SN
SCIENCE NEWS MAGAZINE
SOCIETY FOR SCIENCE & THE PUBLIC
MAY 30, 2015

Picturing Thunder

Why Microbes Hate Fast Food

Ethics of Editing the Genome

More Wave-Particle Trickery

The Eye REWIRED

Optogenetics may offer a way to restore vision

Chicago Doctor Invents

Affordable Hearing Aid

Superb Performance From Affordable Digital Hearing Aid

Board-certified physician Dr. Cherukuri has done it once again with his newest invention of a medical grade ALL DIGITAL affordable hearing aid.

Dr. Cherukuri knew that untreated hearing loss could lead to depression, social isolation, anxiety, and symptoms consistent with Dementia and Alzheimer's disease. In his practice he knew that many of his patients would benefit from new digital hearing aids, but many couldn't afford the expense, which is not generally covered by Medicare and most private health insurance.

Same Digital Technology as $3,000 Hearing Aids

He evaluated all the high priced digital hearing aids on the market and then created his own affordable version — called MDHearingAid® AIR for its virtually invisible, lightweight appearance. This sleek doctor-designed digital hearing aid delivers crisp, clear sound all day long and the soft flexible ear domes are so comfortable you won't realize you are wearing them.

This new digital hearing aid is packed with the features of $3,000 competitors at a mere fraction of the cost. Now most people with hearing loss are able to enjoy crystal clear, natural sound — in a crowd, on the phone, in the wind — without "whistling" and annoying background noise.

Try it at Home with Our 45-Day Risk-Free Trial

Of course hearing is believing, and we invite you to try it for yourself with our RISK-FREE 45-Day home trial. If you are not completely satisfied simply return it within that time period for a full refund of your purchase price.

- **Mini Behind-the-Ear Digital hearing aid**
- **Sleek, nearly invisible**
- **Doctor-Recommended, Audiologist-Tested**
- **FDA-Registered**
- **Thousands of satisfied customers**
- **FREE Shipping in USA**
- **Batteries Included! Comes Ready To Use**
- **100% Money Back Guarantee!**

Satisfied Buyers and Audiologists Agree, *AIR* is the Best Digital Value

"The AIRs are as small and work as well as a $5,000 pair I had previously tried from somewhere else!" —Dennis L., Arizona

"...my mother hasn't heard this well in years, even with her $2,000 Digital! It was so great to see the joy on her face." —Al P., Minnesota

"I would definitely recommend them to my patients with hearing loss"
—Amy S., Audiologist, Indiana

For the Lowest Price Call Today

800-873-0541

Phone Lines Open 24 Hours EVERY DAY

Use Offer Code CB14 to get

FREE Batteries for a Full Year!

100% Money Back Guarantee — 45-DAY RISK-FREE TRIAL

Proudly Assembled in the USA from Domestic & Imported Components.

BBB A+ RATING ACCREDITED BUSINESS

MDHearingAid® AIR

www.MDHearingAid.com

www.MDHearingAid.com

VOL. 187 | NO. 11

ScienceNews

18

Features

News

8

4

Departments

SOCIETY UPDATE
Broadcom MASTERS on *The Tonight Show*

COVER Behind the eye's colorful iris lies the retina. A new technique may restore some sight in people with damaged retinal cells. *Dimitri Otis/Getty Images*

CLOCKWISE FROM TOP LEFT: PHOTKA/ISTOCKPHOTO; L. CALÇADA/ESO; DINOSTAR CO. LTD

Gene therapy, *Gattaca*-style, poses ethical issues

As scientists explore ways to use genetic engineering to battle blindness, it's obvious that gene therapy has gotten more sophisticated.

In traditional gene therapy, scientists "fix" a broken gene by supplying a healthy version to affected cells. Researchers have used this type of gene therapy to treat many diseases, including, as Tina Hesman Saey reports on Page 22, a rare form of inherited blindness. But that's an option for only a select few. Saey describes a new technique that could potentially benefit many more people. It doesn't "fix" a broken gene but rather re-engineers the eye. Transforming nearby nerve cells into cells able to detect light can bypass damaged rods and cones and still get a message to the visual part of the brain.

This approach has already been done with low-resolution electronic implants. But the new technique could boost that resolution 100,000-fold. Human trials have yet to start, but researchers have gotten very good at enabling mice to see.

As Saey was finishing this story, a new report suggested that gains from the current gene therapy method might be short-lived: In some people who regained significant vision after the procedure, the gains later diminished. Disappointing, sure, but the scientists who reported the finding seemed to take it in stride. That's because, in science, not everything works perfectly the first time. You do experiments, you improve things, you figure things out. It's the scientific process.

Which leads me to another story by Saey (Page 16). Chinese researchers have reported editing a gene in a human embryo, stirring a boiling ethical debate about engineering genes in the human germ line, *Gattaca*-style. While the success rate was low, and the embryos would not have been able to grow into fetuses (having been created with three sets of chromosomes), the report still bolstered calls for a ban on tinkering with genes in human eggs, sperm or embryos. That seems premature. These techniques have the potential to help many; seeing if they can ever become "safe and effective" should be scientists' goal. And that requires more research. If the techniques do pan out, we should trust that it's possible to make rules for using that technology appropriately.

— Eva Emerson, Editor in Chief

EDITORIAL, ADVERTISING AND BUSINESS OFFICES
Audit Bureau of Circulations
1719 N Street NW, Washington, DC 20036
Phone: (202) 785-2255
Customer service: member@societyforscience.org
Editorial/letters: editors@sciencenews.org
Sponsor content: snsales@sciencenews.org

Science News (ISSN 0036-8423) is published biweekly by Society for Science & the Public, 1719 N Street, NW, Washington, DC 20036.

Online and iPad access: Activate your subscribing member account, including digital access and the ability to opt out of print, at www.sciencenews.org/activate

Subscribe:
Web www.sciencenews.org/join
For renewals, www.sciencenews.org/renew
Phone (800) 552-4412 in the U.S. or (570) 567-1191 outside of the U.S.
E-mail member@societyforscience.org
Mail *Science News*, PO Box 1205, Williamsport, PA 17703-1205

Subscribing memberships include 26 issues of *Science News* and are available for $50 for one year (international rate of $68 includes extra shipping charge). Single copies are $3.99 (plus $1.01 shipping and handling). Preferred periodicals postage paid at Washington, D.C., and an additional mailing office.

Postmaster: Send address changes to *Science News*, PO Box 1205, Williamsport, PA 17703-1205. Two to four weeks' notice is required. Old and new addresses, including zip codes, must be provided.

SANDY SCHAFFER

Urgent: Special Summer Driving Notice

To some, sunglasses are a fashion accessory…

But When Driving, These Sunglasses May Save Your Life!

Drivers' Alert: Driving can expose you to more dangerous glare than any sunny day at the beach can… do you know how to protect yourself?

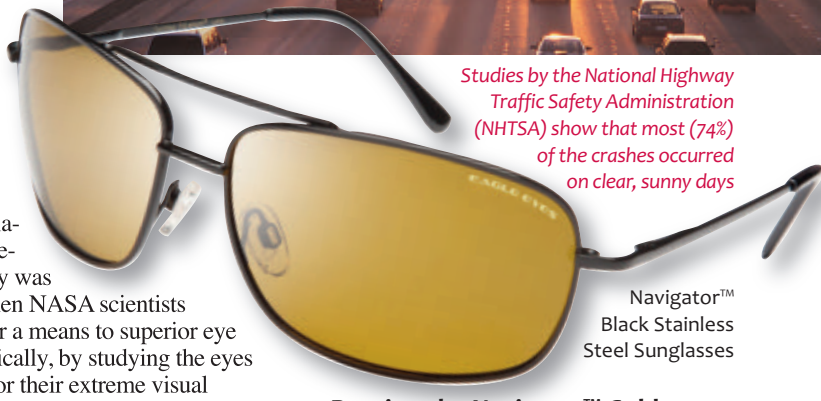
The sun rises and sets at peak travel periods, during the early morning and afternoon rush hours and many drivers find themselves temporarily blinded while driving directly into the glare of the sun. Deadly accidents are regularly caused by such blinding glare with danger arising from reflected light off another vehicle, the pavement, or even from waxed and oily windshields that can make matters worse. Early morning dew can exacerbate this situation. Yet, motorists struggle on despite being blinded by the sun's glare that can cause countless accidents every year.

Not all sunglasses are created equal.
Protecting your eyes is serious business. With all the fancy fashion frames out there it can be easy to overlook what really matters—the lenses. So we did our research and looked to the very best in optic innovation and technology.

Sometimes it does take a rocket scientist. A NASA rocket scientist.
Conventional sunglasses can blur your vision by exposing your eyes to harmful UV rays, blue light, and reflective glare. They can also darken useful vision-enhancing light. But now, independent research conducted by scientists from NASA's Jet Propulsion Laboratory has brought forth ground-breaking technology to help protect human eyesight from the harmful effects of solar radiation light. This superior lens technology was first discovered when NASA scientists looked to nature for a means to superior eye protection—specifically, by studying the eyes of eagles, known for their extreme visual acuity. This discovery resulted in what is now known as Eagle Eyes®.

Slip on a pair of Eagle Eyes® and everything instantly appears more vivid and sharp. You'll immediately notice that your eyes are more comfortable and relaxed and you'll feel no need to squint. The scientifically designed sunglasses are not just fashion accessories—they are necessary to protect your eyes from those harmful rays produced by the sun during peak driving times.

Studies by the National Highway Traffic Safety Administration (NHTSA) show that most (74%) of the crashes occurred on clear, sunny days



Navigator™ Black Stainless Steel Sunglasses

Receive the Navigator™ Gold Sunglasses (*a $99 value*) **FREE! just for trying the Navigator™ Black**

Navigator™ Gold Stainless Steel Sunglasses

The Only Sunglass Technology Certified by the Space Foundation for UV and Blue-Light Eye Protection.
Eagle Eyes® features the most advanced eye protection technology ever created. The TriLenium® Lens Technology offers triple-filter polarization to block 99.9% UVA and UVB—plus the added benefit of blue-light eye protection. Eagle Eyes® is the only optic technology that has earned official recognition from the Space Certification Program for this remarkable technology. Now, that's proven science-based protection.

Certified EAGLE EYES® was developed from original NASA Optic technology and was recently inducted into the Space Foundation Technology Hall of Fame.

The finest optics:
And buy one, get one FREE!
Eagle Eyes® has the highest customer satisfaction of any item in our 20 year history. We are so excited for you to try the Eagle Eyes® breakthrough technology that we will give you a **second pair of Eagle Eyes® Navigator™ Sunglasses FREE—a $99 value!**

That's two pairs to protect your eyes with the best technology available for less than the price of one pair of traditional sunglasses. You get a pair of Navigators with stainless steel black frames and the other with stainless steel gold, plus two micro-fiber drawstring cleaning pouches are included. Keep one pair in your pocket and one in your car at all times.

Your satisfaction is 100% guaranteed.
If you are not astounded with the Eagle Eyes® technology, enjoying clearer, sharper and more glare-free vision, simply return one pair within 60 days for a full refund of the purchase price. The other pair is yours to keep. No one else has such confidence in their optic technology. Don't leave your eyes in the hands of fashion designers, entrust them to the best scientific minds on earth. Wear your Eagle Eyes® **Navigators** with absolute confidence, knowing your eyes are protected with technology that was born in space for the human race.

Eagle Eyes® Navigator™ Sunglasses ~~$99~~†
Offer Code Price **$49** + S&P **Save $50**
***PLUS* receive the Navigator™ Gold absolutely FREE!—2 pairs for the price of one!**

1-800-333-2045

Your Insider Offer Code: EEN102-01
You must use this insider offer code to get our special price.

Stauer®
BBB ACCREDITED BUSINESS
Rating of A+

14101 Southcross Drive W.,
Dept. EEN102-01
Burnsville, Minnesota 55337
www.stauer.com

† Special price only for customers using the offer code versus the price on Stauer.com without your offer code.

Smart Luxuries—Surprising Prices™

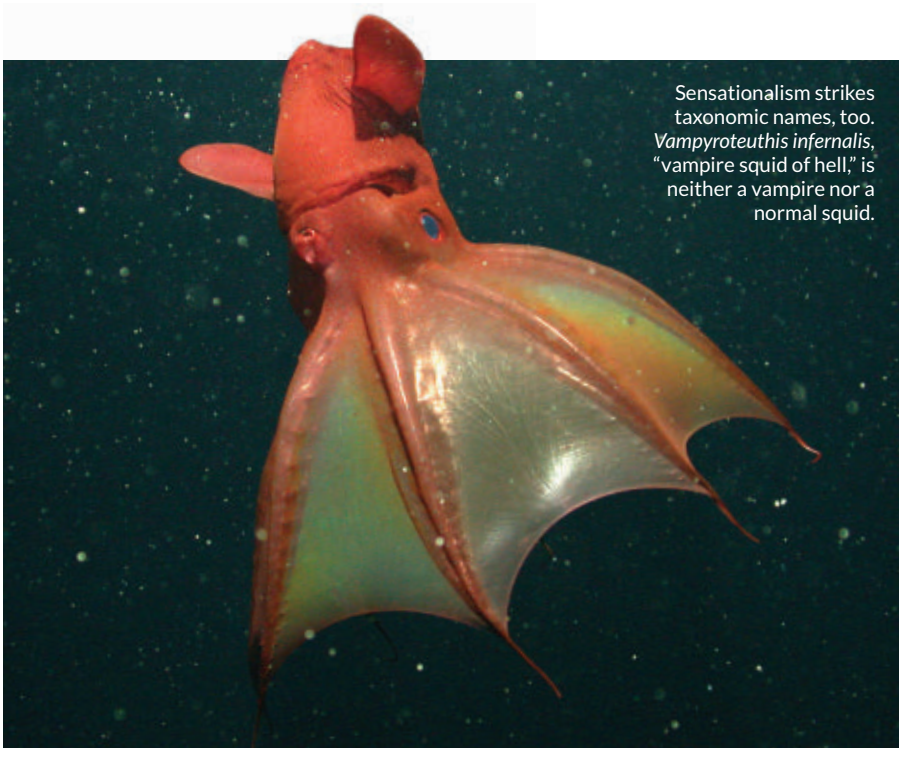
Excerpt from the May 29, 1965, issue of *Science News Letter*

50 YEARS AGO

Dream phase necessary

Dreaming sleep, which seems to be a necessity for man, may also be crucial for cats.... Scientists do not know exactly what function these periods serve in animals. For this reason, they call the phase paradoxical sleep.... [Researchers] have found that a group of cats deprived of paradoxical sleep for a number of days will react the same way humans do. The longer the cats were kept from paradoxical sleep, the more they tried to enter it.

UPDATE: Paradoxical sleep is now called REM sleep. The need for it — in people as in animals — remains mysterious, even though the tools for studying the slumbering brain have increased greatly in sophistication (*SN: 10/24/09, p. 16*). Some researchers argue that REM sleep helps solidify memories. Mammals from platypuses to sloths to giraffes go through REM sleep. Some animals experimentally prevented from REM sleep have learning deficits; others do not.



Sensationalism strikes taxonomic names, too. *Vampyroteuthis infernalis*, "vampire squid of hell," is neither a vampire nor a normal squid.

IT'S ALIVE

Female vampire squid take R&R

Vampire squid live slow. Even their gonads, it turns out, take vacations.

Any information about their reproduction is prized because no one has seen the deep-sea dwellers even swim close enough to each other to flirt. Studies of fished-up specimens offer clues. But the specimens can't solve puzzles such as how sperm gets into female storage pouches, one beside each of her large blue eyes.

What little is known about the biology of *Vampyroteuthis infernalis* suggests a low-speed life scrimping along on modest-at-best resources, says Henk-Jan Hoving of GEOMAR Helmholtz Center for Ocean Research in Kiel, Germany. The species frequents oxygen-starved zones in temperate and tropical waters and has the most sluggish metabolism yet measured among cephalopods. Also, the so-called vampires don't drink blood. They often dumpster dive, gleaning and swallowing wads of sinking fecal pellets and other debris.

This leftovers lifestyle helped Hoving make sense of surprises he and colleagues found in 43 preserved female vampire squid specimens. In many, he found mature ovaries with the expected divots left behind by egg release. Yet some of these obviously working ovaries held no new eggs approaching perfect plumpness for release. The rush to reproduce had paused. In squid, he says, "that's something we had never seen before."

Squid, octopus and cuttlefish typically reproduce in one headlong rush before dying. Called semelparity, this single blowout of procreation also shows up in organisms from salmon to garden squash vines. Among its related species, the vampire looks like the first clear exception, Hoving and his colleagues report in the April 20 *Current Biology*.

"I think they are just not able to mobilize enough energy to have one Big Bang reproduction event," Hoving says.

Not much is known about male vampire squid. But Hoving can say that they don't produce the most famous of squid sperm masses: grenadelike ones that, when free of the male's body, convulse so violently they drill sperm millimeters deep into heads, arms or any soft tissue in range. Vampire sperm clumps instead just turn inside out. Whether males take sabbaticals from producing the sperm parcels, no one knows. — *Susan Milius*

Watch a video of a vampire squid at **bit.ly/SN_vampire**

THE NAME GAME

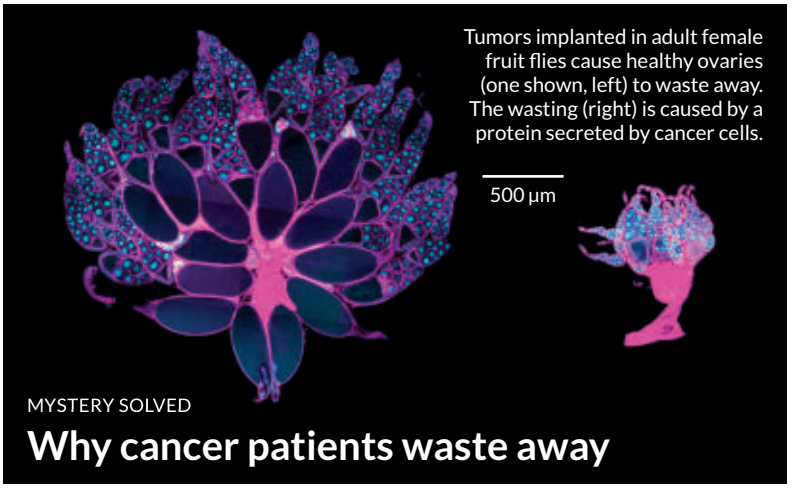
A modest Plutonian proposal

Sad but true: The solar system lacks locales named after canine deities. Fortunately, the New Horizons mission to Pluto might remedy this tragedy. All those undiscovered terrains are going to need names, and researchers offer some recommendations in a paper published online March 29 at arXiv.org.

Aside from a theme of death — Pluto did rule the underworld after all, with Charon ferrying souls there — researchers suggested names from mythology, language and pop culture. Hills and valleys on Pluto's surface might be labeled with the word *cold* in dead or dying languages. Characters from *Star Trek* could be memorialized in craters on the moon Charon. And those neglected canine gods, including Garmr, Asena and Inugami, could finally have a home on terrains of the moon Kerberos, itself named for the multiheaded guard dog of Hades.

Clearly planetary taxonomists will soon have plenty of work. After New Horizons flies past on July 14, a blemish on Kerberos could be named after the Scottish death-hound Cù-Sìth. And Charon might end up with a crater named Scotty. — *Christopher Crockett*

Future interplanetary cruises over the surface of Pluto (illustrated, with its largest moon Charon above the horizon) might include announcements such as "To your left is Wepwawet Crater; to your right, the Julu Valley."



MYSTERY SOLVED

Why cancer patients waste away

A protein released by tumors is what causes cancer patients to waste away, studies of fruit flies suggest.

Fat and other tissues all over the body wither in people with cancer, but the reason for the wasting, also called cachexia, has not been understood. Cancer cells secrete a protein called IMPL2, researchers from Harvard Medical School and the University of California, Berkeley independently report in the April 6 *Developmental Cell*. Both teams came to the conclusion that the protein is responsible for wasting after giving fruit flies cancer.

IMPL2 prevents healthy cells from responding to insulin, a hormone that stimulates cells to import sugar and burn it for energy. When levels of IMPL2 rise, fat, muscle and other tissues can no longer consume sugar and begin to waste away. Lowering IMPL2 levels reduces the amount of wasting, both groups found.

Other factors may also be involved in wasting, the researchers say. — *Tina Hesman Saey*

SCIENCE STATS

Just 1 percent of Amazon's tree species hold half its carbon

The Amazon rainforest holds more carbon than any other ecosystem, but only a small fraction of tree species do most of the work of keeping carbon out of the air. Surveying 530 areas throughout the rainforest, researchers found that roughly 1 percent of Amazonian tree species handle half of the forest's carbon storage.

The Amazon contains about 17 percent of the carbon stored in land plants worldwide. While the forest hosts an estimated 16,000 tree species, the research team identified roughly 150 large, abundant species that remove the most carbon from the air. — *Thomas Sumner*

Amazon trees that carry the most carbon

Species	Percent of total biomass
Eperua falcata	1.93
Eschweilera coriacea	1.87
Bertholletia excelsa	1.31
Qualea rosea	1.27
Chlorocardium rodiei	1.17

SOURCE: S. FAUSET *ET AL/NATURE COMMUNICATIONS* 2015

FROM TOP: L. CALÇADA/ESO; DAVID BILDER AND ALEJANDRA FIGUEROA-CLAREVEGA/UC BERKELEY

LIFE & EVOLUTION

New clues to rise of complex cells

Archaea group may be closest living relatives to eukaryotes

BY SUSAN MILIUS

Cold mud from the seafloor has revealed signs of a new group of microbes that could be the nearest living relatives yet found to the domain of life that includes people and other creatures with elaborate cell structures.

That mud carries DNA of a previously unknown and unusual phylum of one-celled microbes, researchers report online May 6 in *Nature*.

The microbes in this newly named Lokiarchaeota phylum carry the basic DNA of one-celled life in the archaea domain. Yet the new microbes' genes make some 175 proteins that resemble those in eukaryotes, organisms with intricate structures in their cells.

"What was very surprising was the type of function of these genes," says coauthor Thijs Ettema of Uppsala University in Sweden. What the genes do in Lokiarchaeota is a matter of hypothesis. But in eukaryotes, many of these genes help with tasks not seen in archaea, such as changing cell shape and controlling internal compartments called vesicles.

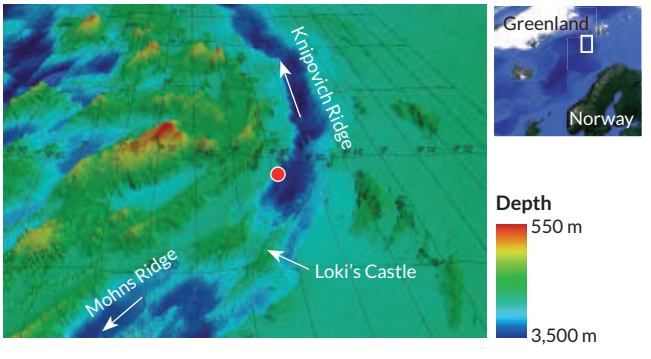
Also, Lokiarchaeota DNA seems to belong within an archaea group called TACK, which some studies predict was a likely source of an ancestor for eukaryotes. (TACK archaea were close relatives of the ancestors of eukaryotes, researchers in France reported online May 11 in *Proceedings of the National Academy of Sciences*.)

The Lokiarchaeota discovery may intensify debates about how cells got complex. In recent decades, biologists have embraced a view that divides organisms into three domains: Two — archaea and bacteria — have single cells with neither nuclei holding DNA nor any other tiny structures enclosed in membranes.

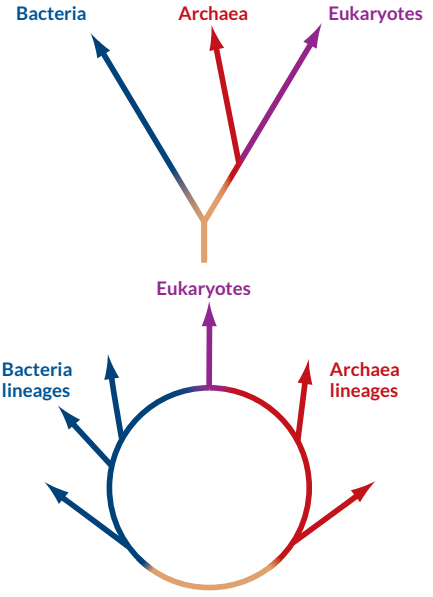
The third domain — eukaryotes — packages DNA inside cell nuclei and furnishes cells with internal nuggets such as mitochondria that specialize in handling energy. How such elaborate cells arose has puzzled biologists, since they have not found clear-cut intermediate forms.

The new find has "genes that might provide a very good starting point to becoming eukaryote," says James McInerney of the National University of Ireland Maynooth. The new work supports a hypothesis, called the ring of life, that eukaryotes arose not from a single lineage but by mingling genes from two domains of less structured cells.

This hypothesis is especially promising in light of the discovery of Lokiarchaeota genes that might allow these organisms' cell membranes to engulf other cells, says evolutionary biologist Mary O'Connell of Dublin City University. One objection to the ring-of-life idea has been the need to explain how genetic merging took place when neither archaea nor bacteria appear able to swallow other organisms.



Visiting the relatives At a deep spot (red dot) on the seafloor between Norway and Greenland, not far from the hydrothermal vents at Loki's Castle, mud has yielded DNA samples from microbes that may be the closest kin found to complex life.



Life stories For decades, variations of a three-domain hypothesis (top) dominated thinking about how major groups of life split. The discovery of Lokiarchaeota lends support to an alternative: a ring diagram (bottom). The ring emphasizes that ancient single-celled archaea and bacteria could have merged genes to give rise to the complex cells of eukaryotes.

It took extreme feats of computing to discover the new genetic mix, Ettema says. The researchers extracted DNA from mud in a sediment core coaxed from the seafloor a few kilometers deep along the Arctic Mid-Ocean Ridge. The scientists censused the DNA fragments with a computer program that sorted bits into separate kinds of life.

The Lokiarchaeota showed up as a blend of genes, some distinctive to archaea and others resembling those from eukaryotes. The more eukaryote-like genes aren't likely to be genetic material snitched from full-fledged eukaryotes, Ettema says. Microbes are known to steal DNA, but these genes were scattered among bona fide archaea DNA instead of appearing in chunks, as stolen goods often do.

Since these conclusions are based on a computer analysis of DNA, "we have huge gaps in our knowledge of what these beasts actually are," McInerney says. "There is a lot of work to do to try to really understand if their relatives 2 billion years ago were important for formation of the eukaryotic cell." ■

FROM TOP: GRAPHICS ADAPTED FROM W. MARTIN AND T.M. EMBLEY/*NATURE* 2004, J.O. MCINERNEY *ET AL*/*NATURE REVIEWS MICROBIOLOGY* 2014; A. SPANG *ET AL*/*NATURE* 2015

Tiny explosions add up to heat corona

Nanoflares may explain high temperature in sun's atmosphere

BY CHRISTOPHER CROCKETT

A relentless onslaught of tiny explosions on the sun's surface blast the solar atmosphere, researchers report. These eruptions, dubbed nanoflares, might help solve the long-standing riddle about why the sun's corona is millions of degrees hotter than its surface.

"This is a real breakthrough to solving one of the most important problems in space science," James Klimchuk, an astrophysicist at the NASA Goddard Space Flight Center in Greenbelt, Md., said at a news conference April 28.

The nanoflares rapidly heat pockets of plasma in the corona, the sun's outer atmosphere, to about 10 million degrees Celsius, said Klimchuk. The plasma then quickly cools to 2 million degrees or so, still much warmer than the roughly 5,500-degree surface of the sun.

Each eruption belches out roughly the same amount of energy as a 10-megaton hydrogen bomb, Klimchuk said. While that amount of energy is enormous by Earth standards, it's just a blip on the sun. These nanoflares have just one-billionth the energy of their much larger cousins, the massive solar flares that hurl bits of the sun into space at millions of kilometers per hour. There are, however, millions of nanoflares erupting every second.

Astronomers can't see individual flares, said Adrian Daw, another astrophysicist at Goddard. Instead they see the superhot plasma in the sun's atmosphere, he said, which is the "smoking gun for nanoflares."

To peer into the corona, Daw and colleagues launched a sounding rocket known as the Extreme Ultraviolet Normal Incidence Spectrograph, or EUNIS, from White Sands, N.M., in April 2013. From about 320 kilometers above Earth's surface, EUNIS observed ultraviolet light streaming from highly ionized atoms in the sun's atmosphere. The amount of energy in the UV light indicated that small pockets of plasma were being heated to about 10 million degrees, the researchers reported at the Triennial Earth-Sun Summit in Indianapolis.

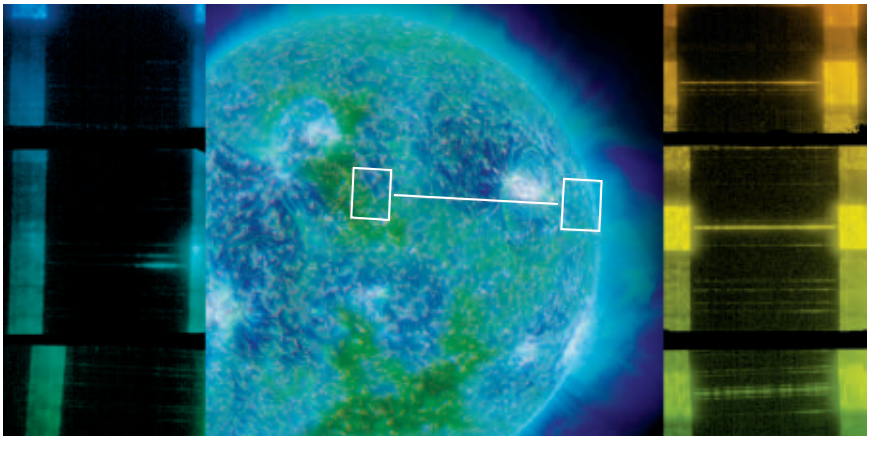
The nature of the corona's thermostat has stumped researchers for 76 years, and nanoflares — first proposed as a solution in 1988 — are just one of many possibilities. One other leading idea is that waves rippling through the plasma transport energy from the solar interior to the corona.

These new results lend significant support to nanoflares, says Amir Caspi, an astrophysicist at the Southwest Research Institute in Boulder, Colo., who was not involved with this research. Caspi detected X-rays from superhot plasma several years ago with another sounding rocket, called X123. The presence of such plasma, he says, suggests something very energetic and impulsive is powering it. Waves, on the other hand, should produce a lot less of the 10-million-degree plasma, he says. There are many open questions about the underlying physics, he adds.

NASA's NuSTAR satellite, which normally looks for X-rays radiating from far-flung black holes and exploding stars, corroborates the EUNIS results. In addition to probing the plasma temperature, NuSTAR looked for high-speed electrons zipping off the sun's surface. Such electrons would indicate that the nanoflares are scaled-down versions of normal flares. No electrons were detected, which could just mean that the signal is swamped by other solar activity. Or nanoflares could be driven by a different mechanism from larger flares.

Researchers suspect nanoflares are triggered by abrupt changes in magnetic fields, which behave like rubber bands, Klimchuk said. If you keep twisting a rubber band, eventually it snaps and releases energy. On the sun, turbulent motion beneath the sun's surface, similar to a boiling pot of water, winds up the magnetic fields, building energy that might eventually erupt in a nanoflare.

More detailed UV spectra of the hot plasma could show how the nanoflares interact with the rest of the corona, said Klimchuk. Visible light observations from the ground, which are much easier to work with than UV data, can also fill in some of the gaps. "What's hard is arranging the moon to block out the sun for you," Daw said. Fortunately, a total solar eclipse will march across the United States in 2017. The eclipse will provide a rare opportunity to view the corona from the ground and track rapid changes better than what's possible from a spacecraft. ■



SDO AND EUNIS/NASA

The EUNIS rocket split ultraviolet light from a region (white boxes and line) of the sun into its component wavelengths (left, right), which revealed emissions from highly ionized atoms in 10-million-degree-Celsius plasma. The data suggest nanoflares help heat the sun's atmosphere.

LIFE & EVOLUTION

Bat-winged dinosaur may have glided

Rodlike bone in forelimb suggests creature took to the skies

BY ASHLEY YEAGER

A dinosaur called *Yi qi* appears to have lifted a page from pterosaurs' flight plan.

Protruding from each of the newly discovered dinosaur's wrists was a pterosaur-like, rod-shaped bone that may have attached to a fleshy wing that helped the dinosaur glide or fly, researchers report online April 29 in *Nature*.

"It's almost like this dinosaur was pretending to be a pterosaur," says paleontologist Sarah Werning of Stony Brook University in New York.

Pterosaurs, flying reptiles that lived at the same time as dinosaurs, had fleshy wings supported by a short bone called the pteroid bone and by a long finger bone. *Y. qi*'s rodlike bone is similar to the pterosaur's but longer. And unlike pterosaurs' pteroid bone, *Y. qi*'s probably sat on the equivalent of humans' pinkie side of the wrist rather than the thumb side, says study coauthor Xing Xu, a paleontologist at the Institute of Vertebrate Paleontology and Paleoanthropology in Beijing.

The fossil find includes membranous tissue preserved between *Y. qi*'s rodlike bone and fingers that may have been part of a wing. But there is only one specimen of *Y. qi* and it's not well preserved. So Xu says it's unclear how the dinosaur's rodlike bone and fleshy wing would have been configured or how well the dinosaur could have glided or flown.

Still, he says, several lines of evidence support *Y. qi*'s aerodynamic capability. The lengths of the pigeon-sized dino's forelimbs and potential wingspan are proportionally longer than those of the dinosaur-like bird *Archaeopteryx*, which glided. Also, rodlike bones covered in tissue membranes tend to accompany flying or gliding, as in bats and flying squirrels, Xu says.

A pigeon-sized dinosaur dubbed *Yi qi* (illustrated) may have glided or flown using fleshy wings similar to those of bats or pterosaurs.

Y. qi lived in China roughly 160 million years ago, 10 million years before *Archaeopteryx*, the team reports. It belonged to the group of dinosaurs called theropods, which slowly transitioned into birds (*SN Online: 7/31/14*). *Y. qi* probably flew nothing like a bird, Xu says, but it may represent an early trial in dinosaur flight. ■

LIFE & EVOLUTION

Plant-munching cousin of *T. rex* found

Bizarre dinosaur roamed South America 150 million years ago

BY ASHLEY YEAGER

Frankensaurus did exist. But don't worry — the sharp-clawed creature with a hodgepodge anatomy was a vegetarian.

Fossils found in Chile reveal a new dinosaur with ancestry that links it to *Tyrannosaurus rex*. But *Chilesaurus diegosuarezi* preferred plants instead of meat, researchers report online April 27 in *Nature*. The discovery supports the idea that not all early *T. rex* relatives were out for blood.

"*Chilesaurus* is ... so drastically different than anything else we've seen before," says Lindsay Zanno of the North Carolina Museum of Natural Sciences in Raleigh.

C. diegosuarezi, which lived 150 million years ago, was about 3 meters long. Its sturdy back legs, thin body and short, stout arms made it a bit like *T. rex*. But its long neck, small head and mouth full of leaf-shaped teeth also gave it a *Brontosaurus*-like appearance (*SN: 5/2/15, p. 14*). The mash-up of features made it hard to know which dinosaur group *C. diegosuarezi* belonged to, says study coauthor Fernando Novas, a paleontologist at the Bernardino Rivadavia Natural Sciences Museum in Buenos Aires.

Using several computer analyses, Novas and colleagues compared *C. diegosuarezi* with many other dinosaurs. The results revealed that it was an early theropod. Scientists once thought that all theropods were carnivores. But studies have begun to suggest that some theropods evolved into plant eaters, says paleontologist Peter Makovicky of the Field Museum in Chicago. A lot of the fossil evidence for this diet switch is based on tooth shape and dates to about 125 million years ago.

"*Chilesaurus* made the switch to plant-eating much earlier," Novas says. "It may have been one of the first experiments in theropod evolution to try the transition." Another theropod, *Limusaurus*, may have made the switch a few million years earlier (*SN: 7/18/09, p. 12*).

"Just a decade ago, no one would have imagined that theropod dinosaurs, Earth's quintessential predators, would turn out to be one of the best evolutionary case studies for how animals adapt to plant-based diets," Zanno says. ■

The 3-meter-long *Chilesaurus diegosuarezi* had a mishmash of anatomy: a body like a *T. rex* with a neck, head and teeth like a *Brontosaurus*.

FROM TOP: DINOSTAR CO. LTD; GABRIEL LÍO

MATTER & ENERGY

Wave-particle duality measured simultaneously

Modified delayed-choice apparatus dissects photon

BY ANDREW GRANT

A new experiment captures a single parcel of light morphing between wave and particle.

Published in the April *Physical Review A*, the experiment dissects a light pulse associated with a single photon to expose wavelike and particle-like components. The results add to recent evidence suggesting that just as Schrödinger's cat can be dead and alive at the same time, light can be a particle and a wave simultaneously at the quantum level. The findings also introduce subtlety to a bedrock tenet of quantum physics about light's apparent unwillingness to display its particle and wave faces at the same time.

Light's ability to switch between wave and particle is well established. Light waves encountering a prism disperse to produce a rainbow of colors. Particles of light, or photons, strike electronic pixels inside digital cameras to create an image. But while light can exhibit the characteristics of particles and waves, Danish physicist Niels Bohr theorized in 1927 that light could behave only as one or the other in a given experiment (*SN Online: 9/15/14*).

Subsequent research has borne out Bohr's idea, even when the choice to observe wave or particle is made after the light has entered the experimental apparatus. In such a delayed-choice experiment, physicists inject a photon into an interferometer. There, the photon encounters a beam splitter, essentially an imperfect mirror. The photon either passes through and exits via one path or gets reflected and exits through a second path. That's perfectly particle-like behavior.

The catch is that once the photon clears the beam splitter, the experimenter may place another beam splitter at a later point where the two paths intersect. When this additional piece of equipment is added, the photon suddenly behaves like a wave. The photon gets divided at the first beam splitter, traverses both paths and then recombines at the second beam splitter, creating interference that identifies it as a wave. Crucially, whether the experimenter chooses to use the second beam splitter or not, the light is either 100 percent particle or 100 percent wave when it exits the interferometer, just as Bohr would have expected.

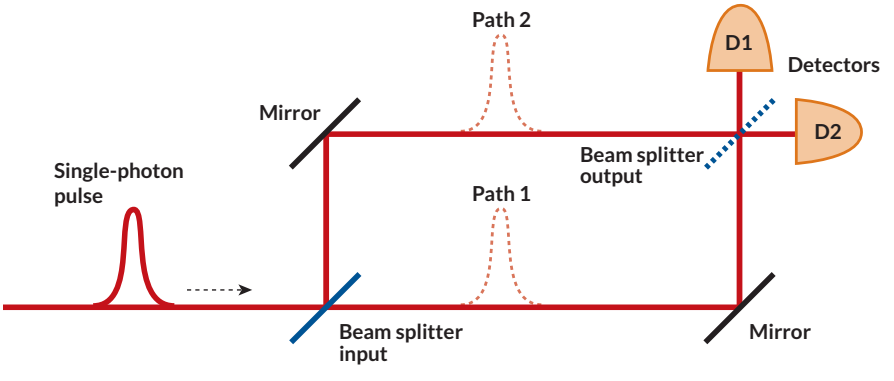
But the new study demonstrates that the distinction between wave and particle is blurrier. Quantum physicist Shi-Liang Zhu of Nanjing University in China and colleagues used the delayed-choice apparatus to measure the specially prepared light pulse of a single photon. The researchers stretched the pulse so that it took roughly 400 nanoseconds to pass a given point. This stretching allowed the researchers to have their cake and eat it too: Instead of choosing early on whether to include the second beam splitter, they could place it or remove it just as the pulse crosses the spot where the paths merge.

Zhu's team showed that for the first 80 nanoseconds of the light's passage, with the second beam splitter in place, a detector situated at the end of one path measured interference, which is characteristic of a wave. But once the researchers removed the beam splitter, the interference disappeared, signaling that the photon was behaving like a particle. Zhu says it's the first time scientists have observed the pulse of a single photon exhibit characteristics of a particle and a wave, a state known as quantum superposition.

The study complements three delayed-choice experiments from 2012 that demonstrated wave-particle superposition. Rather than deciding whether to insert the second beam splitter, these researchers created a quantum switch that determined whether the beam splitter was present, absent or in a superposition of both. In the case of superposition, the researchers showed that the photons were simultaneously particle-like and wavelike. Davide Girolami, a quantum physicist at the University of Oxford, says Zhu's experiment is impressive because it reveals light's wave-particle superposition without the need for that complicated quantum switch.

These recent experiments don't blow up Bohr's theory or physicists' understanding of light's dual nature, Girolami says. Photons will still disperse in prisms as waves and trigger digital cameras as particles, just as Schrödinger's cat isn't partially dead and partially alive. But the results offer useful snapshots of individual photons that will help physicists evaluate light's true nature. "'Wave' and 'particle' are just words," Girolami says. "In quantum physics, those words are imprecise at best." ■

Particle or wave? In the delayed-choice experiment, a photon enters from the left, meets a beam splitter and, like a particle, takes one of two paths. If a second (output) beam splitter is added, even after the photon has passed the first beam splitter, the photon will act like a wave and seemingly take both paths. Researchers showed dual wave-particle behavior by removing the second beam splitter as the photon was passing through it. SOURCE: V. JACQUES *ET AL*/*SCIENCE* 2007



M. TELFER

GENES & CELLS

Bacteria staining long misexplained

Gram technique doesn't work the way biologists thought

BY BETH MOLE

With delicate hues of purple and pink, a lab technique called gram staining has reliably characterized bacteria for more than a century. Yet many scientists are mistaken about why the method works.

Contrary to standard texts, the purple dye called crystal violet, a main ingredient in gram staining, does not actually enter bacterial cells, researchers report online April 27 in *ACS Chemical Biology*. Instead, the dye gets trapped in a tight package of sugar-filled polymers, called peptidoglycan, which envelops bacterial cells. The thickness and integrity of the bacterial armor determines whether crystal violet leaves a cell purple or not. That royal shade, or lack of it, reveals a cell's type of outer structure.

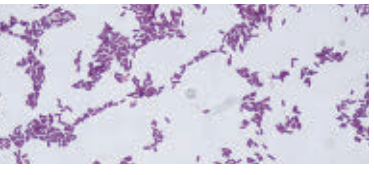
Published by Hans Christian Gram in 1884, gram staining distinguishes gram-positive bacteria (purple) from gram-negative bacteria (pink). Gram-positive critters, such as staph, have a thick peptidoglycan layer that shields an inner cellular membrane. Gram-negative microbes, such as *E. coli*, have a thin peptidoglycan layer sandwiched between a porous outer membrane and an inner membrane.

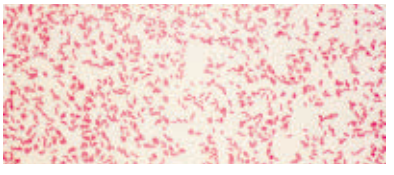
Scientists had thought that crystal violet easily passes through membranes and into both cell types, says microbiologist Moselio Schaechter, an emeritus professor at Tufts University School of Medicine in Boston. A subsequent harsh shower of alcohol then corrodes both cell types' membranes. This particularly clobbers gram-negative cells' outer structures, including the thin layer of peptidoglycan bound to the outer membrane, allowing the purple dye to flush away. Meanwhile, gram-positive cells' sturdier peptidoglycan layer stays largely intact, keeping the microbes purple. The colorless gram-negative cells can then be stained with another dye, such as safranine, tinting the cells pink.

But that explanation is incorrect, says physical chemist Michael Wilhelm of Temple University in Philadelphia. Using a spectroscopy technique that monitors molecules as they traverse membranes, Wilhelm and colleagues found that crystal violet doesn't cross the inner membrane of either cell type.

Instead, the dye seeps into the cracks of peptidoglycan, which acts like a "brick wall of sugar," Wilhelm says. A gram-negative cell's thin wall crumbles in the alcohol wash and releases the dye. In gram-positive cells, crystal violet slowly drains from the thick peptidoglycan barrier, but not quickly enough to leave the cell colorless during the protocol.

"Who'd have thought gram stain lecture material needed an update?" says microbiologist Mark Forsyth of the College of William & Mary in Williamsburg, Va. But, he says, "it may take a while to convince old professors like me to actually change their shtick about how this historic stain works." ■





An old lab technique for differentiating bacteria as either gram-positive (left) or gram-negative (right) has been incorrectly explained for decades.

BODY & BRAIN

Measles raises risk of fatal infections

Virus weakens kids' immune system for years, study finds

BY MEGHAN ROSEN

Preventing children from getting the measles protects them from a slew of other deadly diseases, a new analysis in the May 8 *Science* finds.

Even three years after getting measles, kids are about twice as likely to die from other infectious diseases as children who haven't had measles, the study suggests.

Johns Hopkins University epidemiologist William Moss says the results emphasize the importance of the measles vaccine. Preventing measles seems to come with the unexpected bonus of preventing other infections, too, he says.

"Getting the message out that the measles vaccine has this extra benefit is very important," Moss says. "Particularly in this country, at this time."

After massive vaccination campaigns in the United States and Europe in the 1960s and 1980s, childhood mortality rates dropped more than expected. The reason for this drop was a bit of a mystery, Moss says.

The new study's authors wondered if preventing measles made people less vulnerable to dying from other illnesses. Scientists knew that measles hammered the immune system, but they didn't know quite how hard. So the researchers combed through decades of measles incidence and mortality data from England, Wales, the United States and Denmark to find clues.

In each country, the team found a link between measles cases and children who died from other infectious diseases. Measles infection seems to leave people open to attack from other viruses and bacteria for years — much longer than suspected, says study coauthor Michael Mina, an immunologist and epidemiologist now at Emory University in Atlanta.

"Measles is much worse than people thought," he says. "It has these long-term consequences, and it's gone under the radar for decades."

Mina and colleagues think that measles erases the immune system's memory, as work in monkeys has hinted. So kids' bodies may have trouble recognizing and fending off microbial intruders. ■

BODY & BRAIN

Trip to Mars could damage brain cells

Mimic of cosmic radiation led to memory, learning woes in mice

BY LAURA SANDERS

Like cannonballs slamming into stained glass, high-energy particles can shatter the delicate tendrils that connect nerve cells, a study in mice finds. This neural destruction left mice with memory and learning problems, a finding that has implications for intrepid space explorers.

The result is "worrisome, very worrisome," says neuroscientist M. Kerry O'Banion of the University of Rochester Medical Center in New York. But figuring out the human brain's fate on a long space trip is tricky, he notes.

In the study, Charles Limoli of the University of California, Irvine and colleagues briefly exposed mice to a beam loaded with high-energy versions of either titanium or oxygen, particles an astronaut might encounter in deep space. Six weeks after the quick zap, the mice showed memory deficits. Compared with nonirradiated mice, zapped animals were worse at recognizing new toys and remembering where a particular toy used to be, the team reports May 1 in *Science Advances*.

> "Over the course of a long-term mission, this may become very problematic."
>
> CHARLES LIMOLI

The mice also experienced brain damage. After radiation, complex branches of nerve cells that receive messages were shorter and the cells had fewer branches, the team found.

"We weren't expecting such dramatic effects from these charged particles," Limoli says.

During a long-haul mission to Mars, astronauts would surely encounter these sorts of particles, Limoli says. The study suggests that these encounters could damage astronauts' brains, perhaps leading to cognitive problems during the mission and afterward. "Over the course of a long-term mission, this may become very problematic," Limoli says.

Extrapolating from the mouse study to people is difficult, O'Banion says. "There are big differences in the way the animals are exposed to the radiation versus how astronauts are." Space holds a mixture of particles more complex than those in the study's beam, and the doses differ. The brain's response to radiation is, in many ways, "still a black box," he says.

A deeper understanding of how these particles affect the body and brain may help scientists create better ways to shield vulnerable tissues from space radiation (*SN: 7/26/14, p. 18*). ■

ATOM & COSMOS

Cosmic rays hold clues to lightning

Particles offer way to measure thunderclouds' electric fields

BY ANDREW GRANT

High-speed particles from space are helping to unravel a high-voltage mystery in the clouds.

Astronomers have determined the strength of electric fields in thunderclouds by detecting the radio wave signature of cosmic ray particles striking the atmosphere. Reported April 24 in *Physical Review Letters*, the research offers insight into the confluence of cloud conditions that leads to lightning strikes, which atmospheric simulations cannot explain. The technique could also validate an intriguing hypothesis that cosmic rays provide the spark that triggers lightning.

Cosmic rays constantly pelt the Earth. Occasionally, they strike atoms in the atmosphere and generate a cascade of new particles. LOFAR, an observatory based in the Netherlands, tracks these cosmic ray showers with particle detectors and small antennas. The antennas detect radio waves emitted mainly by the showers' electrons. In fair weather, Earth's magnetic field steers the electrons, leading to a predictable polarization of the radio waves.

But during thunderstorms, LOFAR researchers found, particle showers had distinct radio polarization signatures because the storm clouds' electric fields redirected the electrons. By combining observations with computer simulations, the scientists inferred the structure and electric field strength — 50,000 volts per meter during one storm — of

Scientists at the Netherlands' LOFAR observatory have developed a technique using cosmic rays that may help expose the conditions inside thunderclouds that lead to lightning strikes.

BRYCE BRADFORD/FLICKR (CC BY-NC-ND 2.0)

HUMANS & SOCIETY

Bullying linked to poor mental health

Being picked on worse threat than child abuse, study suggests

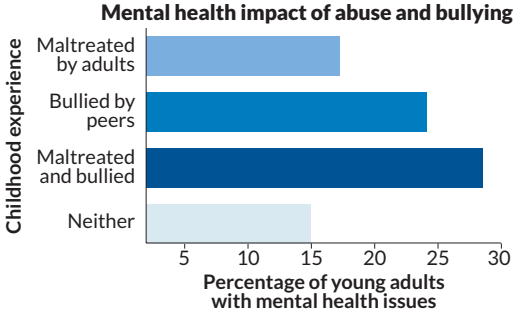
BY BRUCE BOWER

Bullying by peers scars children's mental health over the long haul as much as — or more than — abuse by adults does, a new analysis of U.S. and British kids finds.

By young adulthood, many victims of repeated bullying experience anxiety, depression, self-harm and suicidal thinking and behavior. Their rates of these mental health issues are at least as high as those reported by victims of child abuse who had also been bullied, say psychologist Dieter Wolke of the University of Warwick in Coventry, England, and his colleagues.

Being maltreated by adults — but not picked on by peers — generally leads to fewer long-lasting mental health issues. Abused-but-not-bullied British children display rates of mental problems as young adults comparable to those of kids who were neither maltreated nor bullied, Wolke's team reports online April 28 in *Lancet Psychiatry*. Abused U.S. children have a greater risk of later depression (but not of other mental health problems) compared with kids who were not maltreated or bullied.

"Bullying is not a harmless rite of passage or an inevitable part of growing up," Wolke says. "It has serious, long-lasting, detrimental effects on children's lives." About one in three children worldwide report having been bullied, he says.



Bully bump In a study of British children tracked until age 18, victims of repeated bullying developed mental health problems more often than victims of adult maltreatment did. A combination of bullying and maltreatment proved worst for mental health. A comparable U.S. study found that bullying alone led to the most anxiety and other psychological problems.

SOURCE: ALSPAC, S.T. LEREYA *ET AL*/ *LANCET PSYCHIATRY* 2015

Until now, researchers haven't compared the long-term psychological effects of child maltreatment and bullying, Wolke says.

The new findings underscore the need for parents, schools, child protection services and other agencies to coordinate antibullying efforts, says Corinna Jenkins Tucker, associate professor of family studies at the University of New Hampshire in Durham. But the key question is not whether it's worse to be bullied at school or battered at home, she holds. Children can be victimized in many ways and situations, making simple comparisons misleading, in her view.

In a comment published in the same *Lancet Psychiatry*, Tucker and University of New Hampshire sociologist David Finkelhor advise researchers to study how various forms of victimization add up over time to influence mental health. Cumulative effects of maltreatment by adults, exposure to family violence, having personal property stolen, bullying by a sibling, online bullying and dating violence — to name a few — have yet to be investigated, Tucker and Finkelhor say.

Wolke's findings come from England's Avon Longitudinal Study of Parents and Children and North Carolina's Great Smoky Mountains Study. In the British

thunderclouds pierced by the particles.

Physicist Joseph Dwyer of the University of New Hampshire in Durham says the technique should help scientists piece together how thunderclouds build up the tremendous electric fields required for lightning. Balloons and airplanes can make similar measurements, but they are difficult to maneuver in turbulent air and may trigger lightning.

Cosmic rays may also play a central role in lightning formation by carving out conductive conduits in the air. Study coauthor Pim Schellart, a radio astronomer at Radboud University in Nijmegen, the Netherlands, and colleagues plan to compare the timing of cosmic ray showers and lightning strikes to test that possibility. ■

study, mothers of 3,904 children provided information on abuse and neglect at home from ages 8 weeks to 8.6 years. Children's reports of being bullied were obtained at ages 8, 10 and 13. Mental health interviews were done at age 18.

In the U.S. study, 1,273 children were followed from ages 9 to 16 and incidents of parental abuse and bullying were reported annually. A parent of each child also provided maltreatment and bullying information each year. Final psychiatric assessments occurred as late as age 26.

Mental health problems affected 15 percent of British participants who had not been bullied or maltreated. That figure reached 17 percent in cases of maltreatment only, 24 percent in cases of bullying only and 28 percent for those who had been maltreated and bullied.

In the U.S. study, young adults who had only been bullied displayed the highest percentage of mental health problems, at 36 percent, followed by those who had been maltreated and bullied, at 30 percent.

Regardless of bullying, certain types of child abuse were especially harmful to mental health, Wolke says. In both studies, rates of mental health problems were elevated for those who had experienced sexual and emotional abuse but not physical abuse or harsh parental discipline. ■

GENES & CELLS

DNA disorganization promotes aging

Changes result in cell chaos that causes gray hair, brittle bones

BY RACHEL EHRENBERG

Old cells do not go gently into that good night. In people who age prematurely, changes in the way that DNA is tightly packed in cells lead to mayhem that promotes the aging process, researchers have discovered.

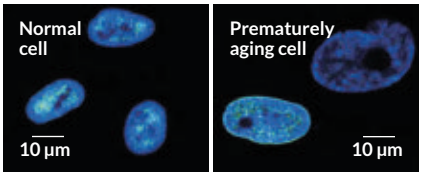
Werner syndrome, a genetic disorder also called adult progeria, leads to graying hair, cataracts, osteoporosis and other signs of aging in people in their 20s. Researchers investigating the syndrome found that mutations associated with the disorder disrupt DNA packaging. These changes unleash genes that shouldn't be turned on, promoting premature aging, an international team of scientists report online April 30 in *Science*.

The scientists also observed some of the same packaging changes in cells of otherwise healthy old people, suggesting that the alterations also drive ordinary aging. Future research into the process could lead to treatments for age-related diseases and may offer ways to slow the advance of old age itself.

Aging is generally thought to result as cells lose the ability to repair and care for their DNA. Two main processes drive this breakdown: damage to caretaker genes and the shortening of telomeres, the caps on chromosome ends that maintain chromosome health and ensure smooth cell divisions. Both processes have been implicated in Werner syndrome.

But the new research points to a third and overlooked mechanism: the disruption of tightly packed bundles of DNA called heterochromatin. When these bundled sections of the genome become disorganized, random stretches of DNA that are supposed to be inaccessible become exposed, resulting in cellular machinery gone wild.

"Aging has been very difficult to understand, and it may be that it happens differently in every person, although we all end up at the same place," says Justine Miller, an expert in age-related diseases at the Neural Stem Cell Institute in Rensselaer, N.Y. Other drivers of aging also lead to random genes being turned on. But those effects pale in comparison with having whole stretches of DNA suddenly accessible and acted on by the cell when heterochromatin becomes disorganized, Miller says.



Tightly packed bundles of DNA in normal human stem cells (left) come undone in cells genetically altered to age prematurely (right), enlarging their nuclei and promoting aging.

In the new work, scientists created a human stem cell line with a faulty Werner gene. The cells aged more quickly than normal cells, and their nuclei, which house the DNA, were enlarged. Compared with normal cells, the elderly cells also had fewer of the chemical signposts that sit on heterochromatin and ensure that DNA isn't accessible. The loss leads to events that disrupt the heterochromatin's ability to stay organized in tidy bundles. This disruption can bring on features of old age: When the team stuck the abnormal cells into the muscles of mice, the muscles atrophied more quickly than those of normal mice.

The disruption of heterochromatin's structure was surprising, says study coauthor Juan Carlos Izpisua Belmonte of the Salk Institute in La Jolla, Calif. The team wondered if disorganized heterochromatin could drive aging in healthy people, too. The team looked for signs in stem cells taken from the dental pulp of six young people (ages 7 to 26) and six older people (ages 58 to 72). Sure enough, there were fewer chemical signposts on the heterochromatin of the older people, suggesting that disorganized heterochromatin may be an important driver of aging in everyone. ■

HUMANS & SOCIETY

Ritual cannibalism occurred in prehistoric England

Archaeologists discover gnaw and butcher marks on 14,700-year-old human bones

BY BRUCE BOWER

A grisly ritual, at least by modern standards, played out in a British cave about 14,700 years ago.

Hunter-gatherers took the bodies of at least six of their deceased comrades to what's now called Gough's Cave and ate them as part of a burial rite, say biological anthropologist Silvia Bello of the Natural History Museum in London and her colleagues. Microscopic analyses show that these bodies, of individuals who presumably died of natural causes, were butchered much like those of nonhuman animals found in the same cave, Bello's team reports in May's *Journal of Human Evolution*.

Human tooth marks consistent with chewing appear on human ribs and other lower-body bones from Gough's Cave, as do stone-tool incisions. Limb bones and other skeletal parts were broken with pounding stones to obtain marrow, the researchers contend.

A Bello-led team reported in 2011 that human remains at Gough's Cave included the top parts of three skulls that had apparently been used as drinking cups. Taken together, these discoveries point to some type of cannibalistic practice related to treatment of the dead, the scientists conclude.

"This is possibly the clearest known archaeological example of ritualistic cannibalism," Bello says.

Her team plans to conduct microscopic studies of scattered human bones at other European sites that, like Gough's Cave, contain artifacts from the Magdalenian culture. That culture lasted from around 15,000 to 12,000 years ago. Many of those bones also bear stone-tool marks and were pounded into pieces. Some researchers have argued that these finds are leftovers of cannibalistic practices. Others say it's more likely that flesh was removed from dead bodies before final burial of bones but not eaten.

> "This is possibly the clearest known archaeological example of ritualistic cannibalism."
>
> **SILVIA BELLO**

Butchered human bones at a roughly 7,000-year-old farming village in Germany have also been interpreted as either remnants of cannibalism or ceremonial reburial (*SN: 1/2/10, p. 10*).

Human tooth marks and related damage on human bones from Gough's Cave provide "compelling evidence for exhaustive butchery and cannibalism of at least six individuals," says Paul Pettitt, an archaeologist at Durham University in England who did not participate in the new study.

The human remains at Gough's Cave date to a time when England's climate was shifting from mild to cold, Pettitt says. If animal prey and edible plants were becoming scarce, cannibalistic burial rituals may have been partly inspired by hunger, he says.

Gough's Cave was discovered in the 1880s. Several excavations in the cave have been conducted since then, with the latest taking place from 1986 to 1992. Workers have unearthed human bones, butchered bones of wild horses and other animals, as well as a variety of stone, bone, antler and ivory artifacts typical of the Magdalenian culture.

Bello's team studied the entire collection of Magdalenian-era human bones from Gough's Cave — 37 skull pieces, four jaw fragments and 164 lower-body fossils. These bones come from three adults, two teenagers and a child.

Microscopic studies of the bones enabled the scientists to produce 3-D digital models of tool and tooth marks. Long cracks, crushed patches, shallow tooth impressions and other bone marks produced by human chewing appeared on 87 lower-body bones. A majority of lower-body bones contained stone-tool incisions characteristic of butchery. Nearly one-third of lower-body bones displayed pounding damage from marrow removal.

Radiocarbon dating of excavated bones indicates that no more than two or three generations of Magdalenian hunter-gatherers practiced ritual cannibalism at Gough's Cave. ■

Human tooth marks and pounding damage (in boxed area) on a 14,700-year-old human leg bone and on dozens of other fossils unearthed in a cave in England indicate that hunter-gatherers there practiced ritual cannibalism, researchers say.

S. BELLO *ET AL/JOURNAL OF HUMAN EVOLUTION* 2015

EARTH & ENVIRONMENT

Current rate of CO_2 rise unparalleled

Human activity dwarfs all other increases since dino extinction

BY THOMAS SUMNER

Humans are dumping extra carbon into the atmosphere at a rate unprecedented since at least the time the dinosaurs went extinct, new research suggests.

Previously, an outpouring of carbon about 56 million years ago had been proposed as faster than the current rate of net increase in atmospheric carbon. But scientists comparing data collected from ocean sediment cores with climate simulations show that this event at most reached only about a tenth of today's carbon increase rate. No direct historical analogs exist to help predict Earth's response to rapidly amassing greenhouse gases, the researchers said May 6.

"Not a single event during the past 66 million years released carbon as fast as we're releasing now," said study coauthor Richard Zeebe of the University of Hawaii at Manoa in Honolulu.

Climate scientists study the past to better predict the future. If a climate simulation can't accurately reproduce historical events, its predictions probably won't be accurate either. With increasing atmospheric carbon dioxide levels shifting climate, scientists have looked for a past event for comparison.

The likeliest contender was the Paleocene-Eocene Thermal Maximum, or PETM, around 56 million years ago. Global CO_2 levels spiked from around 1,000 parts per million to roughly 1,700 to 2,000 ppm, raising global temperatures by several degrees Celsius. While the exact source of this extra carbon is unknown, possibilities range from volcanic eruptions to the release of ice-trapped methane, which breaks down in air to form CO_2. Understanding how long this carbon deluge lasted is key to understanding how fast carbon entered the air.

Scientists can track changes in ancient air by looking at ocean sediments. Calcium carbonate forms layers on the seafloor. As atmospheric carbon levels increase, the ocean becomes more acidic and dissolves more of the calcium carbonate. The amount of calcium carbonate in a sediment therefore serves as a proxy for the level of carbon released at the time the layer formed.

Interpreting ocean sediment cores, researchers proposed in 2013 that the PETM event released its carbon in just over a decade.

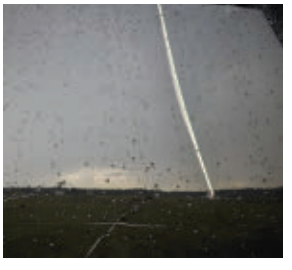
Zeebe and colleagues doubted that finding. Using climate simulations, they calculated the effects of releasing the PETM carbon over various timescales and compared the results with the ocean sediment data. The simulations and sediment data agreed only when the carbon release took more than 4,000 years, Zeebe said. Over that timescale, the carbon release rate was at most 1.1 billion tons a year. That's far less than the roughly 10 billion tons people unleashed via fossil fuel burning in 2013, in addition to carbon released naturally.

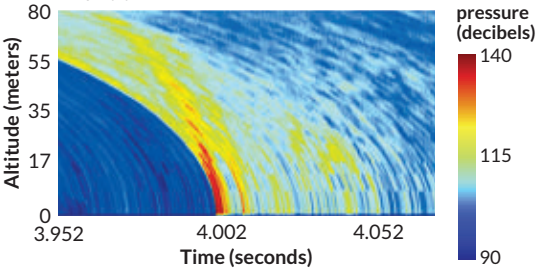
While today's carbon release rate is unmatched, we can still learn from events such as the PETM, said climate scientist Gavin Schmidt of the NASA Goddard Institute for Space Studies in New York City. "When you look back in time, you don't necessarily have to find a direct analog," he said. "You're looking for something that utilizes ... the same Earth processes, but it doesn't have to be in exactly the same way." ■

MEETING NOTE

Visualizing thunder

For the first time, scientists have precisely captured a map of the boisterous bang from a lightning strike. The work could reveal the energies involved in powering some of nature's flashiest light shows.





As electric current flows from a negatively charged cloud to the ground, the lightning rapidly heats and expands the surrounding air, generating sonic shock waves. Scientists have a basic understanding of thunder's origins but lack a detailed picture of the physics.

Maher Dayeh of the Southwest Research Institute in San Antonio and colleagues sparked their own lightning (above, left) by firing a long, Kevlar-coated copper wire into an electrically charged cloud using a small rocket. The lightning followed the conductive wire to the ground. Using 15 microphones laid out 95 meters from the strike zone, the researchers recorded the incoming sound waves. Because sound waves from higher elevations took longer to reach the microphones, the scientists could create an acoustic map (above, right) of the lightning strike with "surprising detail," Dayeh said. He presented the results May 5.

The loudness of a thunderclap depends on the peak electric current flowing through the lightning, the team found. This discovery could one day allow scientists to use thunder to sound out the amount of energy powering a lightning strike, Dayeh said. — *Thomas Sumner*

UNIV. OF FLORIDA, FLORIDA INST. OF TECHNOLOGY, SWRI

Watch a video of scientists triggering lightning at **bit.ly/SN_thunder**

GENES & CELLS

Editing human germline cells debated

New techniques for modifying genes raise ethical questions

BY TINA HESMAN SAEY

Sci-fi novels and films like *Gattaca* no longer have a monopoly on genetically engineered humans. Real research scripts about editing the human genome are now appearing in scientific and medical journals. But the reviews are mixed.

In *Gattaca*, nearly everyone was genetically altered, their DNA adjusted to prevent disease, enhance intelligence and make them look good. Today, only people treated with gene therapy have genetically engineered DNA. But powerful new gene editing tools could expand the scope of DNA alteration, forever changing humans' genetic destiny.

Not everyone thinks scientists should wield that power. Kindling the debate is a report by scientists from Sun Yat-sen University in Guangzhou, China, who have edited a gene in fertilized human eggs, called zygotes. The team used new gene editing technology known as the CRISPR/Cas9 system. That technology can precisely snip out a disease-causing mutation and replace it with healthy DNA. CRISPR/Cas9 has edited DNA in human stem cells and cancer cells. Researchers have also deployed the molecules to engineer animals, including mice and monkeys (*SN Online: 3/31/14*; *SN: 3/8/14, p. 7*). But the method had never been used to alter human embryos.

The team's results, reported online April 18 in *Protein & Cell*, sparked a flurry of headlines because the experiment modified human germline tissue (*SN Online: 4/23/15*). While most people think it is all right to fix faulty genes in mature body, or somatic, cells, tinkering with the germ line — eggs, sperm or tissues that produce those reproductive cells — crosses an ethical line for many. Germline changes can be passed on to future generations, and critics worry that allowing genetic engineering to correct diseases in germline tissues could pave the way for creating designer babies or other potential abuses.

"How do you draw a clear, meaningful line between therapy and enhancement?" ponders Marcy Darnovsky, executive director of the Center for Genetics and Society in Berkeley, Calif. About 40 countries ban or restrict such inherited DNA modifications.

Rumors about human germline editing experiments prompted scientists to gather in January in Napa, Calif. Discussions there led two groups to publish recommendations. One group, reporting in the March 26 *Nature*, called for scientists to "agree not to modify the DNA of human reproductive cells," which would include the nonviable zygotes used in the Chinese study. A second group, writing in the April 3 *Science*, called for a moratorium on the clinical use of human germline engineering, but stopped short of saying the technology shouldn't be used in research. Those researchers say that while CRISPR technology is still too primitive for safe use in patients, further research is needed to improve it. But those publishing in *Nature* disagreed.

"Are there ever any therapeutic uses that would demand ... modification of the human germ line? We don't think there are any," says Edward Lanphier, president of Sangamo BioSciences in Richmond, Calif., and a coauthor of the *Nature* paper. "Modifying the germ line is crossing the line that most countries on our planet have said is never appropriate to cross."

If germline editing is never going to be allowed, there is no reason to conduct research using human embryos or reproductive cells, he says. Sangamo BioSciences is developing gene editing tools for use in somatic cells, an approach that germline editing could render unnecessary. Lanphier says that financial interests play no role in his objection to germline editing.

Other researchers, including Harvard University geneticist George Church, think germline editing may well be the only solution for some people with rare, inherited diseases. "What people want is safety and efficacy," says Church. "If you ban experiments aimed at improving safety and efficacy, we'll never get there."

The zygote experiments certainly demonstrate that CRISPR technology is not yet ready for daily use. The researchers attempted to edit the beta globin gene, or *HBB*. Mutations in that gene cause the inherited blood disorder beta-thalassemia. CRISPR/Cas9 molecules were engineered to seek out *HBB* and cut it where a piece of single-stranded DNA could heal the breach, creating a copy of the gene without mutations. That strategy succeeded in only four of the 86 embryos that the researchers attempted to edit. Those edited embryos contained a mix of cells, some with the gene edited and some without.

In an additional seven embryos, the *HBB* gene cut was repaired using the nearby *HBD* gene instead of the intended single-stranded DNA. The researchers also found that the molecular scissors snipped other genes that the

Genetic gains The development of a series of controversial technologies has led to genetic engineering of human embryos.

July 25, 1978
Birth of Louise Brown, the first baby resulting from in vitro fertilization

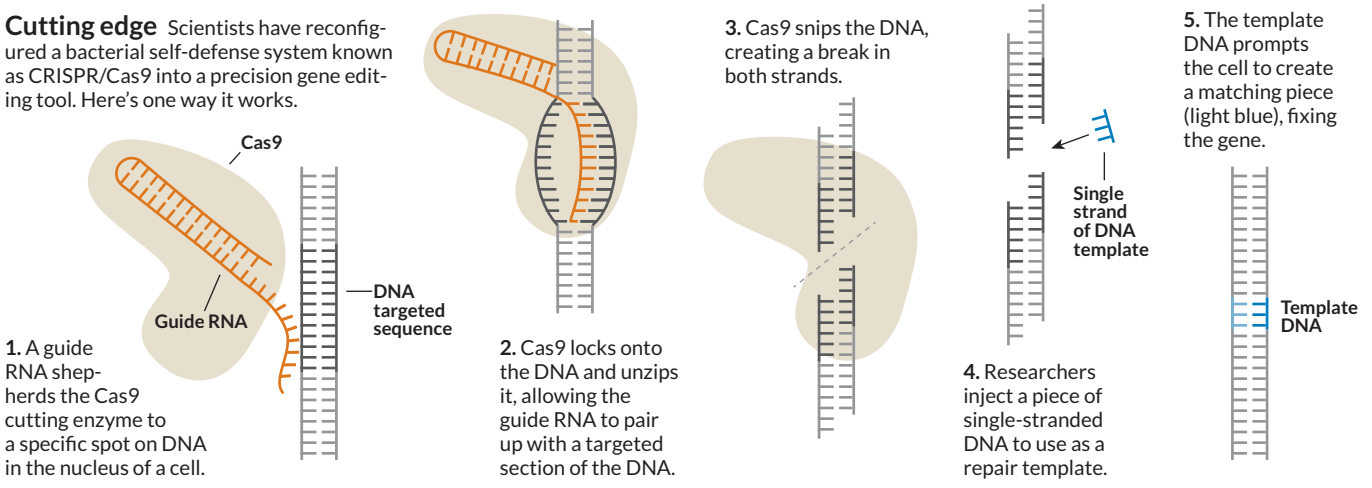
1990
First gene therapy treatment — for girl, 4, with a disease that affects ability to fight infections — begins

July 5, 1996
Birth of Dolly the Sheep, first cloned mammal created by somatic cell nuclear transfer

1998
First report of human embryonic stem cells that have been isolated and grown in lab dishes

2000
Repetitive DNA in bacteria, archaea discovered; later named CRISPRs and found to fight viruses

2009
Successful transfer of nucleus of one egg into donor egg to avert mitochondrial disease in rhesus monkeys



researchers never intended to touch.

"Taken together, our work highlights the pressing need to further improve the fidelity and specificity of the CRISPR/Cas9 platform, a prerequisite for any clinical applications," the researchers wrote.

The Chinese researchers crossed no ethical lines, Church contends. "They tried to dot i's and cross t's on the ethical questions." The zygotes could not develop into a person, for instance: They had three complete sets of chromosomes, having been fertilized by two sperm in lab dishes.

Viable or not, germline cells should be off-limits, says Darnovsky. She opposes all types of human germline modification, including a procedure approved in February in the United Kingdom for preventing mitochondrial diseases. The United Kingdom prohibits other germline editing.

Mitochondria, the power plants that churn out energy in a cell, each carry a circle of DNA containing genes necessary for the organelle's function. Mothers pass mitochondria on to their offspring through the egg. About one in 5,000 babies worldwide are born with mitochondrial DNA mutations that cause disease, particularly in energy-greedy organs such as the muscles, heart and brain.

Such diseases could be circumvented with a germline editing method known as mitochondrial replacement therapy (*SN: 11/17/12, p. 5*). In a procedure pioneered by scientists at Oregon Health & Science University, researchers first pluck the nucleus, where the bulk of genetic instructions for making a person are stored, out of the egg of a woman who carries mutant mitochondria. That nucleus is then inserted into a donor egg containing healthy mitochondria. The transfer would produce a person with three parents; most of their genes inherited from the mother and father, with mitochondrial DNA from the anonymous donor. The first babies produced through that technology could be born in the United Kingdom next year.

Yet another new gene editing technique could eliminate the need to use donor eggs by specifically destroying only disease-carrying mitochondria, researchers from the Salk Institute for Biological Studies in La Jolla, Calf., reported in the April 23 *Cell* (*SN Online: 4/23/15*).

Such unproven technologies shouldn't be attempted when alternatives already exist, Darnovsky says, such as screening embryos created through in vitro fertilization and discarding those likely to develop the disease.

But banning genome-altering technology could leave people with genetic diseases, and society in general, in the lurch, says molecular biologist Matthew Porteus of Stanford University.

"There is no benefit in my mind of having a child born with a devastating genetic disease," he says.

Alternatives to germline editing come with their own ethical quandaries, he says. Gene testing of embryos may require creating a dozen or more embryos before finding one that doesn't carry the disease. The rest of the embryos would be destroyed. Many people find that prospect ethically questionable.

But that doesn't argue for sliding into *Gattaca* territory, where genetic modification becomes mandatory. "If we get there," says Porteus, "we've really screwed up." ■

E. OTWELL

2010–2011
Molecular scissors used for genome editing called TALENs developed by several groups

2012
Ability to program CRISPR/Cas9 system — guide RNA plus enzyme — to precisely edit genes reported

2012
Mitochondrial replacement therapy in human cells reported, paving way for "three-parent" embryos

February 24, 2015
Mitochondrial replacement therapy approved for clinical use in the United Kingdom

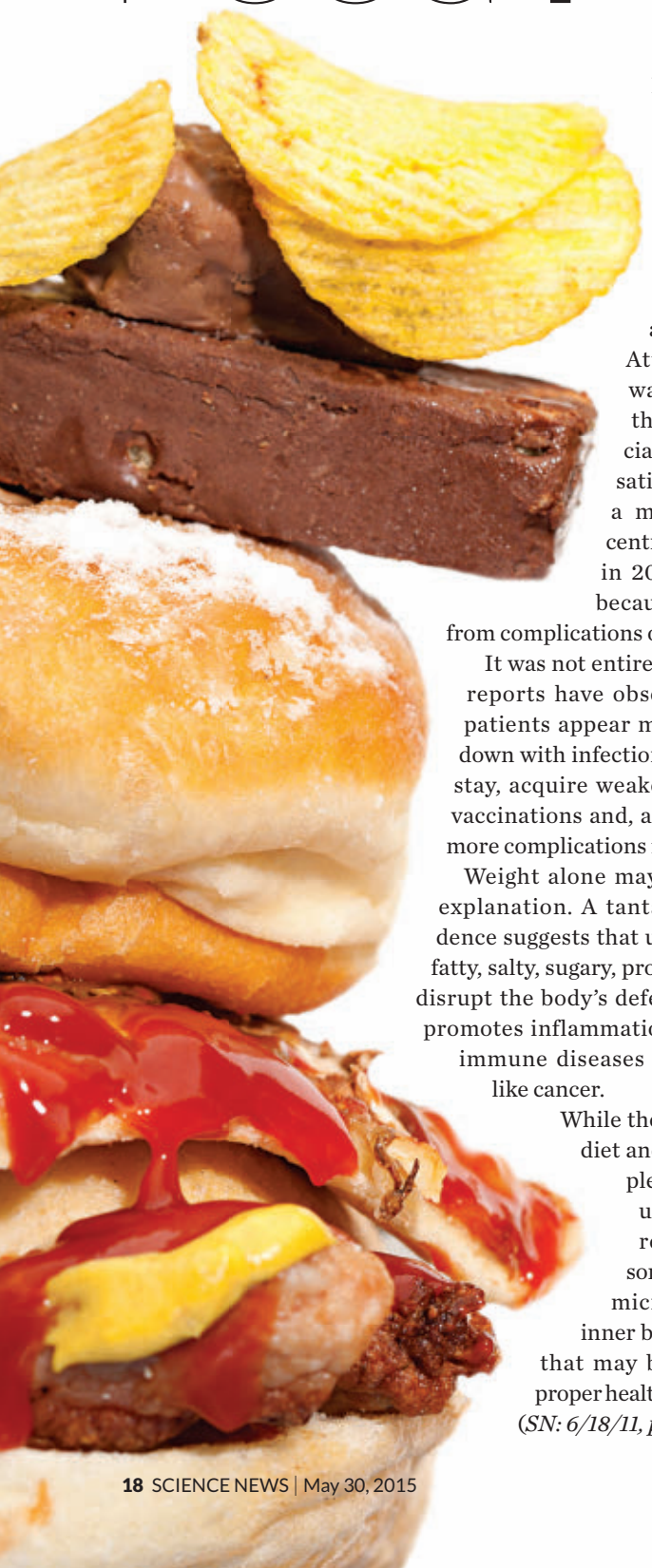
April 18, 2015
Chinese researchers report use of CRISPR/Cas9 to edit a gene in nonviable human zygotes

April 23, 2015
TALENs used to selectively destroy diseased mitochondria in mice, researchers report

Food Fight

Our gut microbes are no fans of junk food

By Laura Beil



Blair River was described as "a big guy with a big heart." The 575-pound former high school wrestler from Mesa, Ariz., became such a fixture at the Heart Attack Grill that he was recruited to be the restaurant's official spokesperson. His satirical ads made him a minor celebrity in central Arizona. He died in 2011 at age 29 — not because of his heart but from complications of influenza.

It was not entirely surprising: Many reports have observed that heavier patients appear more likely to come down with infections during a hospital stay, acquire weaker protection from vaccinations and, as with River, suffer more complications from the flu.

Weight alone may not be the entire explanation. A tantalizing line of evidence suggests that unhealthful foods — fatty, salty, sugary, processed foods — may disrupt the body's defenses in a way that promotes inflammation, infection, autoimmune diseases and even illnesses like cancer.

While the interplay between diet and immunity is complex — and still largely uncharted — many researchers believe some foods perturb gut microbiota, the body's inner bacterial community that may be as important to proper health as any major organ (*SN: 6/18/11, p. 26*).

Among their long list of tasks, gut bacteria may help train the immune system to distinguish between human and microbe so that it can confront what's bad, tolerate what's not, and recruit a diverse army of cellular foot soldiers to stop invading germs. Writing in *Nutrition Journal* in June 2014, one scientist likened gut microbiota to a sparring partner, providing a regular workout that strengthens the contender for a true opponent.

To function at their best, though, gut microbes, like most living things, need to be well fed. And many of the species responsible for immune equilibrium don't seem to care for junk food. In a study reported in *Nature Communications* in April, African-American volunteers who shifted from an American diet to high-fiber, low-fat African cuisine experienced a drop in inflammation in just two weeks.

"It's becoming clear that the typical American diet can damage the immune system," says Ian Myles, a physician and researcher at the National Institute of Allergy and Infectious Diseases in Bethesda, Md. After a lethal infection, most of the blame is understandably leveled against the microbe responsible, Myles says. "It's harder to make the connection that it may have had something to do with what you've been eating the past 30 years."

A slow boil

In a series of experiments over the last decade, scientists have made the case that a poor diet and an out-of-balance intestinal ecosystem can unleash a persistent, low-level immune activation. This is not to be mistaken with the good kind of inflammation, which occurs when bacteria, viruses or anything else foreign slips into the bloodstream. Immune cells rally to surround and eliminate the threat. It's a healthy response necessary for survival.

But this life-saving reaction turns sour when the immune system gets stuck on a slow, endless burn. In an ironic twist of biology, one of the consequences of an immune system that never takes a rest is that it may be asleep when it's really

ZELJKOSANTRAC/ISTOCKPHOTO

Offspring can inherit the effects of parents' lousy food choices

The impact of a Western diet on risk for obesity and cancer can persist for generations, and gut microbes may be responsible, a study published in April suggests. If supported by more research, the findings mean that inherited risk for some diseases is about more than genetics and may be reversible.

In a series of experiments, researchers fed pregnant female mice a high-fat, low-fiber diet representative of typical fast-food fare, while a comparison group ate regular chow. The offspring of the mice on the unhealthy diet were more prone to obesity and cancer — even when the offspring ate normally. The increased risk persisted to the next generation, which also showed signs of premature aging.

The researchers, from MIT and Aristotle University of Thessaloniki in Greece, then zeroed in on the microbiota. Simply transferring intestinal bacteria of mice eating the unhealthy diet to mice eating normal chow that later got pregnant was enough to raise the cancer risk of their pups.

The reverse was true as well: Cancer risk dropped among mice whose mothers ate the fast-food diet but received doses of beneficial bacteria.

"Targeting microbes may be a highly effective population-based approach to lower risk for cancer," the researchers wrote in *Cancer Research*. *— Laura Beil*

needed. Like an annoying car alarm that everyone hears but no one responds to, chronic activation might desensitize the system to actual danger.

There is also evidence that certain kinds of fats and refined sugar, consumed in excess, may compromise the inner lining of the intestine, allowing microscopic leaks that trigger unrelenting immune activation. Also, adipose tissue, or body fat, is so capable of hormone production that it is often referred to as an endocrine organ by itself, able to kindle a low-grade inflammation that stresses tissues and promotes disease.

Taken together, Myles says, the combination of unhealthy diet and obesity explain in part the rise in autoimmune conditions such as celiac disease, type 1 diabetes and other illnesses that occur when the body turns on itself. (For instance, a 2012 report found that the prevalence of inflammatory bowel disease is rising, and rates are highest in Westernized parts of the world.) The connection also suggests avenues for management. One study reported that probiotic foods might help increase insulin sensitivity among diabetics. That's not to say that diet is completely at fault. Why autoimmune diseases occur is a mystery — and theories abound — but a large body of evidence gives some culpability to changes in gut bacteria.

Small world

Until recently, bacteria in the intestine were largely unexplored because they are difficult to grow in a laboratory. Thanks to advances in molecular tools that work outside a petri dish, researchers are getting a better handle on a partnership that serves both host and microbes. While about 1,000 species of bacteria are thought to live in the intestine, only a portion are dominant players. As with ecosystems throughout nature, diversity rules, and an abundance of species signals a healthy environment.

In general, beneficial bacteria appear to prefer certain foods — such as fiber and the complex carbohydrates found in vegetables and whole grains — and dislike others, such as refined sugar and (according to some experiments) saturated fat. Essentially, it's possible to satisfy your own sweet tooth while starving a lot of your microscopic friends.

A landmark experiment in 2009 found that among mice carrying the same microbes found in humans, a high-fat, high-sugar diet caused certain bacterial populations to drop and others to rise. As a result, the microbial community was "dramatically altered over a time scale of hours," researchers from Washington University School of Medicine in St. Louis and the University of Colorado Boulder reported in *Science Translational Medicine*.

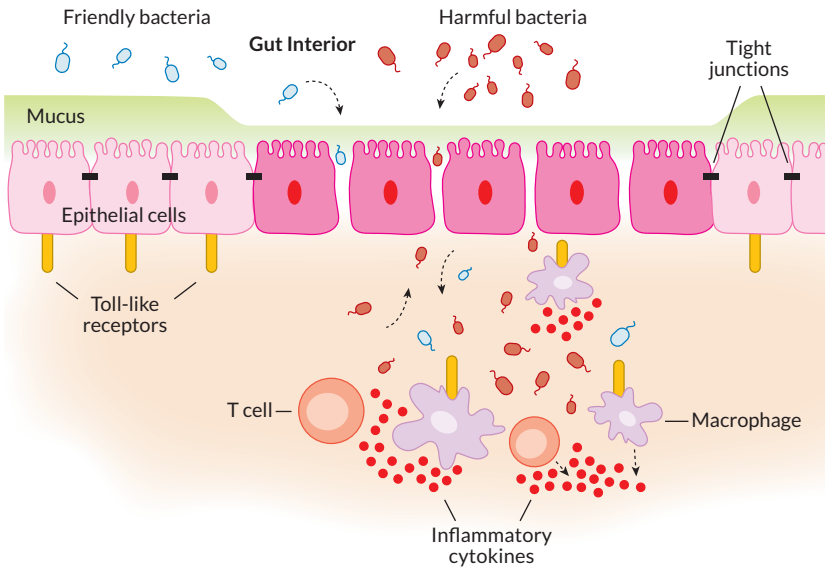
In a follow-up study with human volunteers published last year in *Nature*, a team from Harvard and the University of California, San Francisco found similarly abrupt responses (*SN Online: 12/11/13*). A day after volunteers started a diet dominated by animal products, bacterial diversity in their intestines dropped, which coincided with a bloom in *Bilophila wadsworthia*, a normally minor occupant that has been associated with inflammatory bowel disease.

Pizza and other American favorites are high in fat and sodium, which may be bad for the immune system.

Nutrition Facts
Serving Size 1/2 Pizza (130g)
Servings Per Container 2

Amount Per Serving	
Calories 320	Calories from fat 140
	% Daily Value
Total Fat 15g	**24%**
Saturated fat 4g	**21%**
Cholesterol 0 mg	**0%**
Sodium 560 mg	**24%**
Total Carbohydrate 35g	**12%**
Dietary Fiber 2g	**7%**
Sugars 4g	
Protein 10g	
Vitamin A 6%	Vitamin C 0%
Calcium 10%	Iron 10%

Microbe imbalance causes leaky gut



Break on through

Epithelial cells and mucus that line the intestine protect the rest of the body from pathogens. But a diet heavy in sugars and fats can disrupt the delicate balance of friendly and harmful bacteria and make the gut lining more porous. When bacteria slip through the lining, they set off alarm bells called toll-like receptors. The TLRs signal immune cells, including T cells and macrophages, to produce inflammatory cytokines. The cytokines can weaken the tight junctions between epithelial cells, setting off an inflammatory cycle that might lead to bowel diseases.

"There aren't too many microbes that like to live in the presence of certain types of fat. It's too difficult for them to use as a food source," says Vanessa Leone, a nutrition scientist at the University of Chicago. "So you're selecting for microorganisms that can live in that environment." In 2012, Leone's team reported in *Nature* that a diet high in saturated fat also increases the production of one type of bile. The digestive enzyme made by the liver favors some microbes while killing off others. (And as in other experiments, a major beneficiary appears to be *B. wadsworthia*.)

Nonetheless, researchers caution against singling out saturated fat as a dietary bad boy when other changes in diet have accompanied the rise of obesity. As saturated fats have lost favor, many cooks and food manufacturers have turned to vegetable oils such as corn and canola that are heavy in omega-6 fatty acids. That trend has initiated debate over the role of those acids in inflammation. Deanna Gibson, a gut microbiologist at the University of British Columbia in Kelowna, believes the increased reliance on omega-6 may be promoting the kind of inflammation that contributes to autoimmune disease.

In experiments in mice published in *PLOS ONE* and the *British Journal of Nutrition* in 2013, Gibson and her colleagues reported that diets high in omega-6 fatty acids disrupted gut microbiota. The researchers also saw the kinds of increased inflammatory responses that lead to colitis, a condition resulting from inflammation in the colon.

The mechanisms in the intestine that trigger inflammation are still not fully understood. But toll-like receptors, molecules that sit on certain immune cells, appear to be important players. Once activated, toll-like receptors are capable of setting off alarms. Some respond to the presence of lipopolysaccharides, molecules that are part of the membranes of some bacteria.

Normally, bacteria in the gut hardly come in contact with the rest of the body, because the mucus and epithelial cells lining the intestine keep them in the gut. But diets high in sugar and fat and low in fiber may cause the intestine to become porous. Writing last year in *Cell Metabolism*, researchers from Stanford University pointed out that the calories contained in refined sugars — which constitute an ever-growing part of the American diet — aren't a good food source for the gut's microbiota. In response, evidence suggests, some of the bacteria will eat the mucus lining of the intestine, which may cause the lining to deteriorate.

Eventually, some gut organisms slip through and encounter toll-like receptors on immune cells, setting off an immune reaction. Some experiments also suggest that saturated fat itself may stimulate toll-like receptors.

It's still too early to draw firm conclusions, however. Researchers in South Carolina and Iowa tested the ability of three different diets to activate the toll-like receptors TLR2 and TLR4 on macrophages, key immune cells. All three diets had the same amount of fat, but varied in the proportion of saturated fat: 6 percent, 12 percent or 24 percent. (The diet with 12 percent saturated fat mimicked typical American eating patterns.)

Based on previous studies, the scientists expected the diet with the highest saturated fat to produce the most inflammation. To their surprise, the highest inflammation, and greatest body fat accumulation, occurred with the 12-percent saturated fat diet, the team reported in the *Journal of Lipid Research* in 2013. Since all the diets had the same proportion of total fat, the authors speculate that the combination of different types of fat is responsible for the overall effect.

Salt of the girth

Fat isn't the only thing on the drive-through menu. The average American gets more than twice as much salt as recommended, a problem largely driven by heavy consumption of processed and prepackaged foods. In a study published last year in *Pediatrics*, adolescents who ate the most salt were more likely than others to be obese and have signs of inflammation.

ANDREI IVANOV/VCU, ADAPTED BY T. SHIPLEY AND M. TELFER

Such results are not conclusive; salty foods could also have a high sugar and fat content. But many studies are not reassuring. A multinational research team fed each of six volunteers four diets that differed only in the amount of salt: 12 grams per day, 9 grams, 6 grams, then 12 again. Immune activation spiked on the high-salt diet. More important, in a trend suggesting cause and effect, levels of most immune cytokines gradually fell as salt intake diminished. "These findings raise the possibility that high salt intake might trigger tissue inflammation and autoimmune disease in humans," the scientists wrote last year in *Translational Research*.

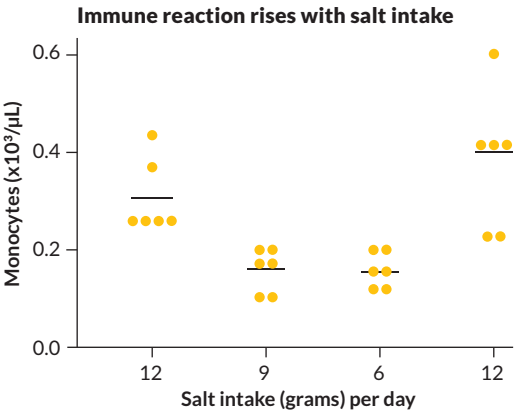
Other studies have raised similar concerns. In 2013, researchers demonstrated that excess salt in the diets of mice could increase activity of certain T cells that have a role in autoimmunity. There's more to learn, cautions immunologist Markus Kleinewietfeld of Yale University and TU Dresden in Germany, lead author of the study, published in *Nature*. "Salt seems to be, in general, a proinflammatory signal for the immune system, but the physiology is not well understood," he says.

There may be situations where the presence of salt is beneficial. He points to a study published this spring in *Cell Metabolism* suggesting that sodium storage in the skin may help protect against infection from a parasite that causes leishmaniasis.

The implications for T cells may be especially important because of their complex role not just in fighting germs, but also in autoimmunity and cancer. T cells may either suppress or promote cancer depending on the circumstances, and their proper balance may rest in part on the health of the gut microbiota.

Consider two types of T cells: Th17 and T-regs (T-regulatory cells). At normal levels, Th17 cells help defend the body against tumors. But in a less-is-more biological irony, if Th17 cells become too numerous, they may actually promote cancer growth. T-regs can keep Th17 under control, but in some situations they can do too good a job and slow the immune system's reaction to cancer cells.

Studies find that the ebb and flow of these cells depends highly on gut bacteria. Germ-free mice — raised in sterile environments with squeaky-clean intestines — are depleted of Th17 cells. But when the mice's insides are populated with certain bacteria, Th17 cells reappear. Furthermore, a study published in *Nature Medicine* in 2009 reported that when mice predisposed to colon cancer were exposed to bacteria that promote Th17 growth, tumor cells grew more aggressively. In short, gut bacteria in the intestine appear to serve as a thermostat for Th17 and T-reg cells.



Don't pass the salt
Healthy volunteers (yellow dots) ate diets with varying levels of salt for about 50 days each. At high salt intake, monocytes, white blood cells that play a role in inflammation, increased. At lower salt levels, the opposite happened. (Black bars are mean values.)

Gut instincts

The good news is that just as a bad diet might promote inflammation, foods that promote healthy microbiota appear to restore order. Microbes that get the food they crave not only maintain normal bacterial ratios, but also produce beneficial fermentation products such as butyrate. (In several studies, butyrate is emerging as a natural compound that tamps down inflammation.)

Other food components also appear to be anti-inflammatory, including omega-3 fatty acids, which are common in fish and nuts. And in one experiment, published in *PLOS ONE* in 2013, Yale's Kleinewietfeld and collaborators used bacteria found in yogurt to reverse the inflammation from a fast food–like diet in mice. The treatment seemed to protect against some of the weight gain such food encourages. Other work by some of the same researchers (see sidebar, Page 19) also raises the possibility that disease risk from microbiota can cross generations.

In any case, it is clear that diet can affect gut microbiota, says cancer biologist Susan Erdman of MIT, senior author of the 2013 study in *PLOS ONE*. "And those changes seem to influence the immune system," she says. "We need a diet that supports the microbes that become an important part of who we are." In other words, choose foods that keep gut bacteria healthy, and odds are, they'll return the favor. ■

Explore more

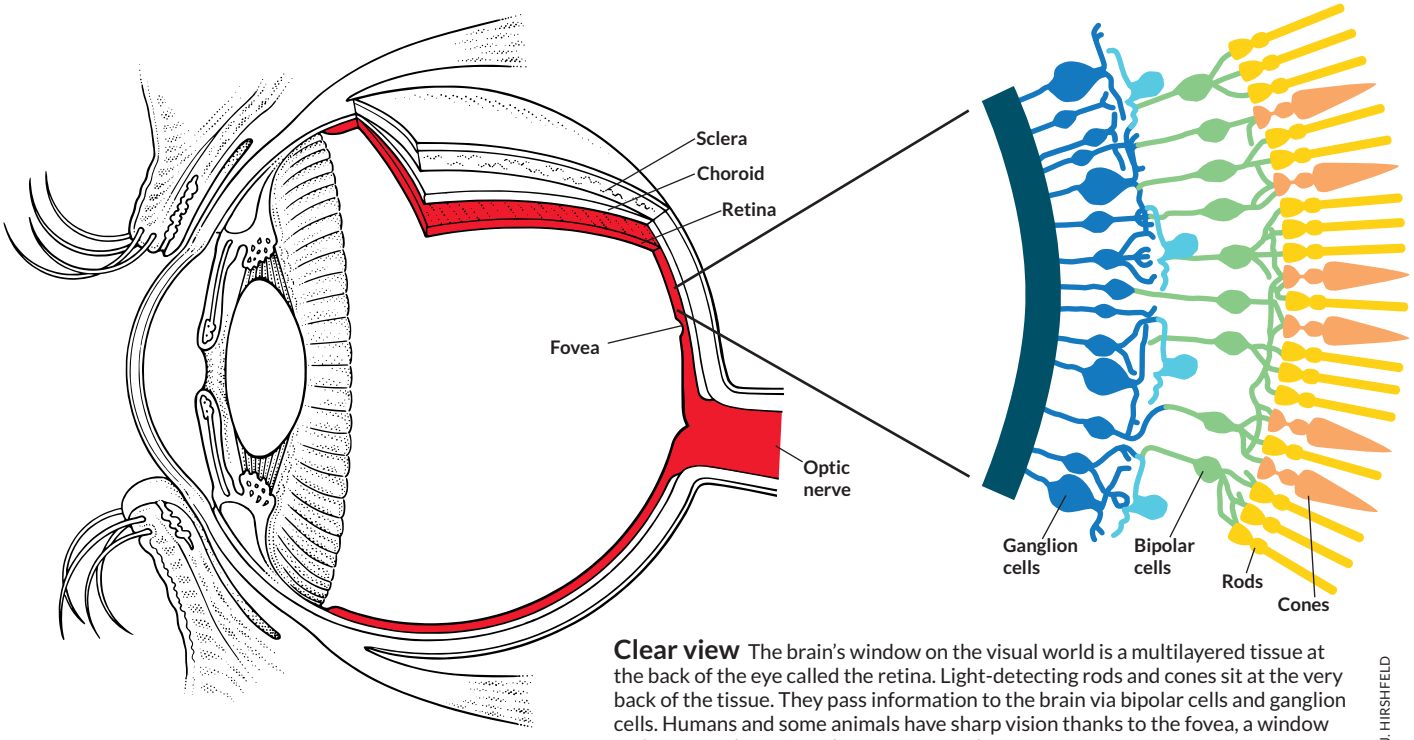
- Silvia Caballero and Eric G. Pamer. "Microbiota-mediated inflammation and antimicrobial defense in the intestine." *Annual Review of Immunolology*. March 2015.

B. YI ET AL/TRANSLATIONAL RESEARCH 2015, ADAPTED BY M. TELFER

SEEING THE LIGHT

Transforming nerve cells into photoreceptors to restore vision

By Tina Hesman Saey



Clear view The brain's window on the visual world is a multilayered tissue at the back of the eye called the retina. Light-detecting rods and cones sit at the very back of the tissue. They pass information to the brain via bipolar cells and ganglion cells. Humans and some animals have sharp vision thanks to the fovea, a window in the retina that offers direct access to the cones.

A man who had been blind for 50 years allowed scientists to insert a tiny electrical probe into his eye.

The man's eyesight had been destroyed and the photoreceptors, or light-gathering cells, at the back of his eye no longer worked. Those cells, known as rods and cones, are the basis of human vision. Without them, the world becomes gray and formless, though not completely black. The probe aimed for a different set of cells in the retina, the ganglion cells, which, along with the nearby bipolar cells, ferry visual information from the rods and cones to the brain.

No one knew whether those information-relaying cells still functioned when the rods and cones were out of service. As the scientists sent pulses of electricity to the ganglion cells, the man described seeing a small, faint candle flickering in the distance. That dim beacon was a sign that the ganglion cells could still send messages to the brain for translation into images.

That 1990s experiment and others like it sparked a new vision for researcher Zhuo-Hua Pan of Wayne State University in Detroit. He and his colleague Alexander Dizhoor wondered if, instead of tickling the cells with electricity, scientists could transform them to sense light and do what rods and cones no longer could.

The approach is part of a revolutionary new field called optogenetics. Optogeneticists use molecules from algae or other microorganisms that respond to light or create new molecules to do the same, and insert them into nerve cells that are normally impervious to light. By shining light of certain wavelengths on the molecules, researchers can control the activity of the nerve cells.

Optogenetics is a powerful tool for probing the inner workings of the brain (*SN: 1/30/10, p. 18*). In mice, researchers have used optogenetics to study feeding behavior (*SN: 3/7/15, p. 8*), map aggression

EYE: TONY GRAHAM/GETTY IMAGES, ADAPTED BY J. HIRSHFELD; WEBVISION.MED.UTAH.EDU, ADAPTED BY J. HIRSHFELD

circuits (*SN: 3/21/15, p. 18*) and even alter memories (*SN: 10/4/14, p. 6; SN: 8/24/13, p. 18*).

After years of work with animals, researchers are now poised to insert optogenetic molecules into the retinal cells of people. The aim is to restore vision in those whose rods and cones don't work.

"It makes sense that the organ that is light sensitive would benefit from [optogenetics] first," says José-Alain Sahel, director of the Vision Institute in Paris. He is involved in one of two efforts to bring optogenetics out of the lab and into the eye clinic.

Studies in people could begin next year.

Circumvent the damage

Optogenetics is, at its heart, a gene therapy. Traditional gene therapy places a healthy copy of a mutated or damaged gene into the cells of a person with an inherited condition. The healthy copy is first packed into a virus. The virus delivers the gene to the "broken" cells and unloads its cargo. Once inside the cell, the gene produces functional copies of the proteins that the original mutations damaged, and the cell starts working again.

This type of gene therapy has famously been used to treat children born with faulty immune systems (*SN: 8/10/13, p. 19*). It has also restored some vision in people with a rare type of inherited blindness called Leber congenital amaurosis (*SN: 5/24/08, p. 8;* see sidebar, Page 25).

That type of blindness, however, is the absolute best-case scenario for gene therapy, says neuroscientist Botond Roska. LCA patients eligible for gene therapy still have light-gathering rods and cones in their retinas but the cells don't work properly because they have a mutation in a gene called *RPE65* (one of a dozen gene mutations that can cause LCA). Introducing the normal version of the gene allows the rods and cones to function again. However, two studies published online this month in the *New England Journal of Medicine* suggest that even in patients who experience vision improvements after gene therapy for LCA, the photoreceptors continue to die and vision deteriorates over time (*SN Online: 5/3/15*). This could mean that, for long-term benefit, another approach is needed.

Most people with inherited blindness don't even have the hope of temporary restoration. Mutations in any of more than 250 genes may lead to blindness, says John Flannery, a cell and molecular biologist at the University of California, Berkeley. Gene therapy is currently impractical or impossible for most of those diseases, he says.

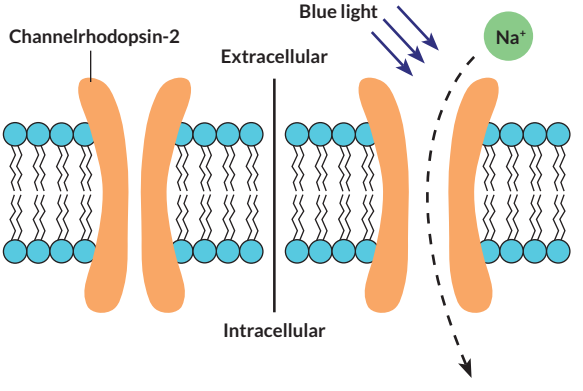
Approximately 200,000 people in the United States have inherited retinal diseases that affect the rods and cones, according to estimates from the Foundation Fighting Blindness. Once those photoreceptors are gone, there's no bringing them back, says Roska, of the Friedrich Miescher Institute for Biomedical Research in Basel, Switzerland.

The optogenetics approach that Pan and others are studying circumvents the missing photoreceptors. That means it differs from traditional gene therapy in important ways: It doesn't fix broken genes, so the therapy should work regardless of which of the 250 genes are causing problems. And instead of trying to resurrect dead or damaged photoreceptors, the scientists aim to transform relay cells into ersatz photoreceptors.

Pan and Dizhoor began kicking around the idea of making bipolar and ganglion cells light sensitive in 2000. In principle it sounded simple: Just move the rods' light-sensing protein, known as rhodopsin, to the other cells. But rhodopsin doesn't work alone. It is part of a light-driven machine in the eye that has more than a dozen parts, says Dizhoor, a molecular biologist now at Salus University in Elkins Park, Pa. "Technically, it's just unfeasible" to move that many cogs, he says. The researchers needed a simple molecule that could make ganglion and bipolar cells sensitive to light.

The breakthrough came two years later when scientists discovered a light-responsive protein called channelrhodopsin in a single-celled algae called *Chlamydomonas reinhardtii*.

Channelrhodopsins form channels in a cell's outer membrane. When certain wavelengths of light hit the protein, the channel opens and lets positively charged ions flow into the cell. That flow of energy is a nerve cell's signal to talk to its neighbors and to the brain. Pan and Dizhoor



Open sesame Optogenetics involves inserting an algae protein into a nerve cell's membrane. In the dark, the protein, called channelrhodopsin-2, or ChR2, is closed. When blue light hits it, the channel opens and allows ions to flow in. That triggers the cell to send a signal to other cells.

JOHN MARTIN BARRETT *ET AL/VISUAL NEUROSCIENCE* 2014, (CC-BY-NC-SA 3.0), ADAPTED BY J. HIRSHFELD

immediately recognized its potential.

"We thought, 'Wow! This is the molecule we've been waiting for,'" Pan says.

They lost little time packing a gene encoding a specific channelrhodopsin, ChR2, into a virus that could infect ganglion cells in blind mice. The researchers reported in *Neuron* in 2006 that the protein could make the cells light sensitive and send a message to the brain in response to blue light shone into the eyes of the mice (*SN: 4/8/06, p. 211*).

A gaggle of ganglion cells

The experiment was just the first step toward restoring vision, though. Researchers have had to wrangle with the issue of which of the cells — ganglion or bipolar — might restore the most vision. Each type of cell has its pros and cons.

To understand the dilemma requires some clarity on how the eye works. Light enters the eye through the pupil and is focused on the retina, a thin, multilayer tissue in the back of the eye.

Light first encounters the retinal ganglion cells. These nerve cells have long tails that bundle together to form the optic nerve and send messages to the brain about what the eye detects. They aren't normally light sensitive. Neither are the bipolar cells, the next layer of cells that light hits. Below both these layers, at the very back of the eye, are the light-detecting rods and cones. Bipolar cells collect light information from these photoreceptor cells and pass it to the ganglion cells, which send it on to the visual processing areas in the brain. Unlike mouse eyes, human eyes have a tiny window called the fovea where bipolar cells and ganglion cells sit off to the side, allowing light to shine directly on the photoreceptors.

The ganglion cells are easiest to reach, which makes them appealing for optogenetics. But human eyes contain about 20 different types of retinal ganglion cells, each of which may convey slightly different visual information to the brain.

Variety may spice up life, but it's potentially the main strike against ganglion cells as a target for optogenetics. That's because the viruses used to ferry optogenetic molecules cannot distinguish between the various ganglion cells. Optogeneticists and gene therapists favor viruses called adeno-associated viruses for delivering their cargo. The viruses come in a variety of packages that determine which types of cells they can infect, but no one has devised a package that will dock only with particular ganglion cell types.

The problem, then, is that optogenetic proteins could be made, and activated, in all 20 ganglion-cell varieties at the same time, including ones that send contradictory information to the brain, says Sahel, in Paris. "It's like saying yes and no to the same thing," he says.

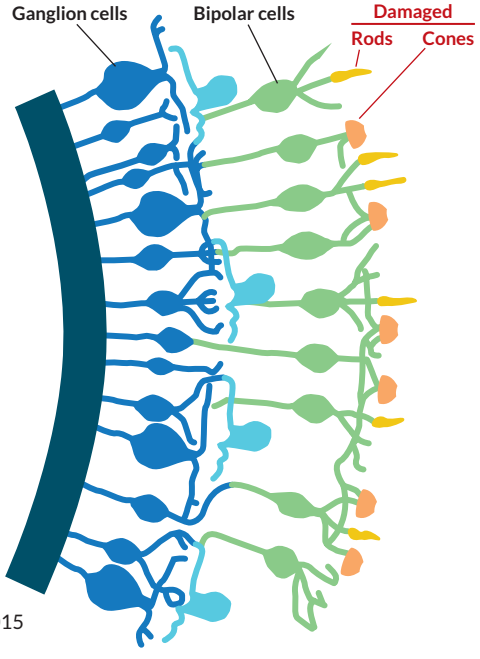
Bipolar expedition

Dizhoor has always thought the bipolar cells were the way to go. After all, they are the natural middlemen between the photoreceptors and the ganglion cells. If their connections with the ganglion cells still hold in degenerated retinas, activating the bipolar cells, which come in two major varieties, should give a less noisy picture of the world than 20 types of ganglion cells chattering at once.

Bipolar cells are described as either ON or OFF. ON bipolar cells are activated when light levels increase, like when you switch on a lamp in a dark room or walk outside into bright sunlight. OFF bipolar cells get excited when light levels decrease. In 2008, Roska and his colleagues put ChR2 into ON bipolar cells in blind mice, enabling them to see patterns about half as well as mice with normal sight (*SN: 5/24/08, p. 8*).

So far, researchers haven't demonstrated that targeting bipolar cells paints a clearer picture of the world than targeting ganglion cells does. Plus, bipolar cells are hard to reach. The viruses have to be injected under the retina, risking detaching the fragile tissue.

No matter which cells they target, researchers have gotten so good at using optogenetics to restore vision in blind mice that every experiment is virtually guaranteed to work, Roska says. New researchers in his lab often mistake blind mice that have had optogenetic therapy for normally



Retina redux
Many inherited blindness conditions, plus eye diseases such as macular degeneration, cause rods and cones in the retina to die or be stripped of their light-detecting parts. The bipolar cells and ganglion cells that remain are functional long after the photoreceptors have gone, making those cells prime targets for optogenetic therapy.

WEBVISION.MED.UTAH.EDU, ADAPTED BY J. HIRSHFELD

sighted mice. Unfortunately, experience with mice doesn't make moving the technology into humans any easier, he says. "You have to reengineer everything you have."

Sahel, Roska and a few other scientists have teamed up to form a company in Paris called GenSight Biologics. The goal is to develop gene therapy and optogenetic therapy for people with one of the most common genetic forms of blindness, a retina-degrading disease called retinitis pigmentosa, and for people with some rare eye diseases. Both GenSight and RetroSense Therapeutics, headquartered in Ann Arbor, Mich., hope to begin clinical trials by the end of 2016. Though they are using similar approaches, they may not end up targeting the same cells.

GenSight is still weighing its options. RetroSense is aiming its optogenetic therapy at the ganglion cells, largely because those cells are easy to access via injection into the center of the eye.

Retinal ganglion cells also have staying power. In retinitis pigmentosa, the photoreceptors are the first to go. Usually, the rods succumb, then the cones. Later, bipolar cells may also die off. But even "very late in the disease process, the ganglion cells are still there," says RetroSense's chief medical officer, Peter Francis. If the approach works, patients who get optogenetic therapy targeted at their retinal ganglion cells may be able to hold on to their new vision for decades, he says.

A monochromatic response

Which cells are made light sensitive isn't the only factor in determining how well people may be able to see. The optogenetic molecules themselves are important.

The photoreceptors that detect light in human eyes operate over a wide range of intensities from dim starlight to a glaring day at the beach, says Flannery of UC Berkeley. And most people can see a rainbow of colors, thanks to the cones' natural light-harvesting proteins. But the light-activated molecules used in optogenetics are far more limited; they are not as sensitive to different degrees of brightness and they detect only certain wavelengths, and thus colors, of light. "It's a bit like a bike with one gear, compared with a bike with 20 gears," Flannery says. The current molecules, he adds, don't work very well at twilight.

Flannery's collaborator Ehud Isacoff, a UC Berkeley neuroscientist, is among researchers devising light-activated molecules to be more flexible than channelrhodopsin and the other proteins that are borrowed from microbes.

Unafraid of the dark

When Allison Corona and her friends played the card game Uno, they had a rule that green and blue were the same color. The rule was instituted because Corona, 23, has an inherited form of blindness called Leber congenital amaurosis.

The light-gathering cells in her retinas don't work properly due to a mutation in a gene called *RPE65*. As a result, Corona's world was indistinct. During the day, she could see light, but not shadows. She saw colors, but had trouble telling them apart. After sunset the world went dark. Corona was afraid to schedule college classes after 3 p.m. because she couldn't navigate unfamiliar surroundings alone.

But Corona has one of the few conditions doctors can treat with traditional gene therapy. In 2012, she participated in a clinical trial at the University of Pennsylvania in which doctors injected viruses carrying healthy copies of the *RPE65* gene into her eyes. The viruses delivered the gene to retinal cells, where it makes a normal version of the protein that Corona otherwise lacks.

Shortly after the surgery, her vision changed. At first, the light was overwhelming and painful.

"I just sat in the dark," Corona says. "I wore sunglasses everywhere. I looked like a rapper."

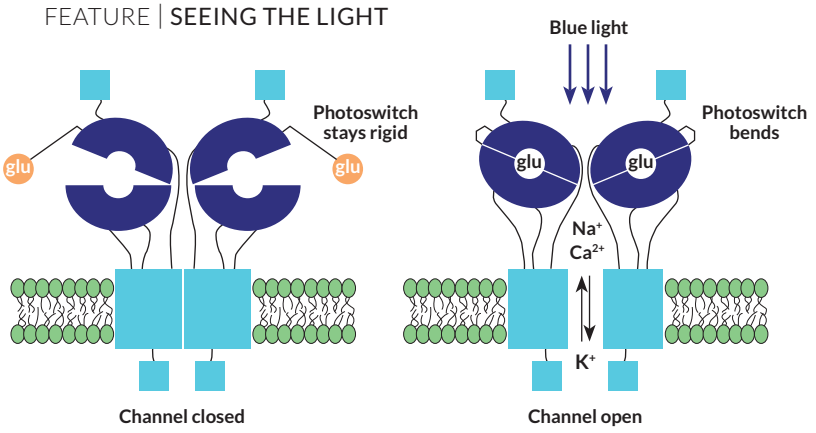
But she adjusted, and her sight improved dramatically, although she is still legally blind.

Today, she plays Uno without the green-equals-blue rule. But she's most excited about having night vision. "Now fear isn't a thing," she says. "I'm able to move around at night. That's a big accomplishment."

Despite recent reports of people with LCA eventually losing their light sensitivity, Jean Bennett, a U Penn molecular geneticist who helped conduct the study that Corona was involved in, says her early data indicate the improvements will last. — *Tina Hesman Saey*

In December, Flannery and collaborators reported in the *Proceedings of the National Academy of Sciences* that they had tested a light-activated version of a protein called an ionotrophic glutamate receptor. In bipolar cells and other cells, when the communication chemical glutamate docks with the receptor, a channel opens and ions flow into the cell. The researchers coupled

FOUNDATION FIGHTING BLINDNESS



Keep away By tweaking a protein involved in nerve cell communication, scientists hope to make a more sensitive photoswitch for optogenetic therapy. In the dark, the engineered protein, a light-gated ionotrophic glutamate receptor, or LiGluR, keeps glutamate out of reach. With light, the photoswitch bends so the protein can grab glutamate and open an ion channel. The cell then signals its neighbors that light exists.

the receptor protein to a molecule called a photoswitch. In the dark, the photoswitch holds glutamate away from its docking port. But when blue light hits the engineered receptor, called LiGluR, the photoswitch allows glutamate to dock and open the channel.

Placing the LiGluR protein in either the retinal ganglion cells or in the ON bipolar cells of blind mice restored the rodents' vision. The mice could see well enough to scurry for the shadows when placed in a brightly lit area. They could also navigate a water maze.

Flannery, Isacoff and colleagues at the University of Pennsylvania also inserted the LiGluR protein into retinal ganglion cells of blind dogs. The cells became responsive to light, but the team is still testing whether the therapy helps the dogs see.

LiGluR has some pros and cons, says Isacoff. It requires a constant supply of the chemical photoswitch, which will probably mean weekly eye injections. On the positive side, patients could receive upgrades as the photoswitch is improved, without needing additional gene therapy. And if optogenetic-powered vision proves to be confusing or uncomfortable for patients, doctors could just stop injecting the chemical. Other optogenetic molecules have no such off switch, Isacoff says.

Because current optogenetic proteins respond only to specific wavelengths of light, people who undergo optogenetic therapy will probably see in shades of gray. Pan and others are tweaking the molecules to extend the range of wavelengths to restore some color vision. That goal is a long way off, Pan says.

Even with improved light-sensing molecules in place, people who get optogenetic therapy will probably need additional help. Special goggles may be needed to boost ambient light levels and tune the wavelengths to optimally activate the proteins.

The brain will probably need some training as well. "We're providing a new language to the retina, and it will take time to learn," Roska says.

RetroSense's Francis isn't worried that patients won't be able to understand what they are seeing. "The brain is extremely good at adapting to visual input and making sense of those images," he says. At least that has been the experience of people who have electrodes implanted on the retinal ganglion cells.

Those early experiments with electrodes that showed the blind man a dim candle eventually evolved into retinal implants. Last year, a company called Second Sight Medical Products of Sylmar, Calif., began selling its "bionic eye" in the United States. The device uses 60 electrodes, each stimulating several ganglion cells in the retina. It's enough to allow blind people to navigate and make out windows and doors. Some say they can see tables in their path and even dishes on the table. It's a big improvement, Sahel says, but it is nowhere near normal vision.

Partial restoration

Optogenetics researchers think they can do better. There are about 1 million ganglion cells and 10 million bipolar cells in the retina. If even 10 percent of those cells can be made light sensitive, that's a potentially huge gain in resolution over the implants. Of course, no one knows if more signal is actually better. It may just increase the noise the brain must decipher.

Even if everything goes swimmingly, patients shouldn't expect to see perfectly, Sahel says. "It won't reach the sophistication of normal vision. It's partial restoration."

The researchers are moving swiftly but cautiously toward trying the therapy in people.

Safety tests in primates look promising so far, but researchers don't have final results and can't move into human trials until they know it is safe. "We want to be first, but we want to be first to do things right," Sahel says. ■

Explore more

- Benjamin M. Gaub *et al.* "Restoration of visual function by expression of a light-gated mammalian ion channel in retinal ganglion cells or ON-bipolar cells." *Proceedings of the National Academy of Sciences.* December 23, 2014.
- Foundation Fighting Blindness: www.blindness.org

STEPHANIE SZOBOTA *ET AL*/*NEURON* 2007, ADAPTED BY J. HIRSHFELD

The Neptune® Bath Lift is the affordable way to regain your independence and safely stay in the home you love!

Now you can safely enter and exit your tub!

For many of us, nothing is more relaxing than a long, luxurious bath. Unfortunately, because of safety concerns, many people, particularly older people, have to forego this simple pleasure. Sure, you can spend big bucks to remodel your bathroom to provide a bathtub you can use, but who wants to do that? Now there's a better way, and it lets you use the bath that's in your home today.

If you or a loved one has given up bathing because of age, mobility or safety concerns, the Neptune® Upright Bath Lift is the answer. It is so simple, you'll wonder why no one thought of it earlier. The battery-operated "chair" safely and steadily lowers you to the bottom of the tub, and when you're done bathing, it gently raises you back to the top of the tub, assuring a comfortable and safe exit from the tub. The remote is fully waterproof, so your bath won't be a "shocking" experience.

The New Neptune® Upright Bath Lift was redesigned making it lighter, more affordable, with more room to bathe—also making it easier to move the lift in and out of the tub. The bath lift features an easy-to-use hand control designed for comfort and safety. If there's not enough power stored for a full lower/raise cycle, it will not operate. The bath lift also features a wide seat and high backrest for maximum comfort and support.

Choose the angle of relaxation you prefer!

Lightweight seat folds flat for easy transport.

Waterproof remote included!

Rediscover the simple pleasure of a warm, relaxing bath with our exclusive home trial. Try the New Neptune® Recliner Bath Lift for up to 30 days, and if you are not completely satisfied, simply return it for a refund of the product purchase price.

Neptune® Recliner Bath Lift

Call now for our lowest price!

Please mention promotional code 100146.

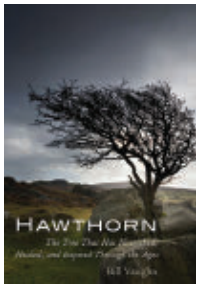
For fastest service, call toll-free 24 hours a day.

1-888-623-2044

52492

BOOKSHELF

The art and science of the hedgerow



Hawthorn
Bill Vaughn
YALE UNIV., $30

It's a testament to doggedness that people through the centuries have extracted any benefit at all from the hawthorn tree. It's a gnarly, unforgiving thing with spikes. It takes real persistence and thick gloves to domesticate. In some places, hawthorns are considered giant weeds. A dense thicket of the bushy trees calls to mind the supernatural hedge that encircled Sleeping Beauty for 100 years.

"A hawthorn is not a tree most people want to hug," writes Bill Vaughn, a writer and graphic artist.

But he shows in *Hawthorn* that outward drawbacks conceal attributes that people have exploited for centuries in iron-forging, weaponry, medicine and above all, the growing of hedgerows. This jack-of-all-trees story makes for a compelling read, spiced with arcane history and Vaughn's own anecdotes.

For starters, readers learn that hawthorn wood is extremely hard and dense. Hawthorn burns hotter than oak or beech and provides ideal charcoal for blacksmith forges — and sword making. Hawthorn branches also snap back into position after flexing, thanks to the wood's fibrous nature. Eastern Native Americans prized it for bows.

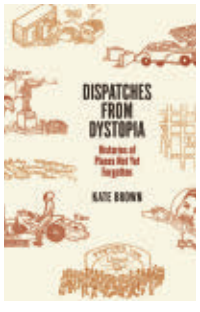
There are many species of hawthorn, all in the genus *Crataegus*. Its fruit, the haw, can range from tasty to arsenic-laden. Native Americans found medicinal value in the haw and other parts of the tree, and studies suggest haw extract can protect against heart failure. Chinese medicine recommends it for digestive ailments.

But hedging has been the crowning achievement of hawthorns. Growing an impenetrable hedge requires skill and patience. Hawthorns are planted and when half-grown, they are "pleached" — gashed partway across the trunk and bent over, inducing them to become a thicket. With other standing trees, hawthorns form the thorny rebar of a living fence, keeping livestock in and predators out.

Hawthorn hedgerows still grow in the green pastures of Normandy and England. The French hedgerows were so high and dense that Allies invading in 1944 needed to weld sharp steel blades on tanks to pierce the barriers.

A century earlier, hedgerow science that had focused on European

---



Dispatches from Dystopia
Kate Brown
UNIV. OF CHICAGO, $25

BOOKSHELF

Histories left behind by the dispossessed

Historians spend lots of time in archives and libraries. But documents often reveal little about people who lived on the margins of society or whose stories are intentionally obscured.

To uncover these people's stories, historian and self-described "professional disaster tourist" Kate Brown ventured into a variety of wastelands. *Dispatches from Dystopia* compellingly chronicles people who were or are living on the edge. For instance, while researching the region surrounding Russia's first plutonium plant, she interviewed people who had once worked inside the remote and secretive facilities. Unsurprisingly, some were tight-lipped. They were far more interested in discussing their myriad medical problems (probably caused by long-term radiation exposure, though the government disputes that) than in talking about the details of the work they had performed.

Brown also went to Chernobyl. Inside the Luxembourg–sized hot zone around the nuclear plant, she had hoped to gain insight into everyday lives. She wanted to sift through belongings that residents had hastily abandoned when they fled the catastrophic release of radiation in 1986. Instead, she found cottages and apartments that had largely been stripped of anything of informational or economic value; whether taken by residents or subsequently pilfered by looters, even the knobs on kitchen cabinets were missing.

In an exceptionally interesting chapter, Brown compares two cities: Billings, Mont. (a town built by railroad entrepreneurs, farmers and miners in the 1880s) and Karaganda, Kazakhstan (a city founded near a Soviet-era prison camp in the 1930s). Because early settlement of these towns was largely driven by distant bureaucracies, the two were laid out on similar rectangular grids despite vast differences between the two home nations' ideas, politics and economic structures. Settlers in both places starved, froze and worked until they dropped from exhaustion; one big difference, Brown notes, is that the unfortunates in Billings had moved there of their own free will.

Many of the tales Brown relates took place in Eastern Europe and central Asia, where she lived. But one of Brown's most poignant investigations unfolded in the United States, in the smallest venue she visited: the basement of Seattle's Panama Hotel. There, a padlocked storeroom held boxes and trunks full of personal belongings abandoned in 1942 by Japanese families who had stayed there on their way to an internment camp in Idaho.

Compiled from fieldwork conducted over a career, *Dispatches from Dystopia* contains powerful tales of the invisible and the overlooked, the exiled and the dispossessed. *— Sid Perkins*

hawthorns had found its way to the Great Plains of North America. Farming there had stalled because settlers lacked access to wood to fence off crops from livestock that would eat them. Vaughn, in one of many readable tangents, reveals that the best hedgerow plant for the Plains wasn't a hawthorn but rather the native Osage orange, a thorny tree that rarely rots and is immune to termites. By 1895, Kansas alone had 72,000 miles of hedgerow.

But the adoption of barbed wire in the late 1800s doomed hedgerows in the Great Plains. The trees died, as did their ability to protect against the wind. Vaughn doesn't say whether Dust Bowl farmers rued their missing hedgerows years later, when winds blew away their topsoil. — *Nathan Seppa*

Buy Books Reviews on the *Science News* website include Amazon.com links that generate funds for Society for Science & the Public programs.

EXPERIENCES

A chemistry card game forges bonds

The new strategy-based card game Ion makes chemistry a diversion. It challenges players to group positively and negatively charged ions to form compounds. Players pass around ion and noble gas cards to win points by building the compounds and collecting sets of noble gases.

New to chemistry? Never fear. The card game relies on basic arithmetic of positive and negative charges to create neutral compounds.

The designs on each card have sleek depictions of atoms' protons, neutrons and electrons. Descriptions teach players to identify elements and compounds in their daily lives. The constant card passing among players adds strategy, as each person tries to boost their point count and foil other players. Action tiles help each player increase their point-earning compounds — and steal from their neighbors.

The chemistry card game Ion pits players against each other to make compounds.

Ion was designed by John Coveyou of Genius Games, which also sells the amusing DNA transcription game Linkage (*SN: 12/27/14, p. 32*). Ion can be ordered online at bit.ly/Iongame. — *Bethany Brookshire*

FEEDBACK

APRIL 18, 2015

Join the conversation

E-MAIL editors@sciencenews.org

MAIL Attn: Feedback
1719 N St., NW
Washington, DC 20036

Connect with us

The bipedal Butcher

In "Fearsome croc dominated northern Pangaea" (SN: 4/18/15, p. 16)*,* ***Ashley Yeager*** *introduced* Carnufex carolinensis*, also known as the Carolina Butcher. This ancestor of crocodiles reigned as a top predator more than 200 million years ago in what later became North America.* Some readers questioned the illustration that accompanied the story, showing the ancient reptile balancing on its hind limbs. "It looks like it's gonna fall flat on its face from the weight of its own head," said commenter **johnGPL**. Reader **Mark S**. asked, "If all they found was the skull, spine and arms, how do they know it was bipedal?"

According to **Lindsay Zanno**, a paleontologist at the North Carolina Museum of Natural Sciences and a coauthor on the study, researchers aren't yet sure whether this species got around on two legs. Some of the Carolina Butcher's close relatives probably walked on their rear limbs while others sprawled on all fours, she says. "We don't have the hind limbs of *Carnufex carolinensis*, but we do have a long skull and short upper arm, so we decided to make this animal rearing up on its hind legs in the illustration."

Nanoparticles washed away

Silver nanoparticles embedded in clothing can guard against infectious bacteria, but they're no match for the washing machine. Some detergents break down these protective particles, as ***Beth Mole*** *reported in "Suds turn silver nanoparticles in clothes into duds"* (SN: 4/18/15, p. 9)*.*
"I suspect that the silver nanofabrics got rushed into production," mused **John Turner**. "When you search the publication servers you find the basic science for making nanosilver fabric was still under way less than eight years ago. Eight years apparently wasn't enough time to test their idea against real-world scenarios like 'I always wash my gym duds with two scoops of OxiClean.'"

Correction

"Stinkin' rich" (*SN: 5/2/15, p. 5*) should have said the approximate value of elements in a metric ton of treated sewage sludge was $500, not $500 million.

JOHN COVEYOU/GENIUS GAMES

ALUMNI

Broadcom MASTERS on 'The Tonight Show'

Sahar Khashayar, winner of the Marconi/Samueli Award for Innovation at the 2014 Broadcom MASTERS, appeared on the April 24 episode of *The Tonight Show Starring Jimmy Fallon* to demonstrate her wildfire detection device during his "Fallonventions" segment. Khashayar's sensor package scouts for three things: heat, smoke and infrared radiation, all warning signs of a wildfire. When one of those signs is detected, her invention sends a message to a smartphone. Her device costs about $60 to build. She believes deploying a network of such fire detectors, especially in areas battling drought, could help prevent or reduce the loss of life and property.

Broadcom MASTERS is a national competition for sixth- to eighth-grade students designed to inspire and encourage the nation's young scientists, engineers and innovators. The 2015 Broadcom MASTERS finals will be held in Silicon Valley in October to celebrate the fifth anniversary of the program.

Watch Khashayar explain how her project works on *The Tonight Show* at **bit.ly/SSP_TonightShow**

PUBLICATIONS

Stories for students now scored for readability

Teachers frequently mention how much they love *Science News*' sibling publication, the online magazine *Science News for Students*. But many have also asked for a way to see at a glance how challenging particular stories might be for their students.

So this year, *SNS* editor Janet Raloff began displaying "readability scores" at the bottom of each *SNS* article and on every post of its companion blog, *Eureka! Lab*. And now, readers can search and filter stories according to readability levels.

For the last two years, *SNS* has published stories at, and usually well below, a ninth-grade reading level. Now the stories display readability scores as a tool for teachers.

"There are several ways to test how accessible the text of a story is for students," Raloff says. "We use the Flesch-Kincaid algorithm. The number it spits out is nominally the minimum average U.S. grade level that should find the story a comfortable read."

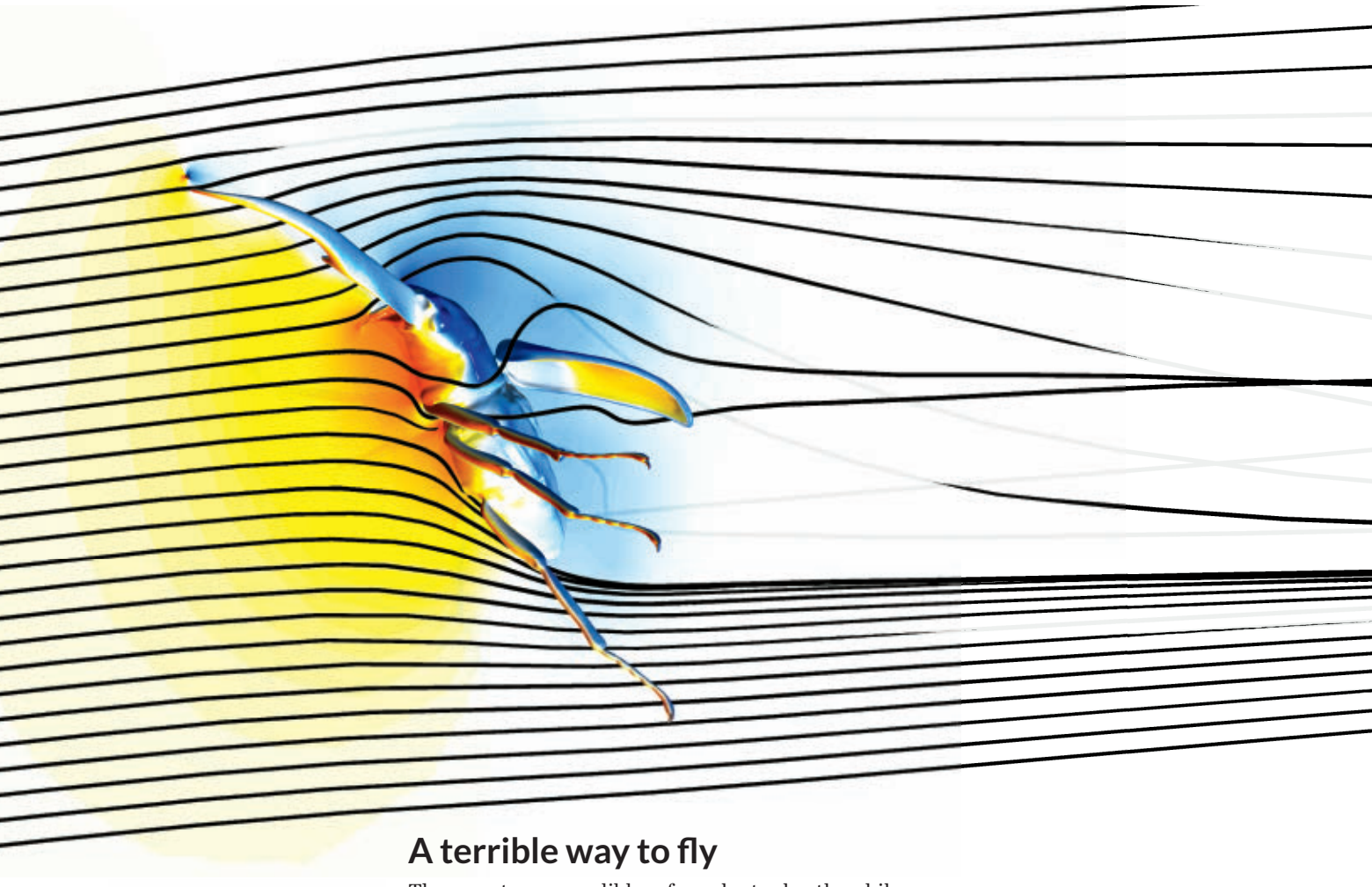
In other words, a score of 6.0 means a story should be fine for at least a sixth-grader. Yet the story's language won't insult anyone older — even an adult. A score of 7.4 (the site's average) is great for kids midway through seventh grade. The very rare story that hits a 9.0 may be somewhat more of a stretch for middle-schoolers, but still accessible.

Clearly, long names, scientific terms and researcher affiliations can conspire to bring scores up. But Raloff says that *SNS* will not forgo this information for the sake of a lower readability score. Instead, sentence structure and length are tweaked and "Power Words" are included to help students parse any research-related terms.

"This allows us to bring even young teens up-to-date developments in everything from zoology and genetics to plate tectonics and quantum physics," Raloff says.

Read *Science News for Students* at **www.sciencenewsforstudents.org**

COURTESY OF S. KHASHAYAR



A terrible way to fly

The monstrous mandibles of a male stag beetle, while useful for mating, come back to bite him during transit.

Flying and running take considerably more work for a male *Cyclommatus metallifer* (left) than they would in a short-jawed world, computer simulations reveal. Saddled with the weight of his giant mouthparts, the beetle flies in an awkward, almost vertical position. The male gets hardly any lift from air pushing against his front (above, orange), and he must overcome diminished air pressure (blue) that sucks him backward. But the sorry aerodynamics have a lower energy cost to the beetle than does lugging his hefty mandible muscles, which account for about 18 percent of his body weight.

A male *C. metallifer* grows some of the most elongated mandibles among beetles. He needs them to fend off other males pursuing, of course, a female. Biophysicist Jana Goyens of the University of Antwerp in Belgium and colleagues used simulations based on scans of real beetles to quantify the energy cost of the mouthparts.

Reporting in the May 6 *Journal of the Royal Society Interface*, the researchers found that males use 26 percent more energy to fly and 40 percent more energy to run than they would with small, female-style mandibles.
—Susan Milius

FROM TOP: J. GOYENS *ET AL./JOURNAL OF THE ROYAL SOCIETY INTERFACE* 2015; UDO SCHMIDT/WIKIMEDIA (CC BY-SA 2.0)

Watch stag beetles fight and run at **bit.ly/stagbeetles**

"The brilliance of the sterling silver setting pairs nicely with the superior fire of the DiamondAura® in the Stauer 5-Stone Ring"
— James T. Fent, Stauer GIA Graduate Gemologist

Receive these scintillating DiamondAura sterling silver stud earrings **FREE**! *Read details below.*

The Fifth C?

Cut, Color, Carat, Clarity... Chemistry?

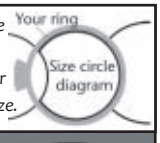
Is it possible that the mind of a scientist can create more beauty and romance than Mother Nature? The laboratories at DiamondAura® were created with one mission in mind: Create brilliant cut jewelry that allows everyone to experience more clarity, more scintillation and larger carat weights than they have ever experienced. So, we've taken 4 carats of our lab-created DiamondAura® and set them in the most classic setting —the result is our most stunning, fiery, faceted design yet! In purely scientific measurement terms, the refractory index of the DiamondAura is very high, and the color dispersion is *actually superior* to mined diamonds.

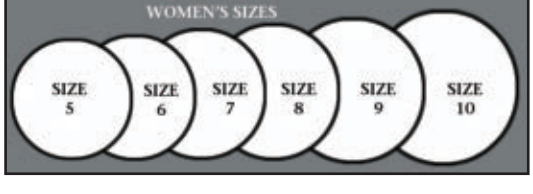
Perfection from the laboratory. We named our brilliant cut stones DiamondAura, because, "they dazzle just like natural diamonds but without the outrageous cost." We will not bore you with the incredible details of the scientific process, but will only say that it involves the use of rare minerals heated to an incredibly high temperature of nearly 5000°F. This can only be accomplished inside some very modern and expensive laboratory equipment. After several additional steps, scientists finally created a clear marvel that looks even better than the vast majority of mined diamonds. According to the book *Jewelry and Gems–the Buying Guide*, the technique used in DiamondAura offers, "The best diamond simulation to date, and even some jewelers have mistaken these stones for mined diamonds."

Compare For Yourself at 4 Total Carats

	Mined Flawless Diamond	DiamondAura® Compares to:
Hardness	Cuts Glass	Cuts Glass
Cut (58 facets)	Brilliant	Brilliant
Color	"D" Colorless	"D" Colorless
Clarity	"IF"	Clear
Dispersion/Fire	0.044	0.066
4 ctw ring	$120,000+	$87.50

Place one of your own rings on top of one of the circle diagrams. Your ring size is the circle that matches the inside diameter of your ring. If your ring falls between sizes, order the next larger size.





The 4 C's. Our DiamondAura **Quinto 5-Stone Ring** retains every jeweler's specification: color, clarity, cut, and carat weight. The transparent color and clarity of DiamondAura emulate the most perfect diamonds—D Flawless, and both are so hard they will cut glass. The brilliant cut maximizes the fire and radiance of the stone so that the light disperses into an exquisite rainbow of colors.

Rock solid guarantee. This .925 sterling silver ring is prong-set with a 1½ carat DiamondAura oval brilliant in the center, showcased between four DiamondAura oval brilliants totaling 2½ carats. Adding to your 4 C's, we will include the DiamondAura stud earrings for **FREE!** Try the DiamondAura **Quinto 5-Stone Ring** for 60 days. If for any reason you are not satisfied with your purchase, simply return it to us for a full refund of the purchase price and keep the stud earrings as our gift.

Not Available in Stores

DiamondAura® Quinto 5-Stone Ring (4 ctw)
Offer Code Price **$87.50** + S&P

FREE stud earrings with purchase of the Quinto 5-Stone Ring—a $99 value!

Available in ring sizes 5-10

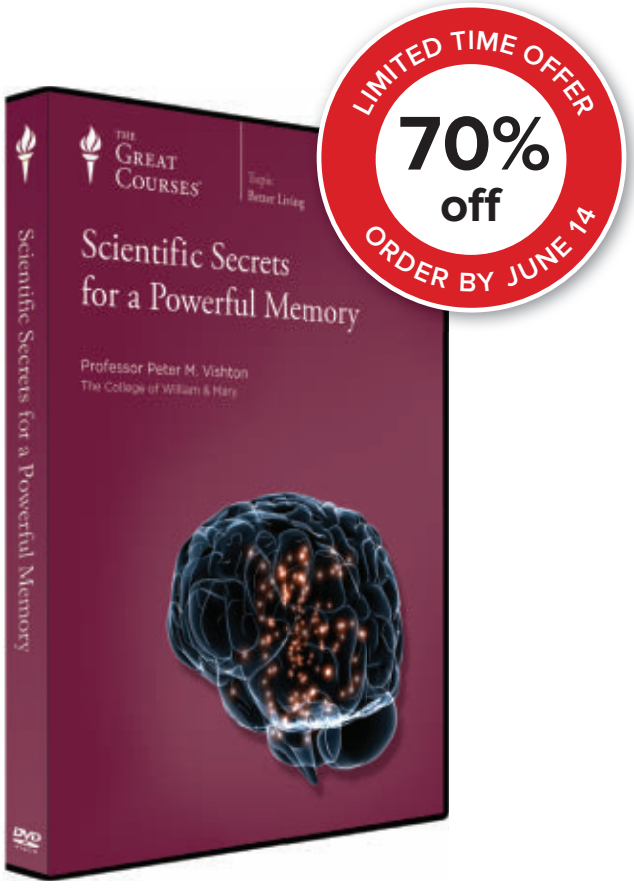
1-800-905-4635

Your Insider Offer Code: DRR190-01
Please use this code when you order to receive your free stud earrings.

Stauer®
14101 Southcross Drive W., Dept. DRR190-01, Burnsville, Minnesota 55337
www.stauer.com

BBB ACCREDITED BUSINESS
Rating of A+

THE GREAT COURSES®



Unlock Your Memory's Untapped Potential

While all of us have an amazing capacity for memory, there are plenty of times when it seems to fail us. Why does this happen? And how can you fix it? **Scientific Secrets for a Powerful Memory** explores the real research on how memory functions, then applies these findings to help you make the best possible use of the capabilities you have.

In just six lectures, award-winning Professor Peter M. Vishton of The College of William & Mary shows you how to remember birthdays and key numbers using the Major System; how to improve how you study for—and perform on—exams; and so much more. By tapping into these scientifically proven strategies, tricks, and techniques, you'll develop the ability to process information more effectively and remember virtually anything you want.

Offer expires 06/14/15

THEGREATCOURSES.COM/6SN

1-800-832-2412

Scientific Secrets for a Powerful Memory

Taught by Professor Peter M. Vishton
THE COLLEGE OF WILLIAM & MARY

LECTURE TITLES

1. Your Amazing Prehistoric Memory
2. Encoding Information with Images
3. Maximizing Short- and Long-Term Memory
4. Why and When We Forget
5. Keeping Your Whole Brain in Peak Condition
6. Human Memory Is Reconstruction, Not Replay

ABOUT YOUR PROFESSOR

Dr. Peter M. Vishton is Associate Professor of Psychology at The College of William & Mary. He taught at Northwestern University and served as the program director for Developmental and Learning Sciences at the National Science Foundation. In 2012, Professor Vishton was named one of the best 300 professors in America by The Princeton Review.

"This is truly a course that should cost much more. Solid information that has application in the real world. Highly recommended."

—J. Candlemann, East Brunswick, NJ

Scientific Secrets for a Powerful Memory
Course no. 1965 | 6 lectures (30 minutes/lecture)

SAVE $70

DVD ~~$99.95~~ NOW $29.95
+$5 Shipping, Processing, and Lifetime Satisfaction Guarantee
Priority Code: 108518

For 25 years, The Great Courses has brought the world's foremost educators to millions who want to go deeper into the subjects that matter most. No exams. No homework. Just a world of knowledge available anytime, anywhere. Download or stream to your laptop or PC, or use our free mobile apps for iPad, iPhone, or Android. Over 500 courses available at www.TheGreatCourses.com.

Special Editions Now Available on verizon FiOS