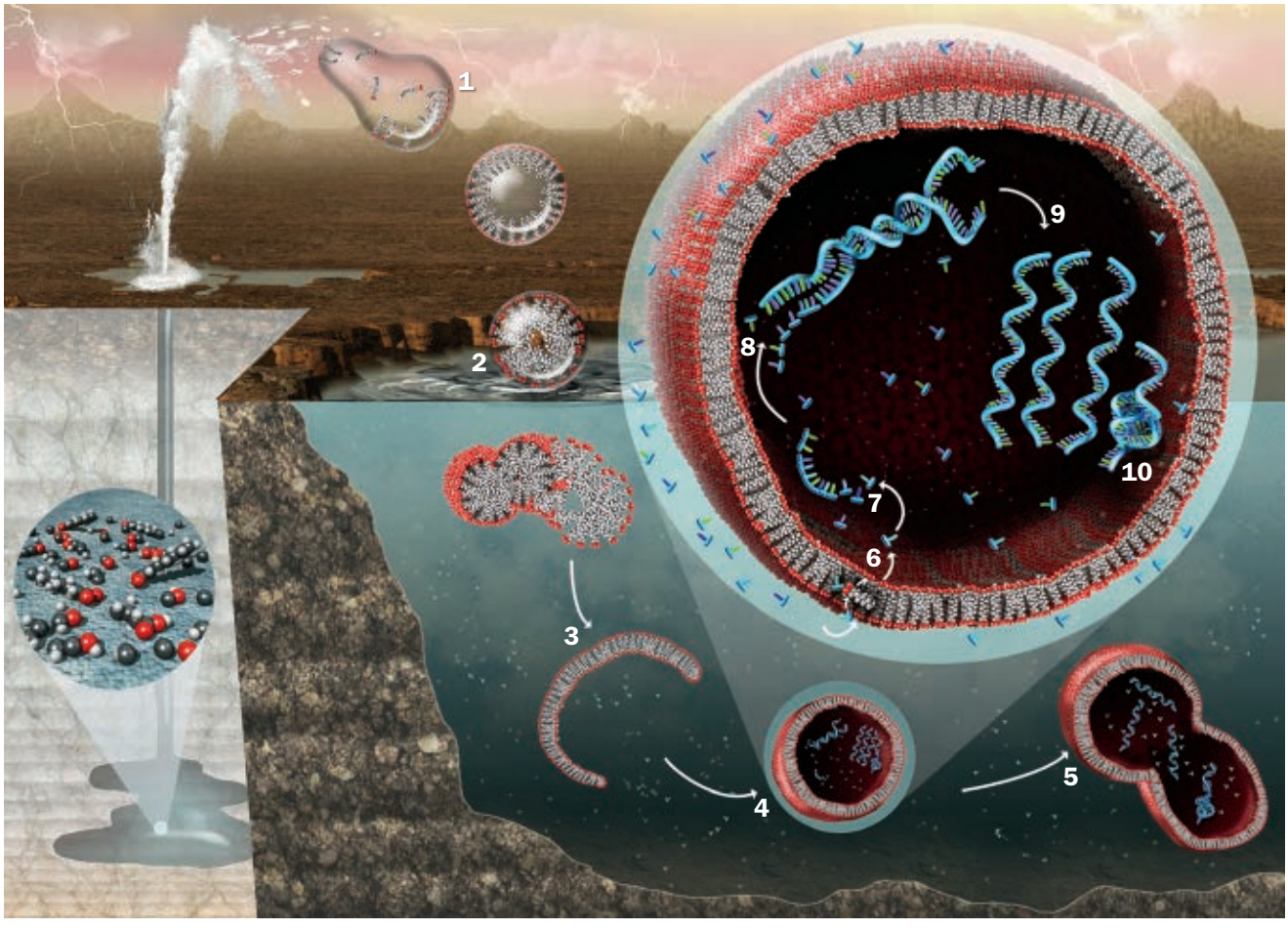
"After we wrote that paper I figured, well, we put these two ideas together, so we'd better do some experiments," Szostak recalled.

Szostak and his team already have a stripped-down system for making tiny hollow spheres, or vesicles, from simple lipid membranes. And lean and mean genetic material is available from simplified versions of today's RNA. The researchers' molecules are showing signs of being able to assemble, replicate, mutate, and be led by the power of natural selection toward complex metabolism.

Such work has helped put Boston and Cambridge among the elite centers of studies of life's origin, and Szostak is now devoted essentially full-time to synthesis of life. "They won't get it done in 2010, but with some good luck and a lot of work it could in the next year or two," says Gerald Joyce, an RNA specialist at the Scripps Research Institute near San Diego, a traditional powerhouse for origins work. Joyce's specialty is the creation of ribozyme systems that replicate and even evolve somewhat — on the verge of life, but not quite there. Of Szostak's work, he says: "They have some really hot chemistry going there. If they can rev it up some more, it — life — will just start working. Once you light Darwinian evolution, it takes off." Others guess it may be five or 10 more years. Szostak won't play that game but says, "How old am I? I think I have time."

Szostak also doesn't care for guessing exactly how life happened the very first time and then duplicating it. Once one system works, he figures the experience will generate clues to other ways. And he doesn't think his work will settle an old argument among pioneers in the field: At what locale did life first take root? "I think it might have taken them all," he

Before the cell By simplifying a cell to only its necessary parts — a fatty acid envelope and RNA-like strands — some scientists are exploring how protocells could have formed from scratch on the early Earth, and then evolved into something more complex.



Vesicle-bound Fatty acids formed in a geyser can be released in water droplets and congregate as the water evaporates (1). By organizing so that their water-loving ends point out, the fatty acids can form organized clumps called micelles (2). Micelles would then join together to form a sheetlike bilayer (3). Random fluctuations could cause that bilayer's ends to connect, eventually forming a hollow sphere (4). As more fatty acids join, the newly formed vesicle will become unstable and could split in two (5).

Roots of genetics The vesicle's fatty acids constantly flip, allowing them to pull in nucleotides (6). Once in the vesicle, single nucleotides can link into strands (7), which can build a double helix as complementary nucleotides attach (8). Temperature fluctuations could cause the strands to split, each again forming another strand, resulting in two copies of two strands (9). In some cases, a strand will fold just right to form a ribozyme, which can speed replication and introduce variation (10).

NICOLLE RAGER FULLER

says. These include a "warm little pond" as Charles Darwin proposed, or the oceanic prebiotic soup suggested independently by Aleksandr Oparin and J.B.S. Haldane in the 1920s, or deep-sea hydrothermal vents, or wave-washed beaches, or lightning-lashed volcanoes, or terrestrial hot springs or other hot spots.

Protolife plays out

Wherever life first flickered into being, it may have moved rapidly to occupy a wide range of environments. In a paper published in April in *Annual Review of Biophysics*, Szostak and his graduate student Itay Budin argue that once even the crudest living system appears, "there is a nearly limitless set of plausible adaptations that would be beneficial for early cells." In a recent speech in Florida at a meeting of the Lasker Foundation, which awarded Szostak, Blackburn and Greider its annual prize for basic medical research in 2006 (an example of why Laskers are tips to future Nobels), Szostak said he expects to see firsthand "an evolutionary arms race" among his first protocells as they compete and evolve ever-superior tools for survival.

For decades, science has known that raw materials for life were surely abundant on early Earth. In a famous 1953 experiment at the University of Chicago, chemist Harold Urey and, chiefly, his graduate student Stanley Miller showed that a primitive atmosphere of methane, ammonia, water vapor and hydrogen, when blitzed by a lightninglike spark, produced a residue rich in amino acids — building blocks of proteins. The demonstration set off a wave of experiments showing that other conditions can do it too. And not only here. Outer space also seems loaded with the Lego blocks of life. Meteorites can contain thousands of organic molecules, including amino acids.

Further and vital reassurance to Szostak's assumption that the needed chemistry was readily available to the first life came early last year. At the University of Manchester in England, chemist John Sutherland and his team reported discovering a natural set of reactions by which ribonucleotides, the fundamental units of RNA, could have built up on the young, lifeless Earth.

"It's almost impossible not to make amino acids, sugars, lipids, alcohols and almost anything you can think of," says geochemist Robert Hazen of the Carnegie Institution for Science in Washington, D.C. "There is almost too much stuff, too many possibilities, to narrow it down to just one idea."

What is firmly supposed is that somehow, somewhere beyond 3.5 billion years ago, before the first signs of microbial life appear in the fossil record, and after about 4.2 billion years ago, when the planet had cooled and became habitable, a vesicle wrapped itself around a few pieces of chained nucleotides. Those nucleotides hooked up into strands as more seeped inside. And, by higgledy-piggledy chance after uncounted similar episodes that went nowhere, some of these innards took the form of a ribozyme with the power to speed reactions, including replication. The protocell divided. It carried the protogenetic material into further generations, the occasional copying mistake opening the door to evolution.

"This is a molecular biology project, but as a practical matter we are almost a full-time organic chemistry synthesis operation."
JACK SZOSTAK

These steps are the ones Szostak's team hopes to watch unfold. The strategy is simple — find molecules that can do the necessary replication quickly. Executing it is difficult — nature had millions of years and an enormous number of ponds, springs, beaches and oceanic hot spring vents to work with. "This is a molecular biology project, but as a practical matter we are almost a full-time organic chemistry synthesis operation," Szostak says.

The project's achievements so far can be broken down in terms of a few larger tasks:

THE MEMBRANE: Modern cell membranes are tough, and highly protective. Bristling with receptors and gates, these layers don't let just anything leak in or out. Their bricks and mortar are phospholipid molecules — so named because of a prominent phosphorus group that provides stability and strength. One end of each molecule is attracted and the other repelled by water molecules. This split personality leads them to form roughly spherical, closed membranes in two layers, with the water-friendly ends all facing the exterior and the water-shy ends tucked along the membrane's interior seam.

Because simpler membranes are more likely to have formed from the natural organic chemistry factory on prelife Earth, the protocell project is working on plainer lipids — chiefly ordinary fatty acids. Like phospholipids, these simpler fatty acids form double-layered protocell walls that keep large molecules in. But unlike phospholipids, they allow nucleotides, the small building blocks of genetic material, to pass through.

THE GENETICS: Making a system that can duplicate itself without modern life's elaborate protein enzymes is the project's biggest hurdle, but progress has been dramatic.

DNA and RNA are made of nucleotides. Each unit is a three-part molecule with a phosphate at one end, a sugar in the middle and a variable nucleobase at the other end. The nucleobases come in various sorts — providing the "letters" of the genetic code and also providing the chemical bonds that let one DNA strand cling to another to form the famed double helix. Szostak's team plans to get a random RNA or similar molecule in the vesicles, let more nucleotides enter one by one and, by linking to the first one, produce two linked strands of an RNA-like molecule. Temperature oscillations or chemical changes should cause complete double helices to come apart, each then rebuilding the whole as fresh nucleotides link on.

But with normal RNA this process is too slow. Ricardo is experimenting with closely related, more reactive and reliable nucleotide systems. "We just tweak

the RNA here and there, change a few atoms," Ricardo says.

One called TNA replaces RNA's ribose sugar with a sugar called threose. Another variant is called morpholino NA. This list goes on, with many more to try. The self-assembly and replication is going better but has not yet been able to reliably and speedily duplicate lengths of genetic material long enough — many dozens of nucleotide units — to fold into long polymers. But the strands are getting longer all the time.

GROWTH AND EVOLUTION: Graduate student Ting Zhu, a native of Shanghai, is the lab's vesicle man. When he joined the group a few years ago the fatty acid lipid vesicles were already standard. Szostak wanted to know how to make solutions of same-size vesicles and to see how fast they could grow. Zhu, a mechanical engineering graduate from MIT, first invented a tiny sorting machine. Szostak suggested he study in detail how the membrane grows by incorporating new fatty acids, a step at a time.

Zhu had a simpler idea: He dumped a high concentration of micelles — small balls of huddled fatty acids — into a suspension of vesicles. He put them in the lab's Nikon TE2000S inverted epifluorescence microscope — a device big enough to fill a closet — and watched a most remarkable thing happen. The vesicles writhed. They grew what looked like wriggling hair. They eventually transformed themselves from round balls into long filaments. Their membranes had grown so fast that they couldn't absorb fluid quickly enough to stay plumped up.

Soon Zhu discovered that simple shaking of the fluid broke the filaments into a new generation of vesicles without spilling their interior contents — and he has shown it can be done while the vesicles are full of nucleotides.

Further work has revealed that simple alcohol buffers can cause each filament to form what looks like a string of pearls that then floats free, forming a new generation. It became clear that the knotty problem of getting protocells to divide might have a simple enough answer: a stew of raw materials to transform the cells into long cylinders and changing turbulence or chemical conditions to split those filaments.

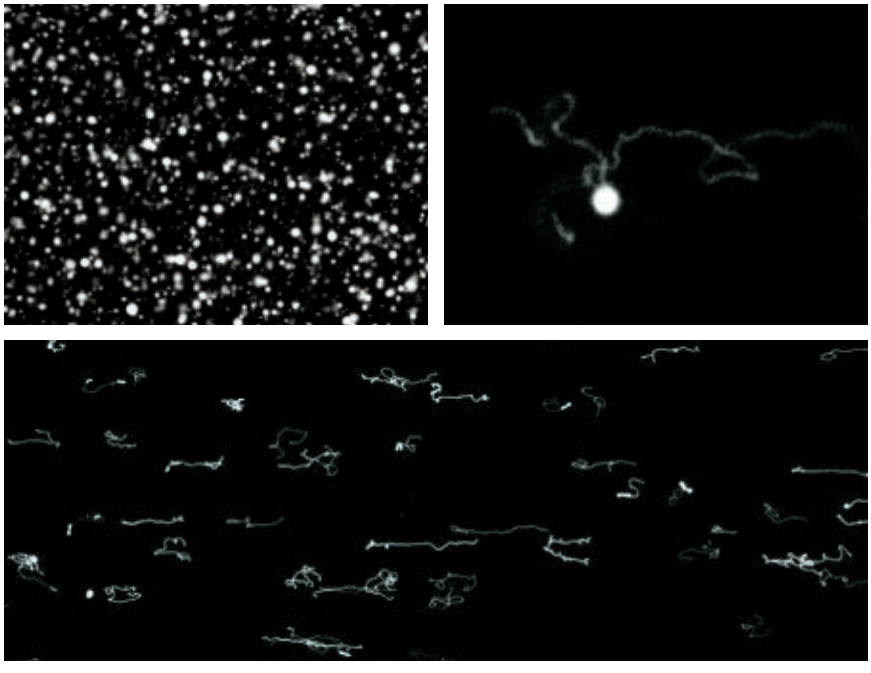
The team sees two ways, if the above steps work, to witness selective, Darwinian pressure alter protocell behavior. For only if the protocells can evolve might they merit status as living cells.

Among discoveries in the lab over recent years is that when a vesicle's cargo of nucleic acid polymers fills up to the point it strains the cell's wall, the membrane will greedily absorb any fatty acids floating by. Cells whose interiors grow the fastest then should, in competition with others, take the lion's share of available lipids, eventually eliminating their competition. Thus, any random changes in the interior, fledgling ribozymes that could speed up nucleic acid replication rates will spread rapidly through an evolving population.

Alternatively, another Darwinian tableau might see the random development of ribozymes able to add phospholipids, the stuff of modern membranes, to outer fatty acid membranes. The result would be more stable structures superior to other cells, also able to hang on more tenaciously to any fatty acids they acquire. Again, those with the most advanced membranes would dominate — up to a point.

This scenario, Szostak says, has a twist. If the phospholipid proportion rises too high, it will prevent small nucleotides from getting inside, starving the genetic machinery of new materials.

The solution for the cell is to find a way to put gates or pores in the membrane that allow fresh nucleotides in. How that might happen is sheer speculation. But Szostak is sure that, given enough time and enough trials, protocells made with his general formula will almost certainly evolve new characteristics that give them an edge over others. And when that happens, Szostak may finally see how, from initial chaos and randomness, something useful emerges. ■



Eerily lifelike Researchers have already seen that a sea of fatty acid vesicles (upper left) can develop spontaneously in the lab. Such vesicles can grow, become unstable and form long filaments (upper right and bottom). Those filaments can break, suggesting a way for protocells to divide.

ALL: T. ZHU AND J. SZOSTAK; TOP LEFT: *PLOS ONE* 2009; BOTTOM: *JACS* 2009

Explore more

- For more on life's origins, visit exploringorigins.org/index.html
- To watch Jack Szostak's lecture to the Lasker Foundation, visit www.laskerfoundation.org/learn/video.htm
- Listen to a talk by Pier Luigi Luisi at www.plluisi.org/research.html (Scroll through the intro, in Portuguese.)

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The Truth Hurts

Scientists question voice-based lie detection

By Rachel Ehrenberg

Truster-Pro and the Vericator may sound like devices Wile E. Coyote would order from the Acme Co., but they are real technologies for detecting lies. Unlike the traditional polygraph, which zeroes in on factors such as pulse and breathing rate, these analyzers aim to assess veracity based solely on speech.

Police departments shell out thousands of dollars on such devices — known collectively as voice stress analyzers — in an attempt to tune in to vocal consequences of lying. Airports are considering versions for security screening purposes, and insurance companies may employ the polygraph alternatives to detect fraud.

But beyond their crime-fighting objective, these tools have something less noble in common with their predecessor: a poor track record in actually telling truth from deception.

Scientists evaluating Truster-Pro, the Vericator and newer analyzer models repeatedly report lackluster results. Now research finds that two of the most commonly used voice stress analyzers can discern lies from truth at roughly chance levels — no better than flipping a coin.

"Quite frankly, they're bogus. There's no scientific basis whatsoever for them," says John H.L. Hansen, head of the Center for Robust Speech Systems at the University of Texas at Dallas. "Law enforcement agencies — they're spending a lot of money on these things. It just doesn't make sense."

A lackluster alternative

Many agencies have been seeking alternatives to the polygraph, especially following a 2003 National Research Council report that concluded that the physiological responses measured, such as increased heart rate, can identify stress but not pinpoint deception. Champions of voice stress analyzers often cite this report among other criticisms of polygraphs as a reason to switch to voice-based lie detection. The National Institute for Truth Verification — a company based in West Palm Beach, Fla., that makes a widely used device called the Computer Voice Stress Analyzer — has a page on its website dedicated to denigrating this traditional lie detector, titled "Polygraph Failures Continue to Mount."

But the institute fails to mention the same report's conclusions about alternatives to the polygraph, including voice analyzers. Research offers "little or no scientific basis for the use of the computer voice stress analyzer or similar voice measurement instruments as an alternative to the polygraph for the detection of deception," the report noted.

As with the old lie detector, creators of voice analyzers usually avoid direct claims that the units detect deception, speech perception expert James Harnsberger said in April in Baltimore at a meeting of the Acoustical Society of America. Instead, the developers contend that physiological changes that occur when someone is lying trigger consistent, readable changes in voice. "There's

an assumption that there's a direct mind-mouth link," said Harnsberger, of the University of Florida in Gainesville.

Speech does in fact change when a person is under stress, both in frequency and in the amount of time spent on segments of words, says Hansen. But, as with the polygraph, distinguishing stress related to deception from stress related to fatigue, anxiety or fear is not so easy.

"No one has identified an acoustic signature that is unique to deception," says Mitchell Sommers, director of the Speech and Hearing Laboratory at Washington University in St. Louis.

Two large studies, one conducted in a jail and another in a lab, suggest that the two most widely used voice stress analyzers haven't pinpointed such a signature, either.

One voice analyzer — Layered Voice Analysis, created by the Israel-based company Nemesysco — purports to use more than 8,000 algorithms to tune in to three states of mind: excitement, stress and cognitive dissonance (the psychological discomfort that comes with holding two conflicting views at once). A second, the Computer Voice Stress Analyzer, claims to detect inaudible changes in "microtremors" in the voice of a lying person. Versions of both systems can cost more than $10,000 with training.

Numbers speak truth

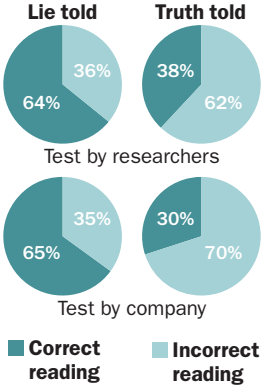
In the jailhouse study, researchers led by Kelly Damphousse of the University of Oklahoma in Norman interviewed a random sample of 319 arrestees during booking in an Oklahoma county jail. The team asked the men about recent use of drugs, including cocaine, marijuana, PCP and methamphetamine, and researchers dissected responses with both voice analyzers. After the interview, the arrestees' urine was tested for actual drug use.

Both voice analyzers got poor marks, write Damphousse and his colleagues in a 2007 report for the Department of Justice. All told, fewer than one-sixth of the lies were detected. LVA spotted about 10 percent of lies, while CVSA got nearly 20 percent. They were better at detecting truths, correctly identifying between 85 and 95 percent. The remaining truths were still falsely labeled as lies.

The technologies didn't fare much better in the lab, Harnsberger reported at the Acoustical Society meeting. As part of a team of Florida researchers, Harnsberger underwent training for both technologies. Working with company representatives, the researchers conducted a study where subjects were video recorded telling the truth and telling lies under various levels of stress. For a very high-stress lie, the participants were asked to make a statement that they strongly disagreed with; topics included sexual orientation and gun control. Participants were also told that the video would be shown to their peers and that they should expect an electric shock during the statement.

Lying in the lab

The Computer Voice Stress Analyzer couldn't reliably detect a lie (left) or the truth (right) in a lab environment.



SOURCE: H. HOLLIEN *ET AL./JOURNAL OF FORENSIC SCIENCE* 2008

One technology caught lies at rates similar to chance, and the other did somewhat better, Harnsberger and colleagues reported at the meeting and in two papers in the *Journal of Forensic Science*. But both detectors also falsely labeled true statements as lies at similar rates. These false positives, which are often unreported in studies and left out of company descriptions of the technologies, are key for evaluating merit, Harnsberger noted.

"A common mistake is to only report how many lies were successfully detected," Harnsberger says. "You could write 'lie' on a piece of paper and hold it up every time someone speaks to you, and you will detect 100 percent of the lies."

Amir Liberman, CEO of Nemesysco, likens the technology to a microscope; it doesn't detect disease per se, but it's a tool for exploration. He adds that the circumstances and the interrogator are crucial to success. Still, Liberman says explicitly that Layered Voice Analysis can do what researchers say it can't: "LVA differentiates between stress and lies," he says. How exactly, he can't disclose. The National Institute for Truth Verification declined requests for an interview.

Harnsberger has repeatedly made the case to policy makers that voice analyzers don't live up to their manufacturers' claims. And currently only two "credibility assessment" devices have been approved for use by the Department of Defense: the good old polygraph and a next-generation version that also evaluates physiological factors.

But manufacturers of the voice stress analyzers continue to lobby for their products, says Harnsberger. Such efforts may not be in vain. In a statement, Defense Department spokesperson René White said the department "continues to conduct research on and evaluate additional potential credibility assessment tools." And according to USAspending.gov, the National Institute for Truth Verification, maker of the Computer Voice Stress Analyzer, has received more than $1.6 million in Defense Department contracts since 2005.

Though the technologies apparently don't tell truth from fiction, they may have merit as props. The jailhouse study followed up work that had asked arrestees about drug use, that time without a lie detector in the room. Comparing the two studies revealed that more than three times as many drug users lied when no device was present than when one was.

"They may be very useful for eliciting admissions," Harnsberger says. "That's not the same as detecting lies." ■

Explore more

- National Research Council report: http://bit.ly/chSa1L
- University of Florida Speech Perception Laboratory: www.clas.ufl.edu/users/jharns/SPL.html

The Invisible Gorilla: And Other Ways Our Intuitions Deceive Us

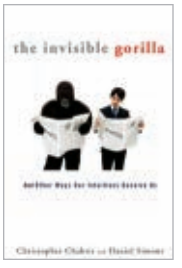
Christopher Chabris and Daniel Simons

People wearing gorilla suits don't always stand out in a crowd. When volunteers were asked to count the number of ball passes made by a basketball team in a video, half never noticed a gorilla-suited intruder walking across the court doing chest thumps.

That experiment, conducted by psychologists Chabris and Simons in 1999, launches their book about the dangers of trusting one's intuitive assumptions, unsullied by rational deliberations, of how the mind works. Invisible gorillas are an example of what the authors call "the illusion of attention," in which people miss objects because their attention is focused tightly elsewhere.

Other mental illusions get similarly assessed. Studies show that personal memories, such as how and where one learned of 9/11, change over time. Even vivid memories that seem true to life can be distorted or entirely false, the authors say. Other evidence indicates that people overestimate their skills and abilities, from competitive chess players to *American Idol* contestants. And people regularly think that they know more than they do, resulting in disastrous financial investments and other catastrophes, Chabris and Simons note.

The authors belong to a psychological school of thought that celebrates methodical, rational analysis over



rapid, intuitive decision making. They say little about research that has revealed intuition's strengths, such as its ability to aid firefighters, pilots and other professionals in making critical decisions under time pressure. Nor do the authors address the possibility that experimentally induced mental illusions stem from thinking strategies that are generally useful, just as optical illusions stem from a largely effective visual system.

Still, the book, unlike the fake gorilla, will not go unnoticed. —*Bruce Bower*

Crown Publishing, 2010, 306 p., $27.

Inside the Outbreaks: The Elite Medical Detectives of the Epidemic Intelligence Service

Mark Pendergrast

In 1951, a group of American men suited up to go to war. This wasn't unusual at the time — the Korean War was on — but this brigade was armed with field notebooks and test tubes, and was trained to take aim at threats to public health. *Inside the Outbreaks* tells the story of

this little-known corps, the Epidemic Intelligence Service of the Centers for Disease Control and Prevention.

Taking a historical approach to the subject, Pendergrast, a science journalist, uses interviews and archival materials to bring to life the people of the EIS, such as the service's founder, epidemiologist Alexander Langmuir, described by his daughter as someone who "people knew when he entered the room."

After training, "Langmuir's boys," as the initial officers were called, traveled the world in search of natural and social causes of cholera epidemics, smallpox outbreaks, food contamination and other ills. The book chronicles these early forays and then recounts the history of the EIS, as it opened its ranks to women, veterinarians, and social scientists and expanded its purview to chronic diseases such as heart disease, diabetes, HIV/AIDS and cancer.

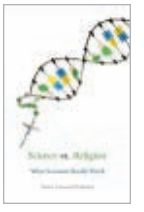
Pendergrast's pace is nearly as frenetic as that of the EIS: Activities in Brazil, Africa and California unfold within a few pages. The effect is jarring at times but does reflect the intensity of those serving at the front lines of public health. —*Rachel Zelkowitz*

Houghton Mifflin Harcourt, 2010, 432 p., $28.

March of the Microbes: Sighting the Unseen

John L. Ingraham

For those who know where to look, microbes abound in daily life. *Belknap Press/Harvard Univ. Press, 2010, 326 p., $28.95.*



Science vs. Religion: What Scientists Really Think

Elaine Howard Ecklund

Through surveys and interviews, a sociologist examines scientists' views on religion. *Oxford Univ. Press, 2010, 228 p., $27.95.*

Green Light: Toward an Art of Evolution

George Gessert

An artist who works with living material considers how aesthetic values influence the ways people breed plants and animals. *MIT Press, 2010, 233 p., $24.95.*

Bright Boys

Tom Green

A writer, producer and playwright tells the story of the first real-time, electronic digital computer and the people who created it. *A.K. Peters, 2010, 327 p., $39.*

A Zeptospace Odyssey: A Journey into the Physics of the LHC

Gian Francesco Giudice

A physicist describes the science behind the Large Hadron Collider, the world's largest particle accelerator, for a general audience. *Oxford Univ. Press, 2010, 276 p., $45.*

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Rick Lovecchio, Doraville, Ga.

We hope so too. Select bookstores across the country now stock Science News. *To find out how to order the braille edition, contact the National Library Service for the Blind and Physically Handicapped at www.loc.gov/nls. —Eva Emerson*

RHIC: No catastrophe

Could you give more details on the measurement reported in the March 13 issue ("Physicists cook cosmic soup to 4 trillion degrees," *SN: 3/13/10, p. 8*)? You quote one researcher as saying that the photons are "brightest when the matter is hottest" and refer to heated iron going from red to white hot. But I thought that classical physics fell precisely due to the "ultraviolet catastrophe" when the Rayleigh-Jeans formula failed to predict the drop in frequency of blackbody radiation at higher temperatures. This led Planck to his equation and started us on the road to quantum mechanics.
David Sole, Detroit, Mich.

The RHIC researchers did indeed measure thermal radiation, but they used the rules of quantum mechanics to extrapolate the temperature, says Steven Vigdor of Brookhaven National Laboratory in Upton, N.Y. As the reader notes, classical physics overestimated the probability of high-frequency radiation, Vigdor says. "But this does nothing to invalidate the steady march of materials toward higher frequency emission as the temperature is increased. Quantum physics still predicts, and experiment confirms, that the intensity of emitted radiation grows at a given frequency with increasing temperature, and that the mean frequency of the entire spectrum of emitted radiation grows with increasing temperature." The PHENIX experiment observed very high-energy photons compared with visible light. —Laura Sanders

Neandertal in you

At last, DNA analysis has shown that we carry Neandertal genetics ("Modern people carry around Neandertal DNA, genome reveals," *SN: 6/5/10, p. 5*). About time. I had long concluded that *Homo sapiens* and *Homo neanderthalensis* must have interbred a lot. There are just too many people who show Neandertal characteristics in their facial bones.
J. Thomas Baylor, Austin, Texas

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Robert Bea

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Explaining the equation behind the oil spill disaster

Catastrophes come in all shapes and sizes, but some basic causative principles underlie most of them. Robert Bea, an engineer at the University of California, Berkeley, has studied system failures from space shuttle explosions to levee breaks during Hurricane Katrina — but as a former oil rig worker he is most familiar with drilling disasters. Bea has thus assumed a key role in analyzing the response to the Deepwater Horizon spill in the Gulf of Mexico (Page 5). He spoke with Science News *contributing editor Alexandra Witze about why the spill could have been foreseen.*

You've looked at failures in more than 600 engineering systems. How does the Deepwater Horizon spill compare?
It is following this road map to disaster exactly. When I came back from Katrina in New Orleans, I got in front of my class and said I have a new equation for disaster. I was trying to be dramatic. But it is A plus B equals C.

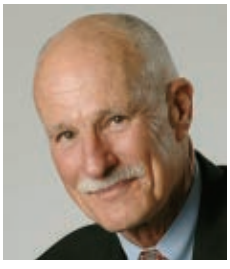
A is important. It's things like extreme pressures, temperatures, darkness, earthquakes, hurricanes, ice that goes bump in the night in the Arctic, volcanoes that spew into the sky. This is Mother Nature doing what she has done for millions and millions of years. B is ... kind of natural too. It includes people's hubris, arrogance, greed, ignorance and a real killer called laziness. C is the disaster that comes sooner or later. This story, as we best know it now, is tracking that equation perfectly.

How big is the role of human fallibility?
Eighty percent of high-consequence accidents fall in the second category, [that] of human-factor uncertainties. Of that, 60 percent traces back to trouble in operations and maintenance — things are designed that can't be built, operated or maintained as intended. When all of these get through, you have a ticking time bomb.

A subcategory is unknown knowables, where information exists but something prevents us from analyzing it properly. No matter how good you are and how much insight you think you have, you can't predict everything. You have to be on constant alert for this category of uncertainty, because it requires a very different set of management tools.

Whose fault was the spill?
The government's responsibility broke down. Industry's responsibility broke down. The only one that didn't is the environment, and unfortunately she's getting treated pretty badly right now. It is a collective set of breakdowns. The crucial one is government — they're the parents in the family. Industry are the children. Here the children told the parents what to do. It's an entire chain: the tool pusher, the rig worker, the company man representing BP, the people in the Minerals Management Service office in New Orleans. Everybody has a share in this one.

The Interior Department is restructuring the Minerals Management Service (the federal agency overseeing offshore drilling) as a result of the spill. Will that make a difference?
Reorganization at the time you've got catastrophes is not a good thing [but it can work]. At the Piper Alpha platform in the North Sea, I went to work three days after it blew up [in 1988], killing 167 people. The United Kingdom found its regulation was part of the causation of the accident. They have completely restructured, and they are leaders in this work today. [In 1980] I went to work a few days after the Alexander Kielland accident in the Norway sector of the North Sea. Today Norway is helping lead the world in regulatory and industrial operations. The U.K. and Norway both took big, strong, intense kicks in the seat of the pants to restructure and refocus.



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What about "quiet failures" — things we don't know about but that could go wrong at any moment? How common are those?
I see these in court. I'm involved in one right now — the failure of the flood protection system for greater New Orleans during Katrina. In Australia, I'm working on a challenge that is so like the Deepwater Horizon it's not funny. The tracks of the Montara blowout [in 2009] are damn near identical to this one. And the American public doesn't know anything about it.

I've worked on an Indonesian deepwater development, 10,000 feet deep. That operator, after two years of intense study of the risk involved, said that the reserve remains underdeveloped today because the technology is not there to prevent failures or to mitigate them.

What can we do now to prevent such catastrophes from happening again?
NOAA [the National Oceanic and Atmospheric Administration] and EPA have mobilized and are planning for 10 years from now. We'll be smarter. We'll have much more information on the environmental impacts and on organizational breakdowns. The knowledge will be there. The question will be, do we react properly to that knowledge? I hope so. ■

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