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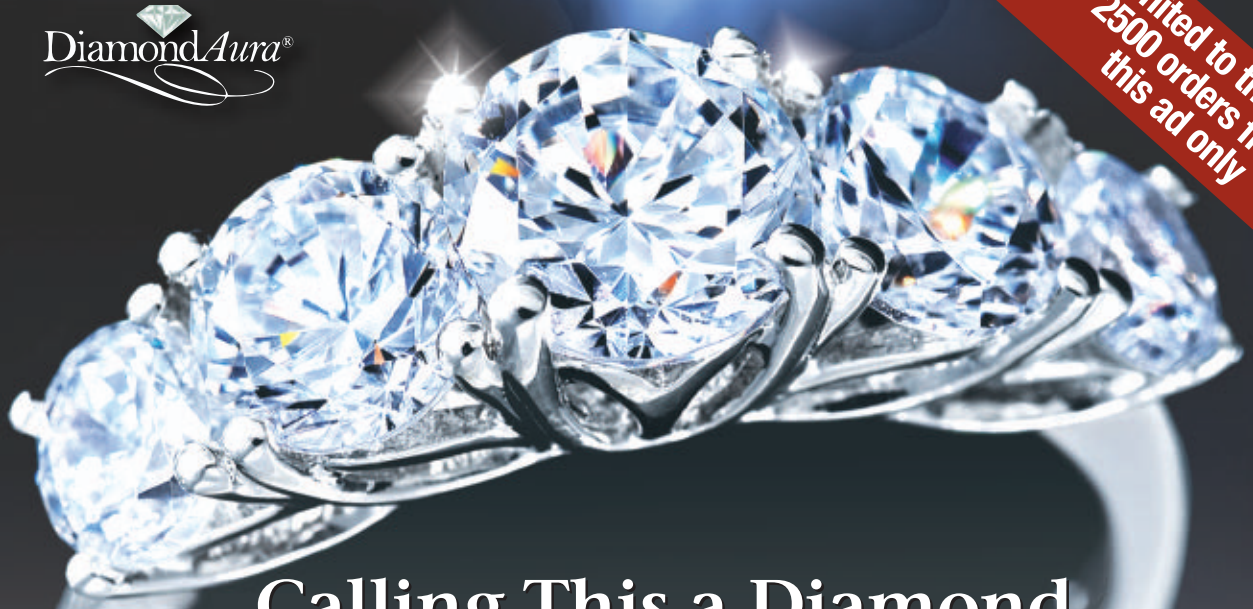

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COVER STORY Scientists question the long-standing notion that adaptation — specifically the evolution of genes that encourage humans to hold on to fat so they can survive times of famine — has driven the obesity crisis. *By Laura Beil*

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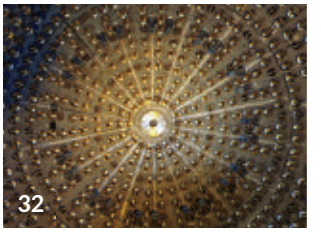

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COVER The debate over the cause of the obesity epidemic rages on as scientists consider the intersection of ancient genes and modern diets. *Cade Martin*

A story about why people get fat may be just that

The mismatch between the modern Western lifestyle and the human body reveals itself with every new statistic about rising U.S. obesity rates. Still, gaining a deeper understanding of the biology behind that mismatch has not been straightforward. Sure, more calories in and less exercise tend to make people fat. But explaining how a propensity for obesity evolved and where exactly it's written in the human genome has been more difficult.

By far the most popular idea of the last 50 years, as contributing correspondent Laura Beil discusses on Page 18, has been the thrifty gene hypothesis. It posits that frequent bouts of famine acted as a force of natural selection among our ancestors, giving an advantage to those whose bodies stored excess calories as fat. The idea makes intuitive sense, but surprisingly little evidence exists to back it up, Beil reports. Efforts to find the genes responsible for this evolutionary edge have largely come up short. That has many researchers in the field embracing a wider palette of explanations. Hopefully, new insights into what primes the body to hold on to calories, and puts many at risk of obesity and diabetes, will soon emerge.

What Beil's article doesn't delve into is advice on how to eat, and whether a protein-rich, low-carbohydrate "Stone Age" diet will help you lose weight and stay healthy, a contested claim made in a number of diet books. Whatever the evolutionary story behind the obesity epidemic, the latest salvo in the diet debate appeared this month in the *Annals of Internal Medicine*. Tulane University researchers and colleagues show that a low-carb diet was better at promoting weight loss and improving cardiovascular risks than was a low-fat diet (go Paleo!). Including genetics in similar studies of diet might allow scientists to map the variation among people and lead to a more complete picture of how evolution has shaped metabolism.

Evolution also acts on disease, as is evident in the update on the Ebola outbreak (Pages 6–7). Read about a plan to fast-track experimental therapies and a genetic report that traces the current epidemic's origins to a single person. On Page 16, you'll find another disease detective story, this one fingering marine mammals for bringing tuberculosis to the Americas a thousand years ago. — *Eva Emerson, Editor in Chief*

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Excerpt from the September 19, 1964, issue of *Science News Letter*

50 YEARS AGO

Rocket Fares to be High for Space Passengers

Passengers traveling to a space station 300 miles above the earth by 1980 will have a speedy but expensive trip.... Such a vehicle could become operational by 1975, if a program were begun by 1967.... The estimated $11,700 round trip fare would apply only after the passenger system has been operating for several years, engineers said. This fare would not cover the original research and development costs, which would raise the per passenger rate to $49,500.

UPDATE: Space tourism took nearly 40 years to become reality, but the price tag of $11,700 is a bargain compared with today's costs. After adjusting for inflation, this 1964 estimate amounts to about $90,000 in 2014. Today, the privately funded Virgin Galactic takes any healthy astroenthusiast that can fork over $250,000 into suborbit. That's a paltry sum relative to the Virginia-based Space Adventures, which transports the astronomically wealthy to the International Space Station for a reported $50 million.

Biologist Sarah Anzick carries a box containing 12,600-year-old bones to be reburied. The bones belong to a toddler whose DNA she examined.

An Antarctic midge lives only about a week as an adult (top) after spending years as a larva (bottom).

THE –EST

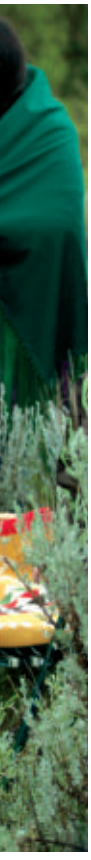
Tiniest insect genome

The smallest known insect genome belongs to an Antarctic midge that has to survive two polar winters as a larva to reach its week or so of sexual adulthood. The DNA instructions for *Belgica antarctica* fit in about 99 million of the paired basic chemical units that encode living creatures. That's a bit more concise than other small genomes among insects, such as the body louse's roughly 105 million base pairs.

The Antarctic midge slims its DNA without giving up protein-coding genes — it has an unremarkable 13,500 or so, in a similar range to nematodes. Instead, midge DNA is missing a lot of the repetitive stretches found between genes. About 19.4 percent of the midge genome carries codes for making proteins versus only about 1.6 percent in the much larger genome of the *Aedes aegypti* mosquito.

Other species, like bladderworts and puffer fish, also minimize genetic repeats. But the midge's truncations might have something to do with life under extreme Antarctic conditions, a team of researchers suggests August 12 in *Nature Communications*. — *Susan Milius*

FROM TOP: SHAWN RAECKE/LIVINGSTON ENTERPRISE; MIDGE IMAGES: RICHARD E. LEE JR.



THE SCIENCE LIFE

The biologist and the bones

Since she was a toddler, molecular biologist Sarah Anzick has had a unique connection to an ancient child.

In 1968, when Anzick was about 2 years old, construction workers in Montana unearthed the 12,600-year-old burial site of a young boy on her parents' land. The 18-month-old's bones are the only human remains ever found of ancient Native Americans known as the Clovis people. For 30 years, archaeologists stored the Clovis child's remains before returning them to the Anzick family for safekeeping in 1998.

As an undergraduate, Anzick worked on the early stages of the Human Genome Project. She later specialized in cancer genetics. "So I had early exposure to the human genome, and could appreciate what we could learn from this book of instructions," she says.

When the Clovis child's bones were returned, she realized she was in a unique position to examine DNA from the remains and "get a glimpse into the ancient past."

Before starting the research project, Anzick consulted with Native American tribes about what should be done with the child's bones. There was no consensus. Many favored reburying the boy unstudied, but, says Anzick, "I felt an obligation to humanity" to unveil the bones' genetic secrets.

She teamed up with researchers who specialize in studying ancient DNA to reconstruct the child's entire genome (*SN: 3/22/14, p. 6*). His DNA revealed that the Clovis people were ancestors of almost all Native Americans alive today.

Once the study was complete, Anzick felt that returning the Clovis child's remains to where his people had placed him was the right thing to do. She says, "These remains belong at rest, not sitting on a shelf."

On a rain-soaked Saturday at the end of June, more than 50 people, including scientists, members of the Anzick family and representatives of six Native American tribes, gathered for the nearly two-hour reburial ceremony. Tribe members said prayers, sang songs, played drums and rang bells to honor the ancient child. The bones were placed in the grave and sprinkled with red ocher, a mineral used in ancient funeral rites. Participants at the reburial ceremony filled in the grave with handfuls, then shovelfuls of dirt and covered it with stones. A stick tied with feathers marked the boy's final resting place.

"At that point, it stopped raining. The clouds opened up and the sun came out. It was an amazing day," Anzick says. Even though the remains are some of the most important ever discovered, Anzick is glad she respected the tribes' wishes to rebury the boy. "It just felt really good.... I needed that closure." — *Tina Hesman Saey*

INTRODUCING

Heat-to-fold robots

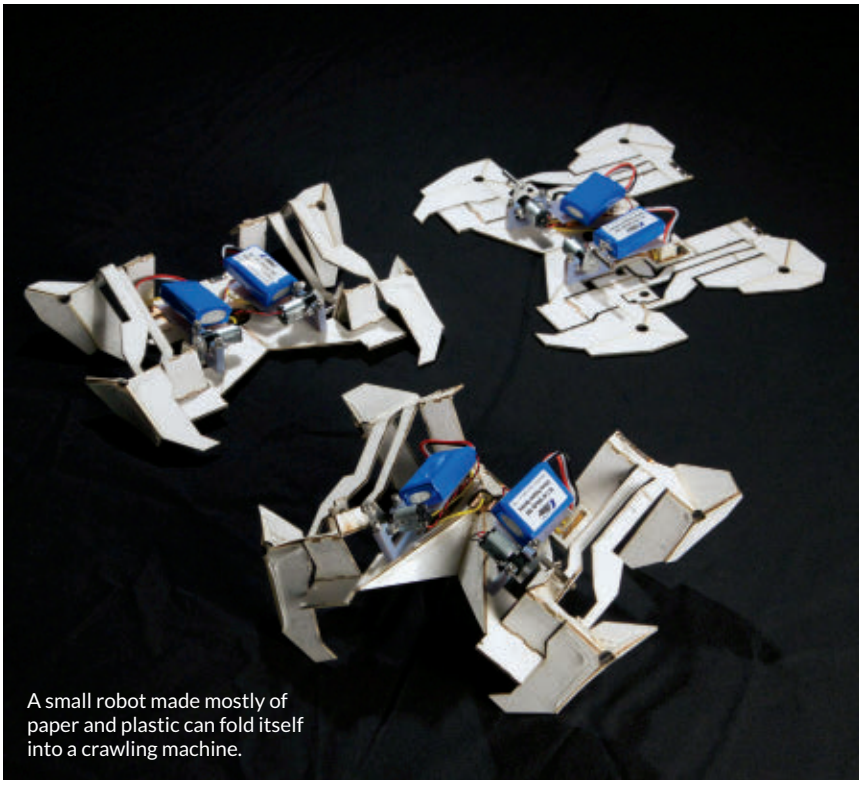
A new crawling robot has taken a page from origami design.

The paper-and-plastic machine can spring from flat to folded just like a pop-up book, researchers report in the Aug. 8 *Science*. And the action is automatic.

Engineers have built self-folding gizmos before, but they still required some hands-on help from humans. By embedding heating elements in the robot's hinges, Harvard engineer Sam Felton and colleagues created a device that can alter shape on its own, using preprogrammed temperature changes.

A tiny onboard computer tells the heaters which hinges to warm first. The hinges, made of a polymer that contracts when heated, pull the robot's parts together. When the polymer cools, the hinges lock in a folded position. Then, two motors jiggle the bot's legs, making the machine shimmy along in a slow crawl.

Self-folding devices like the robot could save room on spacecraft, or even transform into portable shelters for people living in dangerous places.
— *Meghan Rosen*



A small robot made mostly of paper and plastic can fold itself into a crawling machine.

FROM TOP: COURTESY OF S. ANZICK; ETH KROLL, WYSS INSTITUTE

Watch a video of the robots in action at **bit.ly/SN_foldingbots**

News

BODY & BRAIN

Experimental therapies target Ebola

Candidate drugs and vaccines could help end epidemic

BY NATHAN SEPPA

As the Ebola virus outbreak continues to run amok in West Africa, scientists are looking ahead to the possibly pivotal use of experimental drugs and vaccines. It will take months to test, produce and deploy the therapies. But researchers hold out hope that these products — even incompletely vetted — might turn the tide against an illness that has defied public health efforts.

The treatments' use could engender enough hope to encourage people with symptoms — and their close contacts — to come to hospitals, which researchers say would limit the spread of Ebola. Having experimental drugs and especially vaccines in hand could also help in recruiting and maintaining adequate levels of hospital staff, who are at high risk of catching the lethal virus.

Using experimental drugs has downsides: Even if the treatments help some patients, it will be hard to determine their true effectiveness. And failed treatments could exacerbate the despair and distrust already hampering disease control efforts.

Still, public health officials say the situation is dire enough to warrant putting candidate therapies into use after minimal human safety testing. As of September 3, more than 3,500 people had been infected and more than 1,900 had died, the World Health Organization reports. WHO now expects the outbreak will require six to nine months to quell and estimates the caseload could pass 20,000.

The outbreak has hit a thickly populated part of West Africa and spread among Liberia, Guinea, Sierra Leone and Nigeria. None have encountered Ebola before, and the region has been racked by poverty, civil wars, corrupt governments and upheaval. The countries have inadequate health care systems that have suffered even more during the outbreak as some workers abandon their posts.

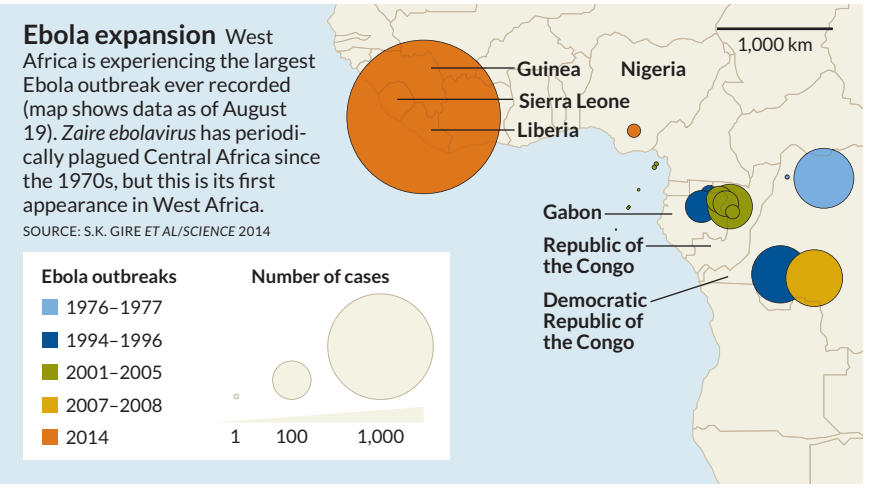
Daniel Bausch, an infectious disease physician at Tulane University in New Orleans, relates an incident in Kenema, Sierra Leone. Dressed in biohazard gear, he and a WHO doctor entered the hospital there in July and found only two workers amid 55 patients. The nurses were gone. Some were demanding higher wages but many had simply left after seeing their colleagues become sick, he says. Patients were in beds and on the floor; the hospital was contaminated.

Unregulated travel in West Africa (border between Guinea and Sierra Leone shown) makes it difficult to contain the Ebola virus.

"You get into a negative cycle in which it becomes a riskier environment for the nurses who do choose to keep working," he says.

Health officials hope experimental treatments and vaccines will turn the situation around. In August, seven people received a test drug called ZMapp, made by Mapp Biopharmaceutical of San Diego. But supplies of that compound are exhausted and making more will take months. WHO Director-General Margaret Chan said scientists were set to convene in Geneva on September 4 and 5 to examine the most promising experimental treatments and vaccines for fast-track testing in people. In the best-case scenario, WHO officials say, some drugs could reach the field later this year after safety trials in human volunteers. All have so far been tested only in animals (*SN Online: 8/29/14*).

Having a drug, even an imperfect one, would have an impact beyond the individuals receiving it, Bausch says. A drug could change the mind-set of people who have been in contact with patients but who are not themselves sick. Many hesitate to get tested because hospitals have no way to treat Ebola, he says. Refusal to get tested extends disease transmission if these contacts turn out to harbor the virus and develop an infection. Word of a drug could induce people to come in for testing. "Getting them out of circulation,"



FROM TOP: KELSEY MIRKOVIC/CDC; REDMAL/ISTOCKPHOTO, ADAPTED BY E. OTWELL

he says, "could end the outbreak."

But the use of experimental drugs might leave scientists with a poor understanding of the drugs' efficacy since about half of Ebola patients in West Africa survive without treatment, says physician Kevin Donovan of Georgetown University.

On the vaccine front, safety testing of an experimental Ebola immunization by U.S. federal researchers and GlaxoSmithKline started the first week of September. Another vaccine, developed at Canada's National Microbiology Laboratory in Winnipeg, appears next. Both tested well in monkeys. WHO will oversee who gets any semi-tested vaccines, with distribution unlikely until 2015.

Health care workers — who account for some 10 percent of deaths in this outbreak — will be at the front of the line for such shots, says William Schaffner, an infectious disease physician at Vanderbilt University in Nashville. Lab workers and those burying the dead would also get priority for vaccination, WHO officials say.

A lightly tested vaccine comes with caveats. It is unknown whether a single dose will protect fully, Schaffner says, so vaccinated health care workers will still need to don biohazard suits and take other extraordinary precautions. If a vaccinated person comes down with Ebola, he says, it would hurt vaccine credibility.

Giving an experimental vaccine to relatives or other contacts of patients would seem like the next logical step, Bausch says, but could be difficult and might not yield much information about the vaccine. Those living amid the Ebola outbreak might distrust the idea of getting a shot they may not need, and might not welcome later visits to their homes to draw blood to test for immune responses, Bausch says. "There's an incredible stigma" attached to having Ebola, he adds. Even survivors "don't admit they had it."

That could complicate work on another treatment approach, called a "convalescent serum." It would be derived from the blood of an infected person who had fended off the virus and developed antibodies against it. That research is still in its early stages, Bausch says. ■

GENES & CELLS

DNA clarifies Ebola outbreak's origin

West Africa's epidemic stems from just one infected person

BY TINA HESMAN SAEY

Genetic data confirm that the Ebola epidemic in West Africa is being spread from human to human, not through contact with infected animals.

The findings emphasize the need for better public health measures to keep the epidemic from spreading more widely among people rather than devoting more resources to tracking down animal reservoirs.

Genetic analyses also reveal that the Ebola virus strains infecting people in Guinea, Sierra Leone, Liberia and Nigeria originated in Central Africa in 2004, an international group of researchers reports August 28 in *Science*. The team sequenced the genomes of 99 Ebola viruses isolated from 78 patients during the first three weeks of the outbreak in Sierra Leone. Patterns of mutations in those viruses confirm epidemiological evidence that a healer who traveled to Guinea brought Ebola back to Sierra Leone. Mourners at his funeral became infected and spread the disease to others.

Analyzing viral DNA this quickly from an ongoing outbreak would have been unthinkable even five years ago, says Anthony Fauci, director of the U.S. National Institute of Allergy and Infectious Diseases in Bethesda, Md. Back then, it might have taken two years to compile and analyze the data, but these researchers did it in two months. "Now, as we're living through it, we're able to trace the spread of the virus," he says.

There is no indication that animals are infecting people, the researchers show. That finding supports evidence that the West African outbreak probably stemmed from a single instance of a person becoming infected, possibly from eating contaminated bushmeat, with the virus passing from person to person since then (*SN Online: 8/11/14*).

Ebola is known to have first struck humans in 1976 in the Congo, and the virus that caused the disease was dubbed *Zaire ebolavirus* for the country's name at the time. Since then, more than a dozen distinct outbreaks have been recorded in Central Africa, Fauci says.

The West African epidemic — the first to strike that part of the continent — started last December in a village on the fringes of Guinea's eastern rainforest, epidemiologists have determined. The version of the virus that made it to Sierra Leone branched from the Guinean strain in February, Stephen Gire, an infectious disease researcher at Harvard University and the Broad Institute, and colleagues report. The virus is a variety of *Zaire ebolavirus* but carries 341 genetic changes not seen in previous outbreaks.

Some virus strains in Sierra Leone have been more pervasive than others. One particular mutation appeared in 50 patients, suggesting those people shared a chain of infection.

But it is not clear whether that mutation or any other makes the virus more transmissible, says Sébastien Calvignac-Spencer, an evolutionary biologist at the Robert Koch Institute in Berlin. Some viral versions may rise to prominence by chance, he says, as might be the case if a mutant virus originated in a very popular person and many people caught the virus at his or her funeral.

But with so many people carrying Ebola — more than 3,000 people have been infected in West Africa — researchers worry the virus may develop mutations that change the structure or function of proteins. Such mutations might make the virus resistant to vaccines or therapies designed to combat it. And any mutation that hits a part of the viral genome used in DNA diagnostic tests might make it difficult to identify the disease.

So far, mutations in the virus haven't impeded treatment or testing, Fauci says. "It doesn't interfere with anything practical now" but could become a problem if the epidemic isn't stopped soon. ■

ATOM & COSMOS

Spacecraft's catch may be dust from interstellar space

Particles nabbed by Stardust could illuminate planets' birth

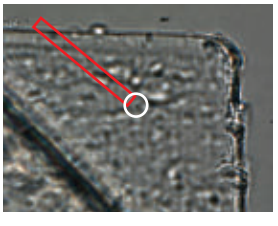
Blasting a particle collected by the Stardust mission with X-rays creates a diffraction pattern, shown in a false-color image. The pattern reveals the particle's internal structure.

BY CHRISTOPHER CROCKETT

Microscopic grains of dust captured by NASA's Stardust spacecraft appear to have come from interstellar space. These fragile particles, perhaps the first directly captured from outside the solar system, could help researchers understand the building blocks of not only Earth and its siblings but also planets around other stars.

The sample — just seven particles — comes after years of collecting dust and more years of thousands of people analyzing the spacecraft's take. "Any sane person asks: Why spend years doing this?" says Andrew Westphal, a planetary scientist at the University of California, Berkeley and a member of the Stardust team. "This is about our origin," he says, "what materials formed the sun, planets and us."

Launched in 1999, Stardust set out to collect interstellar dust, sootlike grains that fill the space between stars, as well as debris from a comet (*SN: 1/10/04, p. 19*). In 2006, the probe swung by Earth and tossed its cargo into a desert in Utah.



A dust particle (circled), about 1 micrometer across, lies embedded in aerogel from the Stardust spacecraft. Its track is marked by the red rectangle.

Eight years later, after sifting through the grains, planetary scientists think they've finally identified certain particles as messengers from outside the solar system. No one description captures all seven particles, Westphal's team reports in the Aug. 15 *Science*. Some are blobs of silicate minerals; others contain crystals of olivine and spinel that appear to have been smoothed over by millions of years of space weathering. To collect samples, Stardust hung a net about the shape and size of a tennis racket out of its door. The net was stuffed with 132 tiles of aerogel, a fluffy silicon-based foam designed to capture high-speed dust particles without damaging them.

Westphal and his team took pictures of both the aerogel and the net's aluminum frame with a powerful microscope. Then, more than 30,000 volunteers — nicknamed "dusters" — pored over the microscope images looking for tracks and craters left behind by anything striking the net. Most of the scans contained nothing of interest. But with 96 particle candidates in hand, researchers measured the composition and structure of the materials embedded in the aerogel and splattered on the frame. Most particles were pieces of the spacecraft, whereas seven appeared to come from space.

Westphal and colleagues argue that the seven samples probably originated in interstellar space because of the orientation of the spacecraft. The aerogel tiles were pointed toward a known stream of dust that comes from the constellation Ophiuchus. The trajectories traced in the aerogel tiles line up with that dust stream.

The grains' oxygen isotopes — forms of the element that vary in mass — look similar to those of material from within the solar system. But that doesn't rule out an interstellar source of the dust, says Bruce Draine, an astrophysicist at Princeton University who was not involved with the study. The gas and dust that surround the solar system probably haven't changed too much in the 4.6 billion years since the sun and planets formed, he says.

The seven particles represent the first opportunity to directly examine debris that is now passing through the solar system, Draine says. Other researchers previously had found possible interstellar particles embedded in meteorites. But, he cautions, those grains have unusual chemical compositions and might not be representative of typical alien dust.

Some of the particles' properties, such as the presence of silicates, match what astronomers deduce from observations of extrasolar dust clouds. But the dust holds a couple of surprises: The speeds of the largest particles were lower than expected, for example, which Draine says suggests either that the particles had some sort of interaction with the solar wind or that their origin was actually within the solar system.

Westphal says his team has produced microscope images of only about half of Stardust's aerogel tiles. In addition to hunting for more dust grains, the researchers plan to figure out how to extract more information out of the seven known particles to clarify their origin.

"We have to be super careful," he says. With so few particles, the researchers don't want to risk damaging a single one. NASA has no plans for a follow-up mission, so these seven may be the only samples from beyond the solar system for a long while. ■

FROM TOP: ZACK GAINSFORTH; NASA, JSC, D. FRANK

HUMANS & SOCIETY

Origin of Egyptian mummies redated

Embalming agents found in body wrappings older than pyramids

BY BRUCE BOWER

Ancient Egyptians' practice of mummifying the dead got its start as early as 6,330 years ago, among groups that farmed and raised animals, a contested new study suggests. The find pushes back the use of embalming agents to about 1,500 years earlier than previously thought, well before the pharaohs and pyramids.

Prehistoric communities that raised cattle, goats and sheep in central Egypt, between the Nile River and the Red Sea, wrapped their dead in linens. The fabric was soaked in the same preservative mixture that was used more than 3,000 years later, when mummification reached its zenith in ancient Egypt, says a team led by archaeologist Jana Jones of Macquarie University in Sydney.

Her team performed chemical analyses of linen wrapping, fragments of reed mats and human skin from 16 ancient bodies recovered at two cemeteries nearly 100 years ago. The researchers conclude August 13 in *PLOS ONE* that experiments with embalming substances started surprisingly early and were forerunners of classic Egyptian mummification.

Many Egyptologists suspect that inspiration for mummification came from observing that rapid drying in extreme heat preserved bodies buried in shallow pits in the desert. The new study suggests artificial mummification began with wrapping corpses with linen coated in a resin preservative. Over time, the practice expanded to involve removing and drying internal organs before placing them back inside the dead.

Woven material placed on an Egyptian corpse over 5,000 years ago contains embalming substances used later to mummify dead pharaohs.

"Our findings of linen impregnated with embalming agents force a fundamental reassessment of the origins of Egyptian mummification," says archaeological chemist Stephen Buckley of the University of York in England, a study coauthor.

Archaeologist and Egyptologist Donald Redford of Penn State is skeptical. It's hard to know why people applied the substance identified in the new study to linens placed over corpses around 6,000 years ago, he says. The earliest confirmed attempts to preserve bodies artificially date to roughly 4,600 years ago, when pharaohs had the first pyramids built, Redford says. Before then, "I know of no other evidence that embalming had even dawned on the thinking of Egyptians."

Linen wrapped around bodies in the two ancient cemeteries contained traces of an embalming mixture, Jones' team asserts. This substance's ingredients included plant oil or animal fat, pine resin, plant gum or sugar, wax and petroleum. The antibacterial properties of pine resin and the plant extract aided tissue preservation, the researchers propose. ■

BODY & BRAIN

Some brain scans may be misleading

Busy neurons don't always draw blood, mouse study finds

BY LAURA SANDERS

A mainstay of many neuroscience labs, functional MRI relies on blood flow changes in the brain to serve as proxies for nerve cell activity. But a new study on mice finds that neurons can be busy with no hint of blood-flow changes.

Many researchers assume that fMRI signals reflect neural activity, says coauthor Patrick Drew, a neuroscientist at Penn State, so that "when neural activity goes up, you should see increases in blood flow." But recently, that cozy relationship has come under scrutiny (*SN: 12/19/09, p. 16*).

The results, published in the Aug. 13 *Journal of Neuroscience*, emphasize the need for caution when interpreting brain-scan results, says neuroscientist Shella Keilholz of Georgia Tech and Emory University School of Medicine in Atlanta.

Functional MRI detects tiny changes in the brain's amount of oxygenated blood, which researchers often interpret as signs of neurons sending off more electrical messages. And in some cases, that interpretation is correct. But by finding a case when neurons are busy with no corresponding change in blood movement, the new study shows that blood flow isn't always a reliable marker of neural activity. "The picture is getting more and more complicated," Keilholz says.

To tease apart the blood flow from neuron firing, Drew and colleagues separately measured each process as mice voluntarily walked on a treadmill. Brain activity in a region called the somatosensory cortex, which is involved in sensing the environment, behaved as expected: Neurons became active while the mice walked, electrodes revealed. And the somatosensory cortex experienced a corresponding surge of blood, experiments on other mice showed.

But in a different part of the brain, the link between neuron activity and blood flow disappeared. Neurons in the frontal cortex, a region involved in complex thinking as well as certain aspects of movement, were active while the mice walked. But blood flow didn't budge, Drew and his colleagues found.

The decoupling shouldn't come as a shock, says Elizabeth Hillman, a neuroscientist at Columbia University who has also found evidence that neural behavior isn't always yoked to blood flow. A study by other researchers on monkeys found that blood flow can increase with no corresponding neural activity, although that result remains controversial. ■

EARTH & ENVIRONMENT

Two oceans implicated in warming lull

Along with Pacific, Atlantic and Southern may stash missing heat

BY BETH MOLE

The Atlantic and Southern oceans may be covering up global warming by hoarding heat. The finding could explain a puzzling plateau in Earth's surface temperatures that many scientists have blamed on the Pacific Ocean.

Since the turn of the century, global average surface temperatures have remained flat despite an unabated rise in greenhouse gas emissions. Scientists have developed several theories to account for the lost heat, including that it is getting trapped in the oceans. Using climate simulations, many studies have pointed to the Pacific Ocean, where unusually strong trade winds may have shoved warm water deep beneath the surface (*SN: 3/22/14, p. 12; SN: 10/5/13, p. 14*).

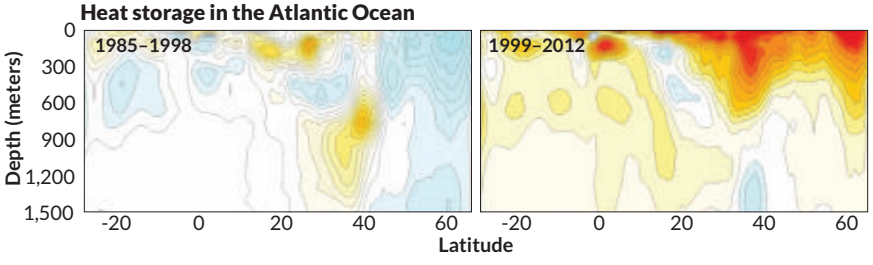
But the new research, reported in the Aug. 22 *Science*, suggests that the Pacific may play only a minor role. Instead, the Atlantic Ocean and the Southern Ocean, which surrounds Antarctica, are stashing most of the warmth. The authors — atmospheric scientist Ka-Kit Tung of the University of Washington in Seattle and oceanographer Xianyao Chen of the Ocean University of China — speculate that cyclical changes in salinity and water circulation can account for the heat grab.

"I still think the Pacific Ocean is playing the lead role in this ocean heat uptake," says climate scientist Matthew England of the University of New South Wales in Sydney. But he says the study is important for pointing out that the Atlantic and Southern oceans are also involved.

Tung and Chen used climate simulations and measurements from global ocean monitoring systems to estimate the inventories of heat in the world's oceans from 1970 to 2012. Since 1999, they found, the oceans as a whole have amassed more heat in deep waters than in previous decades, supporting the idea that oceans are masking global warming. Of the extra heat, the researchers found that nearly all of it sunk into the Atlantic and Southern oceans, with each claiming nearly half the surplus.

It's unclear why the Atlantic and Southern oceans may be taking up extra

What lies below Increased heat storage in the Atlantic Ocean may help explain the recent plateau in global average surface temperatures. From 1999 to 2012, the Atlantic held more heat than average (red) than it did in the previous decade when surface temperatures were on the rise.



LIFE & EVOLUTION

Antarctic lake bursts with life

Multitude of microbes thrives beneath continent's ice

BY THOMAS SUMNER

In January 2013, researchers pulled the first water samples from a dark, frigid lake sealed beneath the Antarctic ice sheet. Within hours, they announced they had found live cells in the water (*SN: 3/9/13, p. 12*). Now, after remaining tight-lipped for 19 months, the team reports in the Aug. 21 *Nature* that the lake doesn't just contain microbial life, it teems with it.

"The number of microorganisms we saw in the water was very comparable with what you'd find in a typical surface lake or in the ocean," says microbiologist and lead author Brent Christner of Louisiana State University in Baton Rouge. "We were very surprised."

Christner's team had used hot water to carefully tunnel hundreds of meters down through the West Antarctic ice sheet to breach the surface of Lake Whillans. The surprising abundance of bacteria and single-celled organisms called archaea, roughly 130,000 cells per thimbleful in the lightless lake, ends a decades-long hunt for evidence of life wedged under the Antarctic ice.

Rivers and more than 400 subglacial lakes kept liquid by geothermal warmth crisscross the land beneath Antarctica's nearly 14-million-square-kilometer ice sheet. In 1999 researchers announced that ice cores collected above Lake Vostok in East Antarctica contained the first evidence of life in a subglacial lake.

Critics, however, suggested that the relatively scarce cells in those cores were contamination from the drilling process. Since then, research teams from Russia, the United Kingdom and the United States have drilled into the ice in hopes of finding definitive proof of life.

At Lake Whillans, Christner and colleagues fabricated a new type of drilling rig to minimize potential contamination. Before drilling begins, the rig heats filtered water, scorching any microbes. The water is then disinfected with hydrogen peroxide and zapped with intense ultraviolet light to kill off any remaining microorganisms. The rig pumps the thoroughly treated water down what Christner describes as a kilometer-long industrial garden hose, melting a 60-centimeter-wide cylindrical hole in the ice.

In January 2013 the team drilled 800 meters into the West Antarctic ice sheet, penetrating the lake's surface (*SN Online: 1/28/13*). Lowering sterilized equipment including a camera into the hole, the researchers retrieved 30 liters of lake

X. CHEN AND K.-K. TUNG/*SCIENCE* 2014

heat, but the researchers think a natural phenomenon may explain how the Atlantic does it. The Atlantic naturally acts as a conveyor belt for heat, Tung says, moving warm waters from the tropics northward. In hot, shallow waters of the tropics, evaporation leaves behind saltier — and denser — water. When that warm, dense water travels north and meets colder, less salty water, it sinks. "And when it goes down it brings the heat along with it," he says. That cycle of heat stashing may currently be stuck on high speed. The conveyor belt could stay that way for a few decades, Tung says, but it will eventually slow down and release heat back into the atmosphere, resuming global warming.

But the theory may be an oversimplification, says oceanographer Igor Polyakov of the University of Alaska Fairbanks. The waters of the North Atlantic are complex and experience other short-term temperature variations and localized cooling cycles, he says, which aren't explained by the conveyor belt idea. "We need to go a long way," he says, "before we will be able to provide a detailed description of the mechanisms driving changes in the oceans and global climate system." ■

water along with sediment cores from the lake bed. Christner says the amount of life in the samples was staggering: The researchers found genetic traces of 3,931 microbial species or groups of species.

Unlike life on the surface, Lake Whillans' microbial inhabitants live a pitch-black existence. Without sunlight for photosynthesis, many of the microbes eat away at the lake bed's rock and produce energy by oxidizing iron or ammonia. These specialized microbes become food for other bacteria and archaea in the lake, the team surmises.

"We found all the elements for there to be a sustained ecosystem," Christner says. "It's perfect."

He suggests that the ecosystem fueled by these rock-chomping microbes supports the idea that life exists elsewhere in the solar system, such as under Mars' polar ice caps or in a subsurface ocean on Jupiter's frozen moon Europa. ■

T. HIGHAM

HUMANS & SOCIETY

New demise date for Neandertals

Study estimates extinction in Europe at 40,000 years ago

BY BRUCE BOWER

Neandertals died out in Western Europe earlier than many scientists thought, between about 41,000 and 39,000 years ago, after interbreeding with modern humans for a few thousand years, a new study suggests.

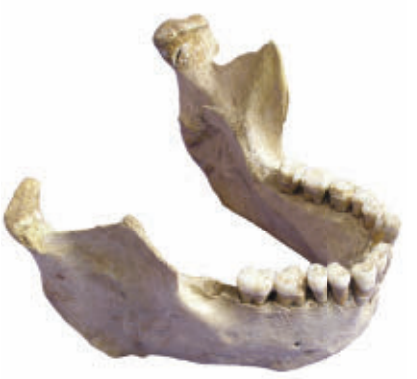
These new findings join a long-standing debate about the fate of the Neandertals that shows no signs of diminishing.

Previous reports that some Neandertals survived in southwestern Europe until about 30,000 years ago hinged on underestimates of the age of carbon in ancient bones and other material, say archaeologist Tom Higham of the University of Oxford and colleagues. Improved methods now indicate that Neandertals disappeared at different times in different regions of Europe before going extinct about 40,000 years ago, the scientists report in the Aug. 21 *Nature*.

The new dates suggest that Neandertals and modern humans (*Homo sapiens*) simultaneously inhabited Western Europe for 2,600 to 5,400 years. While populations of the two hominids overlapped, they could have interbred and exchanged cultural knowledge.

Stone Age sites in Central and Eastern Europe have yet to be dated with the new techniques. That leaves a gap in what's known about how long Neandertals survived and the extent to which they mingled with modern humans, archaeologist William Davies of the University of Southampton in England writes in the same issue of *Nature*. Still, he says, "future researchers will need to try hard to demonstrate Neandertal survival in Europe after 40,000 years ago."

Higham's team dated finds from 40 sites, all in Western Europe except for one in Russia and another in Lebanon. The researchers mainly dated animal bones displaying butchery marks.



European Neandertals, including one represented by this lower jaw excavated in southern Spain, survived no later than about 40,000 years ago, new radiocarbon dates suggest.

Stone tools at most sites had previously been attributed to the Mousterian and Châtelperronian cultures, usually regarded as Neandertals' handiwork.

Neandertals had Europe largely to themselves 45,000 years ago, Higham's team says. They then vanished from different regions at different times. Mousterian tools found in Italy, for instance, were replaced between 44,800 and 43,950 years ago by cutting tools attributed to modern humans.

Châtelperronian finds in France, thought by some experts to represent a final phase of Neandertal culture and by others to be modern human creations (*SN: 5/13/06, p. 302*), were made no later than 40,000 years ago, the scientists say.

Higham adds that sites in southern Spain, reported to have hosted Neandertals until roughly 30,000 years ago (*SN: 9/23/06, p. 205*), also don't break the 40,000-year-old barrier in his analysis.

Higham's results confirm suspicions that Europe's last Neandertals and first modern humans inhabited parts of the continent at the same time, comments paleoanthropologist Jean-Jacques Hublin of the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. In their last millennia, Neandertals made tools and personal ornaments based on techniques picked up from modern humans, he proposes.

João Zilhão, an archaeologist at the University of Barcelona, disagrees. It's more likely that Neandertals were variants of *H. sapiens* that achieved cultural advances long before being genetically swamped by large numbers of incoming modern humans from Africa, he asserts. ■

GENES & CELLS

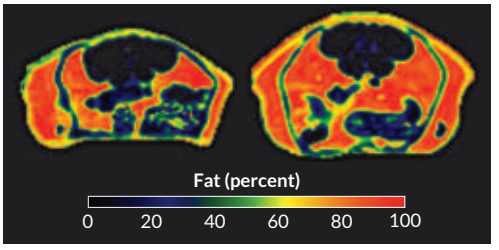
Early antibiotics may lead to fat later

Altered microbiome in infant mice promotes adulthood obesity

BY TINA HESMAN SAEY

Antibiotics given to infants may have lifelong consequences, a study of mice suggests.

Low doses of antibiotics given to pregnant mice and to their newborns led baby mice to become obese as adults, researchers report August 14 in *Cell*. The effect was not due to the drugs themselves but to the disruption of the gut microbiome, the community of microbes in the mice's intestines.



Mice treated as babies with low doses of penicillin (right, MRI image of abdomen shown) nearly doubled their body fat when fed high-fat diets as adults. Mice that didn't get antibiotics as babies (left) didn't pack on as much fat on the same diet.

"We're using antibiotics as if there were no cost," says microbiologist Martin Blaser of New York University, who led the study. "The costs are not immediate but may be long-term."

Blaser's team has previously found that antibiotics alter the microbiome, which can lead to inflammation and weight gain in animals. In the new study, the researchers investigated what happens if a baby takes antibiotics when the microbiome is establishing itself. The microbiome helps train the immune system.

Microbiologist Laura Cox in Blaser's lab and colleagues treated pregnant mice with low doses of penicillin until pups were born and the baby mice weaned, at about 4 weeks old. That is equivalent to about the first year of a human baby's life. Other mice slurped the drug in drinking water until they were 8 weeks old, while others were on penicillin for life or never.

Gut bacteria compositions in antibiotic-treated mice were different from those of mice that didn't get the drug. When the drug was halted at weaning, the microbes became normal by the time the pups were about 8 weeks old.

But those animals had changes in their immune system and metabolism that lasted into adulthood. Antibiotic-treated baby mice grew up to be plump adults. And when adults ate a high-fat diet, those that got penicillin as babies became obese: Female mice nearly doubled their fat mass. Mice that never got penicillin gained less fat on the same diet.

Epidemiologist Anita Kozyrskyj of the University of Alberta in Edmonton, Canada, thinks early antibiotic exposure may affect human children similarly. She and colleagues report August 5 in the *International Journal of Obesity* that boys who got short courses of antibiotics before they were a year old weighed more as middle schoolers than kids who didn't get antibiotics early on.

Patrick Seed, a pediatrician and microbiologist at Duke University, worries that parents may refuse life-saving antibiotics for their children because the drugs might lead to obesity. "We have to have restraint with antibiotics, but we can't completely fear them," he says. ■

BODY & BRAIN

Study may explain how pain saps will

Motivation in mice drained by molecule muffling reward cells

BY LAURA SANDERS

Relentless pain can curb a person's will to exercise, work or socialize. The constant hurting may drain motivation by quieting nerve cells in a brain area involved with pleasure, a study in mice suggests. The results, published in the Aug. 1 *Science*, could help explain why people who chronically ache could have trouble with treatments that require action, such as physical therapy.

"The natural response to the pain experience is to avoid and withdraw," says psychologist Laura Simons of Boston Children's Hospital. And nursing a wound can help acute injuries heal. But when pain turns chronic, that tendency to withdraw can persist beyond the point of being useful. By illuminating one way that long-term pain reduces motivation, the new study "legitimizes what chronic pain patients experience," Simons says.

The results may ultimately lead to better drugs for disorders that involve reductions in motivation, such as depression, says study coauthor Robert Malenka of Stanford University School of Medicine.

Malenka and colleagues tested motivation in mice by making them work for food. The mice had to poke their noses into a hole over and over to get a food reward. After seven to 21 days of chronic paw pain, the animals' ambition waned and they earned less food, the researchers found. When food came for free, the mice in pain ate just as much as pain-free mice, suggesting that the trouble came only when extra effort was needed.

Temporary pain relievers didn't eliminate the motivation drain triggered by pain from nerve injury and inflammation. "It isn't that the animal is experiencing pain and can't physically do the nose poke," Malenka says. It's that chronic pain made the mice unwilling to work for food.

Studies on the brains of these motivation-sapped mice revealed a difference in a group of nerve cells in the nucleus accumbens, an area involved in the pursuit of rewards. In response to chronic pain, these nerve cells showed signs of a weakened reaction to incoming signals. This lackluster response involved a protein snippet called galanin, a molecule thought to play a role in pain sensation.

Galanin and the nucleus accumbens cells probably aren't the only factors in reducing motivation, Malenka says. ■

L.M. COX AND SUNGHEON G. KIM

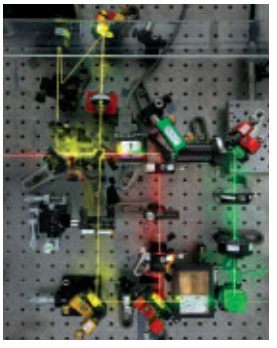
Blind quantum camera snaps images of Schrödinger's cat

Laser experiment takes photographs without detecting light from feline subject

BY THOMAS SUMNER

Exploiting the quantum quirk that spawns Schrödinger's undead cat in the famous thought experiment, a research team snapped a portrait of a cat-shaped cardboard hole without collecting any light bouncing off the two-dimensional kitty. This blind camera, made up of lasers and crystals, could help scientists illuminate microscopic worlds difficult to picture using existing techniques, the team reports in the Aug. 28 *Nature*.

"We didn't detect any photons coming from the object, but we got a surprisingly clear image," says physicist and lead author Gabriela Lemos of the University of Vienna.



In the quantum camera, light travels from the green laser through crystals and reflectors. The cat cutout sits in the middle of the red light beam.

The experiment marks the first time an image has been produced using light that never interacted with the subject. Existing quantum imaging techniques such as ghost imaging still require light from the object to complete the picture (*SN: 9/12/09, p. 12*).

Lemos acknowledges that the physics that powers her team's new experiment seems counterintuitive. "It doesn't really make much sense to us either, and I love these things that challenge our imagination and intuition."

The team's tabletop experiment begins with a green laser beam randomly divided into two separate beams. This split creates two possible destinies for each photon. Just as Schrödinger's cat is both dead and alive in an unopened box, each photon follows both possible paths simultaneously, a phenomenon called superposition. As long as neither pathway adds information to the photons, each photon will interact with its alternate-reality doppelgänger when the two paths come back together, producing a recognizable pattern of light. In this case, that pattern formed a silhouette of a cat.

Photons in the left half of the split beam enter a special crystal that transforms each incoming green photon into two photons, a yellow and a red. A special reflector directs the red photons toward a piece of cardboard with a 3-millimeter-tall, cat-shaped hole cut into it. While some light passes cleanly through the opening, other photons smack into the cardboard.

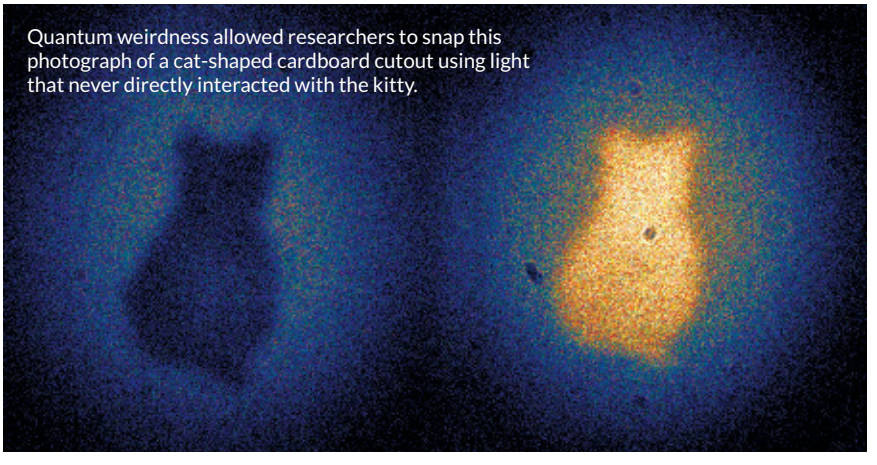
The setup then aligns the remaining red photons and the right half of the green laser beam into another crystal that converts green light into red and yellow light. Where the incoming red photons blocked by the cardboard would have been in the crystal, the newly created yellow photons appear, having unwittingly been created with information about the size of the cat hole. It's at this moment that information jumps from the red beam to some of the newly created yellow photons.

Finally, mirrors direct the left and right yellow light beams together and toward a camera (the red photons are tossed out). On the outside of the camera where the yellow photons from the second crystal carry information, the two beams don't interact. Inside the cat outline, however, the photons interfere with one another, creating a telltale light pattern. Although the red photons interacted with the cat, it's the yellow light that paints the image — photons that never went near the cardboard kitty.

Lemos says the image quality took her by surprise. "I was expecting something horrible, something completely blurred," she says. "We didn't even need long exposure times; it just took a second. When we moved around the cardboard, we immediately saw the change in the image."

The method could provide benefits for scientists imaging tiny structures such as integrated circuits and living cells, Lemos suggests. The quantum camera can use one wavelength of light to interact with an object and then shift the information to a light range that's more easily measurable, she says.

Seth Lloyd, a mechanical engineer at MIT, says that while the setup seems promising, time will tell whether it outperforms existing imaging techniques. For now, "the work is extremely cool, it uses quantum mechanics to do things that sound extremely improbable, if not impossible," Lloyd says. "It makes the science elegant." ■



Quantum weirdness allowed researchers to snap this photograph of a cat-shaped cardboard cutout using light that never directly interacted with the kitty.

FROM TOP: LOIS LAMMERHUBER; G.B. LEMOS

BODY & BRAIN

Mice do mimic people, or don't

New study disputes earlier report on genes, inflammation

BY TINA HESMAN SAEY

In direct conflict with a controversial 2013 study, researchers say that mice may be better mimics of human inflammation than previously suggested.

The earlier study found that inflammation brought on by burns, sepsis and trauma changed gene activity very differently in mice and people (*SN: 3/23/13, p. 10*). But two scientists have now reanalyzed data from that study using different statistical methods. Mice and humans actually have very similar gene activity changes, the researchers report August 4 in the *Proceedings of the National Academy of Sciences*.

In addition to using distinct statistical methods, the two teams analyzed different subsets of data. Each group accuses the other of bias in their data choices.

The original study's conclusion jeopardized funding for studies that use mice as human stand-ins, says Klaus Schughart, a geneticist and infectious disease researcher at the Helmholtz Centre for Infection Research in Braunschweig, Germany. Schughart, who was not involved in either study, thinks the new report better represents how useful mice are as imitations of human diseases.

Animal mimics — or models, as scientists call them — are never perfect representations of people. But mice have been shown time and again to react as humans do to many diseases and injuries, Schughart says.

In last year's report, though, researchers examined changing patterns of gene activity in response to traumas, such as burns and sepsis, in mice and humans and then measured how well those patterns matched up. The answer appeared in the paper's title: poorly.

In humans, more than 13,000 genes altered activity when a person was burned, for instance. But in mice, activity changed in only a few more than 4,000 genes. Of the genes with altered activity, only a fraction — not quite 2,300 genes — changed in both species, and even fewer of those reacted in the same way.

Tsuyoshi Miyakawa, a neuroscientist at Fujita Health University in Toyoake, Japan, and Keizo Takao of the National Institute for Physiological Sciences in Okazaki, Japan, thought that the approach used in that study stacked the deck against mice. The Japanese scientists reanalyzed the data, looking at the subset of genes with altered activity in both species.

For example, among genes for which burns changed activity in both species, 940 became more active in both mice and humans, 852 decreased activity in both and 664 increased activity in one species but decreased it in the other, Miyakawa and Takao found. The fact that so many genes changed expression in the same direction, up or down, is meaningful, Miyakawa says. The title of his and Takao's paper differs by only one word from the 2013 title: Mice "greatly" instead of "poorly" mimic humans.

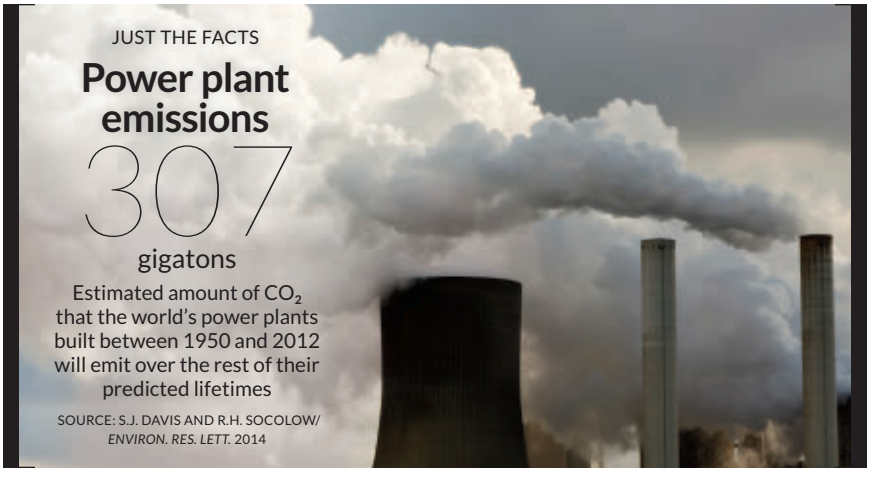
But Ronald Tompkins, a surgeon at Massachusetts General Hospital in Boston who coauthored last year's study, says Miyakawa and Takao "cherry-picked" their data, giving a view biased toward mice. The 1,500 or so genes with activity changes in the same direction represent only about 11 percent of all changes in humans, Tompkins says. That's a poor mimic, he says.

Assuming that mice and humans are essentially the same and examining only shared responses hasn't led to effective therapies for trauma, burn and sepsis victims, Tompkins says. More research should focus on people. "We're confident about our dataset and our interpretation," he says.

The difficulty is that the two groups are asking fundamentally different biological questions, says George Tseng, a biostatistician at the University of Pittsburgh. The first group asked whether mice can fully represent human disease. The second group asked whether mice could partially mimic the gene activity changes. "Both papers were right, but the second paper, in my opinion, is scientifically more meaningful for animal model research," Tseng says.

That's because Miyakawa and Takao's study used a different statistical method, one that is better because it makes fewer assumptions, he says. He would have liked the Japanese researchers to apply their improved analytical method to the biological question asked in the first study. He suspects that the method would detect greater overlap between human and mouse reaction to trauma than the 2013 paper reported.

If there is a moral to the story, Tseng says, it's that different biological questions and analyses can produce wildly different conclusions. "Mathematics cannot lie, but problematic study design... and interpretation can create unintentionally misleading conclusions." ■



BRONSWERK/ISTOCKPHOTO

ATOM & COSMOS

Gamma rays stump astronomers

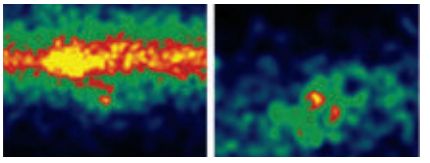
How novas generate flashes of high-energy light unclear

BY CHRISTOPHER CROCKETT

There's a newfound source of gamma rays: explosions on the surfaces of stars. Figuring out how these novas generate such high-energy light might help astronomers understand the life cycle of certain stars and whether they evolve into more powerful explosions such as supernovas.

Over the last six years, the Fermi satellite has seen gamma radiation coming from a menagerie of sources such as pulsars and remnants of exploding stars, says Fermi team member Teddy Cheung of the U.S. Naval Research Laboratory in Washington, D.C. But in 2012 and 2013, Fermi detected something new — three gamma-ray bursts associated with novas.

Novas occur when a dead star's core, called a white dwarf, orbits a star and sucks gas off it. The mass builds up and triggers a thermonuclear detonation on the white dwarf's surface.



The Fermi satellite has discovered instances of gamma rays coming from novas (two shown). How these stellar explosions produce such high-energy light is a mystery.

Fermi detected gamma rays from a nova once before, but that was an unusual situation. In 2010, the satellite observed a white dwarf orbiting a red giant star, which blows gas into space. The shock wave from the white dwarf's detonation most likely ran into debris from the red giant, which would have allowed electrons and protons to accelerate to the speeds needed to produce gamma rays.

But the three new detections, reported in the Aug. 1 *Science*, are from "classical" novas: a white dwarf orbiting a star more like the sun, which belches out relatively small amounts of gas. That leaves nothing for the shock wave to run into. So it's unclear what generates the gamma rays.

"We don't know quite how to interpret it," says astrophysicist Brian Metzger of Columbia University, who was not part of the study. One possibility, he says, is that the nova's shock wave runs into itself. If a slow-moving eruption was followed by a faster eruption, the second shock wave might catch up to the first and stir up the particles enough to generate gamma rays.

Unraveling the mystery requires simultaneous observations of novas at many wavelengths, Metzger says. Such data will also reveal whether some novas graduate to become type 1a supernovas, where the explosion destroys the white dwarf. Supernova measurements led to the discovery of dark energy, the repulsive force that's accelerating the expansion of the cosmos. "There's this whole rung of understanding," says Metzger, and novas are the starting point. ■

NASA, DOE, FERMI LAT COLLABORATION

BODY & BRAIN

Collagen-making cells curb sepsis

Experimental treatment halts mice's immune overreaction

BY NATHAN SEPPA

Injections of an obscure cell best known for making collagen might quell the runaway inflammation that underlies lethal sepsis. Mice getting fibroblastic reticular cells were much more likely than others to survive sepsis — a finding that brightens prospects for developing an effective treatment for the condition.

Sepsis is an immune overreaction, typically triggered by bacterial infections reaching the blood. Even with antibiotics to address the infection, sepsis is fatal roughly 25 to 40 percent of the time, killing some 7.3 million people every year. Options for treating sepsis are limited because suppressing immunity amid an infection is ill-advised.

Fibroblastic reticular cells build and maintain connective tissues in lymph nodes and the spleen and guide the actions of immune cells that inhabit these tissues. In the Aug. 13 *Science Translational Medicine*, researchers report that injections of cultured reticular cells seem to keep the immune response in check in mice with sepsis. The cells block inflammation and preserve needed immune cell populations in the spleen.

"I'm encouraged by these results," says Ron Daniels, a physician and sepsis expert at the Heart of England NHS Foundation Trust. "The concept of using a cell itself is a fairly novel one."

In experiments, researchers injected mice with a bacterial toxin or punctured the animals' colons. Both led to sepsis. Since sepsis in humans is more common in old people, the team induced the condition in 16 older mice. Five of six mice getting the reticular cells four hours after infection survived, whereas only one of 10 control mice getting a saline injection did.

The researchers also tested the therapy on young mice 16 hours after an intestinal puncture. While all control mice died, four of nine receiving the reticular cells survived.

The researchers found that the reticular cells trigger the release of nitric oxide using an enzyme called nitric oxide synthase-2. In a separate experiment, reticular cells from mice lacking the enzyme failed to protect mice from sepsis. Mice getting normal reticular cells not only fared better but had lower levels of trouble-making inflammatory proteins called cytokines, says study coauthor Biju Parekkadan, a Harvard bioengineer. The reticular cells seem to defuse the "cytokine storm" that shows up in the blood in severe sepsis.

"This was a really remarkable dampening" of cytokines, Parekkadan says. The researchers hope to test the cells in larger mammals or in patients so ill with sepsis that they have little to lose. ■

GENES & CELLS

Seals gave ancient Americans TB

Marine mammals transported tuberculosis from Africa

BY TINA HESMAN SAEY

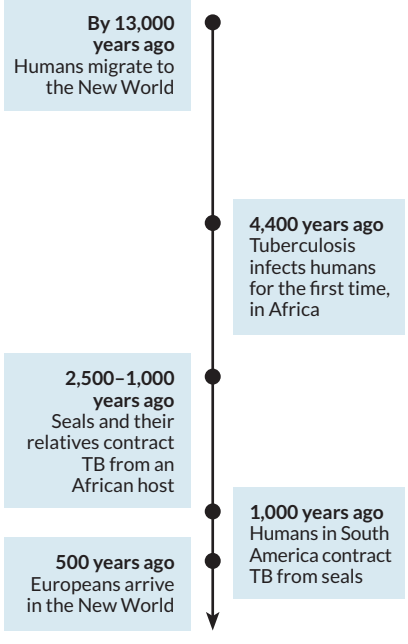
Seals brought tuberculosis to South America long before Columbus sailed to the New World, a new study shows.

Tuberculosis DNA recovered from three roughly 700- to 1,000-year-old Peruvian skeletons doesn't match the strain of TB brought to the New World by European explorers. Instead, it closely resembles a strain that infects seals in the Southern Hemisphere, researchers report August 20 in *Nature*.

Scientists had long thought that *Mycobacterium tuberculosis*, the bacterium that causes TB, originated in cattle as *M. bovis*, jumped to humans after dairy cows were domesticated and then came to the Americas with Europeans.

Several pre-Columbian skeletons found in the New World, however, have spine and rib deformities suggestive of TB infections, says Christina Warinner, a molecular anthropologist at the University of Oklahoma in Norman who was not involved in the new study.

TB timeline New genetic research suggests the earliest cases of tuberculosis in the New World predate the arrival of Europeans.



Those skeletons sparked speculation that TB was even more ancient than thought, perhaps migrating out of Africa as humans colonized the globe. In that scenario, scientists reasoned, TB would have crossed the Bering land bridge with the first people to settle the Americas.

The new study supports a very different idea: that all animal strains of TB are actually derivatives of a human-infecting subspecies that originated in Africa. "Africa really seems to be the cradle of tuberculosis, not just humankind," says study coauthor Johannes Krause, a paleogeneticist at the University of Tübingen in Germany. He and his colleagues used genetic data from many types of mycobacteria to construct a tuberculosis family tree. The human-infecting strains formed seven major branches. All of the animal-infecting strains, including the seal one, are related to *M. tuberculosis* from one of those branches.

Using the Peruvian skeletons as a time stamp, Krause and colleagues calculated that TB mutates 10 times as rapidly as indicated by previous estimates based on humans' migration out of Africa. Using the new mutation rate, the researchers determined that TB first infected humans about 4,000 to 4,400 years ago in Africa. That's a much younger date — by at least 5,000 years — than previously suggested.

The date had the researchers scratching their heads about how an African strain could get to South America, says study coauthor Anne Stone, an anthropological geneticist at Arizona State University in Tempe. The Bering land bridge had been drowned 1,000 years or more before the bacterium jumped into humans, so TB couldn't have come to the New World with the first Americans. And the ancient Peruvian strain clearly arrived before Europeans. Instead, says Stone, "It swam to the Americas."

She and her colleagues speculate that people in Africa passed TB to mice, hyraxes or some other animal, which then infected seals. Seals gave the disease back to humans living in South America.

Seals may have contracted tuberculosis from an animal in Africa and then carried the disease to the New World, where the marine mammals gave it to early South Americans.

BRIAN GRATWICKE/FLICKR (CC BY 2.0)

All the human skeletons in the new study belonged to the Chiribaya culture and were excavated from sites near Peru's southwestern coast. Seal-bone tools found at the sites indicate that these people probably had contact with seals. But the ancient Peruvians' TB was slightly different than the *M. pinnipedii* that infects modern seals. The researchers estimate that the bacterium may have started to infect humans at least 100 years before those ancient Peruvians died. The timing suggests the bacterium may have adapted to humans, enabling person-to-person transmission.

Pre-Columbian skeletons from Illinois also show signs of TB infection, Warinner says. She wonders whether the seal bacterium caused an epidemic throughout the Americas, or if people in Illinois caught the disease from a different animal source. "They weren't getting it from seals," she says.

The modern-day seal bacterium doesn't usually infect people, and the strain that infected ancient Peruvians is no longer around. European strains now cause most TB infections in the Americas, says Eric Rubin, a microbiologist at the Harvard School of Public Health. The seal version may have petered out because pre-Columbian American populations were too sparse to sustain its spread, or because it wasn't virulent enough to compete with the European strains. ■

Pink bulges indicate this coral is about to release gametes for open-water fertilization. Where the resulting babies live may depend on how much reefs stink.

DANIELLE DIXSON

LIFE & EVOLUTION

Corals, fish can 'smell' bad reefs

Young corals and fish can tell bad neighborhoods from good ones in part by the stink of overgrown seaweeds. This distaste for compounds leaching from seaweeds shows how "bad habitat" chemical cues discourage youngsters from even trying to make their homes on degraded reefs, says Mark Hay of Georgia Tech. There, seaweeds can outcompete corals and offer few rocky hidey-holes for reef fishes. Exactly how the youngsters sense the taint of seaweeds isn't clear. But in tests with paired water samples, young corals and fishes preferred seawater dipped from coral-rich protected reefs from Fiji — but not when certain reef-choking seaweeds had been in those water samples for an hour. Surveys on the reefs themselves revealed more young corals settling in seaweed-sparse areas than in overgrown ones. And when researchers set out tiles on poles above seaweeds, coral larvae settled there but not in the thick growth at the bottom, Hay and colleagues report in the Aug. 22 *Science*. — *Susan Milius*

HUMANS & SOCIETY

Richard III ate like a king

England's infamous King Richard III hit the banquet circuit during the years leading up to his violent 1485 death. Details of Richard's killing and medical problems have emerged since his skeleton was excavated in 2012 (*SN: 3/9/13, p. 14*). Because different bones regrow tissue at different rates, studying the chemical makeup of key bones revealed what types of food Richard ate at various stages of life. Chemical comparisons of two teeth, a rib and an upper leg indicate that, as king, Richard ate luxury foods such as game birds and freshwater fish that he washed down with wine, say Angela Lamb of the British Geological Survey in Keyworth and colleagues. Before taking the throne in 1483, Richard consumed humbler items such as bread, barley, ale and water, the scientists propose August 16 in the *Journal of Archaeological Science*. — *Bruce Bower*

EARTH & ENVIRONMENT

Humans tripled ocean's mercury

Human activities such as fossil fuel burning may have tripled the amount of mercury in the ocean's surface since preindustrial times. Carl Lamborg of the Woods Hole Oceanographic Institution and colleagues used seawater samples from around the world to estimate the amount of anthropogenic mercury in the oceans. Along with tripling in the ocean's surface, mercury increased by a factor of 2.5 in waters between 100 and 1,000 meters deep, compared with the preindustrial era, the team found. In total, humans have dumped up to 74,000 metric tons of mercury into the ocean, the team estimates in the Aug. 7 *Nature*. It's unclear, however, what the mercury pollution means for humans. In the sea, mercury can transform into methylmercury, a toxic substance that can build up in fish and poison people. How that conversion happens is mostly a mystery, Lamborg says. "If you tripled the amount of mercury in the ocean, do you triple the amount of mercury in fish as well?" he asks. "We don't know." — *Beth Mole*

BODY & BRAIN

Prosthesis uses arms to tell legs to move

The motion of swinging arms may someday drive the movement of paralyzed legs. A device, described in the Aug. 13 *Journal of Neuroscience*, creates an artificial connection between arms and legs that could bypass spinal damage. In an experiment, 10 healthy men lay on their sides while wires supported their legs horizontally, which allowed scientists to see subtle movements. Electrodes detected activity in shoulder muscles as the men swung their arms. A computer converted these signals into stimulations delivered to the spine and a nerve near the ankle. The men relaxed their legs, allowing them to become puppets controlled by the stimulation. When the men swung their arms, their legs started a walking motion, Syusaku Sasada of the National Institutes of Natural Sciences in Okazaki, Japan, and colleagues report. The legs sped up when the men pumped their arms faster and then gradually stopped when the men rested their arms or the device was turned off. — *Laura Sanders*

MATTER & ENERGY

Magnets get flipped by light

A laser zap can alter the magnetism of a host of materials, researchers report August 21 in *Science*. The finding opens the possibility of using lasers to speed up data storage. Computer hard drives read and write data by flipping the magnetization of memory cells with magnetic fields. In 2007, researchers discovered that for a limited set of materials, they could replace the relatively slow, energy-hogging magnetic fields with laser light. The clockwise or counterclockwise polarization of the light determined the direction of the materials' magnetization. Eric Fullerton of the University of California, San Diego and his team shined a rapid-pulse laser at a wider variety of materials. Many of the materials could also be tuned by the polarization of the light. One material, a film containing iron and platinum, is already being tested in hard drive prototypes that use lasers as a heat source to make storing data easier. Upgrading the lasers to emit quick polarized pulses could enable the laser light to read and write data as well. — *Andrew Grant*

Watch a video of the arm-controlled prosthesis at **bit.ly/SN_prosthesis**

Ancient Genes,

CADE MARTIN

Poking holes in the thrifty gene hypothesis

By Laura Beil

The obesity crisis has given prehistoric dining a stardom not known since Fred Flintstone introduced the Bronto Burger. Last year, "Paleo diet" topped the list of most-Googled weight loss searches, as modern Stone Age dieters sought the advice of bestsellers like *The Paleo Solution* or *The Primal Blueprint*, which encourages followers to "honor your primal genes."

The assumption is that America has a weight problem because human metabolism runs on ancient genes that are ill equipped for contemporary eating habits. In this line of thinking, a diet true to the hunter-gatherers we once were — heavy on protein, light on carbs — will make us skinny again. While the fad has attracted skepticism from those who don't buy the idea whole hog, there's still plenty of acceptance for one common premise about the evolution of obesity: Our bodies want to stockpile fat.

For most of human history, the theory goes, hunter-gatherers ate heartily when they managed to slay a fleeing mastodon. Otherwise, prehistoric life meant prolonged stretches of near starvation, surviving only on inner reserves of adipose. Today, modern humans mostly hunt and gather at the drive-thru, but our Pleistocene genes haven't stopped fretting over the coming famine.

The idea that evolution favored calorie-hoarding genes has long shaped popular and scientific thinking. Called the "thrifty gene" hypothesis, it has arguably been the dominant theory for evolutionary origins of obesity, and by extension diabetes. (Insulin resistance and diabetes so commonly accompany obesity

Modern Meals

that doctors have coined the term "diabesity.") However, it's not that difficult to find scientists who call the rise of the thrifty gene theory a feat of enthusiasm over evidence. Greg Gibson, director of the Center for Integrative Genomics at Georgia Tech in Atlanta, calls the data "somewhere between scant and nonexistent — a great example of crowd mentality in science."

Support for the thrifty gene theory may be eroding in scientific circles, even while it's still going strong on the Internet. One analysis published in February in the *American Journal of Human Genetics* found no consistent association between 65 variations in possible thrifty genes and survival. Another study published in January in *Biology Letters* concluded that prehistoric hunter-gatherers actually ate more often, not less, than later societies that grew their own food. In describing the results, anthropologists from the University of Roehampton and University of Cambridge wrote that their finding challenges popular assumptions about the evolution of diet and today's epidemic of obesity and diabetes.

It's not that obesity has nothing to do with genetics, Gibson says. Of the 21,000 or so genes that make up the human genome, he estimates that perhaps hundreds influence body weight. Where the genetics get controversial, he says, is with the assumption that the overriding influence is a throwback to starvation — leaving humans at the mercy of genes that encourage overeating and the rapid accumulation of fat.

Instead, thrifty genes, if they exist, are just part of a complex genetic picture that contributes to the obesity epidemic, says Hertzel Gerstein, director of endocrinology and metabolism at McMaster University in Hamilton, Canada. The interaction between any one person's predisposition and the calorie-dense Western smorgasbord is still not well understood. "People are looking for an explanation," he says. "The thrifty gene hypothesis might be a piece of an explanation.

However, if you accept it too uncritically, you close your mind and thinking to possibly better explanations."

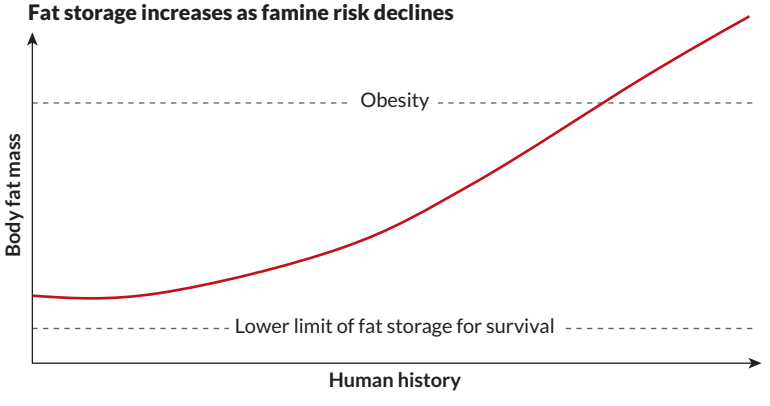
And there are plenty of lesser-known theories that address the origins of obesity, including those rooted in the complex nature of evolution, the migration patterns of early humans and changes in gene function that don't involve mutations. Some explanations even stand out for their creativity, such as one from a team of researchers in India who propose that obesity is a consequence of declining human aggression.

Survival of the fattest

For all its recent fame, the thrifty gene hypothesis isn't new. Geneticist James Neel of the University of Michigan Medical School proposed it in 1962 in the *American Journal of Human Genetics*. "It must be remembered," he wrote, "that during the first 99 percent or more of man's life on Earth, while he existed as a hunter and gatherer, it was often feast or famine." The human who gorged and then held an extra pound or two in reserve when food was scarce was better able to survive. Thus, he concluded, the development of insulin resistance (a propensity for diabetes) conferred some physiological advantage that continues to exert itself.

In the five decades since its debut, the theory has "gone off in all sorts of directions," says Andrew Prentice, who studies international nutrition at the London School of Hygiene & Tropical Medicine. Prentice supports the general concept Neel proposed — that the genetic influences on body weight are the product of natural selection from lean times — but not in the way people commonly interpret it. For one, he doesn't think the advantage of fatness had much to do with mortality.

Holding on to calories As time progressed, the threat of famine among early humans decreased, according to the thrifty gene hypothesis. But genes that once helped humans survive starvation remain and bodies are programmed to stockpile fat, even in times of increasing abundance. SOURCE: J.R. SPEAKMAN/*ANNU. REV. NUTR.* 2013



His research, particularly focused on women in Gambia, in West Africa, suggests that food shortage affects fertility, and that women with the highest body weight have greater reproductive success. (Alternatively, women who become dangerously thin cease to ovulate.) Once human societies became agricultural, they went through periods of both blight and plenty — much like the population Prentice studies today. He's found that plumpness is an advantage not because thinner members of a population are more likely to die, but because they are less likely to bear children and pass their genes to the next generation.

One of the most vocal critics of the thrifty gene hypothesis is John Speakman, who heads the Energetics Research Group at the University of Aberdeen in Scotland. If being fat offered a benefit over the course of human evolution, Speakman reasons, then even more of the population would be obese, in the same way humans have universally developed large brains and upright postures. With the thrifty gene idea and its different incarnations, "the common thread is that some time in our evolutionary history it was advantageous to be fat," he says. If this were true, and genes for obesity conferred a survival benefit, "the real problem is to explain why so few people get fat," he says. "Even in America, 60 to 70 percent of people are not obese. How come so many of us didn't inherit thrifty genes?"

Speakman became disenchanted with the thrifty gene hypothesis about a decade ago, deciding that it was based on simplistic assumptions about evolutionary dynamics. He set out to test it. Among other studies, in 2013 in *Disease Models & Mechanisms*, he published a mathematical analysis gauging how 32 known gene variations associated with body mass index would help people survive near starvation — and found that they made only a tiny difference.

The basic problem with the thrifty gene idea, he says, is that "it's based on a naïve view of how evolution works." Natural selection, though powerful, isn't the sole architect of human DNA. Nature favors genes that help survival, but genes that are simply along for the ride will also be handed down through generations. In early human history, Speakman says, being overweight was a disadvantage. When a species is subject to predators, the slower, fatter members who are prone to overheating are the unlucky ones who will be picked off first. As humans developed fire and weapons, and grew less vulnerable to being hunted, he argues, heavier individuals survived — not because

Why are some modern humans hefty?

Theory	Authors	Description
Thrifty gene hypothesis	James Neel, U.S., 1962	Human history was marked by feast or famine. Humans who had fat reserves — who were exceptionally efficient at storing fat — were more likely to survive.
Drifty gene hypothesis	John Speakman, U.K., 2008	A counter to the thrifty gene theory. Fatness was not a survival advantage. It just stopped being a disadvantage when humans no longer had to run from predators, so obesity drifted into the population.
The thrifty phenotype	Several authors	A handful of hypotheses that revolve around the idea that poor nutrition in the womb encourages the development of diabetes when food is abundant in adulthood.
Genetically unknown foods	Riccardo Baschetti, Italy, 1998	Obesity and diabetes occur when populations are introduced to new foods they have not adapted to.
Aggression control	Prajakta Belsare *et al*, India, 2010	As humans relied less on fighting and aggression to survive, a propensity for obesity emerged. Overindulgence becomes less of a problem when being docile is no longer a life-or-death calamity.
Climate adaptations	Dyan Sellayah *et al*, U.K., 2014	Survival in cold parts of the world favored genes that help preserve body temperature — a higher metabolic rate meant lower obesity and diabetes. In hot spots, lower metabolism meant the opposite.

being fat was good, but because upper limits on body weight didn't matter as much any more.

He calls this idea the "drifty gene" hypothesis, a name he first proposed in 2008 in the *International Journal of Obesity* to reflect that obesity might not have been actively selected for, just passively allowed to float into the human genome. "If you have a mutation that happens but doesn't create a selective advantage, its influence is not going to be strong, but it's not going to be actively removed either," Speakman says.

Nifty, not thrifty

Speakman's isn't the only alternative to the idea that obesity is a holdover from ancient times of starvation. Elizabeth Genné-Bacon, a graduate student in genetics at Yale University, recently compiled evidence for the thrifty gene hypothesis along with other theories in a paper in the *Yale Journal of Biology and Medicine*.

"I see textbooks with [the thrifty gene hypothesis] all the time," she says. "It's elegant and it makes sense. It's easy to understand." However, she found the support to be surprisingly thin, writing that, "Obesity researchers are often not aware that there is, in fact, limited evidence to support the thrifty gene hypothesis."

For example, if thrifty genes were influencing metabolism, populations that most experienced famine should be particularly prone to diabetes. She points out that Europe has a lengthy history of war, disease and frequent food shortages, but European descendants have a lower incidence of diabetes than the indigenous people of the Americas and Pacific Islands who may have had less food turmoil in their distant past.

In her article, she lists other theories that might explain the evolution of obesity and diabetes:

The thrifty phenotype theory: This one exists in several forms. In a version proposed in 1992, researchers from University of Cambridge and Southampton General Hospital said the "thrifty phenotype" offers a survival advantage related primarily to obesity. Poor nutrition in the womb encourages the development of diabetes when food is abundant in adulthood even if it doesn't cause direct changes to DNA, according to the theory. The reason, as they later explained in 2001 in the *British Medical Bulletin*, is that undernourishment in early development produces changes that include both reduced insulin secretion and insulin resistance — hallmarks of diabetes.

Genetically unknown foods: This theory rests on the observation that obesity and diabetes occur rarely in native populations, such as those who live on the Pacific Island of Nauru or the Pima Indians of Arizona — until they develop a taste for the Western diet. First published in the late 1990s by Riccardo Baschetti, a physician in Padua, Italy, the theory proposes that humans evolved to eat foods with the natural sweetness of fruit (not Froot Loops), leaving bodies that are not able to cope with sugar and fat at every checkout stand.

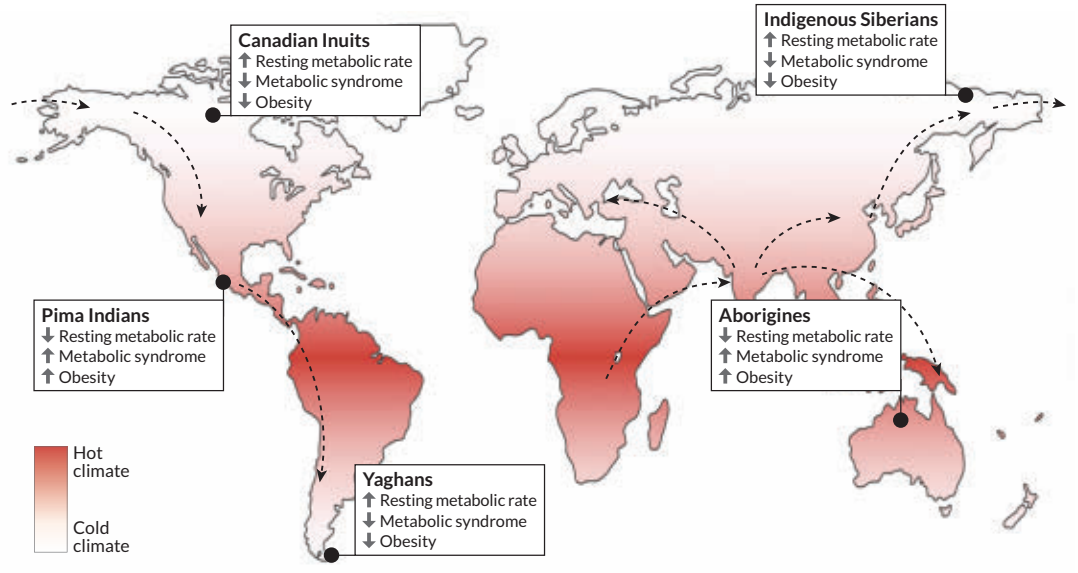
Aggression control: A full male is a docile male, researchers from University of Pune, India and colleagues wrote in 2010 in *Medical Hypotheses*. As humans relied less on fighting to survive, a propensity for obesity emerged. (When lethargy won't get you killed, overindulgence isn't so bad.)

Climate adaptations: One of the latest hypotheses appeared in the journal *Endocrinology* in May. A team of researchers from the United Kingdom noted that diabetes and obesity are unevenly distributed among populations throughout the world; the highest risk for both diseases occurs among people adapted for warmer temperatures (such as native populations in the tropics or Central and South America). As humans migrated out of Africa beginning around 70,000 years ago, they

Menu of options

Alternative theories to the thrifty gene hypothesis abound. So far, no single hypothesis on the genetic origins of obesity has gained universal acceptance.

SOURCES: E. GENNÉ-BACON/*YALE J. BIOL. MED.* 2014; D. SELLAYAH *ET AL/ENDOCRINOLOGY* 2014



Warm climates, slow metabolism
A new theory for the evolution of obesity proposes that as early humans migrated out of Africa and around the globe (dotted lines) and their bodies adapted to survive in different climates, those in warm climes ended up with a lower metabolic rate to help remain cool. That left them more vulnerable to metabolic syndrome, the cluster of symptoms that include high blood sugar and excess fat.

settled in varied climates. Survival in colder parts of the world amplified genes that help preserve body temperature — a higher metabolic rate that keeps the body warm would confer some resistance to obesity. Genes adapted for warmer climates would lower metabolic rate, burn calories at a slower pace and make the body more inclined to accumulate fat.

Evolution through thick and thin

None of the theories could be right, or several could, says Jeffrey Friedman, the Rockefeller University geneticist best known for discovering leptin, the hormone that helps regulate appetite. When he announced the finding in 1994, many researchers thought Friedman had found a link to a long-sought thrifty gene because of leptin's direct relationship to appetite. Friedman himself went looking into the origins of obesity by conducting genetic studies on the island of Kosrae in Micronesia, where the native population was reduced to only 300 in the late 1880s, following a typhoon and an influx of communicable diseases from the West. Such a small, confined population creates a genetic bottleneck that makes isolating specific genes easier. As the island's eating patterns fell under Western influence, diabetes and obesity emerged as serious health threats.

So far, though, he hasn't found the answers he would like, including insight into evolutionary influences on diabetes. One of the largest studies of the Kosrae, published by Friedman and colleagues in *PLoS Genetics* in 2009, found that common gene variants associated with cholesterol and insulin response could not explain why some people became diabetic while others did not.

Despite the gaps in knowledge, it's a mistake to think that any one theory would explain an evolutionary tendency to become obese, Friedman says. He points out that humans in different parts of the world experienced different evolutionary pressures, so what's true in one population might not apply to the next.

"People think about evolution of this sort taking place over millennia or eons. Evolution can be evident in a single generation," he says. "Populations walk around with a spectrum of alleles, or genetic variants, and the frequency of one variant versus another can be changed dramatically even in a single generation."

Just as being overweight might have affected survival, so would being too thin. "You could draw the lines wherever you want, but the point is if you were infinitely fat or infinitely thin it wouldn't be good," he says. "And there's a biological system that's evolved that allows you balance in a particular environment."

The genetics of obesity has many influences, not just starvation or abundance or cold climates or warm, says obesity geneticist Claude Bouchard at Pennington Biomedical Research Center in Baton Rouge. Human DNA is a genetic soup that reflects any number of prehistoric dinner challenges. "Evolution must have been zigzagging," he says. "That's why it's hard to put all the pieces together." ■

Explore more

■ John Speakman. "Evolutionary perspectives on the obesity epidemic: adaptive, maladaptive, and neutral viewpoints." *Annual Review of Nutrition* July 2013.

D. SELLAYAH ET AL/ENDOCRINOLOGY 2014, ADAPTED BY E. OTWELL

At first and even second blush, you will think this is the famous brand "X" watch that sells for over $1,000. But this is the Rodell-7 Argonaut™ Watch, the last watch you will ever need to buy. And, incredibly, it can be yours for only ~~$129~~ $69 (or even less) – read on!*

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A Unique and Wonderful Watch

We won't insult your intelligence with such fantastic stories. But we will tell you one very important and sobering fact: Virtually all those fancy watches have mechanical movements. And mechanical movements have scores of tiny parts. Their wear and tear are mitigated by jeweled bearings. But that will help only for so long. Ultimately – and that sometimes may be as little as a year – these movements will wear out and that is the end of them. Because it is not possible or practical to repair them you have to throw those watches away or keep them as a reminder of your folly.

The Argonaut™ Watch is powered by the finest Japanese quartz movement. The only moving parts are the hands and the date disk. Nothing will wear out. We guarantee its proper functioning for three years. Once every two years or so, you'll need a new battery. It shouldn't cost you more than $5.

The Watch is of quality solid stainless steel construction. It is as watertight as an oyster. It is tested to 10atm, and that means that it will be water-tight to about 330 ft. It comes with a most elegant stainless steel band that is virtually indestructible.

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Less is MORE

Simple rules of thumb may be best for complex financial decisions

By Bruce Bower

In Shakespeare's *Hamlet*, Polonius gives his son Laertes some famous advice: "Neither a borrower nor a lender be." That's easier said than done. These days, many people, businesses and governments accumulate mountains of debt. Average credit card debt in 2013 reached $15,480 per household in the United States, according to financial website NerdWallet. Overall U.S. national debt is rapidly approaching $18 trillion.

There are ways to borrow and lend money wisely, even if Polonius would disapprove. The trick, according to an upstart group of researchers, is to design simple approaches that work reasonably well, even if imperfectly, in particular financial situations.

These investigators explore how simple rules of thumb that focus on key pieces of information and ignore all other evidence can improve money management. These strategies are known as heuristics. In a business world where managers are often pressured to throw as much information as possible at a complex decision, such as how to price their products, rules of thumb are viewed as flawed products of lazy thinking.

Yet heuristics can outperform number-crunching exercises in fields such as business, where many interconnected, often unknown factors can trigger unpredictable perils, says economist Shabnam Mousavi of Johns Hopkins Carey Business School in Washington, D.C. Given gigabytes of data, bankers and business managers want to use as much of it as possible to make crucial financial forecasts. Studies suggest, however, that less mental effort produces better judgments than complex calculations do in comparably uncertain situations, including deciding whom to vote for (*SN: 7/5/08, p. 22*) and how to invest money (*SN: 6/4/11, p. 26*).

Psychologist Gerd Gigerenzer of the Max Planck Institute for Human Development in Berlin refers to less-is-more decision-making tactics as "fast-and-frugal heuristics."

KELSEY DAKE

In choosing between two soaps, for instance, a consumer may simply grab the most recognizable brand. Or, a shopper might use one good reason to make a purchase — say, opting for a lower-priced computer over a more expensive model. If there's no price difference, the buyer may simply select the better-reviewed model.

Now researchers are focusing heuristic investigations on developing uncomplicated budgeting tools for credit card users, based on the type of user they are. Investigators have also uncovered rules of thumb favored by business managers and have started to develop simpler, more effective regulations to govern how banks loan money.

"In an uncertain world, heuristics are indispensable tools for making decisions, not second-best solutions," Mousavi says.

Credit styles

To use credit cards wisely in our pay-as-you-go culture, simple strategies are essential, researchers say.

More than two-thirds of people contacted in a 2009 national survey said they possessed credit cards. About half of them reported sometimes carrying a balance and paying interest on it, according to FINRA, a nonprofit firm that regulates U.S. stockbrokers and firms.

New evidence, in the August *Journal of Business Research*, describes simple online budgeting strategies tailored to different types of credit card users.

Economist Hersh Shefrin of California's Santa Clara University and market researcher Christina Nicols, formerly of Ketchum Public Relations in Washington, D.C., used data from 2009 surveys of U.S. credit card holders to identify four ways in which people use credit cards. Shefrin and Nicols then worked with the credit card firm Chase, which funded the project, to design heuristics-based online tools for each type of credit card user, ranging from risky to responsible.

Customers with a "make it easy" outlook are most likely to fall into debt, Shefrin says. These folks express little concern about controlling their finances and usually or always make the minimum payment on their balances. A "make it easy" stance characterized 27 percent of cardholders surveyed nationally.

For them, Shefrin suggests, online systems developed by several credit card firms to monitor monthly expenses make sense. In Chase's system, customers can also create a plan that charges no interest on everyday expenses in return for making regular payments.

"Control seeking" card holders typically pay the minimum amount but almost half try to limit purchases to emergencies or big-ticket items. They could benefit from the same online options, Shefrin says. "Financially savvy" card holders, who pay most or all of their monthly charges on two or more credit cards, often track monthly expenses on their own. Members of this group — which represented 20 percent of the national sample, as did control seekers — might consider shifting to a single credit card with online monitoring of monthly expenses, Shefrin says.

Online expense monitoring could also help those already paying all monthly charges on a single credit card, he adds. This "confident and in control" group made up 33 percent of the national sample.

A simple heuristic such as reducing spending when monthly expenses exceed a preset limit works well for reducing debt with little effort, Shefrin says. That strategy isn't foolproof. Some people don't set their spending limit low enough or lose track of their expenses. But since September 2009, 91 percent of nearly 3 million users of Chase's online budgeting system have managed to make more than their minimum monthly payments. "Heuristics are imperfect, but it is critical not to let the perfect be the enemy of the good," Shefrin says.

Strategies for Credit Users

1. Make it easy
27% of credit card users surveyed nationally
Worry about it later; pay it off eventually.

Budget strategy: Set up plan to repay debt over time. Monitor expenses online; set warnings when spending limits exceeded.

2. Control seeking
20% of credit card users
Tend to make minimum payments but try to control spending.

Budget strategy: Same as above.

3. Financially savvy
20% of credit card users
Often pay off monthly balances on two or more cards.

Budget strategy: Shift to a single credit card and monitor monthly expenses online.

4. Confident and in control
33% of credit card users
Use a credit card for the convenience or rewards. Pay off charges each month.

Budget strategy: Set up online monitoring of monthly expenses.

SOURCE: H. SHEFRIN AND C. M. NICOLS/ *J. BUSINESS RESEARCH* 2014

Managing complexity

Many business managers take the good over the perfect when grappling with how best to sell their products, suggest two investigations also published in the August *Journal of Business Research*.

Big companies often determine prices for what they sell based on calculations that incorporate a product's relative quality, the number of competing products and many other factors. But successful brand managers often prefer to go simpler, setting prices based on one good hunch: their opinions of their brands' strength in the marketplace, says Alexander Rusetski, a marketing professor at York University in Toronto.

In an online survey of 116 brand managers at U.S. companies, Rusetski presented hypothetical situations in which volunteers described how their team would determine

prices for a new product just after a major competitor had launched a similar product.

Two out of three managers favored setting prices by comparing the strength of their companies' brands to that of major competitors' brands. Managers of products with superior market shares, profitability and growth rates — or strong brand strength, as defined by Rusetski — set prices higher than those of competitors. Managers of products with relatively weak brand strength favored lower or equal prices.

Researchers know little about how pricing based on brand strength, which can lead to overcharging for powerful brands, influences overall sales relative to another simple tactic — pricing based purely on a product's quality.

More is known about the effectiveness of rules of thumb used by businesspeople to identify active customers — those likely to continue buying services from a company. Bankers in Sweden and four nearby countries discern active clients by using principles as simple as "an active customer should have had contact with us during the past year and have at least 20 euros on their account," economists Andreas Persson of the Hanken School of Economics in Helsinki and Lynette Ryals of Cranfield University in England concluded in a second study. German researchers reported in 2008 that similar rules did surprisingly well at identifying which customers of a clothes retailer, an airline and an online CD seller spent the most on those products over as many as four years.

Bank on it

Even the Bank of England, the central bank of the United Kingdom, is intrigued by heuristics. Working with Gigerenzer and his colleagues, Bank of England economists have conducted simulations based on historical data. Their findings indicate that, when calculating how much money to keep in reserve to cover potential loan defaults, simple measures may be the way to go. Decisions based on the amount of a bank's capital relative to total loans to households and businesses — known as the leverage ratio — can sometimes do better than calculations that consider many factors, including the rate of asset growth, the amount of retail loans relative to retail deposits and many other variables.

Several simple measures of banks' financial strength from 2006, including leverage ratios, slightly outperformed complex calculations in predicting which of 116 international banks ended up failing during the global financial crisis in 2007 to 2009, the researchers reported in May in a paper published by the Bank of England. That crisis, partly fueled by bad bank loans justified by complex risk formulas, did much to inspire the bank's interest in simpler approaches.

The Max Planck-Bank of England team is exploring the extent to which decision trees consisting of a few crucial questions can complement other approaches in detecting financially vulnerable banks, says study coauthor Sujit Kapadia of the Bank of England.

Business heuristics are not without risks; a rule of thumb that works well at first can misfire as circumstances change, cautions economist Colin Camerer of the California Institute of Technology in Pasadena. Asking one's waiter in a restaurant to recommend a dish may be a simple, effective strategy to enjoy a good meal in Europe, where waiters in some countries get no or minimal tips and can afford to be honest, Camerer says. In the United States, though, waiters get fairly substantial tips based on the cost of the meal and are more likely to push the most expensive entrée when asked.

In a volatile financial world, today's handy investing heuristic can turn sour overnight, Camerer predicts. With so much financial data now available at a keystroke, it can be dangerous to ignore too much information, he says.

Follow your instincts

Still, astute investors apparently exploit an emotional heuristic in which a sense of impending danger motivates them to sell stocks headed for a tumble in value, Camerer and his colleagues reported July 22 in the *Proceedings of the National Academy of Sciences*.

Business heuristics are not without risks; a rule of thumb that works well can misfire as circumstances change.

Over 50 rounds of a lab game, 320 volunteers in groups of 11 to 23 could buy or sell one share of a stock that randomly paid a modest or large dividend after each round. Or, players could buy nothing and earn a small interest rate on their stash of experimental money.

In 12 of 16 games, the average price volunteers were willing to pay for a share of stock rose sharply before plunging by at least 50 percent.

Those who made the most money sold their stock near its peak value, several rounds before declining demand for the stock among players caused its value to plummet. Brain scans obtained from 44 volunteers during the games showed enhanced activity in an area called the insula among high earners shortly before dumping their stocks. Insula activity rises during physical and emotional discomfort.

"A biological early-warning system for danger that includes the insula probably evolved to be overly panicky," Camerer suggests. Those who heed internal early warnings about stock prices or anything else react to lots of false alarms, which is inconvenient in the short run but ensures that actual threats won't be missed in the long run, he suspects.

That hair-trigger tendency suggests a rule of thumb fit for Polonius: "Better safe than sorry." ■

Explore more

- S. Mousavi and G. Gigerenzer. "Risk, uncertainty and heuristics." *Journal of Business Research*. Vol. 67, August 2014, p. 1671.
- H. Shefrin. "Born to spend? How nature and nurture impact spending and borrowing habits." April 2013. Report for Chase Blueprint: bit.ly/SN_Chase

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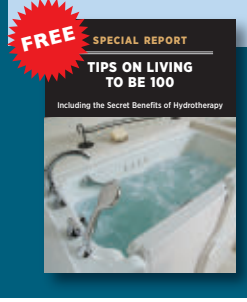

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FILM

‘Dinosaur 13’ relives fight over famous fossils

The morning of August 12, 1990, didn’t seem like a good one for fossil hunting. Thick fog clung to the South Dakota prairie where Peter Larson and other collectors had been digging for dinosaur bones, and their ’75 Suburban had a flat tire.

But while Larson fixed the truck, Susan Hendrickson hiked through the murk and struck dino gold. There, embedded in the side of a cliff, she found a *Tyrannosaurus rex* skeleton — the biggest, most complete specimen ever discovered.

It may also be one of the most fought-over. *Dinosaur 13* chronicles the *T. rex*’s tumultuous journey from its burial spot to its eventual display in Chicago’s Field Museum and dives into the dino’s tangled custody battle.

After Hendrickson’s discovery, the team dubbed the dinosaur Sue, eased her bones from the cliffside and hauled them back to Larson’s Black Hills Institute of Geological Research, which sells and displays fossils. For two years, the team cleaned the fossils and organized the delicate, jumbled bones.

“We were riding on top of the world,” says Larson’s brother Neal. Then, he says, “All hell broke loose.”

On May 14–16, 1992, the FBI and the National Guard swooped in and carted the dinosaur away.

Larson’s team spent over a decade fighting to get her back. On the other side of the battle stood the Cheyenne River Sioux tribe, a landowner portrayed as looking to get rich quick and the federal government, who held the land of Sue’s discovery site in a trust. Both sides vied to convince a judge of Sue’s rightful owner, and whether or not Larson’s team conspired to steal the federal government’s property.

The documentary highlights the rift between collectors like Larson, who sell fossils, and academic paleontologists, whose interests may not be as commercial. Though the film is more *Law & Order* than *Jurassic Park*, and dinosaur lovers may wish for more information, we do get a few bits about Sue’s life. Violent injuries, including a broken skull and a lower jaw ripped from the socket, suggest that she may have died in an attack by another *T. rex*, Larson says.

But *Dinosaur 13*’s focus isn’t so much Sue as it is her discoverers. We’re primed to see the story from their perspective and feel the agony of lost research and a dinosaur wrenched from her home. — *Meghan Rosen*
Lionsgate, 2014

BOOKSHELF

Virtual Unreality

Just because the Internet told you, how do you know it’s true?

Charles Seife

Reality and the virtual world “can no longer be completely disentangled,” argues Seife. A science journalism professor at New York University, he documents how this entanglement is altering what we know — or think we know. It’s an unsettling read, all the more because he gives concrete examples of how unscrupulous people can exert undue influence on the public, politics and the economy.

The Internet makes it easy to hide an author’s identity. It can also mask the source of information presented as fact. Take Wikipedia and its crowdsourced “knowledge.” Wikipedia attempts to shield the sources of its entries. Yet one *New Yorker* investigation turned up a 24-year-old who wrote or edited 16,000 Wikipedia entries — but lacked the credentials and expertise he had claimed. Wikipedia’s founder seemed unconcerned when confronted with this information. Concludes Seife: “Loyalty and hard work for Wikipedia earn one an authority that transcends anything that mere subject-matter expertise can give you.” Bottom line: User beware.

Another new role of the Internet is the global soapbox. For the first time in history, the voice of a few can move across the world at the speed of light and at virtually no cost. This certainly democratizes speech. But if that speech is hateful or dangerously uninformed, Seife argues, it risks becoming a virtual “virus” that infects “a dispersed but digitally interconnected group.”

That information, like so much else on the Web, is free. Seife warns we often get what we pay for. Too often, he notes, news outlets parrot (some might say loosely plagiarize) the posts of others because original reporting is slow and expensive. And online news sites now tailor much of what they cover for search engines — not people. It’s those algorithms, after all, that decide what pages top our search lists. The Internet has begun “changing what news is” and devaluing quality throughout the media industry, Seife suggests.

His book is not an indictment of digital technology. Society reaps tremendous benefits from it. But the digital landscape is evolving so quickly that few of us fully appreciate our vulnerability to being snookered. Seife’s mission is to open our eyes to how the superpowers of the Internet can be misused. By pointing to signs of digital shenanigans, he shows us how to try and avoid them. — *Janet Raloff*
Viking Press, $26.95

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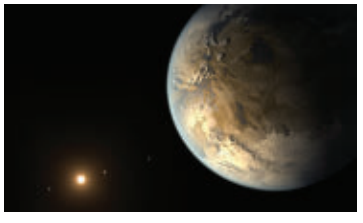
AUGUST 9, 2014

SOCIAL MEDIA

Mourning for exoplanets

Readers were disappointed that the two potentially habitable exoplanets orbiting the star Gliese 581 may not exist after all (*SN: 8/9/14, p. 11*). But there are still plenty of planets out there — including others that may be life-friendly, like Kepler 186f (below).



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Meditations on meditation

People get uncomfortable when they're left alone with their thoughts. In fact, as ***Bruce Bower*** *reported in "People find solitude distressing" (*SN: 8/9/14, p. 12*), college students willingly gave themselves electric shocks to avoid quiet contemplation.*

Readers chimed in with lots of explanations for the findings. **Nancy Martsch** wondered about family dynamics. "As an only child, I've noticed that people from large families or with very close siblings tend to depend on the company of others for entertainment." **John Day** asked, "Why would the authors imagine even for a millisecond that the pathological behavior of modern Americans is a result of Darwinian evolution, as opposed to a reaction and plastic adaptation to a crazy-paced, out-of-control civilization?"

Gretchen Dean warned against using the study to make sweeping generalizations about human behavior. "Perhaps a statement attributed to the French philosopher Pascal should be considered: 'The trouble with Western man is he does not know how to be content in an empty room.' Maybe the study simply lends validity to this observation and does not apply to all human minds. Culture can influence far more than just dress and customs. Before declaring a behavior universal, perhaps researchers should take it on the road."

Tracing Tibetans' genetic past

Tibetans may have gained a genetic adaptation for high-altitude living from extinct human relatives known as the Denisovans. Analyses show a match between distinctive DNA variations in both groups, wrote ***Tina Hesman Saey*** *in "Tibetan high life aided by old DNA" (*SN: 8/9/14, p. 8*).*

Jim LeSire wasn't convinced that the Denisovans were the source of the variations. "I see no reason why it should be necessary for Tibetans to have inherited their genes from the Denisovans; they could just as easily have developed their own." **Tim Cliffe** countered that the gene variants weren't just similar; they were an exact match. "That doesn't happen by accident or coincidence."

Even if the variants are virtually the same, **LeSire** argued, "no one knows where either group got it. The Denisovans might have inherited it from the same source as the Tibetans."

Both readers have a point, says **Saey**. "Tibetans and Denisovans could have inherited the adaptation from an unknown extinct hominid group — the researchers can't rule out that possibility. But the exact match is what makes it seem unlikely that Tibetans and Denisovans acquired so many identical DNA mutations independently."

The Sahara's less-dusty interludes

*In "Dust helped build up the Bahamas" (*SN: 8/9/14, p. 18*),* ***Thomas Sumner*** *explained how airborne particles from the Sahara Desert may have provided nutrients to the carbonate-producing microbes that erected the islands.*

But the Sahara hasn't always been a desert, as **John Compton** pointed out. "Did dust continue to blow across the Atlantic during the ice ages to promote the growth of the calcium carbonate layer, or was this an intermittent process, depending upon the climate?"

Geochemist **Peter Swart** of the University of Miami in Florida replies that while the desert was periodically less arid or less extensive than it is now, it's likely that some dust was always blowing across the Atlantic from the Sahara and nearby regions. During less-dusty periods, **Swart** notes, the Bahamas' growth would have slowed.

Corrections

The right-hand graph in "Dead zone shrank as winds declined" (*SN: 9/6/14, p. 11*) shows a repeated number. The second barometric pressure from the top should be 200 pascals, not 250 pascals.

Due to an editing error, words were omitted from "Gene methylation can lead to cancer" (*SN: 9/6/14, p. 15*). The sentence should read: "Lanlan Shen of Baylor College of Medicine in Houston and colleagues studied whether cancer could result from attaching a chemical tag called a methyl group to the DNA building block cytosine."

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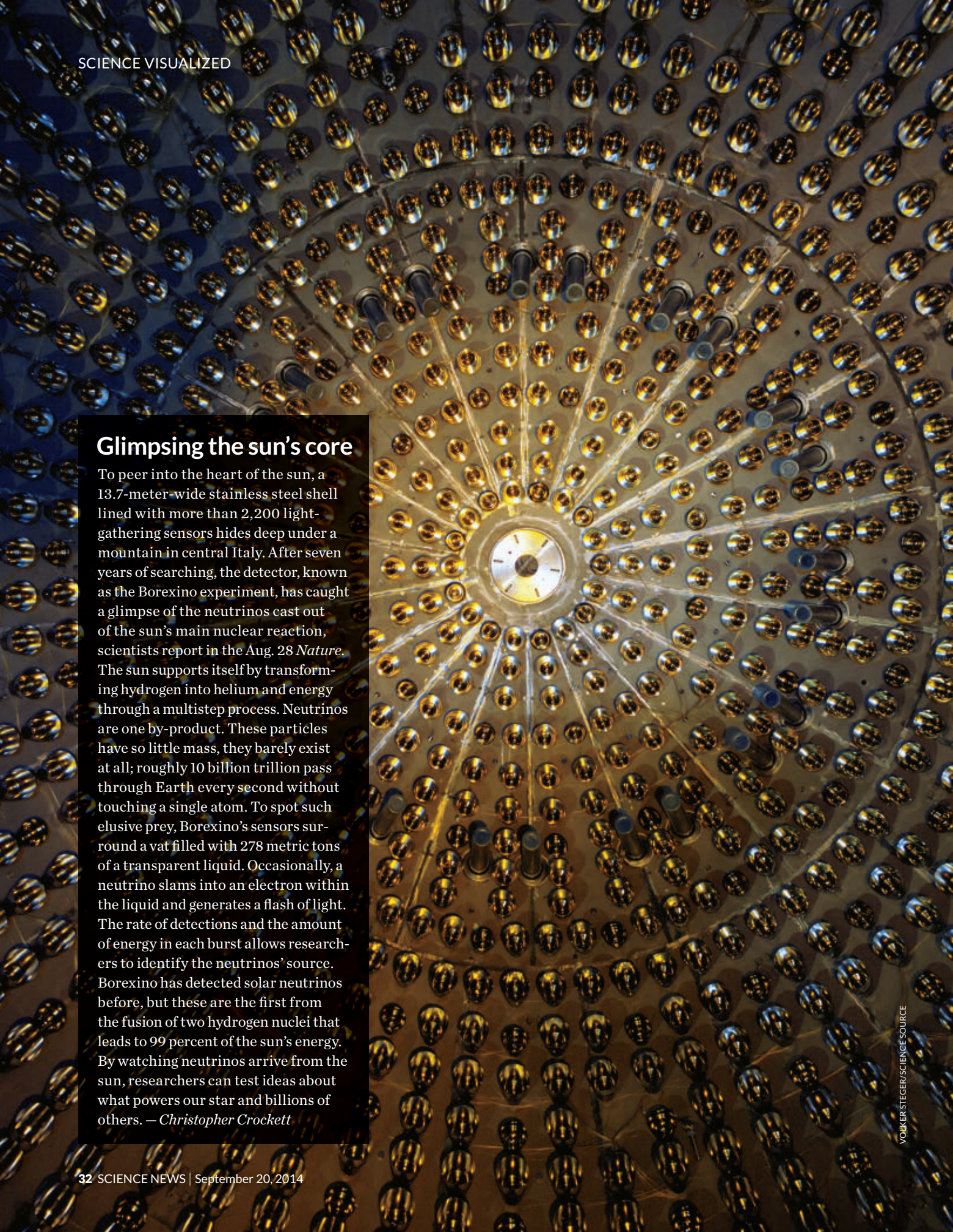

Glimpsing the sun's core

To peer into the heart of the sun, a 13.7-meter-wide stainless steel shell lined with more than 2,200 light-gathering sensors hides deep under a mountain in central Italy. After seven years of searching, the detector, known as the Borexino experiment, has caught a glimpse of the neutrinos cast out of the sun's main nuclear reaction, scientists report in the Aug. 28 *Nature*. The sun supports itself by transforming hydrogen into helium and energy through a multistep process. Neutrinos are one by-product. These particles have so little mass, they barely exist at all; roughly 10 billion trillion pass through Earth every second without touching a single atom. To spot such elusive prey, Borexino's sensors surround a vat filled with 278 metric tons of a transparent liquid. Occasionally, a neutrino slams into an electron within the liquid and generates a flash of light. The rate of detections and the amount of energy in each burst allows researchers to identify the neutrinos' source. Borexino has detected solar neutrinos before, but these are the first from the fusion of two hydrogen nuclei that leads to 99 percent of the sun's energy. By watching neutrinos arrive from the sun, researchers can test ideas about what powers our star and billions of others. — *Christopher Crockett*

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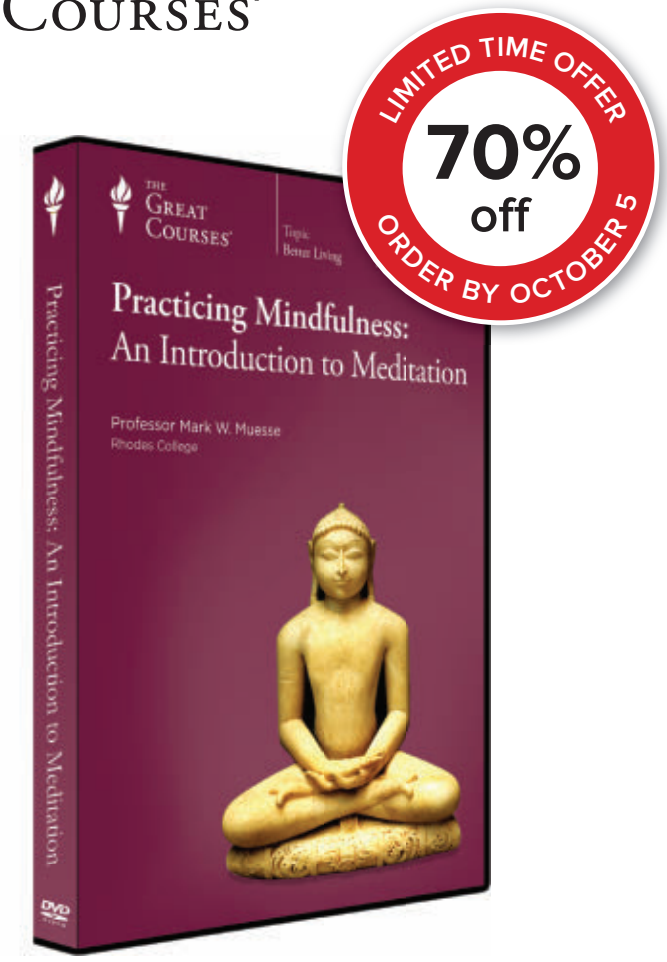

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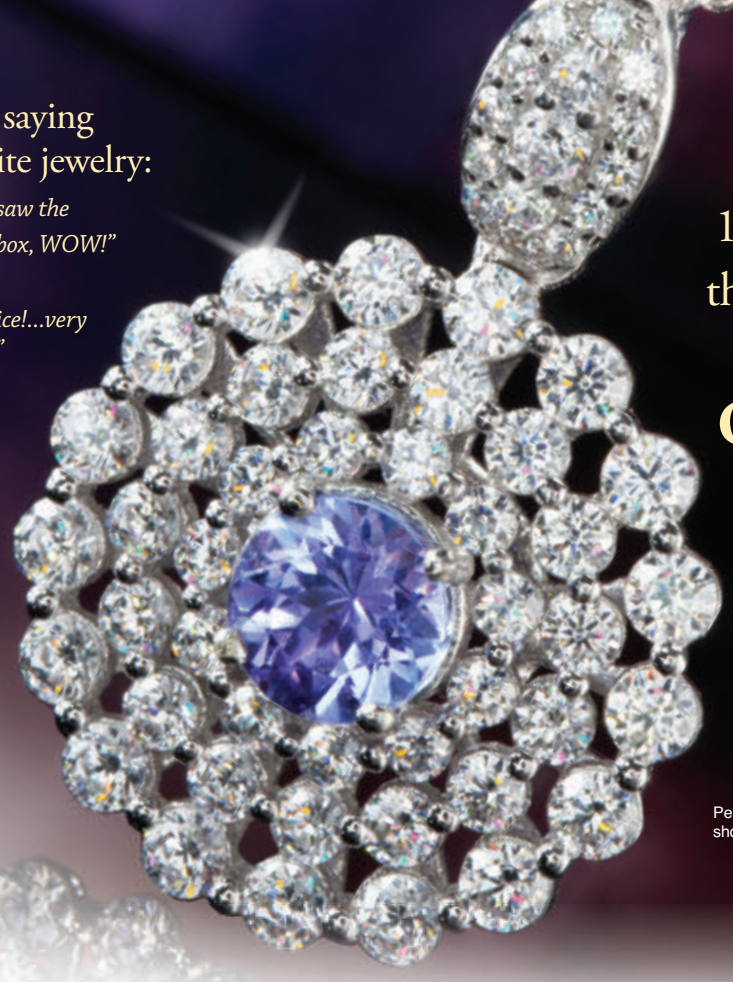

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