Reflection Perfection | Monster Virus | Taken with Trinitite

www.sciencenews.org

ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ AUGUST 10, 2013

The Neuroscience of Anorexia

Down Syndrome Chromosome Silenced

Skirmishing Over War's Origins

Odd specimen

South African find could be a human ancestor or an evolutionary dead end

Curse of the Perfect Gift

OWN THE 15-CARAT EMERALD NECKLACE THAT CHANGES EVERYTHING

Our story begins in the sweltering jungles of South America. My search for precious emeralds hit a dead end, so my partner suggested we expand our horizons. I agreed. When he asked where we should go, I said, "everywhere."

We journeyed through Africa and Europe's greatest cities. We crossed into India and hopped from one Southeast Asian paradise to the next. I was on the verge of giving up when one last detour led to the spectacular gems in our ***Stauer Spring Emerald Necklace***. As you can see, it was worth the effort. And today, they can be all yours for **ONLY $59!**

You get to share the emerald find of a lifetime. Each natural gem radiated with the vivid green color of spring buds. The stones looked spectacular and the price was almost too good to be true. But my stroke of luck had unforeseen consequences…

The "Emerald Curse" strikes. Back home, I strung those exquisite emeralds and wrapped the necklace as a gift for my wife. That's when my trouble began. She LOVED it. Absolutely adored it.

Every 18" necklace features 15 carats of polished, enhanced genuine emeralds with gold-finished links.

In fact, she rarely goes anywhere without the necklace and has basked in compliments from strangers for months now.

So what's the problem? I'm NEVER going to find an emerald deal this good again. By giving her such a perfect gift, *I've made it impossible to top myself.*

Bring yours home today for 80% OFF. Our ***Stauer Spring Emerald Necklace*** features 15 carats of emerald beads, hand-wired together with delicate gold-finished links. Some curses you don't wish on your worst enemies, but this is one I want to share with friends. That's why today you have the chance to own this stunning collection of stones (available online for $299) for **ONLY $59!**

Your satisfaction is guaranteed. If you are not completely thrilled by this rare emerald find, send the necklace back within 30 days for a full refund of your purchase price. This stunning necklace at this price is available only while supplies last. So please call today to make sure you don't let the perfect gift slip away!

Stauer®
14101 Southcross Drive W., Dept. SEN148-01
Burnsville, Minnesota 55337
www.stauer.com

Call now to take advantage of this fantastic offer.
1-800-721-0373

Promotional Code SEN148-01
Please mention this code when you call.

Spring Emerald Necklace
Available Online for ~~$299~~
Exclusive Call-In Price
Only $59

AUGUST 10, 2013 • VOL. 184 • NO. 3

ScienceNews

In The News

Features

Departments

COVER The skull of *Australopithecus sediba* bears features that suggest it may belong on the human lineage, its discoverer claims. *Courtesy of L. Berger/ Univ. of the Witwatersrand*

ScienceNews
MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC

EDITORIAL, ADVERTISING AND BUSINESS OFFICES
1719 N Street NW, Washington, DC 20036 Phone (202) 785-2255
Audit Bureau of Circulations
Subscriptions subs@sciencenews.org Editorial/Letters editors@sciencenews.org
Advertising/Business snsales@sciencenews.org
Texterity Digital edition provided by Texterity, www.texterity.com
Science News (ISSN 0036-8423) is published biweekly, for $54.50 for 1 year or $98 for 2 years (international rate $80.50 for 1 year or $161 for 2 years) by Society for Science & the Public, 1719 N Street NW Washington, D.C. 20036. Preferred periodicals postage paid at Washington, D.C., and an additional mailing office.
Subscription Department: PO Box 1205, Williamsport, PA 17703-1205. For new subscriptions and customer service, call 1-800-552-4412.
Postmaster: Send address changes to *Science News*, PO Box 1205, Williamsport, PA 17703-1205. Two to four weeks' notice is required. Old and new addresses, including zip codes, must be provided.

FROM THE EDITOR

A thinking problem, sculpted into the brain

SANDY SCHAEFFER

To the uninitiated, anorexia nervosa may appear to be a problem of faulty thinking: People with the eating disorder have just gotten stuck in a bad pattern of relating to food, it seems. If they could just stop thinking the way they do, they would get better and regain the often dangerous amount of weight that they have lost. But full-fledged anorexia nervosa is actually a very difficult disorder to treat, and physicians have had little insight into why something so basic as eating can go so wrong for some people.

Some scientists now believe that anorexia has roots in the way the brain works in some people, writer Meghan Rosen reports on Page 20. The latest neuroimaging studies hint that people who have anorexia may respond differently from most people to rewards like sweets. They may also be overly sensitive to sugar, and relatively insensitive to other sensory cues such as pain. Other studies, as well as the experience of those who work with patients, suggest that people with anorexia tap deep wells of self-control to curb the eating impulse. While this extraordinary willpower may help in other areas of their lives (many teens with anorexia earn straight A's, for instance), it can become harmful for people stuck in a struggle with food. One study also showed a more robust working memory in people with anorexia, which may help keep them focused on their misguided weight loss goals.

People Rosen interviewed on the front lines of anorexia treatment were in surprising agreement in the belief that it is a brain-based disorder. Others might greet the brain-imaging data with more skepticism. While these studies clearly have many limits — it's impossible to know, for example, whether the differences in brain activity existed to begin with or developed as part of the eating disorder — being able to visualize the brain differences can clearly help patients and families battling the disorder. Just knowing there is some biological component to anorexia has been healing for many patients, lessening their need to blame themselves or their families.

Rosen's article is an intriguing consideration of how we view the physical versus the mental, with one being somehow more real and legitimately beyond our control than the other. But the more we learn about the brain, the clearer it is that the two are tightly intertwined, with our thoughts and mental processes sculpting the cells and circuits that give rise to them, and vice versa. — *Eva Emerson, Editor in Chief*

Actual size 19 mm

The ONLY Confederate Cent Ever Struck

Hidden from Abraham Lincoln. Now authorized by the Smithsonian Institution

There was only one Confederate cent design ever commissioned and struck during the Civil War. Surprisingly, the Confederate coin maker was actually a Northerner living in Philadelphia: Robert Lovett, Jr. Although Lovett at first designed the coin with dreams of glory, he soon lost his nerve and realized that he could be hanged for treason if Abraham Lincoln's federal forces ever discovered his Southern secret. In order to hide the evidence, Lovett hid the few Confederate coins he had struck, and buried the coin dies in his basement. He then silently kept his treasonous secret until long after the Civil War was over.

A Civil War Treasure of the Smithsonian Institution

When Lovett's secret was finally revealed in 1873, his coins and original dies began a colorful saga. Today, one of his original 1861 Confederate Cents is highly sought-after by both coin collectors and Civil War enthusiasts. One sold at a 2005 auction for more than $74,000! Lovett's original hand-engraved dies for the coins eventually came to be housed in the prestigious collection of the Smithsonian Institution®.

Own the 1861 Confederate Cent Proof—in Pure Silver!

Now, in honor of the 150th anniversary of the Civil War, New York Mint is proud to be releasing this 1861 Confederate Cent Silver Proof carefully created from the original dies. Struck in stunning, deeply mirrored and frosted 99.9% pure proof silver, each is officially licensed by the Smithsonian Institution.

Your 1861 Confederate Cent Silver Proof is certified Gem Proof by Numismatic Guaranty Corporation (NGC) and comes in a protective acrylic holder.

Triple Guaranteed! Order Risk Free

Your 1861 Silver Proof is actually triple guaranteed! First, it is officially authorized by the Smithsonian Institution. Second, it has been certified Gem Proof by NGC. Thirdly, you are protected by our 30-day Satisfaction Guarantee. You must be completely satisfied or return your 1861 Silver Proof within 30 days for a prompt refund (less all s/h).

This is an unprecedented opportunity to hold Civil War history literally in your hands. Don't miss out!

1861 Confederate Cent Certified Silver Proof
$189.00 *(plus s/h)*

Call Toll Free 24 hours a day

1-866-286-7546

Offer Code: CPS141-01
Please mention this code when you call

New York Mint®

14101 Southcross Drive W., Dept CPS141-01
Burnsville, Minnesota 55337
www.NewYorkMint.com

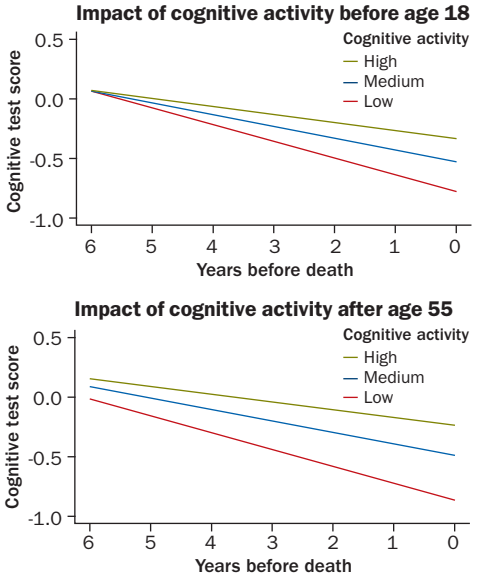
Prices and availability subject to change without notice. Past performance is not a predictor of future performance. NOTE: New York Mint® is a private distributor of worldwide government coin and currency issues and privately issued licensed collectibles and is not affiliated with the United States government. This product was minted privately and is not legal tender in the U.S. Facts and figures deemed accurate as of June 2013. ©2013 New York Mint, LLC.

Say What? | BRÛLÉ \BROO-lay\ n.

A vegetation-free zone that forms around a tree sharing a symbiotic relationship with certain species of truffle. Brûlés get their name from the burnt appearance of the ground that develops as the truffle wages biochemical warfare against plants and other fungi growing near the tree. The compounds released by the truffle may even have an impact on bacteria lurking in the soil. An international team of scientists analyzing the dirt around four French brûlés produced by the black truffle *Tuber melanosporum* (left) found different microbial communities living inside and outside the dead zone. While some types of bacteria may thrive on compounds produced by the truffle, others retreat, the team suggests April 30 in *PLOS ONE*. —*Allison Bohac*

Science Stats | READING MINDS

When it comes to the brain, "use it or lose it" applies no matter your age. Research has shown that people who read more perform better on cognitive skill tests late in life, but hasn't established cause and effect. In a new study, researchers used surveys to estimate reading and other cognitive activities throughout life for 294 Chicago-area residents, then autopsied the volunteers' brains after death (at an average age of 89). Brain workouts, whether performed at an early or late age, slowed cognitive decline in the last six years of life (right), supporting the idea that cognitive workouts may delay the effects of age-related lesions. SOURCE: R.S. WILSON *ET AL*/*NEUROLOGY* 2013



50 Years Ago

Excerpt from the August 10, 1963, issue of *Science News Letter*

FORECASTING BY COMPUTER

Future weather satellites and improved methods of using computers to forecast weather are keys to the continued expansion of the Weather Bureau under its new head.... The computer program is expected to lead to improved long-range weather forecasts and to help bring closer to reality the control of weather.... Use of weather satellites to give photographs of the earth's cloud cover has already given weathermen valuable information on otherwise unknown storms, including hurricanes, and has shown changes in large-scale weather patterns.

UPDATE: The ability to control the weather remains a dream, but the fleet of Earth-monitoring satellites and sophisticated computer programs now available to predict the weather has greatly improved forecasting. Warnings for hurricanes, for instance, are now given more than 50 percent earlier than in the 1960s.

Introducing | CITY-SAVVY BIRD

A routine sweep for avian flu has turned up a new bird hiding in plain sight in and around Cambodia's capital city of Phnom Penh. This never-before-seen species (right) belongs to the tailorbird genus, a group of Old World songbirds known for "stitching" together nests of leaves by pulling plant fibers through holes punched with their needlelike beaks. Described by an international team of researchers in the August *Forktail*, *Orthotomus chaktomuk* takes its name from a Khmer word meaning "four faces," a reference to the rivers that converge to form an "X" shape at Phnom Penh. Seasonal flooding of these rivers creates the humid scrubland habitat that *O. chaktomuk* calls home. Weighing in at about 7 grams, both males and females sport cinnamon-colored heads and gray and white bodies. The team suspects that the dense vegetation of the floodplains, as well as *O. chaktomuk*'s similarity to more common native tailorbirds, allowed it to elude discovery for so long. —*Allison Bohac*

SAY WHAT?: CRACKPHOTOS/GETTY IMAGES; INTRODUCING: ASHISH JOHN/WCS

“When we do see remote triggering, it seems to foreshadow larger induced earthquakes. It shows the faults are reaching a tipping point.” —NICHOLAS VAN DER ELST, PAGE 16

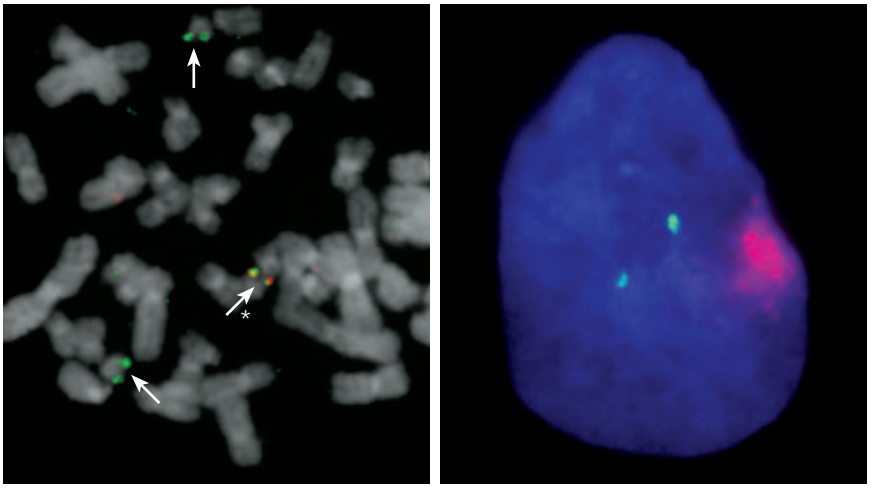
In the News

STORY ONE

Technique inactivates Down-causing chromosome

Early-stage research could eventually lead to gene therapy

By Tina Hesman Saey



Chromosome quieter To shut down the extra chromosome that leads to Down syndrome, researchers inserted additional genetic material (left, in red on chromosome indicated by arrow with asterisk) into one of three copies of chromosome 21 (indicated by arrows). The addition (right, in red) switches the chromosome off. The other two copies (green) function normally.

J. JIANG *ET AL*/*NATURE* 2013

Borrowing a trick from nature, researchers have switched off the extra chromosome that causes Down syndrome in cells taken from patients with the condition.

Though not a cure, the technique, reported July 17 in *Nature*, has already produced insights into the disorder. In the long run it might even make the flaw that causes Down syndrome correctable through gene therapy.

“Gene therapy is now on the horizon,” says Elizabeth Fisher, a molecular geneticist at University College London. “But that horizon is very far away.”

Down syndrome, also called trisomy 21, occurs when people inherit three copies of chromosome 21 instead of the usual two. It is the most common chromosomal condition, affecting around one in every 700 babies born in the United States. People with the disorder typically have both physical and cognitive complications of having an extra chromosome.

“Down syndrome has been one of those disorders where people say, ‘Oh, there’s nothing you can do about it,’ ” says Jeanne Lawrence, a chromosome biologist and genetic counselor at the University of Massachusetts Medical School in Worcester, who led the study with colleague Jun Jiang.

The researchers decided to see whether they could shut down the extra chromosome by drawing on a biological process called X inactivation. Women have two X chromosomes and men have only one X and a Y. To match the amount of X chromosome products made by males, female cells shut down one copy. Cells do that using a chunk of RNA called *XIST*, which is made by one X chromosome but not the other. The RNA works by pulling in proteins that essentially board up the chromosome like an abandoned building. The other X remains active by making a different RNA.

1 in 700

Frequency of Down syndrome in U.S.-born babies

Lawrence’s team thought that if they added *XIST* to another chromosome, it might shut that one down too. So Jiang put the gene for *XIST* onto one of the three copies of chromosome 21 carried by stem cells grown from a man with Down syndrome. That copy of the chromosome got switched off.

“It’s kind of surprising that it wasn’t done before. I’m smacking my own forehead and saying, ‘duh,’ ” says Roger Reeves, a geneticist at Johns Hopkins University.

One idea about why an extra chromosome 21 causes cognitive problems is that it may slow down the growth of brain cells. Jiang grew nerve cells from the Down patient’s stem cells to see how cells with one shut-down chromosome developed compared with cells bearing three active copies. The cells with only two working chromosomes grew faster, forming clusters of nerve cell precursors within two weeks, while the uncorrected cells needed four or five additional days.

For the latest news,
visit **www.sciencenews.org**

The work is an enormous step forward in Down syndrome research, Fisher says, and "may take us much closer to understanding the molecular basis of the disorder." The technique could allow researchers to figure out which genes are most involved in Down syndrome and how extra copies affect cells and ultimately the body, she says.

Reeves wants to use the technology in animal experiments, a crucial step in determining whether it could find use as gene therapy for people with Down syndrome. He plans to work with Lawrence's group to switch off the extra chromosome in mice engineered to have a disorder that simulates some features of Down syndrome.

But Reeves doubts that scientists could use the method to switch off the extra chromosome in every cell in the body. Doing so would probably require gene therapy at a very early stage of pregnancy, something scientists don't know how to do. "I just don't see how we would get there from where we are today," Reeves says.

Such universal silencing of the extra chromosome would be necessary to forestall the developmental problems caused by Down syndrome. But other problems associated with the condition might be prevented or reversed by shutting down the extra chromosome after birth. For instance, people with Down syndrome are at high risk of developing childhood leukemia and of getting Alzheimer's disease. Gene therapy to turn off the extra chromosome in the bone marrow or the brain might prevent those problems.

These therapeutic possibilities are still far in the future and may never pan out, says William Mobley, a neurologist and neuroscientist at the University of California, San Diego. "We have to move cautiously and deliberately and not say that a cure for Down syndrome is on the horizon — it's not true," he says. "But gosh is there excitement that progress is being made." ■

Back Story | A DNA CHALLENGE OF STUPENDOUS SIZE

The idea came easily, says Jeanne Lawrence: Mimic nature by using RNA to shut down chromosome 21. But executing the vision was a feat that has left other scientists in awe.

Part of the problem is that the piece of RNA, called *XIST*, is huge. Previously, researchers had spliced into a chromosome pieces of genetic material shorter than about 8,000 DNA units, or nucleotides, says Jun Jiang, the University of Massachusetts Medical School colleague Lawrence says "worked night and day for five years" to pull off the project. At about 21,000 nucleotides, *XIST* dwarfed the previous record holder. "Getting something that big onto a chromosome had never been done before," Lawrence says.

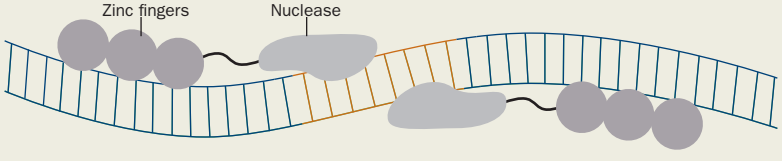
But Jiang did it. She customized proteins called zinc finger nucleases (*SN: 7/30/11, p. 9*) to accomplish the goal. Zinc fingers are structures that recognize and grab onto a specific sequence of DNA letters. Jiang engineered fingers to seek out a particular spot on chromosome 21. She tethered nucleases, enzymes that work in pairs to cut DNA, to the zinc fingers.

Cells repair breaks in DNA using a process called homologous recombination. The process normally uses the matching chromosome as a guide for sealing the breach. Instead, Jiang supplied a template containing the enormous *XIST* gene sandwiched between two bits of DNA that matched sequences on either side of the gap. So when cells healed the wounded copy of chromosome 21, they also incorporated the *XIST* gene.

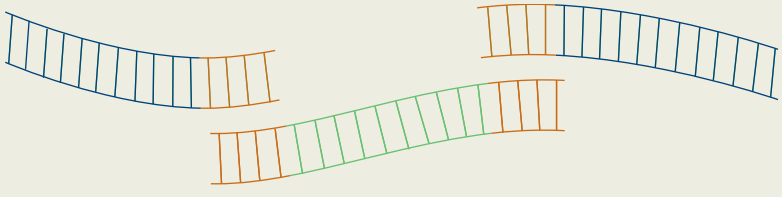
If the technique works for chromosome 21, it might apply to lethal conditions that are caused by having three copies of other chromosomes. The research really goes beyond gene therapy to chromosome therapy, says Montserrat Anguera, a biologist at the University of Pennsylvania. —*Tina Hesman Saey*

Cut and paste In recent years, scientists have developed a way to cut DNA using proteins called zinc finger nucleases. Carefully orchestrated cuts can induce cellular processes to add in desired chunks of DNA.

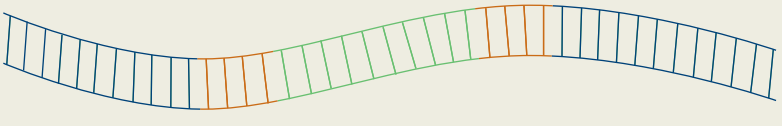
Zinc finger nucleases recognize certain locations on a chromosome's DNA.



The nucleases cut the DNA, triggering a cell repair response.

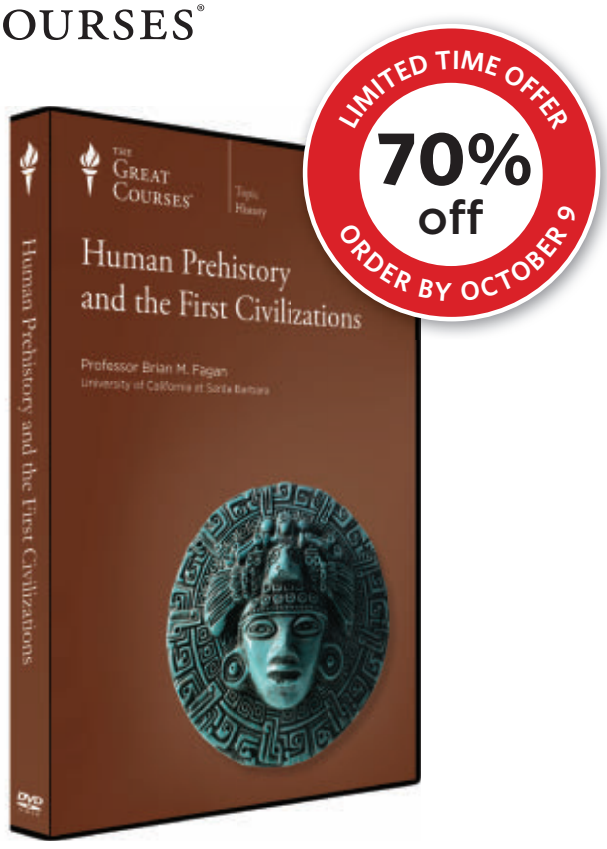


Scientists take advantage of the repair process to insert a new gene.



E. OTWELL

THE GREAT COURSES®



Uncover the Thrilling Story of Human Origins

Where do we come from? How did our ancestors organize into the first great civilizations of the world? What does this shadowy past reveal about who and what we are? **Human Prehistory and the First Civilizations** brings you the startling answers to these and other questions about the dawn of humanity.

These 36 lectures reveal what recent scientific and archaeological research and theorizing suggests about human origins, how populations developed, and the ways that civilizations spread around the globe. Guided by award-winning Professor Brian M. Fagan—an expert in anthropology and the recipient of a Public Education Award from the Society for American Archaeology—you investigate dozens of archaeological sites all over the world. As you explore man's earliest origins, you learn about stone-tool making, mammoth hunting, temple building, ancient farming, and more. A riveting world tour of prehistory, this illuminating course reveals the profound links between how we lived in the past and today.

Offer expires 10/09/13

1-800-832-2412

WWW.THEGREATCOURSES.COM/3SN

Human Prehistory and the First Civilizations

Taught by Professor Brain M. Fagan
UNIVERSITY OF CALIFORNIA AT SANTA BARBARA

LECTURE TITLES

1. Introducing Human Prehistory
2. In the Beginning
3. Our Earliest Ancestors
4. The First Human Diaspora
5. The First Europeans
6. The Neanderthals
7. The Origins of *Homo sapiens sapiens*
8. The Great Diaspora
9. The World of the Cro-Magnons
10. Artists and Mammoth Hunters
11. The First Americans
12. The Paleo-Indians and Afterward
13. After the Ice Age
14. The First Farmers
15. Why Farming?
16. The First European Farmers
17. Farming in Asia and Settling the Pacific
18. The Story of Maize
19. The Origins of States and Civilization
20. Sumerian Civilization
21. Ancient Egyptian Civilization to the Old Kingdom
22. Ancient Egypt—Middle and New Kingdoms
23. The Minoan Civilization of Crete
24. The Eastern Mediterranean World
25. The Harappan Civilization of South Asia
26. South and Southeast Asia
27. Africa—A World of Interconnectedness
28. The Origins of Chinese Civilization
29. China—Zhou to the Han
30. Southeast Asian Civilizations
31. Pueblos and Moundbuilders in North America
32. Ancient Maya Civilization
33. Highland Mesoamerican Civilization
34. The Origins of Andean Civilization
35. The Inka and Their Predecessors
36. Epilogue

Human Prehistory and the First Civilizations
Course no. 380 | 36 lectures (30 minutes/lecture)

SAVE UP TO $275

DVD ~~$374.95~~ NOW $99.95
+$15 Shipping, Processing, and Lifetime Satisfaction Guarantee

CD ~~$269.95~~ NOW $69.95
+$10 Shipping, Processing, and Lifetime Satisfaction Guarantee

Priority Code: 77676

Designed to meet the demand for lifelong learning, The Great Courses is a highly popular series of audio and video lectures led by top professors and experts. Each of our more than 400 courses is an intellectually engaging experience that will change how you think about the world. Since 1990, over 10 million courses have been sold.

Matter & Energy

For more Matter & Energy stories, visit **www.sciencenews.org**

Light trap makes perfect mirror

Perforated material that reflects light could improve lasers

By Andrew Grant

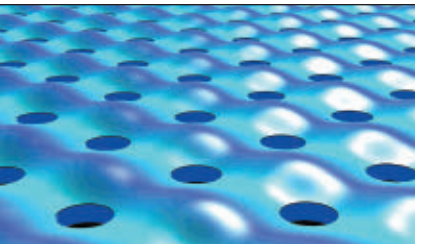
A new type of mirror that reflects light perfectly has been constructed, a feat many scientists thought wasn't possible. The mirror could find its way into powerful lasers and other devices.

Many modern technologies rely on trapping light and shuttling it around, whether to carry data over the Internet or to play a DVD. Engineers prevent light from escaping by directing it to bounce off reflective materials, but there are drawbacks to that process. Many mirrors absorb some of the light that strikes them, and the glass in fiber-optic cables reflects only if light grazes it at a very low angle.

Physicist Chia Wei Hsu and colleagues at MIT weren't looking to invent a mirror when they started studying photonic crystals, slabs of material drilled with holes small enough to manipulate individual light waves. Most of the time, light penetrated at least partially into the team's crystal, a block of perforated silicon nitride. But when the researchers shined a specific frequency of red light at a 35-degree angle to the surface of the slab, they were surprised to find that it bounced back completely — none of it leaked away or got absorbed.

The cause of this quirky phenomenon, Hsu's team reports in the July 11 *Nature*, is light interfering with itself. A bunch of combined factors — including the light's wavelength, the angle at which it hits the surface and the pattern of drilled holes — orients the light so that waves trying to enter the slab cancel each other out. Only reflecting waves can propagate.

Computer scientist and physicist John von Neumann proposed a similar phenomenon in 1929, but all previous attempts to demonstrate it experimentally had failed. "No one had thought of a practical method to do this," says Douglas Stone, a physicist at Yale University.



Holes drilled in a new type of mirror (illustrated) help it perfectly reflect a particular wavelength of light.

Now that physicists know this mirror is real, Stone says it may prove useful in lasers, which are concentrated beams of light at a single wavelength. Specially designed photonic crystals tuned to specific wavelengths could enable engineers to amp up the energy of lasers without sacrificing efficiency, he says.

In addition, Hsu notes that this mirror may have uses beyond optics. At least in theory, he says, crystals with distinct patterns of drilled holes should be able to perfectly reflect sound waves and even water waves. ■

Under magnet's sway

Dollops of magnetic fluid can assemble themselves into simple structures and dynamic complex formations, researchers report in the July 19 *Science*. Proteins can warp and fold themselves into new arrangements; scientists want to create synthetic self-assembling structures as changeable and versatile as the natural ones that drive life. Physicist Jaakko Timonen at Aalto University in Finland and colleagues experimented with ferrofluids, liquids containing suspended magnetic nanoparticles that behave strangely when exposed to magnetic fields. The researchers placed a ferrofluid droplet on a nonstick surface and gradually moved a magnet toward the surface from below. The strengthening field caused the droplet to split into simple, evenly spaced daughter droplets (above). Then Timonen and his team oscillated the magnet horizontally, moving it increasingly faster and over longer distances. At certain thresholds of speed and distance, the droplets suddenly coalesced into elongated globules (below) that changed shape as the magnet yanked them back and forth. Timonen says the work should help scientists better understand and exploit dynamic self-assembly. —*Andrew Grant*

FROM TOP: BO ZHEN; MIKA LATIKKA & JAAKKO TIMONEN

Atom & Cosmos

"People claim too often that the rings we see are due to planets." — WLADIMIR LYRA

How molecules hook up in space

Reactions in the cold sped up by quantum quirk

By Andrew Grant

Molecules floating in the dark, cold vacuum of interstellar space can exploit quantum mechanics to react and produce more complex chemicals, a new study suggests. Researchers explain the reactions using a quirky property of quantum physics, which may be a key cog in a cosmic assembly line that churns out intricate organic molecules, including some necessary for life.

Astronomers have long known that stars manufacture chemical elements, but it's only recently that researchers have discovered complex organic molecules floating around in clouds of gas and dust in space (*SN: 1/30/10, p. 26*). The formation of these chemicals, which include alcohols, sugars and even an ingredient found in tar, is hard to explain because molecules in space should rarely collide.

Last year astronomers discovered a molecule called methoxy, or CH_3O, in a gas cloud. It forms when hydroxyl (OH) and methanol (CH_3OH) react. Yet that reaction requires more energy than is available in space, where temperatures hover just above absolute zero.

While not specifically pursuing this mysterious reaction, Dwayne Heard and his team at the University of Leeds in England were exploring the reactivity of hydroxyl with other molecules, including methanol. In the course of the work, the researchers placed the two reactants together in a cryogenic vessel. To their surprise, they found that the reaction was about 50 times faster at −210° Celsius than at room temperature, even though the chilled molecules had far less energy to work with.

"A particle can go right through the bottom of the mountain, almost as if the mountain weren't there." ERIC HERBST

Writing in *Nature Chemistry* June 30, Heard's team explains its finding with a phenomenon called quantum tunneling. Ordinarily, a chemical reaction occurs only if the reacting molecules have enough energy to overcome a threshold known as the energy barrier, which is like a hill. But a peculiar consequence of quantum mechanics is that molecules can occasionally bypass that hill without the requisite energy. "A particle can go right through the bottom of the mountain, almost as if the mountain weren't there," says Eric Herbst, an astrochemist at the University of Virginia.

Heard and his colleagues found that the chances for quantum tunneling improve at low temperatures because slow-moving hydroxyl and methanol molecules are more likely to stick together rather than bounce off each other when they collide. This temporary bond provides more opportunity for tunneling through the energy barrier, hastening the reaction. Heard estimates that about 1 in 10 hydroxyl-methanol collisions in space produce methoxy; without quantum tunneling, that would drop to about 1 in 10 million.

Other interstellar molecules may owe their existence to quantum mechanics, says Stephen Klippenstein, a theoretical chemist at Argonne National Laboratory in Illinois. "People will definitely find other reactions like this," he says. "This will not be a unique case." ■

Planets not needed in stars' rings

Gas and dust together may form elliptical patterns

By Jessica Shugart

Rings around distant stars aren't necessarily a sign of orbiting planets. That's the conclusion of a simulation that challenges a tantalizing notion in planetary science: that elliptical voids in a star's dusty debris disk betray a planet's presence. Instead, the rings could result from interactions between the dust and gas.

"People claim too often that the rings we see are due to planets," says planetary astrophysicist Wladimir Lyra of Caltech.

As young star systems evolve, the surrounding gas dissipates and dust particles collide and clump together. The hardiest of those blobs carve out ring-shaped pathways in the star litter and, astronomers think, form planets. While distant planets are difficult to image directly, those rings have been found circling several stars (*SN: 7/27/13, p. 8*).

But around older stars, traces of gas within the debris disks may subsist but fall below the limits of detection, Lyra says. "People always thought that the effect of gas would be negligible," he says.

Predicting the outcome of the dance between gas and dust requires powerful computer simulations. Lyra and colleague Marc Kuchner, of NASA Goddard Space Flight Center in Greenbelt, Md., created a simulation that accounted for that interaction. In their model, published in the July 11 *Nature*, dust warmed by a parent star transfers heat to the surrounding gas. This warmed area attracts the dust and gas into clusters that expand sideways and form a ring, Lyra says.

The work will affect astronomers' interpretations of rings in star disks, says planetary astrophysicist Thayne Currie of the University of Toronto. "We have to be careful about taking every single one of these rings as signposts of planets." ■

Humans

For more Humans stories, visit **www.sciencenews.org**

War arose recently, analysis claims

Cross-cultural survey suggests Stone Age was peaceful

By Bruce Bower

A battle has broken out among scientists trying to untangle the origins of war.

The fighting is over whether hunter-gatherer communities in recent centuries have tended more toward war — defined as banding together in groups to kill people in other populations — than toward one-on-one attacks within their own communities. A second front has broken out over how to extrapolate from modern behavior to the Stone Age. Some anthropologists regard the nomadic groups as helpful if imperfect models of Stone Age human behavior. Others suspect that too much evolutionary change and irregular contact with outsiders make hunter-gatherers unreliable guides to the past.

Lethal attacks on one community by another rarely occurred during the 19th and 20th centuries, according to a new analysis of data previously gathered from nomadic hunter-gatherer populations. Murders of one person by another in the same group accounted for a majority of intentionally caused deaths, anthropologists Douglas Fry and Patrik Söderberg of Åbo Akademi University in Vasa, Finland, report in the July 19 *Science*.

Ten of the 21 hunter-gatherer groups studied had no recorded killings involving more than one attacker, effectively making those societies no-war zones, Fry and Söderberg say.

The new evidence suggests that humans have evolved a tendency to avoid killing in general, the researchers contend. War originated only within the past 10,000 years, in their view, with armed conflicts intensifying as the first states expanded between 6,000 and 4,000 years ago.

"Fry and Söderberg go against the popular tide in science ... and win hands down," says anthropologist R. Brian Ferguson of Rutgers University in Newark, N.J.

Archaeological evidence from Europe, the Middle East and western Asia contains relatively few signs of murder and war until after 10,000 years ago, he says.

But the new study has attracted fire from other investigators. "Fry and Söderberg use the hunter-gatherer record inappropriately to push the idea that because many modern hunter-gatherers were not seen to have war, ancestral hunter-gatherers also did not often have war," says Harvard anthropologist Richard Wrangham.

Wrangham and others say that the new paper ignores relatively high homicide rates previously documented in hunter-gatherer groups, including some in the study. Critics also point to reports of regular fighting among neighboring hunter-gatherer communities; the groups that Fry and Söderberg studied were largely isolated. From critics' perspective, war probably goes back tens of thousands of years and stoked the evolution of intense cooperation within, but not between, human groups.

Murders cause more deaths than war in both traditional and modern societies, with exceptions coming during the 20th century's two world wars, says Harvard University neuroscientist Steven Pinker. Given war's rarity, researchers are unlikely to observe raids and other attacks on rival groups when studying small hunter-gatherer samples such as those in the new study, he says. Rates of violent death are higher among hunter-gatherers and in other non-state societies than in state societies, he adds.

Fry and Söderberg's finding that mobile hunter-gatherer bands infrequently organize warlike attacks does not surprise anthropologist Polly Wiessner of the University of Utah. But raiding and war does take place in a few such groups, as well as among sedentary hunter-gatherers that live year-round in bountiful settings near coasts or rivers. The great unanswered question, Wiessner says, concerns "how different societies harnessed and tamed aggression to build larger societies throughout human evolution."

A cross-cultural analysis finds that nomadic hunter-gatherer populations rarely organize to attack other groups, with the exception of Australia's indigenous Tiwi society (shown).

© BILL BACHMAN/ALAMY

Fry and Söderberg identified data on 148 killings in 21 mobile hunter-gatherer groups. Just over half of those killings were committed by lone perpetrators. Almost two-thirds resulted from disputes within families, executions of group members, competition among men over women and other conflicts within groups.

About one-third of killings involved attacks by one group on another. Reasons included disputes over resources, thefts of women and revenge attacks for past stealing or other offenses.

Australia's Tiwi had an exceptionally large number of killings, 69, and accounted for most of the lethal attacks across groups.

Economist Samuel Bowles of New Mexico's Santa Fe Institute criticizes Fry and Söderberg for choosing relatively peaceful groups, including the Tiwi, that mostly live in places where state-run armies discourage intergroup conflict. In his 2009 analysis of eight hunter-gatherer societies, the Tiwi ranked near the bottom in estimated rates of war-related deaths. None of the other seven groups he studied were part of Fry and Söderberg's work.

Mind & Brain

"This is one of the most important steps that I have seen in recent years." —LARS OLSON

Injured rats regain bladder control

Nerve cells regenerated in animals with severed spinal cords

By Meghan Rosen

Paralyzed rats can now decide for themselves when it's time to take a leak. Animals in a new study regained bladder control thanks to a new treatment that coaxes severed nerves to grow.

Instead of dribbling urine, the rodents squeezed out shots of pee almost as well as healthy rats do, researchers report in the June 26 *Journal of Neuroscience*. The study is the first to regenerate nerves that control bladder function in animals with severely injured spinal cords.

Unlike paralyzed rats, severely paralyzed humans can't leak urine to relieve a full bladder. Unless injured people are fitted with a catheter, urine backs up into the kidneys. "These people get kidney failure all the time," says study leader Jerry Silver, a neuroscientist at Case Western Reserve University in Cleveland. "It's a terrible problem. If they didn't have the catheter, they would die."

Silver's team has spent years refining a technique to tear down scar tissue and encourage damaged nerve cells to grow. The researchers snip out a healthy nerve bundle from between rats' ribs, graft it into a damaged section of spinal cord and then add an enzyme that chews up scar tissue. In 2006, the technique returned some limb control to rats with one paralyzed forepaw. And in 2011, it helped paralyzed rats regain the ability to breathe.

In both cases, the rats retained bladder control because researchers had snipped only halfway through the bundle of spinal cord nerves. For the new study, the researchers severed the entire nerve bundle and leaving a gap about the width of a pencil. They then pumped up the treatment protocol from the earlier studies by adding to the injury site a molecule that boosts nerve growth.

Over several months, the damaged nerves slowly inched down through the grafted nerves, and then, says Silver, "they kept going and going like little Energizer bunnies." After six months, the rats could mostly control their bladders and could even wiggle their legs a bit.

The method may have potential beyond restoring bladder control. It could also restore sensation to the skin, which could help paralyzed people detect and avoid bedsores, says neuroscientist Lars Olson of the Karolinska Institute in Stockholm. "This is one of the most important steps that I have seen in recent years," he says.

Life

For more Life stories, visit **www.sciencenews.org**

Liver fuels white shark migration

Stored energy enables swim from California to Hawaii

By Susan Milius

A white shark's big fat liver, which can plump up to more than a quarter of an animal's body weight, turns out to be the fuel tank for extreme migrations.

White sharks (*Carcharodon carcharias*) in the eastern Pacific take a springtime swim from California to Hawaii and return in late summer. A one-way, 4,000-kilometer trip takes about a month.

By combining data from two kinds of tracking tags attached to the animals, an unusual analysis shows that sharks fatten up for the demands of migration much the way birds do, says Gen Del Raye of the University of Hawaii in Manoa.

A white shark's liver is its largest visceral organ, and, at its maximum, 90 percent of its volume comes from high-energy lipids. As the sharks use up calories stored as oily lipids, their livers shrink and their bodies lose buoyancy. The tracking data revealed signs that sharks progressively sink faster when gliding as they travel to Hawaii, Del Raye and his colleagues report in the Sept. 7 *Proceedings of the Royal Society B*. Tagged sharks on the California coast didn't change in glide trajectories.

The data give researchers their first evidence that sharks rely on stored lipids in the liver for the ordeal of migration, says M. Aaron MacNeil of the Australian Institute of Marine Science in Townsville.

One of the tag types that the researchers analyzed logs indicators of location and dive depth. It eventually pops loose from the animal, bobs to the surface and uploads its data to a satellite. Another kind of tag records the animal's acceleration. Del Raye used these data to select episodes when sharks stopped swimming and just drifted downward at a steady, shallow angle.

Waters off California and Mexico offer a great opportunity to bulk up on elephant seals and other marine mammals. But prey become scattered and scarcer on the trip to Hawaii. Making a long journey into food-poor waters is a risky endeavor, so biologists expect sharks get some kind of big payoff, perhaps in mating.

Marine ecologist Nigel Hussey would like to know whether white sharks out in the central Pacific reload their livers to some extent to fuel the journey back to California. Hussey, of the University of Windsor in Canada, notes that another recent paper argues that white sharks may need to feed more often than biologists thought. ■

Right antenna is bee's caller ID

Sensory neuron asymmetry affects how insects interact

By Jessica Shugart

To avoid a scuffle, a wayward honeybee might do best to stay on a stranger's left. That's because honeybees preferentially use their right antennae to distinguish between friends and intruders, researchers report June 27 in *Scientific Reports*.

Scientists knew that the bees' left and right antennae picked up different sensory cues, but the new work makes clear that this asymmetry extends into how bees navigate social situations.

The study also helps scientists understand a "big and interesting question: Why are our brains asymmetric?" says honeybee physiologist Julie Mustard of Arizona State University in Tempe. "The idea is that asymmetries allow the brain to have more area for processing complex information."

Honeybee antennae are blanketed with a jungle of hairlike sensilla, microscopic protrusions housing neurons that transmit sensory information to the brain. Compared with the left antenna, the right contains more sensilla dedicated to smell, known to play a key role in honeybee communication.

To find out whether lopsidedness would influence behavior, researchers led by Giorgio Vallortigara of the University of Trento in Italy snipped bees' right or left antennae and then paired off the clipped bees in petri dishes. When both members of the pair came from one hive, couples with intact right antennae responded quickly with a French kiss of sorts: They used their tongues to sample each other's fluids. But leftie hive-mates held back the friendly overtures, sometimes exposing their jaws or pointing stingers at each other.

In pairs of bees from two different colonies, the right-antennaed bees were more likely to act aggressively toward strangers than the lefties, which mounted fewer stinger-baring displays toward the foreign bees.

A honeybee that's missing its right antenna is less likely than intact bees to get aggressive with strangers.

ELISA RIGOSI

The right and left sides of the bees' brains perform different functions, Vallortigara says, making their brains more like humans' than scientists had expected. The open question is whether a common genetic recipe leads to brain asymmetry across species, Vallortigara says. ■

"It's the first time I've heard of good proof of the microbiome playing a role in maintenance of species." —SUSAN PERKINS

Microbes may separate species

Removing bacteria in wasps spares crossbred offspring

By Susan Milius

The microbes teeming inside creatures may be an overlooked but vital part of what divides host organisms into species.

Two species of jewel wasp (*Nasonia girault*i and *N. vitripennis*) stay separate largely because most male larvae die when the species crossbreed, say Seth Bordenstein and Robert Brucker of Vanderbilt University in Nashville. Biologists have long blamed the demise on lethal incompatibilities in DNA. Yet using antibiotics to kill off the gut bacteria in the supposedly doomed hybrids rescued many of them, Bordenstein reported June 23 at a meeting and July 18 in *Science*. In this lab test, the germ-free hybrid larvae survived about as well as purebred germ-free larvae.

Parasitic jewel wasps (female shown) maintain their species barriers with help from microbes.

In a further test, Brucker gave microbe-free hybrid larvae two kinds of gut bacteria from regular hybrids. Survival rates plummeted.

"I would never have predicted that," said Corrie Moreau, an ant taxonomist at the Field Museum in Chicago. "We were blown away."

Susan Perkins, microbiology curator at the American Museum of Natural History in New York, agreed. "It's the first time I've heard of good proof of the microbiome playing a role in maintenance of species," she said.

The experiments don't mean genetics is irrelevant for separating these species, Bordenstein said. But maybe biologists have missed part of the story. Two species may split not only because their genes change but also because their communities of resident microbes differ, he said.

To see how those communities might have diverged, Brucker and Bordenstein compared the kinds of microbes flourishing in four species of jewel wasp. The researchers used the comparison to create an evolutionary family tree, and the relationships it showed are the same ones that scientists had deduced from the wasps' genes.

The fatal mismatch of microbes divides species that diverged long ago in the family tree, but not a more recently separated pair. Both genetic and microbial trees show that *N. girault*i's closest relative is *N. longicornis*. Lab-bred hybrids of the two don't die off in great numbers. And rendering hybrids of the more recently divided species germ-free didn't make a noticeable difference in survival. The genetic and microbial barriers are both lower in this more recent species split than across the ancient wasp divide.

Just how wasp genetics and microbes interact to kill hybrids remains to be seen. Bordenstein pointed out that the dying larvae darken in color, as if secreting melanin — a common response to inflammation. Some of the parts of animal genomes that evolve most rapidly are those dealing with immune systems, so perhaps genes separating species end up welcoming or repelling different microbes.

The experiment presents a good reason to look at what's called the hologenome, the combined DNA of an organism and its many microscopic residents, Bordenstein said. "That's kind of a controversial term right now," he said, because there aren't many studies showing when the host-microbe combination matters. Looking at combined DNA certainly expands the notion of which genes matter. A human has roughly 20,000 genes, Bordenstein said, but resident microbes add some 8 million. ■

COURTESY OF R.M. BRUCKER

MEETING NOTES

Terrapins show off

Human eyes may not do justice to the spectacle of terrapins flirting. Male and female diamondback terrapins (*Malaclemys terrapin*) in eastern North America gather in shallow water during breeding season. The first study of how these terrapins might perceive their potential mates finds that, unlike humans, terrapins see ultraviolet wavelengths as well as blue, green and red light, Abby Dominy of Drexel University in Philadelphia reported on June 22. Terrapin shells don't reflect UV, but the reptiles' skin does. In shallow water, enough UV penetrates for terrapins to show off their contrasting patches of shell and skin. Whether female terrapins find male displays alluring is Dominy's next question. —*Susan Milius*

Might be giants

By getting creative in defining "island," scientists have found a new way to test why creatures evolve giant forms when they move onto islands. Brian Langerhans of North Carolina State University and his colleagues studied the Bahamas mosquitofish (*Gambusia hubbsi*) around Andros Island. There, mosquitofish colonize small, isolated inland bodies of water called blue holes. On average, mosquitofish in the 23 blue holes studied are now nearly 20 percent longer than their offshore relatives, Langerhans said June 23. Milder competition may have made a difference, he said: The mosquitofish tended to evolve larger bodies in blue holes with fewer other kinds of fish competing. —*Susan Milius*

Health & Illness

Microbes put brakes on colitis

Gut bacteria's compounds tame immune system

By Jessica Shugart

Common molecules made by bacteria in the gut may act as chill pills for the immune system. These molecules prevent misplaced immune attacks in inflammatory bowel diseases like colitis, a new study finds.

"It is a huge advance," says Sarkis Mazmanian of Caltech. "This opens up the notion that a very easy and potentially very safe therapy for inflammatory bowel disease could exist."

Years of research have hinted that microbes play a role in obesity, allergy, inflammatory bowel disease and colon cancer, diseases linked to immune dysfunction. But scientists have had difficulty pinpointing direct links between the bacteria in the gut and the army of immune cells living there.

Some researchers have focused on individual microbial species among the gut's teeming hordes to see how they affect the immune system. But Wendy Garrett's team at Harvard University decided to look instead for possible immune tamers among molecules common to many different species of bacteria. The team chose to investigate short-chain fatty acids because bacterial species that make large amounts of these molecules are in short supply in some people with inflammatory bowel disease.

To see whether the microbial molecules play a role in quieting the immune system, the researchers added them to mice's drinking water. The animals developed elevated levels of inflammation-dousing regulatory T cells in their colons, the team reports July 4 in *Science*. The cells work like wet blankets, dampening autoimmune flare-ups before they burn out of control.

The team also found that those short-chain fatty acids protected mice from an experimental form of colitis, an inflammatory disease that can destroy the colon.

Garrett hopes that the acids play the same role in tamping down inflammation in people. Many bacterial species that inhabit the guts of humans also produce the acids. And people might not even have to take the fatty acids as drugs to boost their supply. Eating higher amounts of dietary fiber might also do the trick, because the microbes consume fiber to make the acids.

Studies have shown that, compared with people in some developing countries, Westerners tend to consume lower amounts of dietary fiber and have lower amounts of short-chain fatty acids in their guts. They also have higher — and increasing — rates of inflammatory bowel disease.

The study suggests that a lack of dietary fiber could reduce levels of short-chain fatty acids in the gut and might explain that elevated prevalence of inflammatory bowel disease, says gut microbiologist Justin Sonnenburg at Stanford University. "A really good hypothesis at this point is that reduced short-chain fatty acid production over time is bad for colonic health." ■

Four-question test ID's depression

Uncomplicated tool is quick way to identify mood disorders

By Bruce Bower

A surprisingly simple decision-making tool shows promise as a way for physicians to identify people with depression.

An answer to the first of four questions was all that researchers usually needed to identify women who weren't depressed, say psychologist Mirjam Jenny of the Max Planck Institute for Human Development in Berlin and her colleagues. Using all four questions, this tool spotted depressed women about as well as two more complex methods, Jenny's team reports June 24 in the *Journal of Applied Research in Memory and Cognition*.

If the findings hold up in other studies, physicians and other professionals with no mental-health training could use this brief technique to tag individuals who need thorough depression evaluations. "This decision tree can be used to screen for depression, but not to reach a final diagnosis," Jenny says.

Her team drew on data from 1,382 German women who completed a 21-item screening questionnaire for depression on two occasions, separated by 18 months. Based on this measure, depression initially affected 3.6 percent of the sample, or 50 individuals, and later appeared in 1.9 percent of the sample, or 26 individuals. Women's initial responses to a handful of items that best predicted whether they would rank as depressed 18 months later were used to create a four-question decision tree.

The first question in the tree — "Have you cried more than usual in the last week?" — led the pack in identifying cases of depression. A "no" response to this or any of the other three questions — which inquired about feelings in the last week of disappointment or self-hate, discouragement about the future and personal failure — exempted women from being categorized as depressed. Those who responded "yes" to all four questions were classified as depressed.

The tool is impressive, remarks physician and health care researcher Glyn Elwyn of the Dartmouth Center for Health Care Delivery Science in Hanover, N.H. But it may not be sensitive to depression in men, he says. ■

24.8 per 100,000 | New U.S. cases per year of type 1 diabetes in whites under age 10

22.6 per 100,000 | New U.S. cases per year of type 1 diabetes in whites ages 10–19

First foods linked to diabetes risk

Timing may be important in babies predisposed to condition

By Nathan Seppa

Infants at risk of type 1 diabetes who receive their first solid food between ages 4 months and 6 months appear less likely to develop the condition than those starting solid food outside that time window.

Type 1 diabetes, which can strike children at any age, occurs when an aberrant immune reaction kills cells in the pancreas, requiring a person to take insulin shots. Two studies in 2003 found an association between early first foods and the presence of rogue antibodies, a warning sign of type 1 diabetes. The new findings go an important step further, tracking babies long enough to see who developed diabetes, says Kendra Vehik, an epidemiologist at the University of South Florida in Tampa.

The new study, which appears July 8 in *JAMA Pediatrics*, included 1,835 children in the Denver area who had reached at least age 7. They were at high risk because they either carried a genetic variant associated with the disease or had a parent or sibling with type 1 diabetes. Of the 53 children with diabetes, 28 had had their first exposure to solid food before age 4 months. Diabetes risk in that group was roughly double that of kids who had started eating food at age 4 to 6 months.

Babies who had eaten their first foods later than age 6 months had a tripled risk. But very few children were started on solid foods that late, so study coauthor Jill Norris, an epidemiologist at the University of Colorado Denver, says she's unsure of the reliability of that result.

The study also suggests an increased risk from introducing fruit before 4 months and rice and oats after 6 months, but those findings aren't statistically strong enough to implicate the timing of introducing those particular foods in diabetes risk, Norris says.

More interesting, she says, was a finding that babies who were breast-fed when they were introduced to wheat were about half as likely to develop type 1 diabetes as were infants not breast-fed while starting on wheat. Researchers know that infants' immune systems are still a work in progress. One hypothesis holds that first solid foods might overstimulate the immune system, Norris says. How that would affect the complex immune reaction that causes type 1 diabetes, or whether breast-feeding might prevent it in some cases, remains unknown. ■

Earth

21 Average annual number of earthquakes greater than or equal to magnitude 3.0 in central and eastern U.S., 1967–2000

100+ Average annual number of earthquakes greater than or equal to magnitude 3.0 in central and eastern U.S., 2010–2012

Huge quakes foretell smaller ones

Ominous activity triggered at wastewater injection sites

By Erin Wayman

Giant, distant earthquakes may help scientists identify places where humans are liable to set off smaller tremors when they inject fluid deep into geologic deposits.

Scientists have known for decades that injecting huge volumes of liquid underground — such as waste from hydraulic fracturing, or fracking — can set off quakes. But in most cases it doesn't, and scientists can't predict when or where such earthquakes will happen.

In the July 12 *Science*, seismologists report that massive earthquakes unleash seismic waves that can trigger tremors near wastewater disposal wells half a world away. The tiny quakes may warn that a fault is close to rupture.

"When we do see remote triggering, it seems to foreshadow larger induced earthquakes," says coauthor Nicholas van der Elst of Columbia University's Lamont-Doherty Earth Observatory in Palisades, N.Y. "It shows the faults are reaching a tipping point."

Concerns over human-caused quakes have grown in recent years as earthquake activity has shot up in unexpected places. From 1967 to 2000, the central and eastern United States experienced an average of 21 earthquakes per year of magnitude 3.0 or greater. From 2010 to 2012, the region saw more than 300 such quakes, reports William Ellsworth, a seismologist at the U.S. Geological Survey in Menlo Park, Calif., in the same issue of *Science*.

The rise in earthquakes in Arkansas, Colorado, Ohio, Texas and elsewhere coincides with an increase in extracting natural gas and oil from shale formations in those states. Oil and gas don't easily flow through impermeable shale, so petroleum companies drill horizontal wells and pump in pressurized fluid to fracture a small section of rock (*SN: 9/8/12, p. 20*). This fracking process itself doesn't cause earthquakes, Ellsworth says. But the fracking fluid comes to the surface along with the oil or gas. Contaminated by a mix of chemicals, the fluid is disposed of by injecting it deep underground, where it puts pressure on faults.

"Any time we change the fluid regime underground, we could bring a fault closer to failure," Ellsworth says.

The United States is home to tens of thousands of wastewater disposal wells. "The vast majority," Ellsworth says, "don't appear to induce earthquakes." Scientists don't understand why only some pose a risk.

The discovery by van der Elst and colleagues may help identify spots where quakes will pop up. In reviewing seismic activity in the Midwest from 2003 to 2013, the researchers discovered that distant earthquakes appeared to initiate small tremors near some wastewater disposal wells, which in turn presaged larger quakes.

At each of three sites in Oklahoma, Texas and Colorado, the team saw seismic activity spike in the days following at least one massive earthquake: a 2010 magnitude 8.8 quake in Chile, a 2011 magnitude 9.1 event in Japan and a magnitude 8.6 earthquake in Sumatra in 2012. Six to 20 months after the initial tremors, each of the three sites experienced quakes ranging from magnitude 4.3 to 5.7.

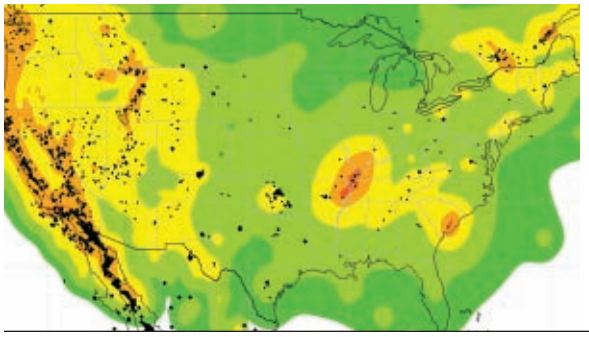
The seismic waves from the giant temblors probably perturbed fluids in the faults, increasing the pressure, van der Elst says.

If operators can identify when a fault is about to rupture, they can adjust how much fluid they are injecting into a well or stop the injection altogether. However, the usefulness of this foreshadowing is limited because big earthquakes that can remotely trigger tremors occur only rarely, says Cliff Frohlich, a seismologist at the University of Texas at Austin. They happen roughly once a year.

Another limitation is that not all wastewater sites that produced earthquakes during the study responded to remote triggering. Van der Elst suggests that when a well is very close to a fault, just a few months — rather than years or decades — of fluid injection can build pressure and cause a fault to slip. In these cases, there's little chance of a big earthquake happening before the fault reaches its tipping point.

Even with caveats, the work helps scientists better understand the nature of induced quakes. For the most part, earthquakes related to wastewater disposal have been small to moderate and not caused much damage. When such wells were limited to remote places like West Texas, small rumbles in the ground didn't bother anyone, Frohlich says. Now that they are being built near densely populated areas like Dallas, people are rethinking the hazards.

Increasingly shaky Over the last few years, the number of earthquakes in the central and eastern United States has increased. So has the number of wastewater disposal wells associated with fracking. The map shows all of the magnitude 3.0 or greater earthquakes that occurred in the United States in 2009–2012. Places with the highest natural earthquake hazards are shown in red and orange while areas with lower risk are shown in yellow and green.

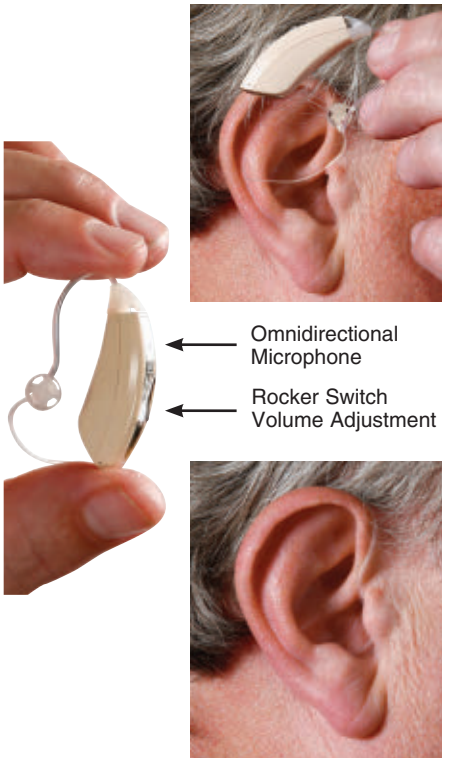


COURTESY OF *SCIENCE*/AAAS

Personal Sound Amplification breakthrough

Our sound engineers understand acoustics so you can understand conversations

Perfect Choice HD™ features world-class processors for natural sound quality in a wide spectrum of listening situations.



If you are considering personal sound amplification, you probably know that louder isn't always better. If it were, you could just get one of those ear horns that were popular back in the 19th Century... as long as you weren't concerned with your appearance. Fact is, human conversation- the main reason people get amplification, occupies a relatively small range in the overall sound spectrum. Sound quality and speech intelligibility are much more important than sheer volume. That's why the sound engineers behind the Perfect Choice HD™ focus on optimal listening in challenging situations. The latest algorithms are running on advanced processors that are super small, yet super powerful. Best of all, Perfect Choice HD™ is comfortable and discreet. The lightweight case (you can choose between Silver and Beige) nestles in behind your ear. The clear hearing tube (good luck spotting it in the picture to the right... look closely, it's there) is connected to a flexible tip that fits comfortably into your ear canal. No one needs to know you are using it, and no one needs to know it didn't cost you thousands of dollars. The unit comes pre-programmed and is ready to use right out of the box, so it's the hassle-free choice that you can begin enjoying immediately.

Superior engineering makes it better

- Mosaic™ multi-layered noise reduction accentuates voices and seeks to lower general background noise from the environment.

- Advanced adaptive feedback suppression delivers high-quality sound even in demanding situations. Reducing feedback while delivering high-frequency gain is the hallmark of a professional grade personal amplifier.

- Expansion feature automatically acts to further reduce low-level noise while in particularly quiet environments.

- Wide Dynamic Range Compression brings soft voices into focus and reduces unexpected loud sounds so it can be worn all day long. It automatically changes, in real time to optimally fit your environment.

Why Perfect Choice HD is the best choice !	
Lightweight / Inconspicuous	Less than 1 ounce
Sound Quality	Excellent – Optimized for speech
Test and Fitting Required	No
Free Batteries for Life	Yes, ask for details
One-on-One Personal Set Up	Free
Friendly Return Policy	60 Days

Are you or a loved one frustrated in these situations?

• Restaurants • Dinner parties
• Outdoor conversations
• Lectures • Sermons
• Meetings
...and other times where you need to turn up the volume

Affordable, Simple to use, Virtually impossible to see

Why spend another day wondering if amplification is right for you. Perfect Choice HD™ comes with our exclusive home trial- if you are not completely satisfied simply return it for a refund of the product purchase price. Call now, you'll be glad you did... and so will your friends and family.

Perfect Choice HD™

Personal Sound Amplification Product

Call now and find out how you can get **FREE Batteries for Life!**

Please mention promotional code 46982.

1-877-566-0345

Perfect Choice HD™

1998 Ruffin Mill Road
Colonial Heights, VA 23834

80785

Perfect Choice HD is not a hearing aid. If you believe you need a hearing aid, please consult a physician.

Molecules

For more Molecules stories, visit **www.sciencenews.org**

Surgical tool smokes out cancer

Method sniffs for molecular sign of certain tissue types

By Cristy Gelling

A new tool could tell surgeons within seconds whether they are slicing through cancerous or healthy tissue. The tool, which analyzes smoke produced by electric currents used to cut or destroy tissue, was about 95 percent accurate in identifying cancers and other human tissues during surgery.

Testing the smoke could help surgeons identify the outer margins of a tumor and remove as much of it as possible, leaving healthy tissue intact. Currently, if doctors need information about the extent of a cancer during a surgical procedure, they must wait 20 to 30 minutes for a tissue sample to be examined under a microscope. The new tool, called the iKnife, delivers a diagnosis in 2.5 seconds or less, researchers report July 17 in *Science Translational Medicine*.

The iKnife consists of an electric blade hooked up to an instrument that performs chemical analysis. "They are basically blowing up tissue, making smoke out of it and then sampling that smoke with a mass spectrometer," says Nicholas Winograd, a chemist at Pennsylvania State University. "I don't think it's at all obvious that this kind of thing would work, and I give them a lot of credit for developing it."

Mass spectrometry is an analytical method that converts molecules in complex biological mixtures into electrically charged particles and then identifies them based on their mass and charge.

Because tools that use electric currents to cut tissue generate a haze of charged particles from human tissues, the researchers realized they could directly analyze the smoke. In surgery, these electrical cutting tools "are as common as scalpels," says study leader Zoltán Takáts of Imperial College London.

One type of charged particle in the surgical smoke is fat. Takáts' team found that smoke from each type of tissue and cancer had characteristic proportions of different fat molecules. The researchers discovered this when they created a database of mass spectrometry results from nearly 3,000 tissue samples from 302 patients. When analyzing a new sample, the iKnife can compare its mass spectrum to the ones in the database and predict its tissue type.

Surgeons removing many kinds of cancers then used the iKnife to test the database's predictions. For approximately 95 percent of samples from 91 surgeries, the iKnife gave a diagnosis that matched the results from standard postoperative tests. In 11 patients, the iKnife revealed that the preoperative cancer diagnosis had been incorrect.

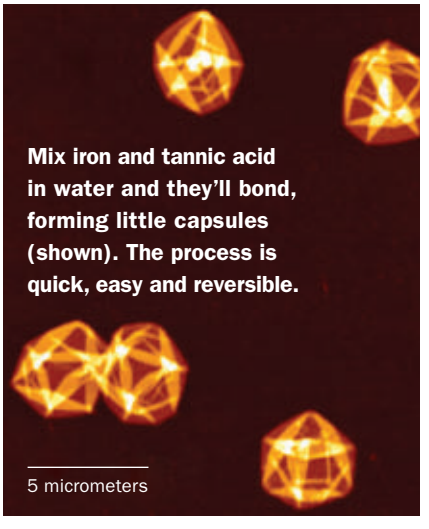
To get regulatory approval, Takáts and his team need to complete formal clinical trials. These trials will test whether the new approach improves outcomes for patients going under the knife. ■

A simple recipe for coatings

Cheap approach may prove useful in foods, medicines

By Rachel Ehrenberg

It's not often that chemists find a quick, simple and cheap method for making things with widely available ingredients, but researchers have done just that: They've created elegant little capsules and coatings in water simply by mixing iron and a compound from plants called tannic acid. The soft coatings form on their own around whatever else is in the water — glass beads, bacteria, gold nanoparticles and more. Just changing the solution's acidity can prompt the coatings to disassemble.



Mix iron and tannic acid in water and they'll bond, forming little capsules (shown). The process is quick, easy and reversible.

COURTESY OF *SCIENCE*/AAAS

The coatings' ingredients are considered safe — tannic acid is found in wine, while iron is an important element for living things. That means the capsules might help in delivering drugs in the body or find use in cosmetics or foods, says bioengineer Gregory Payne of the University of Maryland in College Park.

The work fits with an ongoing effort to find biologically friendly, useful materials, Payne says, and it takes advantage of materials that are right under everyone's noses. "It opens up a lot of opportunities."

Using ordinary lab equipment, the research team, led by materials scientist Frank Caruso of the University of Melbourne in Australia, create the tiny coatings at room temperature. When the researchers add tannic acid to water, it tends to congregate around surfaces, whether pieces of polystyrene or *E. coli* bacteria. When the researchers add iron ions to the mix, the iron latches on to the tannic acid molecules, connecting them into a thin film. At a pH of 7.4, the capsules were still intact after 10 days; at a pH of 3, they disassembled within four hours, the team reports in the July 12 *Science*. ■

Genes & Cells

For more Genes & Cells stories, visit **www.sciencenews.org**

Gene therapy made safer

Six kids with rare diseases stay healthy up to three years

By Tina Hesman Saey

A virus derived from HIV can safely fix broken immune systems and correct genetic diseases, suggest two new studies involving children with rare conditions.

For both studies, researchers put healthy genes into the children's own DNA using lentiviruses, in this case genetically engineered versions of HIV that can no longer cause disease. Earlier gene therapy trials using different viruses had a flaw: When the viruses plunked themselves into the patient's DNA, they sometimes amped up activity of neighboring cancer-causing genes, leading to leukemia. That side effect, along with the death of a young man participating in another clinical trial, nearly halted gene therapy in the United States in the early 2000s.

Now, researchers led by Luigi Naldini of the San Raffaele Telethon Institute for Gene Therapy in Milan have altered the lentiviruses so that they won't accidently turn on nearby genes. The researchers then infect bone marrow stem cells with lentiviruses carrying the appropriate gene and transplant the stem cells into patients.

In one study, three boys with Wiskott-Aldrich syndrome, an inherited disease that disables the immune system, received gene therapy. Now, two to three years after the therapy, the former "bubble boys" have healthy immune systems, Naldini and colleagues report July 11 in *Science*. The boys also show no signs of developing leukemia — which should help allay concerns about the team's gene therapy approach, says Todd Rosengart, a surgeon and gene therapy researcher at Baylor College of Medicine in Houston.

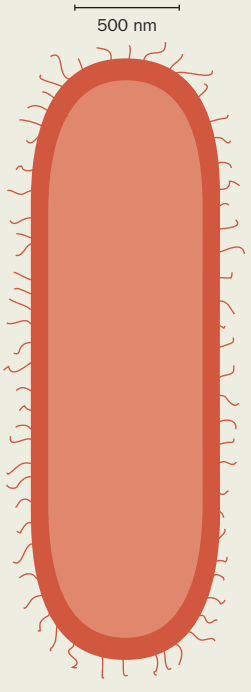
In the second trial, Naldini and his colleagues treated three children with a metabolic disease called metachromatic leukodystrophy. Children with the disease lack an important enzyme. As a result, they gradually become paralyzed and suffer cognitive damage, dying within a couple of years. Up to two years after the therapy, the children in the study are still making enough of the enzyme to keep their brain and spinal cord working normally with no sign of leukemia, the researchers report in another paper published at the same time in *Science*.

The results are encouraging, says Uta Griesenbach, a gene therapist at Imperial College London. "Even after fairly long-term follow-up, it appears to be safe and effective." The boys aren't out of the woods yet — some of the patients in the original gene therapy trials didn't develop cancer until up to five years after treatment. But Griesenbach says that the children in the new studies don't have warning signs of cancer.

Because the lentiviruses appear safe and work so well, scientists may start doing gene therapy for more common conditions such as Parkinson's disease, says Senlin Li, a medical researcher at the University of Texas Health Science Center at San Antonio. ■

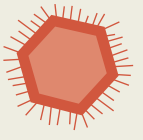
Giant virus is notable not just for size

The largest virus ever identified has been found on the seafloor off the coast of Chile. At 1,000 nanometers long, *Pandoravirus salinus* is about twice the length of the previous record holder for biggest virus, *Megavirus chilensis*, and has a genome twice as large. That makes *P. salinus* larger than the smallest bacteria. Beyond its impressive size (see scale comparisons below to other viruses and at left, the bacterium *E. coli*), the *Pandoravirus* has some other strange qualities. Rather than reproducing by first making a viral coat and then filling it in or by building its coat around genetic material, *P. salinus* builds its insides and outsides simultaneously, starting at one end of the horseshoe-shaped viral particle and finishing at the other. What's more, only 7 percent of the virus's genes match any known gene sequences, researchers report in the July 19 *Science*. The authors suggest a controversial hypothesis for why the *Pandoravirus* is so odd: It could have evolved from a type of free-living ancient cell that no longer exists. Its discovery is likely to add fuel to the heated debate about the evolutionary origins of viruses. —*Cristy Gelling*



Escherichia coli
Base pairs: 4.6 million
Length: 3,000 nm
Diameter: 1,000 nm

Pandoravirus salinus
Base pairs: 2.5 million
Length: 1,000 nm
Diameter: 500 nm



Megavirus chilensis
Base pairs: 1.26 million
Diameter: 500 nm

Influenza type A
Base pairs: 13,500
Diameter: 100 nm

E. OTWELL

The Anorexic Brain

Neuroimaging improves understanding of eating disorder

By Meghan Rosen

In a spacious hotel room not far from the beach in La Jolla, Calif., Kelsey Heenan gripped her fiancé's hand. Heenan, a 20-year-old anorexic woman, couldn't believe what she was hearing. Walter Kaye, director of the eating disorders program at the University of California, San Diego, was telling a handful of rapt patients and their family members what the latest brain imaging research suggested about their disorder.

It's not your fault, he told them.

Heenan had always assumed that she was to blame for her illness. Kaye's data told a different story. He handed out a pile of black-and-white brain scans — some showed the brains of healthy people, others were from people with anorexia nervosa. The scans didn't look the same. "People were shocked," Heenan says. But above all, she remembers, the group seemed to sigh in relief, breathing out years of buried guilt about the disorder. "It's something in the way I was wired — it's something I didn't choose to do," Heenan says. "It was pretty freeing to know that there could be something else going on."

Years of psychological and behavioral research have helped scientists better understand some signs and triggers of anorexia. But that knowledge hasn't straightened out the disorder's tangled roots, or pointed scientists to a therapy that works for everyone. "Anorexia has a high death rate, it's expensive to treat and people are chronically ill," says Kaye.

Kaye's program uses a therapy called family-based treatment, or FBT, to teach adolescents and their families how to manage anorexia. A year after therapy, about half of the patients treated with FBT recover. In the world of eating disorders, that's success: FBT is considered one of the very best treatments doctors

LUKE LUCAS

have. To many scientists, that just highlights how much about anorexia remains unknown.

Kaye and others are looking to the brain for answers. Using brain imaging tools and other methods to explore what's going on in patients' minds, researchers have scraped together clues that suggest anorexics are wired differently than healthy people. The mental brakes people use to curb impulsive instincts, for example, might get jammed in people with anorexia. Some studies suggest that just a taste of sugar can send parts of the brain barrelling into overdrive. Other brain areas appear numb to tastes — and even sensations such as pain. For people with anorexia, a sharp pang of hunger might register instead as a dull thud.

The mishmash of different brain imaging data is just beginning to highlight the neural roots of anorexia, Kaye says. But because starvation physically changes the brain, researchers can run into trouble teasing out whether glitchy brain wiring causes anorexia, or vice versa. Still, Kaye thinks understanding what's going on in the brain may spark new treatment ideas. It may also help the eating disorder shake off some of its noxious stereotypes.

"One of the biggest problems is that people do not take this disease seriously," says James Lock, an eating disorders researcher at Stanford University who cowrote the book on family-based treatment. "No one gets upset at a child who has cancer," he says. "If the treatment is hard, parents still do it because they know they need to do it to make their child well."

Pop culture often paints anorexics as willful young women who go on diets to be beautiful, he says. But, "you can't just choose to be anorexic," Lock adds. "The brain data may help counteract some of the mythology."

Beyond dieting

A society that glamorizes thinness can encourage unhealthy eating behaviors in kids, scientists have shown. A 2011 study of Minnesota high school students reported that more than half of girls had dieted within the past year. Just under a sixth had used diet pills, vomiting, laxatives or diuretics.

But a true eating disorder goes well beyond an unhealthy diet. Anorexia involves malnutrition, excessive weight loss and often faulty thinking about one of the body's most basic drives: hunger. The disorder is also rare. Less than 1 percent of girls develop anorexia. The disease crops up in boys too, but adolescent girls — especially in wealthy countries such as the U.S., Australia and Japan — are most likely to suffer from the illness.

As the disease progresses, people with anorexia become intensely afraid of getting fat and stick to extreme diets or exercise schedules to drop pounds. They also misjudge their own weight. Beyond these diagnostic hallmarks, patients' symptoms can vary. Some refuse to eat, others binge and purge. Some live for years with the illness, others yo-yo between weight gain and loss. Though most anorexics gain back some weight within five years of becoming ill, anorexia is the deadliest of all mental disorders.

46 percent

Anorexic patients who fully recover

H.C. STEINHAUSEN/*CHILD AD. PSYCH. CLIN. N. AMERICA* 2009

0.6 percent

Portion of U.S. adult population who will suffer from anorexia in their lifetimes

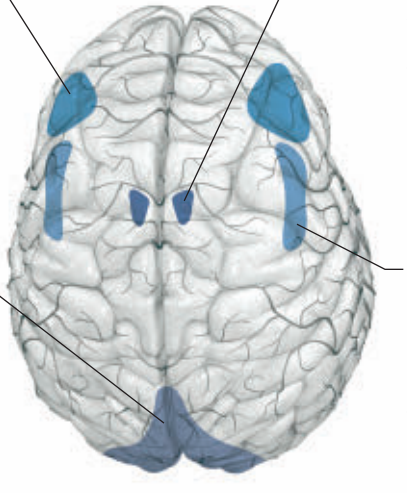
NATIONAL INSTITUTE OF MENTAL HEALTH

Though anorexia tends to run in families, scientists haven't yet hammered out the suite of genes at play. Some individuals are particularly vulnerable to developing an eating disorder. In these people, stressful life changes, such as heading off to college, can tip the mental scales toward anorexia.

For decades, scientists have known that anorexic children behave a little differently. In school and sports, anorexic kids strive for perfection. Though Heenan, a former college basketball player, didn't notice her symptoms creeping in until the end of high school, she remembers initiating strict practice regimens as a child. Starting in second grade, Heenan spent hours perfecting her jump shot, shooting the ball again and again until she had the technique exactly right — until her form was flawless.

"It's very rare for me to see a person with anorexia in my office who isn't a straight-A student," Lock says. Even at an early age, people who later develop the eating disorder tend to exert an almost superhuman ability to practice,

Different wiring Studies of the brains of people with anorexia have revealed a number of complex brain circuits that show changes in activity compared with healthy people.



Dorsolateral prefrontal cortex
A self-control center of the brain. The DLPFC acts like a brake system to curb impulsive behaviors. In anorexics, the DLPFC may work overtime to keep people from giving in to the temptation to eat.

Visual cortex
Processes visual information. Compared with healthy people, the visual cortices of anorexics may be more active when thinking about eating food or performing cognitive tasks.

Ventral striatum
Part of the brain's reward circuitry. The ventral striata of anorexics may be hypersensitive to flavors healthy people find pleasurable, such as sugar. This oversensitivity could affect patients' enjoyment of food.

Insula
Involved in self-awareness of body states, such as pain and hunger. The insula is the first brain region to register the taste of sweets. In anorexics, the insula may not correctly detect sweets and other signals.

MEDICALRF, ADAPTED BY M. ATAROD

focus or study. "They will work and work and work," says Lock. "The problem is they don't know when to stop."

In fact, many scientists think anorexics' brains might be wired for willpower, for good and ill. Using new imaging tools that let scientists watch as a person's mental gears grind through different tasks, researchers are starting to pin down how anorexic brains work overtime.

Control signs

To glimpse the circuits that govern self-control, experimental neuropsychologist Samantha Brooks uses functional magnetic resonance imaging, or fMRI, a tool that measures and maps brain activity. Last year, she and colleagues scanned volunteers as they imagined eating high-calorie foods, such as chocolate cake and French fries, or using inedible objects such as clothespins piled on a plate. One result gave Brooks a jolt. A center of self-control in anorexics' brains sprung to life when the volunteers thought about food — but only in the women who severely restricted their calories, her team reported March 2012 in *PLOS ONE*.

The control center, two golf ball–sized chunks of tissue called the dorsolateral prefrontal cortex, or DLPFC, helps stamp out primitive urges. "They put a brake on your impulsive behaviors," says Brooks, now at the University of Cape Town in South Africa.

For Brooks, discovering the DLPFC data was like finding a tiny vein of gold in a heap of granite. The control center could be the nugget that reveals how anorexics clamp down on their appetites. So she and her colleagues devised an experiment to test anorexics' DLPFC. Using a memory task known to engage the brain region, the researchers quizzed volunteers while showing them subliminal images. The quizzes tested working memory, the mental tool that lets people hold phone numbers in their heads while hunting for a pen and paper. Compared with healthy people, anorexics tended to get more answers right, Brooks' team wrote June 2012 in *Consciousness and Cognition*. "The patients were really good," Brooks says. "They hardly made any mistakes."

15–19 years

Peak age of onset of anorexia

A.R. LUCAS *ET AL/AM. J. PSYCH.* 1991

12 times

Factor by which the annual death rate for young women with anorexia is higher than that for young women in the general population

NIMH

A turbocharged working memory could help anorexics hold on to rules they set for themselves about food. "It's like saying 'I will only eat a salad at noon, I will only eat a salad at noon,' over and over in your mind," says Brooks. These mantras may become so ingrained that an anorexic person can't escape them.

But looking at subliminal images of food distracted anorexics from the memory task. "Then they did just as well as the healthy people," Brooks says. The results suggest that anorexic people might tap into their DLPFC control circuits when faced with food.

James Lock has also seen signs of self-control circuits gone awry in people with eating disorders. In 2011, he and colleagues scanned the brains of teenagers with different eating disorders while signaling them to push a button. While volunteers lay inside the fMRI machine, researchers flashed pictures of different letters on an interior screen. For every letter but "X," Lock's group told the teens to push a button. During the task, anorexic teens who obsessively cut calories tended to have more active visual circuits than healthy teens or those with bulimia, a disorder that compels people to binge and purge. The result isn't easy to explain, says Lock. "Anorexics may just be more focused in on the task."

Bulimics' brains told a simpler story. When teens with bulimia saw the letter "X," broad swaths of their brains danced with activity — more so than the healthy or calorie-cutting anorexic volunteers, Lock's team reported in the *American Journal of Psychiatry*. For bulimics, controlling the impulse to push the button may take more brain power than for others, Lock says.

Though the data don't reveal differences in self-control between anorexics and healthy people, Lock thinks that anorexics' well-documented ability to swat away urges probably does have signatures in the brain. He notes that his study was small, and that the "healthy" people he used as a control group might have shared similarities with anorexics. "The people who tend to volunteer are generally pretty high performers," he says. "The chances are good that my controls are a little bit more like anorexics than bulimics."

Still, Lock's results offered another flicker of proof that people with eating disorders might have glitches in their self-control circuits. A tight rein on urges could help steer anorexics toward illness, but the parts of their brain tuned into rewards, such as sugary snacks, may also be a little off track.

Sugar low

For many anorexics, food just doesn't taste very good. A classic symptom of the disorder is anhedonia, or trouble experiencing pleasure. Parts of Heenan's past reflect the symptom. When she was ill, she had trouble remembering favorite

Food alert Images of high-calorie foods (left) switched on a self-control center in the brains of anorexic women. Pictures of objects on plates kept the control center quiet.

COURTESY OF S. BROOKS

dishes from childhood, for example — a blank spot common to anorexics. "I think I enjoyed some things," she says. Beyond frozen yogurt, she can't really rattle off a list.

After Heenan started seriously restricting her calories in college, only one aspect of food made her feel satisfied. Skipping, rather than eating, meals felt good, she says. Some of Heenan's symptoms may have stemmed from frays in her reward wiring, the brain circuitry connecting food to pleasure. In the past few years, researchers have found that the chemicals coursing through healthy people's reward circuits aren't quite the same in anorexics. And studies in rodents have linked chemical changes in reward circuitry to under- and overeating.

To find out whether under- and overweight people had altered brain chemistry, eating disorder researcher Guido Frank of the University of Colorado Denver studied anorexic, healthy-weight and obese women. He and his colleagues trained volunteers to link images, such as orange or purple shapes, with the taste of a sweet solution, slightly salty water or no liquid. Then, the researchers scanned the women's brains while showing them the shapes and dispensing tiny squirts of flavors. But the team threw in a twist: Sometimes the flavors didn't match up with the right images.

When anorexics got an unexpected hit of sugar, a surge of activity bloomed in their brains. Obese people had the opposite response: Their brains didn't register the surprise. Healthy-weight women fit somewhere in the middle, Frank's team reported August 2012, in *Neuropsychopharmacology*. While obese people might not be sensitive to sweets anymore, a little sugar rush goes a long way for anorexics. "It's just too much stimulation for them," Frank says.

One of the lively regions in anorexics' brains was the ventral striatum, a lump of nerve cells that's part of a person's reward circuitry. The lump picks up signals from dopamine, a chemical that rushes in when most people see a sugary treat.

Frank says that it's possible cutting calories could sculpt a person's brain chemistry, but he thinks some young people are just more likely to become sugar-sensitive than others. Frank suspects anorexics' dopamine-sensing equipment might be out of alignment to begin with. And he may be onto something. Recently, researchers in Kaye's lab at UCSD showed that the same chemical that makes people perk up when a coworker brings in a box of doughnuts might actually trigger anxiety in anorexics.

Mixed signals

Usually a rush of dopamine triggers euphoria or a boost of energy, says Ursula Bailer, a psychiatrist and neuroimaging researcher at UCSD. Anorexics don't seem to pick up those good feelings.

When Bailer and colleagues gave volunteers amphetamine, a drug known to trigger dopamine release, and then asked them to rate their feelings, healthy people stuck to a familiar script. The drug made them feel intensely happy, Bailer's team described March 2012 in the *International Journal of Eating Disorders*. Researchers linked the volunteers' happy feelings to a wave of dopamine flooding the brain, using an imaging technique to track the chemical's levels.

But anorexics said something different. "People with anorexia didn't feel euphoria — they got anxious," Bailer says. And the more dopamine coursing through anorexics' brains, the more anxious they felt. Anorexics' reaction to the chemical could help explain why they steer clear of food — or at least foods that healthy people find tempting. "Anorexics don't usually get anxious if you give them a plate of cucumbers," Bailer says.

Beyond the anxiety finding, one other aspect of the study sticks out: Instead of examining sick patients, Bailer, Kaye and colleagues recruited women who had recovered from anorexia. By studying people whose brains are no longer starving, Kaye's team hopes to sidestep the chicken-and-egg question of whether specific brain signatures predispose people to anorexia or whether anorexia carves those signatures in the brain.

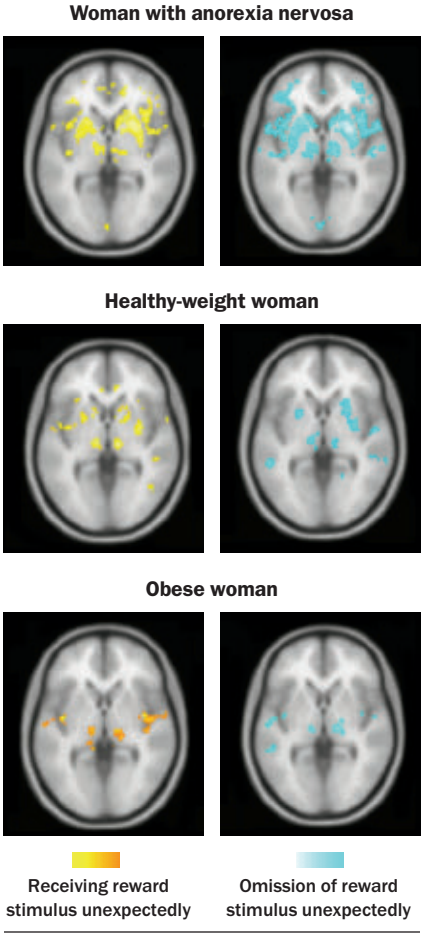
Though Kaye says that there's still a lot scientists don't know about anorexia, he's convinced it's a disorder that starts in the brain. Compared with healthy children, anorexic children's brains are getting different signals, he says. "Parents have to realize that it's very hard for these kids to change."

Kaye thinks imaging data can help families reframe their beliefs about anorexia, which might help them handle tough treatments. He thinks the data can also offer new insights into therapies tailored for anorexics' specific traits.

Sensory underload

One trait Kaye has focused on is anorexics' sense of awareness of their bodies. Peel back the outer lobes of the brain by the temples, and the bit that handles

Sugar high When an anorexic woman unexpectedly gets a taste of sugar (yellow) or misses out on it (blue), her brain's reward circuitry shows more activity than a healthy-weight or obese woman's. Anorexics' reward-processing systems may be out of order.



G. FRANK *ET AL*/*NEUROPSYCHOPHARMACOLOGY* 2012

body awareness pops into view. These regions, little islands of tissue called the insula, are one of the first brain areas to register pain, taste and other sensations. When people hold their breath, for example, and feel the panicky claws of air hunger, "the insula lights up like crazy," Kaye says.

Kaye and colleagues have shown that the insulas of people with anorexia seem to be somewhat dulled to sensations. In a recent study, his team strapped heat-delivering gadgets to volunteers' arms and cranked the devices to painfully hot temperatures while measuring insula activity via fMRI.

Compared with healthy volunteers, bits of recovered anorexics' insulas dimmed when the researchers turned up the heat. But when researchers simply warned that pain was coming, other parts of the brain region flared brightly, Kaye's team reported in January in the *International Journal of Eating Disorders*. For people who have had anorexia, actually feeling pain didn't seem as bad as anticipating it. "They don't seem to be sensing things correctly," says Kaye.

If anorexics can't detect sensations like pain properly, they may also have trouble picking up other signals from the body, such as hunger. Typically when people get hungry, their insulas rev up to let them know. And in healthy hungry people, a taste of sugar really gets the insula excited. For anorexics, this hunger-sensing part of the brain seems numb. Parts of the insula barely perked up when recovered anorexic volunteers tasted sugar, Kaye's team showed this June in the *American Journal of Psychiatry*. The findings "may help us understand why people can starve themselves and not get hungry," Kaye says.

Though the brain region that tells people they're hungry might have trouble detecting sweet signals, some reward circuits seem to overreact to the same cues. Combined with a tendency to swap happiness for anxiety, and a mental vise grip on behavior, anorexics might have just enough snags in their brain wiring to tip them toward disease.

Now, Kaye's group hopes to tap neuroimaging data for new treatment ideas. One day, he thinks doctors might be able to help anorexics "train" their insulas using biofeedback. With real-time brain scanning, patients could watch as their insulas struggle to pick up sugar signals, and then practice strengthening the response. More effective treatment options could potentially spare anorexics the relapses many patients suffer.

Heenan says she's one of the lucky ones. Four years have passed since she first saw the anorexic brain images at UCSD. In the months following her treatment, Heenan and her family worked together to rebuild her relationship with food. At first, her fiancé picked out all her meals, but step by step, Heenan earned autonomy over her diet. Today, Heenan, a coordinator for Minneapolis' public schools, is married and has a new puppy. "Life can be good," she says. "Life can be fun. I want other people to know the freedom that I do." ■

Searching for treatments

The bowl of pasta sitting in front of Kelsey Heenan didn't look especially scary.

Spaghetti, chopped asparagus and chunks of chicken glistened in an olive oil sauce. Usually, such savory fare might make a person's mouth water. But when Heenan's fiancé served her a portion, she started sobbing. "You can't do this to me," she told him. "I thought you loved me!"

Heenan was confronting her "fear foods" at the Eating Disorders Center for Treatment and Research at UCSD. Therapists in her treatment program, Intensive Multi-Family Therapy, spend five days teaching anorexic patients and families about the disorder and how to encourage healthy eating. "There's no blame," says Christina Wierenga, a clinical neuropsychologist at UCSD. "The focus is just on having the parent refeed the child." Therapists lay out healthy meals and portion sizes for teens, bolster parents' self-confidence and hammer home the dangers of not eating. Heenan (shown at left with husband Dennis) compares the experience to boot camp. But by the end of her time at the center, she says, "I was starting to see glimpses of what life could be like as a healthy person."

Treatment options for anorexia include a broad mix of behavioral and medication-based therapies. Most don't work very well, and many lack the support of evidence-based trials. Hospitalizing patients can boost short-term weight gain, "but when people go home they lose all the weight again," says Stanford University's James Lock, one of the architects of family-based treatment. That treatment is currently considered the most effective therapy for adolescent anorexics.

In a 2010 clinical trial, half of teens who underwent FBT maintained a normal weight a year after therapy. In contrast, only a fifth of teens treated with adolescent-focused individual therapy, which aims to help kids cope with emotions without using starvation, hit the healthy weight goal.

Few good options exist for adult anorexics, a group notorious for dropping out of therapy. New work hints that cognitive remediation therapy, or CRT, which uses cognitive exercises to change anorexics' behaviors, has potential. After two months of CRT, only 13 percent of patients abandoned treatment, and most regained some weight, Lock and colleagues reported in the April *International Journal of Eating Disorders*. Researchers still need to find out, however, if CRT helps patients keep weight on long-term. —*Meghan Rosen*

Explore more

■ W. Kaye *et al*. "Nothing tastes as good as skinny feels: The neurobiology of anorexia nervosa." *Trends in Neurosciences*. 2013.

GRACE BEEKMAN

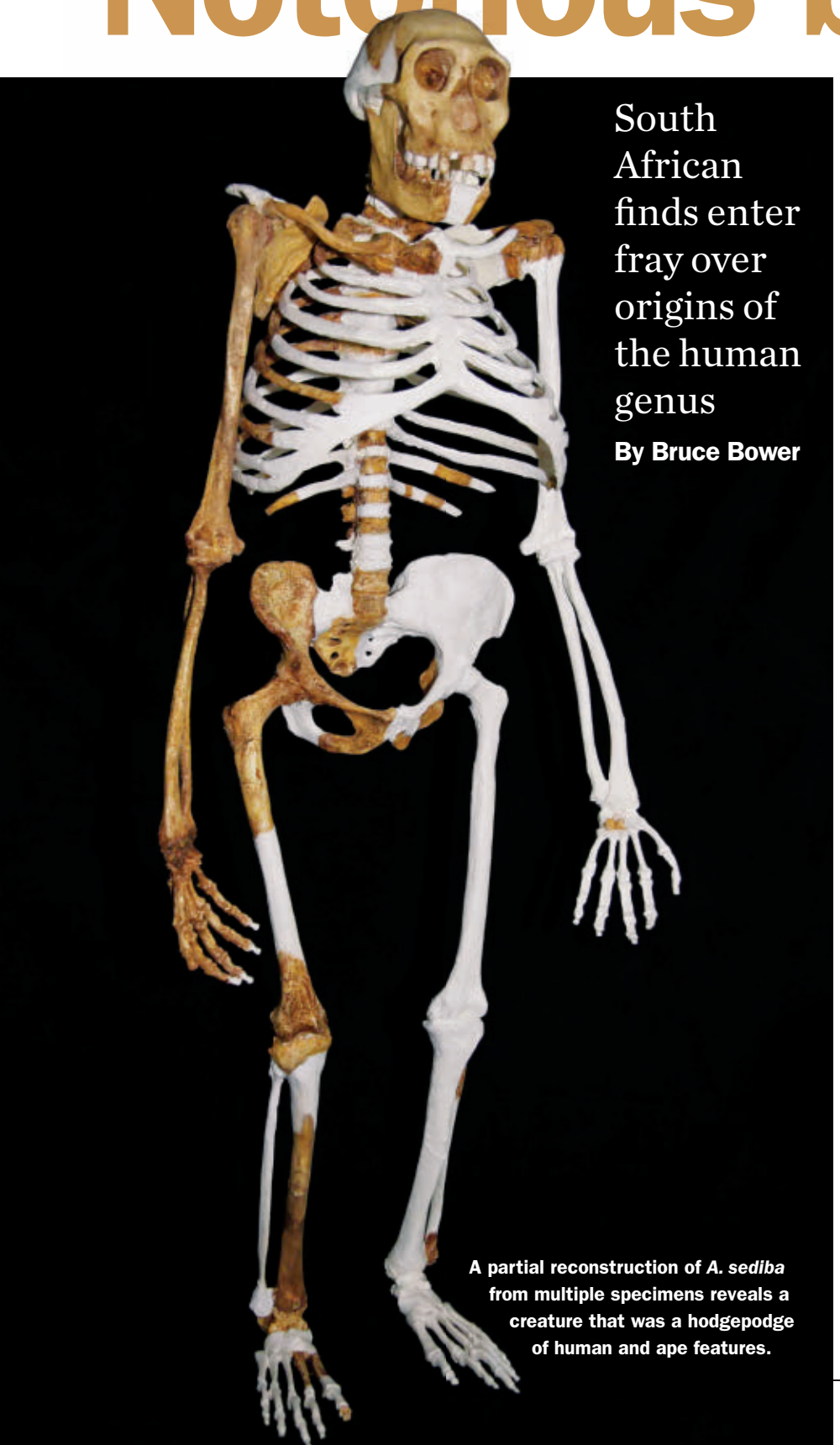
Discovery is all around them—not just in their textbooks. Whether you're a teacher, parent or grandparent, visit and share *Science News for Kids* and rekindle the joy of discovery in your future scientist.

www.sciencenewsforkids.org

SN KIDS

Science News for Kids is a free online publication of the Society for Science & the Public, publisher of *Science News* magazine and *Science News Prime*, a tablet magazine for iPad. SSP is a non-profit 501(c)3 organization dedicated to public engagement in the advancement of human discovery. See societyforscience.org for details.

Notorious bones



South African finds enter fray over origins of the human genus

By Bruce Bower

A partial reconstruction of *A. sediba* from multiple specimens reveals a creature that was a hodgepodge of human and ape features.

COURTESY OF L. BERGER/UNIV. OF THE WITWATERSRAND

Almost 2 million years ago in what's now South Africa, a boy and a woman fell through a hole in the ground into an underground cave, tumbling about 50 meters to their deaths.

Then things got interesting. A storm soon washed the partly decomposed bodies a few meters into a subterranean lake or pool. Much like quick-setting concrete, moistened soil rapidly hardened around the corpses and preserved the unfortunate duo's bones.

A couple of thousand millennia later, partial skeletons of the roughly 9-year-old boy and 30-year-old woman were discovered where they had rested for so long. Anthropologist Lee Berger of the University of the Witwatersrand spotted the cave at South Africa's Malapa site by scrutinizing satellite photos on Google Earth. Excavations of the two skeletons and bones from other ancient individuals started in late 2008.

Now the fallen Stone Age kid and his elder have plunged into a long-standing scientific dispute about the evolutionary roots of the *Homo* genus, a group of upright-walking, large-brained species that led directly to people today. Researchers generally agree that small-brained members of the human evolutionary family, known as australopithecines, evolved into the first representative of the *Homo* line between 3 million and 2 million years ago.

But so few fossils dating to that stretch of time have been unearthed that the era of early *Homo* evolution is considered a "muddle in the middle" of the hominid family tree.

Enter the Malapa skeletons, by far the most complete finds from that perplexing period. In 2010, Berger's team identified these fossil folk as members of a previously unknown australopithecine species, *Australopithecus sediba*. In six papers published this April in *Science*,

Two Africas East Africa (sites 1–5) is widely considered the birthplace of the human lineage. But with the discovery of *A. sediba* in the Malapa Valley of South Africa (sites 6–10), Lee Berger and his colleagues argue that southern Africa is a more credible cradle of humanity.

● East African sites
● South African sites

Homo erectus 2 3 10
Homo habilis 2 4
Homo rudolfensis 2
Australopithecus afarensis 1 5
Australopithecus africanus 6 8 9
Australopithecus sediba 7

the scientists delivered a complete head-to-toe analysis of the specimens.

Berger takes two big swipes at status quo thinking about *Homo* evolution with his analysis of the Malapa fossils. First, he nominates *A. sediba* as the most likely ancestor of the first *Homo* species. Forget the popular notion that the *Homo* genus arose in East Africa. Southern Africa was where the evolutionary action was, Berger contends.

Second, he rejects previous contentions that a handful of fragmentary, mainly East African skull and jaw fossils dating to as early as 2.4 million years ago belong to the *Homo* line. *A. sediba* features an odd mix of humanlike and apelike skeletal traits. Considering only skull, hand and hip fossils, it would have been easy to misclassify the Malapa discoveries as a *Homo* species, Berger says. The same danger applies to the East African finds, in his view.

"*Australopithecus sediba* should be considered as likely a candidate ancestor for the earliest members of the genus *Homo* as any other presently available fossil specimens, and perhaps the best candidate," Berger says.

Anthropologists aren't lining up to endorse *A. sediba* as a major evolutionary player. But the South African finds have generated new interest in the muddle in the middle.

"For the next decade, questions about the origins of the *Homo* genus will be in the forefront of hominid research," says anthropologist Susan Antón of New York University.

A weird mix

Berger and his collaborators never would have predicted that hominids living in southern Africa almost 2 million years ago were put together like the two Malapa individuals. Neither would any other researcher.

A. sediba possessed a brain only slightly larger than a chimpanzee's. Adult members of the ancient species reached heights intermediate between full-grown people and chimps. Yet the Malapa skulls also display *Homo*-like traits such as small front teeth, rounded brain cases and narrow faces with slight chins.

Comparisons of tooth measurements known to be largely influenced by genetics show that *A. sediba* differed greatly from East African hominids, says anthropologist Joel Irish of Liverpool John Moores University in England. That includes *Australopithecus afarensis*, a species that lived in East Africa from about 4 million to 3 million years ago. The famous partial skeleton of Lucy discovered in 1974 belongs to *A. afarensis*, which many researchers suspect was a direct ancestor of the *Homo* line.

Tooth sizes and shapes tie *A. sediba* most closely to *Australopithecus africanus*, another southern African hominid that lived from around 3.3 million to 2.1 million years ago, Irish concludes. But the Malapa individuals' teeth also display similarities to early *Homo* species.

The same goes for *A. sediba*'s lower jaws, which in some ways resemble those of *A. africanus* and in other ways align with fossil chops from *Homo habilis* and *Homo erectus*. *H. habilis*, or handy man, lived in eastern and possibly southern Africa from 2.4 million to 1.4 million years ago. *H. erectus* inhabited Africa and Asia from about 1.9 million to perhaps 143,000 years ago.

Anthropologist Darryl de Ruiter of Texas A&M University in College Station estimates that *A. sediba*'s jaws markedly changed shape from childhood to adulthood, in a developmental shift much like one previously calculated for *H. erectus*.

Unlike early *Homo* species, *A. sediba*'s long arms were built for tree climbing and possibly hanging from branches, says anthropologist Steven Churchill of Duke University. Yet the Malapa pair had humanlike hands capable of gripping and manipulating objects.

A relatively narrow, apelike upper rib cage that fanned out like an inverted funnel supported tree scaling by *A.sediba*, says anthropologist Peter Schmid of the University of Zurich in Switzerland. A cone-shaped chest interferes with arm swinging while walking and running, so the Malapa folk probably didn't move as adeptly on the ground as early *Homo* species did.

Still, *A. sediba* had a relatively narrow, humanlike lower rib cage and pelvis.

Preserved spinal bones indicate that the Malapa hominids had longer and more flexible lower backs than people today do. Inward curving of *A. sediba*'s lower back recalls that of a 1.5-million-year-old *H. erectus* skeleton previously found in Kenya, says anthropologist Scott Williams of New York University.

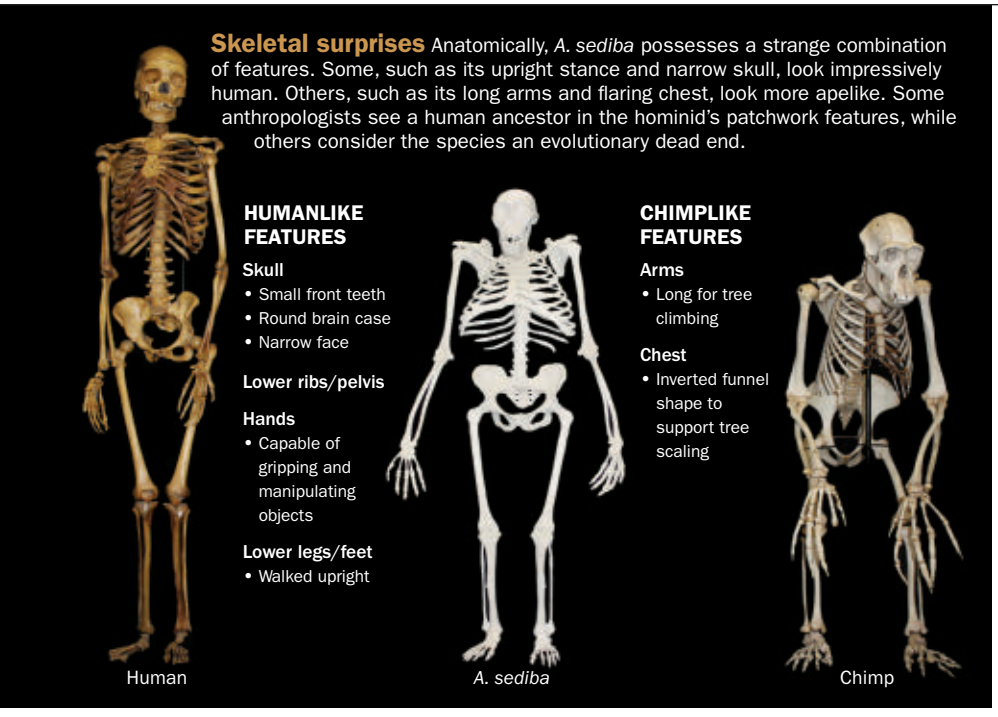
Finally, *A. sediba's* leg and foot bones show that the species walked upright, but with an unusual, pigeon-toed gait. Some people walk this way, but they tend to develop problems with their feet, knees, hips and back, says Boston University anthropologist Jeremy DeSilva. Thanks to expanded knee bones and other lower-body adjustments, *A. sediba* had no such troubles, DeSilva explains. But the hominid wasn't walking anywhere fast.

"The Malapa fossils look more like *Homo erectus* than anything else," says de Ruiter, a coauthor of Berger's on four of the April *Science* papers. "*A. sediba* could be a transitional type of hominid on the way to the *Homo* genus."

Bad timing

Many researchers outside of Berger's group regard *A. sediba* as an evolutionary bridge to nowhere. Malapa hominids

GEOATLAS/GRAPHI-OGRE, ADAPTED BY E. OTWELL



evolved too late to have been ancestors of a *Homo* genus that, given other fossil finds, must have originated at least 2.3 million years ago, they argue.

By 2 million years ago, several lines of hominids with various humanlike traits had emerged in eastern and southern Africa, says anthropologist Christopher Stringer of the Natural History Museum in London. Only one of those groups could have carved out a path to the *Homo* genus. He doubts it was *A. sediba*.

"The Malapa line may have died out as a failed experiment in how to evolve an upright stance and humanlike features," Stringer says.

His critics are the ones who have failed, Berger responds, by assuming that a few fragmentary fossils represent the earliest members of the *Homo* genus.

Consider perhaps the crown jewel of early *Homo* fossils, an upper jaw and palate discovered in Ethiopia in 1994. This fossil was found on the surface of a small hill and may not have eroded out of the 2.3-million-year-old soil layer its discoverers say it came from, Berger contends.

Also, he argues, humanlike features of *A. sediba*'s jaws and skull illustrate the danger of diagnosing any find as a member of *Homo* based on such fragmentary evidence. Echoing Stringer's point but from a different perspective, Berger argues that because different hominids evolved distinctive blends of apelike and humanlike traits, fossils from one body part are unreliable guides to a specimen's place on the hominid family tree.

Berger asserts that a pair of alleged early *Homo* species — *H. habilis* and *Homo rudolfensis* — possessed large teeth typical of australopithecines and apelike feet. Lacking more complete skeletons, he suspects that those East African species were actually australopithecines.

If that's the case, it's more likely that *A. sediba* originated somewhere in Africa before 2 million years ago and was a direct ancestor of the first true *Homo* species, *H. erectus*, Berger says. Previous fossil discoveries suggest that *H. erectus* reached western Asia 1.77 million years ago, shortly after appearing in Africa.

That's the evolutionary story with the strongest fossil support, mainly from the two partial Malapa skeletons and a previously unearthed skeleton of an *H. erectus* boy, de Ruiter says.

Fossils previously proposed as early *Homo* representatives are too few and incomplete for his taste. "Every single scrap of fossil evidence for early *Homo* before 2 million years ago could fit in a shoe box, along with one shoe," de Ruiter says.

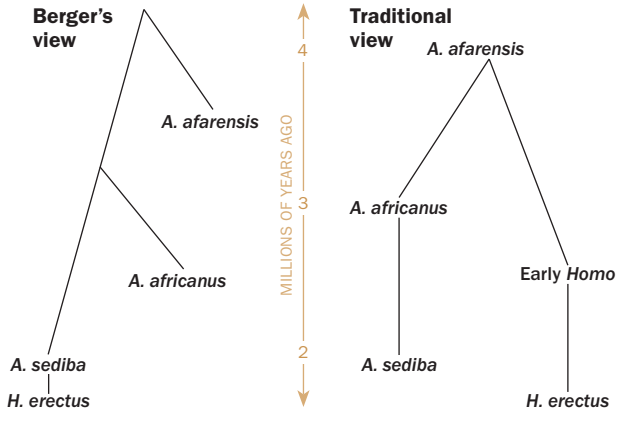
Miffed mentor

In a big way, Berger has the famous anthropologist Donald Johanson to thank for his Malapa discoveries. Johanson, who led the excavation of Lucy's skeleton at Ethiopia's Hadar site in 1974, was Berger's hero and inspired him to pursue anthropology. As an undergraduate in Georgia, Berger invited the famous anthropologist to have breakfast with him when Johanson was in the area to give a talk. Johanson advised the young man to do graduate work at Witwatersrand and investigate South Africa's rich fossil sites.

Now, 25 years later, Johanson finds himself exasperated at Berger's rejection of early *Homo* in East Africa and insistence that *A. sediba* was an evolutionary skeleton key that opened the door to human evolution.

"It's wonderful that Berger found the

Feuding family trees Anthropologist Lee Berger and his colleagues argue that the discovery of *A. sediba* necessitates redrawing the branches of the evolutionary tree leading to *Homo erectus*, the supposed ancestor of modern humans. Berger and his colleagues consider *A. sediba* ancestral to *Homo erectus* (left), while others might favor putting the species on a side branch, as at right.



FROM TOP: COURTESY OF L. BERGER/UNIV. OF THE WITWATERSRAND; E. OTWELL

Malapa fossils, but he wants to sweep evidence for early East African *Homo* under the rug," says Johanson, now at Arizona State University in Tempe.

Johanson coauthored a 1996 report on an upper jaw and palate that many researchers outside Berger's group regard as the oldest known *Homo* specimen.

That specimen was broken in half along the roof of the mouth when it was discovered on the surface of a low, steep hill at the Hadar site in Ethiopia. The wide, deep palate and relatively small teeth place it squarely within the *Homo* genus, Johanson asserts.

Soil clinging to the two pieces enabled researchers to identify a section of the hill from which the fossils had eroded, presumably weeks or months before members of a survey team noticed them. A layer of volcanic ash just above the fossil's presumed original resting spot puts the jaw at about 2.3 million years old, Johanson says.

Fossils from Lucy and other members of her kind, as well as preserved footprints at Tanzania's Laetoli site, make it clear that upwards of a million years earlier, *A. afarensis* walked around the same part of Africa on humanlike feet. That evidence makes Lucy's species a good candidate ancestor for early *Homo* at Hadar, Johanson says.

In contrast, *A. sediba* walked on feet and legs that were more apelike and less able to stride efficiently than those of Lucy's kind. The South African species appears to have been an evolutionary offshoot of *A. africanus* with a body design unlike that of any other hominid, in Johanson's view.

"At the moment, it looks like *Homo* evolved somewhere in East Africa," he says.

Hominids capable of traveling long distances evolved in Africa by 2.5 million years ago, so it's hard to know where on the continent *Homo* first appeared, comments anthropologist Brian Richmond of George Washington University in Washington, D.C.

Like Johanson, Richmond sees *A. sediba* as a likely descendant of *A. africanus* in a now-defunct line of southern African hominids.

For now, too few fossils have been found to determine precisely where Berger's discoveries stand in the grand scheme of human evolution. So Berger and his colleagues returned to Malapa in September 2012. Based on previous observations of fossils poking out of parts of the cave, they suspect Malapa to yield at least three more hominid skeletons.

Even the most momentous discoveries there aren't likely to explain the origin of the *Homo* genus to everyone's satisfaction. Any further finds will be welcomed, nonetheless, to the hominid fossil record's sparsely populated muddle in the middle. ■

Little Foot steps up

Up to a million years before an *Australopithecus sediba* boy and adult female perished in a cave at South Africa's Malapa site, one of their evolutionary relatives fell to his death through a narrow shaft into the nearby Sterkfontein Caves. With much less fanfare than that triggered by Lee Berger's Malapa finds, the Sterkfontein hominid's nearly complete skeleton has been excavated by University of the Witwatersrand anthropologist Ronald Clarke (shown above with the fossil's skull).

Clarke's take on hominid evolution in southern Africa differs radically from Berger's. Clarke's opinion is informed by his own discovery, which he assigns to a new species: *Australopithecus prometheus*.

"*A. sediba* has nothing to do with the origin of the *Homo* genus," Clarke says. "I don't claim *A. prometheus* does, either."

Berger and Clarke have a tense relationship. As a Witwatersrand graduate student in anthropology, Berger befriended Phillip Tobias, the head of the university's human biology and anatomy departments. Tobias then groomed Berger as his academic successor at the university over the more senior Clarke.

Clarke and Tobias realized in 1997 that Sterkfontein held an ancient hominid's skeleton. A couple of years before, they had uncovered only the lower legs with parts of the feet and a skull. Tobias playfully dubbed the find "Little Foot," a name that stuck. Clarke has now freed much of the skeleton from its hard shell.

Hundreds of *Australopithecus* fossils, mainly jaw and skull pieces, have been uncovered at Sterkfontein since 1936. Most researchers lump all of them into the species *Australopithecus africanus*.

But Little Foot's relatively large teeth and flat face demonstrate that another hominid species lived alongside *A. africanus* in southern Africa, Clarke contends.

Soil carried into the cave at various times by rains has complicated attempts to pin down when these hominids lived. Clarke estimates that, based on its position in the cave, Little Foot lived around 3 million years ago. Other fossils of *A. prometheus* and *A. africanus* come from a Sterkfontein section dating to around 2.5 to 2.1 million years ago, he says. Some scientists who have worked at Sterkfontein suspect Little Foot lived closer to 2 million years ago.

Where in Africa *Homo* originated is far more mysterious than Little Foot's age, Clarke says. Though fossils of 3-million- to 2-million-year-old African hominids come almost exclusively from eastern and southern parts of the continent, he observes, "the *Homo* genus could have first developed in central Africa for all we know." —*Bruce Bower*

Explore more

- Lee Berger's website: www.profleeberger.com
- Institute of Human Origins website: iho.asu.edu

KATHLEEN KUMAN AND RON CLARKE

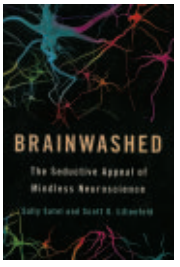
Brainwashed

Sally Satel and Scott O. Lilienfeld

By reducing human thought and behavior to colorful images of excited neurons, neuroscientists have turned brain scans into brain scams, write psychiatrist Satel and psychologist Lilienfeld. The argument that thinking involves more than brain activity is not new, but the authors give it an up-to-date, provocative treatment.

Satel and Lilienfeld take aim at functional MRI scans that have been used by researchers and media to claim that specific brain areas represent the seats of love, hate and other human experiences. At best, the authors say, these scans detect a fraction of brain activity that occurs when people perform mental tasks. Such brain measures can neither fully predict nor explain people's thoughts and feelings, they assert.

That hasn't dimmed the cultural appeal of research that explains desires and actions as products of the brain that have little or nothing to do with personal responsibility or free will. One offshoot of brain-centered science is the burgeoning business of neuromarketing, in which advertising consultants use fMRI and brain wave data to tell Google, Facebook and other companies — for a price — whether consumers will buy or ignore new products. Brain data may eventually identify attention-grabbing products, Satel and Lilienfeld suggest, but there's no evidence that neural information reveals people's product preferences.



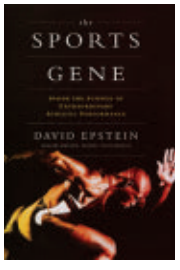
The authors similarly challenge popular arguments that the brain controls drug addiction, criminal activities and moral reasoning. Their skepticism does not extend to psychology, though; they uncritically accept the controversial idea, now in vogue, that people typically make decisions with error-prone, split-second intuitions and occasionally opt for more accurate, logical deliberations. But that's a topic for another book. — *Bruce Bower*

Basic Books, 2013, 226 p., $26.99

The Sports Gene

David Epstein

Sprinter Usain Bolt's website proclaims him "arguably the most naturally gifted athlete the world has ever seen." But is the speed that propelled Bolt to Olympic gold really a product of his genes, or do the secrets of his success lie in rigorous training and support from Jamaica's rich sprinting tradition? Epstein, a sports writer, former scientist and competitive runner, explores the variables for building the perfect athlete in his new book.



One popular theory holds that 10,000 hours of practice can make anyone an expert in a given field. But Epstein offers caveats. Some people are genetically endowed to benefit from training. Others struggle to make even marginal improvements, partially because their genes cause them to plateau physiologically or make their body types fundamentally unsuitable for their sports.

Some controversial topics that Epstein tackles are pachyderms other writers might tiptoe uncomfortably around. He examines the roles of race and gender in athletic performance, presenting a wealth of evidence for each theory about why some people become sports stars while others never get out of the beer leagues. He sometimes takes a side so convincingly that the reader is in danger of whiplash when he switches to make a competing case.

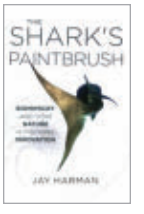
But hear him out. By the time his tale comes to an end, Epstein will have persuaded you that most athletic traits are "a braid of nature and nurture so intricately and thoroughly intertwined as to become a single vine." — *Tina Hesman Saey*

Current, 2013, 352 p., $26.95

The Human Spark

Jerome Kagan

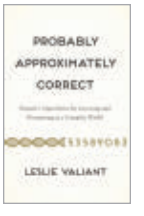
A psychologist takes a new look at the nature versus nurture debate by examining research on human development from infancy on. *Basic Books, 2013, 333 p., $28.99*



The Shark's Paintbrush

Jay Harman

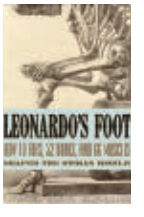
Learn how scientists and engineers are using nature's designs to create new medicines and materials. *White Cloud Press, 2013, 339 p., $26.95*



Probably Approximately Correct

Leslie Valiant

By looking at human decision-making processes, a computer scientist proposes an algorithm-based approach to understanding how living things learn and evolve. *Basic Books, 2013, 195 p., $26.99*



Leonardo's Foot

Carol Ann Rinzler

An in-depth look at the anatomy and history of feet reveals their often overlooked importance in human evolution, medicine and art. *Bellevue Literary Press, 2013, 208 p., $16.95*

Golf Science

Mark F. Smith, ed.

This colorful illustrated guide describes the physics, neuroscience and anatomy behind the perfect swing. *Univ. of Chicago, 2013, 192 p., $30*

How to Order To order these books or others, visit www.sciencenews.org/bookshelf. A click on a book's title will transfer you to Amazon.com.

Not-so-smart perception

Researchers studying associations between IQ and selected visual tasks ("Less is more for smart perception," *SN: 6/29/13, p. 18*) report that tracking small moving foreground objects, a task at which high-IQ subjects excelled, is often more important than detecting large-object motion or attending to background activity. They suggest that for driving or walking in busy areas, high-IQers have an advantage over people with lower IQs, who are better at monitoring large objects. I will have to take their word for it, but I can't quite get out of my mind an image of all those geniuses, deftly swatting away mosquitoes and flies while being run over by trucks and trains whose approach in the background they didn't notice.

Holly J. Massey, Livermore, Calif.

Intelligence is not memorized

Practice on memory tasks ("Memory training questioned," *SN: 6/15/13, p. 12*) did not make people smarter on intelligence tests. Such studies are based on the theory that intelligence is some quality of the mind-brain that can be strengthened by mental practice. There is another concept of intelligence as composed of learned basic repertoires, such as language, that make possible learning other repertoires. A child with a good language repertoire will do better in school than a child with a less good repertoire. There is experimental evidence that supports this theory as well as naturalistic evidence such as the Flynn effect (the observation that since the 1930s, intelligence has generally increased).

Arthur W. Staats, via e-mail

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

The Science Life

For more of The Science Life, visit People at **www.sciencenews.org/people**

Atomic ant sand

During his first visit to New Mexico's Trinity Site, where the world's first atomic bomb test occurred, polymer scientist Robb Hermes could feel the military police watching him. Or maybe it was just his nagging conscience. Milling around with other tourists, he had to fight the urge to bend down, pretend to tie his shoes and swipe a piece of Trinitite — a glassy, mildly radioactive mineral created by the explosion 68 years ago.

Removing Trinitite from the site is a federal crime. But Hermes was fascinated by the strange material and wanted to figure out how the little bits formed in the heart of an atomic blast. So he hatched a scheme. He returned to his office at Los Alamos National Laboratory, called up officials at the U.S. Army's White Sands Missile Range (home to Trinity) and asked for a box of ant sand. Ants, he knew, build their mounds from mineral grains gathered up to 15 meters from their homes.

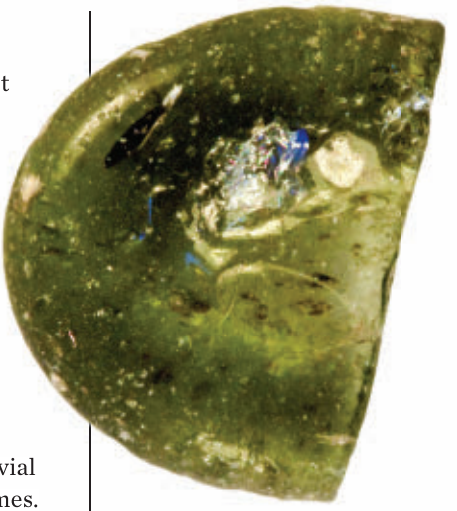
"I thought if I could get some ant sand, maybe I'd find at least a vial of little Trinitite pieces collected from around the site," says Hermes.

When the sand arrived in the mail, Hermes and a geology club friend did indeed discover beads of Trinitite. The pieces were surprisingly spherical, which turned out to be the key to piecing together how the mineral formed.

Waves of heat rippling outward from the plutonium bomb didn't just sear the sand into glass like the surface of a crème brûlée, as many people had thought. Instead, Hermes found, the blast tossed sand up and melted it to form Trinitite, flinging droplets up to 1,800 meters from ground zero. Some drops solidified before hitting the ground, and some collected into puddles of molten material. In analyzing Trinitite's makeup, Hermes even found colorful traces of steel and copper from the tower that held the bomb and from the wires connected to the instruments.

He has since gotten the Army's blessing to do his Trinitite research. Hermes, now retired, supplies the ant sand to geologists who study meteorites. Microscopic spheres found at sites around the world resemble the Trinitite beads, evidence perhaps for a controversial theory that a meteor broke up in the atmosphere about 13,000 years ago and bombarded Earth with stones that burst in the air like miniature nuclear warheads. One theory holds that such an impact might have wiped out most of North America's large animals, along with the Clovis culture that depended on them for food. — *Devin Powell*

Though removal of the bomb-created mineral Trinitite from the blast site is a federal crime, this area open to the public has been picked clean compared with nearby spots.



Atomic age mineral
Trinitite is a glassy material found at the Trinity atomic test site near Alamogordo, N.M. It was formed when sand was tossed into the air and melted by the first atomic bomb test, on July 16, 1945.

Trinitite

- **Also known as:** atomsite or Alamogordo glass
- **Color:** often green, sometimes black, blue or red
- **Composition:** arkosic sand (feldspar, quartz and clay)
- **Radioactivity:** low, from isotopes including strontium-90 and plutonium-239
- **Temperature of Trinity atomic blast:** 8,430 kelvins (14,710° F)

TOP LEFT AND BOTTOM: MARY CAPERTON MORTON/THE BLONDE COYOTE; TOP RIGHT: R. HERMES

TECHNOLOGY SIMPLIFIED

Wow! The Computer Designed for You, Not Your Grandchildren!

Easy to read. Easy to see. Easy to use. Just plug it in!

NEW
Touch Screen Technology

One-touch "zoom" magnification

Big Bright Screen

No bulky tower

U.S. Based Customer Service

Simple navigation so you never get lost

***FREE* Automatic Software Updates**

"I love this computer! It is easy to read and to use! I get photo updates from my children and grandchildren all the time."
– Janet F.

Have you ever said to yourself "I'd love to get a computer, if only I could figure out how to use it." Well, you're not alone. Computers were supposed to make our lives simpler, but they've gotten so complicated that they are not worth the trouble. With all of the "pointing and clicking" and "dragging and dropping" you're lucky if you can figure out where you are. Plus, you are constantly worrying about viruses, spam and freeze-ups. If this sounds familiar, we have great news for you. There is finally a computer that's designed for simplicity and ease of use. It's the WOW Computer, and it was designed with you in mind. This computer is easy-to-use, worry-free and literally puts the world at your fingertips. From the moment you open the box, you'll realize how different the WOW Computer is. The components are all connected; all you do is plug it into an outlet and your high-speed Internet connection. Then you'll see the screen. This is a completely new touch screen system, without the cluttered look of the normal computer screen. The "buttons" on the screen are easy to see and easy to understand. All you do is touch one of them, from the Web, Email, Calendar to Games– you name it… and a new screen opens up. It's so easy to use you won't have to ask your children or grandchildren for help. Until now the very people who could benefit most from E-mail, and the Internet are the ones that have had the hardest time accessing it. Now, thanks to the WOW Computer, countless older Americans are discovering the wonderful world of the Internet every day. Isn't it time you took part? Call now, and a patient, knowledgeable product expert will tell you how you can try it in your home for 30 days. If you are not totally satisfied, simply return it within 30 days for a refund of the product purchase price. Call today.

- Send & Receive Emails
- Have video chats with family and Friends
- Surf the internet:
  Get current weather and news
- Play games on line:
  Hundres to choose from!

Call now and find out how you can get your own WOW! Computer.

Mention promotional code 46983
for a special introductory pricing.

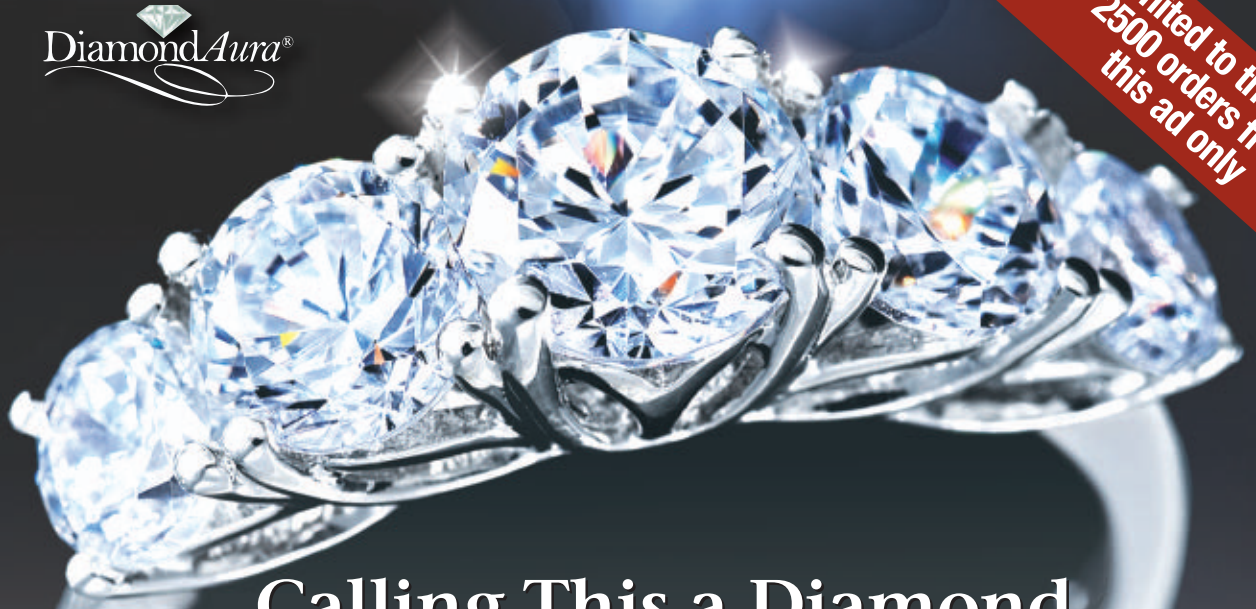
1-877-805-0652

*first*STREET®

80755

DiamondAura®

Limited to the first 2500 orders from this ad only



Calling This a Diamond Would Be an Insult

*Experts warn that millions of rings may be "romantically defective" when compared to the stunning Diamond*Aura® *5-Stone Ring for* ***ONLY $99!***

She loves natural diamonds. She loves you even more. But when even 1-carat stones can sell for as much as $9,000, it's time to reconsider your relationship... with diamonds. Compared to the Stauer **Diamond*Aura*® *5-Stone Ring*** with nearly 2 ½ carats, a skimpy diamond is "romantically defective." Have you overpaid only to be underwhelmed? Send it back. You can do better. You can do bigger. And you can absolutely take her breath away for ***under $100.***

When "cute" is a four-letter word. If you want to make a romantic impression, go big. Cute doesn't cut it. Your love deserves to be wowed. If you're a billionaire with money to burn, turn the page. Everyone else? What you read next just might change your life. There's only one way to find out...

Science not snobbery. Thanks to the advanced science behind our exclusive Diamond*Aura*, you can experience superior clarity and larger carat weights without the outrageous cost of natural diamonds. Diamond*Aura* is crafted using an incredibly complex process that involves heating rare minerals to temperatures of nearly 5000°F. After expert cutting and polishing, every lab-created Diamond*Aura* retains the classic jeweler's specifications, including color, clarity, cut and carat weight. Diamond*Aura* doesn't emulate the world's most perfect diamonds... it surpasses them. The color dispersion is ***actually superior to mined diamonds***. You get more sparkle, fire and flawless beauty for a fraction of the price.

EXCLUSIVE BONUS OFFER!
*Order today to get these FREE 1-Carat Diamond*Aura® *sterling-silver studs* ***PLUS*** *$300 in Stauer Gift Coupons!***

Scintillating in sterling silver. Our quest for perfection doesn't end in the lab. Passionate designers bring inspiration from history books and Fifth Avenue showrooms to every inch of the ***5-Stone Ring***. The ring features a classic quintet of round-cut, lab-created Diamond*Aura*, 2 1/3 carats prong-set in the finest .925 sterling silver. A spectacular alternative for those occasions when a lonely solitaire will simply not suffice.

Romantic satisfaction guaranteed. If for any reason she doesn't completely adore the ***5-Stone DiamondAura Ring,*** return it within 30 days for a full refund of your purchase price. We're so confident you'll fall for Diamond*Aura*, that we'll include a stunning $99 pair of 1-carat Diamond*Aura* studs in sterling silver... absolutely FREE. **Plus, when you call today, you'll also get $300 in Stauer Gift Coupons ($25 to use every month for 12 months with no minimum purchase required)! You played by the old rules and the big jewelry stores emptied your pockets. Our job is to fill them back up!

JEWELRY SPECS:
- 2 1/3 ctw brilliant white Diamond*Aura*®
- .925 sterling-silver setting

Diamond*Aura*® 5-Stone Ring
(2 1/3 ctw) **$99**
Include $99 Stud Earrings (1 ctw) ~~$198~~
NOW BOTH ONLY — ***$99*** + S&P

Specify ring whole size 5-10 when ordering.
Call now to take advantage of this extremely limited offer.

1-888-870-7382

Promotional Code FSR341-03
Please mention this code when you call.

Stauer®
14101 Southcross Drive W.,
Dept. FSR341-03
Burnsville, Minnesota 55337
www.stauer.com

ACCREDITED BUSINESS
Rating of A+

Smart Luxuries—Surprising Prices