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DECEMBER 9, 2017

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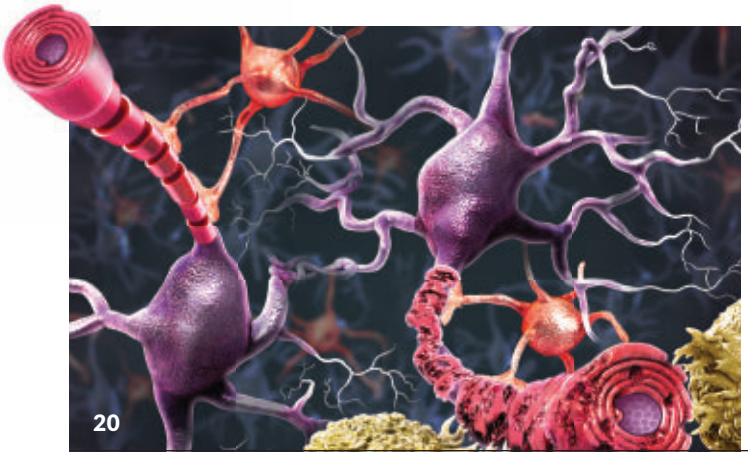
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COVER On Canada's Ellesmere Island (shown), researchers dig up sediments to study warming trends. *Wayne Lynch/Alamy Stock Photo*

Would you opt to see the future or decipher the past?

Wouldn't it be brilliant if every scientist had a crystal ball? It's a question that came to me while reading Alexandra Witze's story "Lessons from the Pliocene," beginning on Page 24. Witze discusses how scientists are studying a warming period some 3 million years ago to try to understand how Earth will handle rising temperatures. The geologic epoch, known as the Pliocene, isn't a perfect crystal ball, she notes. But paleoceanographer Heather Ford says, "It's our closest analog for future climate change."

A true crystal ball could answer many climate change questions: Which cities might be underwater in the future? Which regions will be suitable for farming, and which will become desert? Which diseases should we watch for? More generally, we could all find out just how bad extreme heat and weather might be. Perhaps such insights would give us the kick we need to change our current behavior and appropriately plan for a dramatically different world.

With such science-focused clairvoyance, we could also improve our lives in other ways: Those treatments we hope to find for multiple sclerosis (Page 20), we could find them today instead. We could predict problems with infrastructure, including the potential for swaying bridges (Page 14) or worse, bridge collapse. And we could explore when it would be smart to use gene drives, if ever, and when they'd be a colossal mistake (Page 16).

It would be enticing to peek into the future, but much of the past is also obscured. Wouldn't it be neat to know when human ancestors first started making stone tools (Page 7) or to study past pandemics and their sources with crystal-clear clarity (Page 12)? I'd be fascinated to watch how water striders (Page 4) or leafhoppers (Page 5) evolved.

In an informal poll, I asked *Science News* staffers if they'd prefer to see into the past or the future. Life sciences writer Susan Milius, among the first to reply, said exquisite vision that worked only backward would drive her crazy. "Plain old 20/20 hindsight is annoying enough," she said. A couple of other writers voted for the future too, so they could scoop competitors on stories. And assistant art director Chang Won Chang wanted to know how technology will change how humans live.

But most people would choose to look back instead of forward. "The origins of life and the origins of the universe are more intriguing questions to me than their respective ends," said audience engagement editor Mike Denison. Associate editor Emily DeMarco thought that knowing the future could hinder creativity, and design director Erin Otwell said thinking about the future comes with too much worry. Added astronomy writer Lisa Grossman, "I don't think I would trust anything I saw in the future." Then she circled back to a sentiment similar to that expressed by the scientists studying the Pliocene. "Seeing the past might help me understand the present," Grossman said, "which would help me prepare for the future."

And what about our readers? What scientific answers would you seek if you had a crystal ball? Let us know via e-mail at editors@sciencenews.org.

— Elizabeth Quill, Acting Editor in Chief

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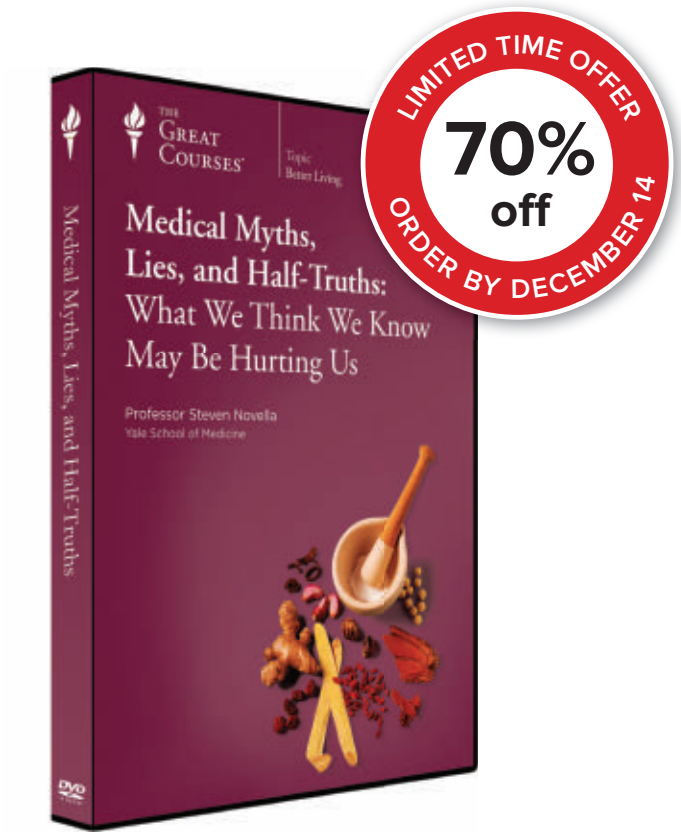

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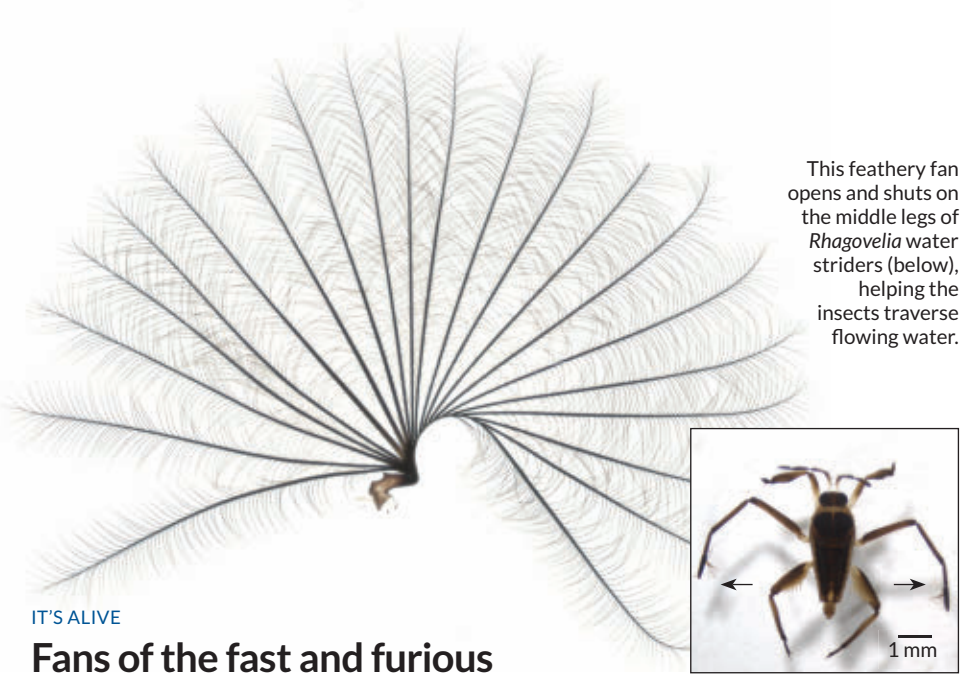
Excerpt from the December 9, 1967 issue of *Science News*

50 YEARS AGO

Folic acid

Pregnant women who do not have enough folic acid — a B vitamin — in their bodies can pass the deficiency on to their unborn children. It may lead to retarded growth and congenital malformation, according to Dr. A. Leonard Luhby.... "Folic acid deficiency in pregnant women could well constitute a public health problem of dimensions we have not originally recognized," he says.

UPDATE: Folic acid — or folate — can prevent brain and spinal cord defects in developing fetuses. Since the U.S. Food and Drug Administration required that all enriched grain products contain the vitamin starting in 1998, birth defects have been prevented in about 1,300 babies each year. But some women still don't get enough folate, while others may be overdoing it. About 10 percent of women may ingest more than the upper limit of 1,000 micrograms daily — about 2.5 times the recommended amount, a 2011 study found. Too much folate may increase a woman's risk for certain cancers and interfere with some epilepsy drugs.



This feathery fan opens and shuts on the middle legs of *Rhagovelia* water striders (below), helping the insects traverse flowing water.

IT'S ALIVE

Fans of the fast and furious

For an animal already amazing enough to walk on water, what could growing feather fans on its legs possibly add?

These fans preoccupy Abderrahman Khila, who keeps some 30 species of bugs called water striders walking the tanks in his lab at the University of Lyon in France.

"Walk" may be too humdrum a word. The 2,200 or so known species of water striders worldwide can zip, skim, skate and streak, without getting their long legs wet. Among such damp-defying acrobats, however, only the *Rhagovelia* genus grows fans of delicate plumes on the middle pair of its six legs. Even little hatchlings pushing their way out of underwater eggs have a pair of feathery microfluffs ready for cruising at the water's surface.

A first guess at a function — maybe plumes help support bigger adults — would be wrong, Khila says. *Rhagovelia* is not a giant among water striders. In a jar of alcohol in his lab, Khila treasures a specimen of a much bigger species, with a body the length of a peanut shell and a leg span that can straddle a dinner plate. Yet this King Kong of striders, found in Vietnam and China, slides over the water as other species do, cushioned by air trapped in dense hydrophobic leg bristles. No froufrou feathers needed.

The insects don't use the fans for their action-packed, often violent interactions either. "In the lab, they eat each other all the time," Khila says. A newly molted strider, still soft and weak after wriggling out of its old external skeleton, can get mobbed by cannibals. Any other insect, such as a mosquito, landing on the water surface also triggers a frenzy. Small striders "start to attack the legs of the mosquito," Khila says, "and seconds later there are 50 water striders gathered around." With their tubelike mouthparts, the striders stab holes in the victim and inject enzymes to liquefy flesh into a meat shake to suck out.

For *Rhagovelia*, Khila sees the fans as a "key evolutionary innovation," a trait that pops up in evolutionary history with no clear line of precursor forms.

Now he and colleagues have identified a fan benefit. When they removed plumes from the bugs or suppressed genes needed for fan formation, the striders couldn't turn as deftly or run upstream against the current as well as fully fanned *Rhagovelia* can, the researchers report in the Oct. 20 *Science*. Striders in a closely related but fanless genus were likewise hampered. The innovative fan opened new territory, helping the insects navigate flowing water, the researchers conclude.

Fan-maker genes are intriguing in another way. Evolutionary biologists have long debated whether such evolutionary innovations just repurpose and recombine old developmental genes or actually rely on new ones. In the case of the fans, two genes are unique to this genus, but three others are repurposed. So in a twist on an old debate, Khila says, "neither hypothesis is wrong." — *Susan Milius*

FROM LEFT: A. KHILA; ANTONIN CRUMIERE

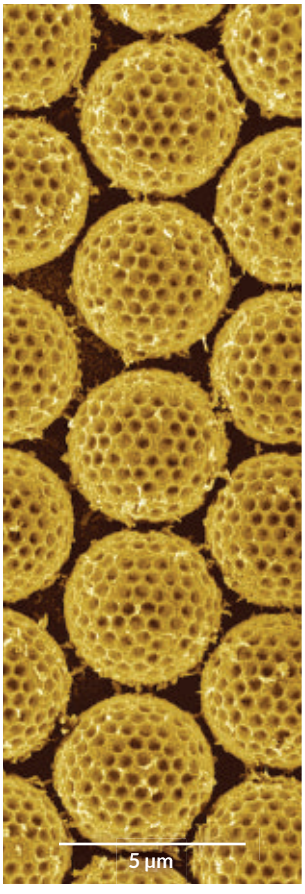
THE –EST

Spiral galaxy is most ancient observed

Astronomers have spotted the oldest known spiral galaxy. Dubbed A1689B11, the galaxy emitted its light 11 billion years ago, when the universe was less than 3 billion years old. Tiantian Yuan, an astronomer at Swinburne University of Technology in Melbourne, Australia, and colleagues found the new record-holder, reported October 31 at arXiv.org, thanks to a closer cluster of galaxies, which acted as a gravitational lens to yield two magnified images of A1689B11.

About 70 percent of galaxies in the modern universe display spiral arms. These gas-filled limbs are often where new stars form. A1689B11 has characteristics of both ancient and modern spiral galaxies: Like spiral galaxies of a similar age, it formed stars about 10 to 20 times faster than modern spirals do. But A1689B11 rotated calmly with very little turbulence, more like the spiral-armed Milky Way than its ancient peers. Astronomers think this mix of traits could suggest A1689B11 marks a transition point in galactic evolution.

The further back in the universe's history astronomers look, the more rare spiral galaxies become (*SN: 9/2/06, p. 157*). Studying galaxies like A1689B11 can help reveal when and how galaxies started to grow arms, and how those arms influenced the galaxies' later evolution. — *Lisa Grossman*



These human-made brochosomes have nanoscale indentations that interfere with light. Leafhopper-made brochosomes may have the same antireflective properties.

HOW BIZARRE

Leafhoppers use nano camo

Nature has no shortage of camouflage tricks. One form of deception, used by plant-eating insects called leafhoppers, relies on a smidge of nanotechnology.

Leafhoppers are found worldwide. Most of the 20,000 or so described species produce small quantities of microspheres called brochosomes — tiny, honeycombed spheres. Researchers previously learned that the brochosomes, which leafhoppers rub on their bodies, make the insects water-repellent. But why the bugs also cover their eggs with the nanoballs was a mystery.

Now able to manufacture brochosomes in large quantities, engineers have found that the microparticles have the exact shape and size to prevent reflection of light in any direction. As a result, surfaces covered with brochosomes appear similar to a leaf in the spectrum of light that is visible to at least one predator, the ladybird beetle, report mechanical engineer Tak-Sing Wong and his team at Penn State on November 3 in *Nature Communications*.

The finding suggests that the antireflective property of the spheres hides and protects leafhopper eggs from would-be predators. Manufactured brochosomes have many potential applications, the researchers say, such as harvesting solar energy, where antireflective surfaces are needed. — *Viviane Callier*

FROM TOP: GABRIELLA LUONGO; SHIKUAN YANG, BIRGITT BOSCHITSCH STOGIN, T.-S. WONG

This Mediterranean sea slug is a hunter and a thief.

SAY WHAT?

Kleptopredation \KLEP-toe-preh-DAY-shun\n.

A food-gathering strategy of eating an organism and the meal it just ate

A wily sea slug gets two meals in one: It gobbles up smaller predators soon after they've gulped in their own prey.

"Kleptopredation" is the term Trevor Willis of the University of Portsmouth in England and his colleagues propose for this kind of food theft by well-timed predation.

Researchers already knew that the small Mediterranean nudibranch *Cratena peregrina*, with a colorful mane of streamers atop its body, climbs and preys on skinny, branched colonies of *Eudendrium racemosum* hydroids, which are distant relatives of corals. The nudibranchs devour the individual hydroid polyps and, new tests show, prefer them well fed.

In experimental buffets with fed or hungry polyps, the nudibranchs ate faster when polyps were fat with just-caught plankton. In this way, at least half of a nudibranch's diet is plankton. This quirk explains why some biochemical signatures good for distinguishing predators from prey get murky for nudibranchs and hydroids, the researchers report in the November *Biology Letters*.

A weird echo of this meal-stealing strategy appears in some jumping spiders. The arachnids don't have the biology to drink vertebrate blood themselves. Instead, they catch a lot of female mosquitoes that have recently tanked up (*SN: 10/15/05, p. 246*). — *Susan Milius*

GENES & CELLS

Gene therapy fixes rare skin disease

Scientists replace 80 percent of ailing boy's epidermis

BY TINA HESMAN SAEY

In a last-ditch effort to save a dying 7-year-old boy, scientists have used stem cells and gene therapy to replace about 80 percent of his skin.

The procedure's success demonstrates that the combination therapy may be effective against some rare genetic skin disorders. The study also sheds light on how the skin replenishes itself, researchers report in the Nov. 16 *Nature*.

In 2015, a boy with a rare genetic skin condition called junctional epidermolysis bullosa had lost most of his skin and was close to death. Children with the condition have mutations in one of three genes — *LAMA3*, *LAMB3* or *LAMC2* — that produce parts of the laminin 332 protein, which helps attach the top layer of skin, the epidermis, to deeper layers.

People with the condition are sometimes called "butterfly children" because their skin is as fragile as the insect's wings. Even mild friction or bumps can cause severe blistering. The blistering can also affect mucus membranes inside the body, making breathing, swallowing and digesting food difficult. About 1 in every 20,000 babies in the United States is born with the condition. More than 40 percent die before adolescence.

Doctors thought the boy would also perish, says plastic surgeon Tobias Hirsch of Ruhr University Bochum in Germany, who helped care for the boy. Surgeons gave him a skin graft from his father, but the child's body rejected the transplant.

Hirsch's team turned to stem cell researcher Michele De Luca of the University of Modena and Reggio Emilia in Italy. In clinical trials, De Luca's group had grown patches of gene-repaired skin for kids with the same condition. Together, those cases had replaced 0.06 square meters of tissues, about the area of letter-sized paper. But the boy, who has a mutation in *LAMB3*, needed about 0.85 square meters of skin — 14 times as much.

In September 2015, the team took a 4-square-centimeter patch of unblistered skin from the boy's groin and grew skin stem cells from that sample. Then De Luca and colleagues used a retrovirus to insert a healthy copy of *LAMB3* into DNA in the lab-grown skin stem cells.

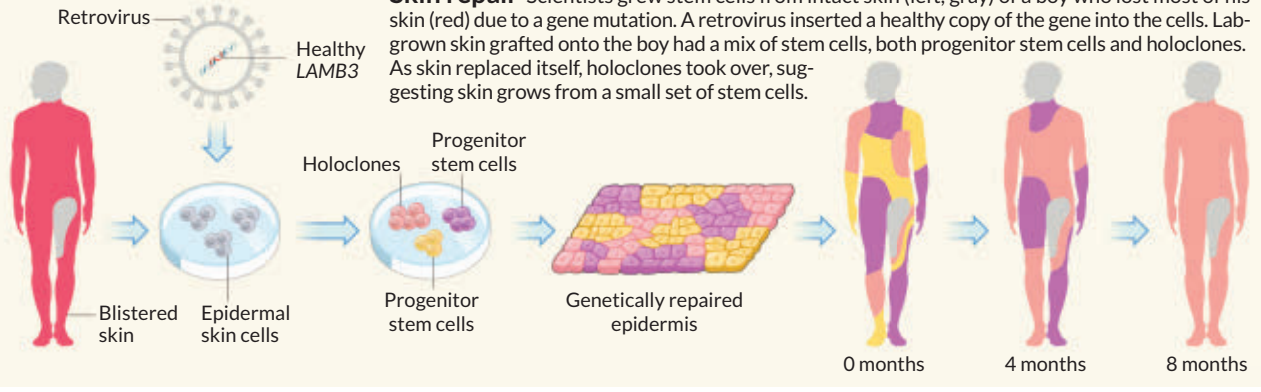
The genetically corrected skin cells grew into sheets that were grafted onto the boy in two surgeries in October and November 2015. After one additional surgery, he was released from the hospital in February 2016.

"The kid is now back to school. He plays soccer," Hirsch says. The boy's new skin is fully functional. He still has some blistering in untreated areas, and his doctors are considering replacing more skin. Meanwhile, some of the corrected stem cells may be spreading into the untreated epidermis, and may eventually replace all of his skin. But researchers can't take many samples to find out. "He's a patient," De Luca says. "He's not a mouse."

The case is a landmark in stem cell therapy, says stem cell researcher Elaine Fuchs of Rockefeller University in New York City. "It makes considerable headway in resolving a brewing controversy in the epidermal stem cell field" over exactly how skin regenerates.

One possibility is that a large number of stem cells populate the skin. Each stem cell can then either copy itself or morph into a variety of different types of mature skin cells. The other possibility is that only a small number of long-lived stem cells — known as holoclones — give rise to short-lived progenitor cells that are forerunners to mature skin cells.

When researchers inserted *LAMB3*, it landed in different places in each lab-grown stem cell. De Luca's group used the different insertions like bar codes to track the boy's holoclones and other skin cells. At first, his skin was a patchwork of cells, with about 91 percent of progenitor cells having different insertions than the holoclones. After four months, only 37 percent of the progenitor cells were different from holoclones. By eight months, nearly all skin cells had come from the holoclones. That pattern indicates that most of the progenitor cells had died and were replaced by offspring of the long-lived holoclones. The findings suggest that a small number of stem cells replenish the skin. ■



Skin repair Scientists grew stem cells from intact skin (left, gray) of a boy who lost most of his skin (red) due to a gene mutation. A retrovirus inserted a healthy copy of the gene into the cells. Lab-grown skin grafted onto the boy had a mix of stem cells, both progenitor stem cells and holoclones. As skin replaced itself, holoclones took over, suggesting skin grows from a small set of stem cells.

M. ARAGONA AND C. BLANPAIN/*NATURE* 2017

Croc bites mimic ancient tool marks

New study questions claim of butchery 3.4 million years ago

BY BRUCE BOWER

Recent reports of African and North American animal fossils bearing stone-tool marks from butchering a remarkably long time ago may be a crock. Make that a croc.

Crocodile bites damage bones in virtually the same ways that stone tools do, say paleoanthropologist Yonatan Sahle of the University of Tübingen in Germany and colleagues. Animal bones allegedly cut up for meat about 3.4 million years ago in East Africa (*SN: 9/11/10, p. 8*) and 130,000 years ago in what's now California (*SN: 5/27/17, p. 7*) come from lakeside and coastal areas. Those are places where crocodiles could have wreaked damage now mistaken for butchery, the scientists report online November 6 in *Proceedings of the National Academy of Sciences*.

Larger samples of animal fossils, including complete bones from various parts of the body, are needed to begin to tease apart the types of damage caused by stone tools, crocodile bites and trampling of bones by living animals, Sahle's team concludes. "More experimental work on bone damage caused by big, hungry crocs is also critical," says coauthor Tim White, a paleoanthropologist at the University of California, Berkeley.

In a field where researchers reap big rewards for publishing media-grabbing results in high-profile journals, such evidence could rein in temptations to overinterpret results, says David Braun, an archaeologist at George Washington University in Washington, D.C., who did not participate in the new study. "There's a push to publish extraordinary findings, but evolutionary researchers always have to weigh what's interesting versus what's correct."

The researchers who conducted the original ancient butchery studies agree that bone marks made by crocodiles deserve closer examination and careful comparison with proposed stone-tool marks. But the researchers stand their ground on their original conclusions.

Microscopic investigations in the 1980s led some researchers to conclude that carnivores such as hyenas leave U-shaped marks on bones. In contrast, stone tools leave V-shaped incisions with internal ridges. And hammering stones create signature pits and striations.

Sahle's group expanded on previous research by paleoanthropologist Jackson Njau of Indiana University Bloomington. In his 2006 doctoral dissertation, Njau reported that bone damage produced by feeding crocodiles looks much like stone-tool incisions and pits, with a few distinctive twists such as deep scratches. Njau studied cow and goat bones from carcasses that had been eaten by crocodiles housed at two farms in Tanzania.

In the new study, the scientists used Njau's findings to reassess marks on fossils excavated in Ethiopia and dating to about 4.2 million, 3.4 million and 2.5 million years ago. Damage to these fossils has generally been attributed to butchery with stone tools.

Incisions and pits on arm bones from ancient *Australopithecus* hominids and similar marks on a horse's leg probably resulted from crocodile bites and not stone-tool use, as initially suspected, the team says. If tools had indeed damaged the hominid remains, that would raise the possibility of cannibalism — a difficult behavior to confirm with fossils. Tellingly, Sahle's team argues, the bones come from what were once waterside areas. Some bones were found in the same sediment layer as crocodile remains. Marks on the bones include deep scratches consistent with croc bites.

The horse fossil comes from a spot along an ancient lakeshore where no stone tools have been found, a further clue in favor of damage from croc bites.

Jagged pits, incisions and other marks scar a leg fragment and lower jaw from a hoofed animal. But microscopic analyses could not definitively attribute the damage to stone tools or crocodile bites.

Ancient crocodiles had teeth (one shown, left) capable of gouging chunks out of a prey's bones. Some damage on fossils, such as this horse leg (right), previously attributed to tools may instead be the result of crocodile bites.

In light of these findings, the bones from California and the 3.4-million-year-old bones from East Africa should be reexamined with the possibility of croc damage in mind, White says. For now, the earliest confirmed stone-tool marks occur on bones from two East African sites dating to around 2.5 million years ago (*SN: 4/17/04, p. 254*), he adds.

The crocodile marks described in the study don't look "especially like" damage to the 130,000-year-old mastodon bones on California's coast, says paleontologist Daniel Fisher. Fisher, of the University of Michigan in Ann Arbor, was one of the researchers who proposed those bones had been butchered. No fossil evidence indicates crocs lived there at that time, he adds. Several lines of evidence, including pounding marks and damage near joints, point to stone-tool use at the West Coast site, says Richard Fullagar, an archaeologist at the University of Wollongong in Australia and Fisher's collaborator.

Further studies of the 3.4-million-year-old African bones will statistically compare the probability of various causes for particular marks, including crocodile bites, says Shannon McPherron, who led the investigation that concluded the bones displayed signs of butchery. In that way, researchers can assess whether any one cause stands out as the strongest candidate, says McPherron, an archaeologist at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. ■

Y. SAHLE ET AL/PNAS 2017

ATOM & COSMOS

Odd star explodes again and again

Years-long supernova may be iPTF14hls' third outburst

BY LISA GROSSMAN

A shocking supernova refuses to die.

This exploding star, named iPTF14hls, has erupted continuously for the last three years, and it may have had two other outbursts in the past, astronomers report in the Nov. 9 *Nature*. Such a tireless supernova could be the first example of a proposed explosion that involves burning antimatter in a stellar core — or it could be something new altogether.

"A supernova is supposed to be a one-time thing — the star explodes, it's dead, it's done, it can't explode again," says astrophysicist Iair Arcavi of the University of California, Santa Barbara. "It's the weirdest supernova we've ever seen.... It's like the star that keeps on dying."

When discovered in September 2014 by a telescope at the Palomar Observatory near San Diego, iPTF14hls looked like an ordinary type 2 supernova in a galaxy about 500 million light-years away. Such explosions mark the death throes of a star eight to about 50 times the mass of the sun (*SN: 2/18/17, p. 24*), and typically glow for about 100 days before starting to dim.

The first sign that iPTF14hls was unusual came a few months after its discovery, when it started growing brighter. That turned out to be one of five irregular cycles of brightening and dimming.

Even stranger, data collected from September 2014 to June 2016 show that the supernova remained bright for more than 600 days, Arcavi and colleagues report. The eruption, which is just now showing signs of winding down, may have already been in progress when it was discovered, so it could have persisted even longer.

"That's just unheard of," says Stanford Woosley, a theoretical physicist at the University of California, Santa Cruz who was not involved in the discovery. "Ordinary supernovae don't do that."

Normally, layers of gas kicked out of an exploding star slow and cool as they expand. But iPTF14hls maintained a high temperature — between 4,700° and 5,700° Celsius — for the entire time it was observed, and its outer gas layers did not slow down as they should have. That means that this gas may have already cooled and slowed, suggesting it had been expelled in an earlier, superpowerful eruption that occurred unseen between 2010 and 2014, the team says.

> "It's the weirdest supernova we've ever seen.... It's like the star that keeps on dying."
>
> IAIR ARCAVI

Historical data on photographic plates from the Palomar Observatory showed yet another bright burst in the same part of the sky in 1954. One theory suggests that stars between 95 and 130 times the mass of the sun can explode several times, though these cyclic deaths have never been seen before. Such stars get so hot that they convert gamma rays, with high energy that helps keep the star from collapsing under its own gravity, into electrons and their antimatter counterparts, positrons. Without that internal energy, the star's core collapses and gets even hotter. That collapse can trigger a partial explosion, in which the star blows off a large amount of mass. But after the explosion, the electrons and positrons can recombine into gamma rays and hold up the remaining stellar core.

The star can blow off steam several times, the idea goes, before finally dying in a supernova. Eventually, the remains of such a supernova would collapse into a black hole with about 40 times the mass of the sun.

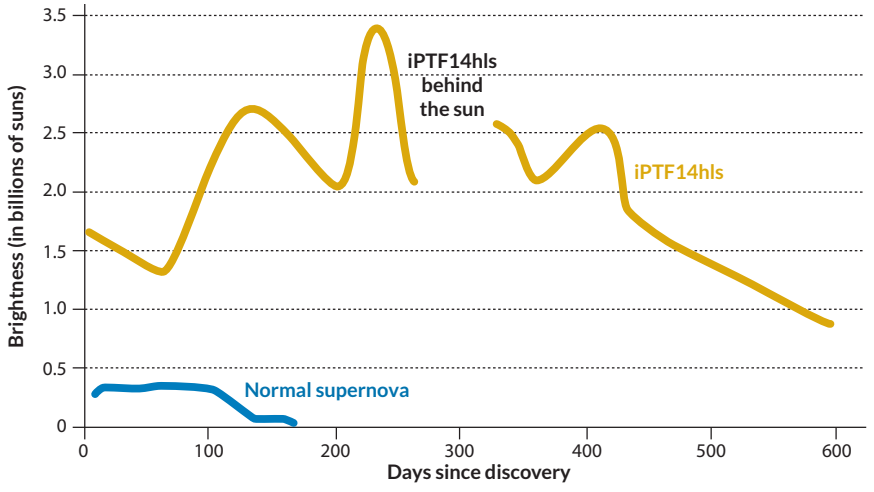
But this theory also predicts that the star would blow off all its hydrogen in the first explosion. That doesn't fit here: iPTF14hls expelled 50 times the mass of the sun in hydrogen in 2014. The amount of energy in the most recent explosion is also greater than it should be.

Woosley thinks that a magnetar, a highly magnetized rapidly rotating stellar corpse (*SN Online: 11/3/10*), could glow continuously for around two years, though that wouldn't explain the 1954 eruption. He hopes the most recent data will help determine which theory explains iPTF14hls, or if physicists need to come up with something new.

The show may be ending. The latest data indicate that iPTF14hls is finally fading, Arcavi says. As the outer layers of gas cool and become transparent, they could reveal whatever is at the explosion's core. The team plans to just keep watching.

"I am not making any more predictions about this thing," Arcavi says. "It surprised us every time." ■

Persistent supernova The star iPTF14hls has erupted continuously since its discovery in 2014 and has fluctuated in brightness (yellow) at least five times (first 600 days of observation shown). Typical supernovas (blue) fade after about 100 days. SOURCE: S. WILKINSON/LCO



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BODY & BRAIN

Study casts doubt on nerve cell claim

Adult brains may stop making new neurons in memory region

BY LAURA SANDERS

In stark contrast to earlier work, new findings suggest adults don't produce new nerve cells in a brain area important to memory. Scientists came to that conclusion after scrutinizing 54 human brains spanning the age spectrum.

The results, described November 13, are preliminary. If confirmed, they would overturn the widely accepted idea that the hippocampus churns out new neurons in adulthood.

Animal studies have hinted that boosting the birthrate of neurons, a process called neurogenesis, in the hippocampus might enhance memory or learning, combat depression and even stave off the mental decline that comes with old age (*SN: 9/27/08, p. 5*). In rodents, exercise, enriched environments and other tweaks can boost hippocampal neurogenesis and memory performance. But the new study may temper those ambitions for people.

Shawn Sorrells of the University of California, San Francisco and colleagues studied human brain samples that ranged from fetal stages to age 77, acquired either postmortem or during surgery. These samples were cut into thin slices and probed with molecular tools that can signal dividing or young cells — signs that neurons are being born.

Fetal and infant samples had evidence of dividing cells and young neurons in the hippocampus. But the numbers declined with age. In tissue from a 13-year-old, there were only a handful of young neurons. In adults, there were none.

The researchers also found no physical signs of dividing cells and young neurons — such as small, elongated nuclei — in adults, Sorrells reported. The absence of young hippocampal neurons conflicts with some earlier work.

A definitive answer has eluded scientists in part because there's no easy way to study these cells in living people. And the methods used to study tissue from people after they've died can be finicky. Molecular markers of neurogenesis in adult brain tissue can be difficult to see, says Jonas Frisén of the Karolinska Institute in Stockholm, who has used a different method to spot new neurons in adults.

It's also possible that the birthrate of neurons varies widely from person to person. "There are huge differences between different mouse strains," Frisén says. It's not unreasonable that similar differences might exist in people, he says. In the new study, subjects' health could have declined before their deaths, reducing neurogenesis at the end of their lives.

This preliminary study is not the last word, says Gerd Kempermann of Technische Universität Dresden in Germany. The body of evidence in favor of human neurogenesis in the adult hippocampus, including results from his lab, is quite strong, he says. "I am convinced enough that an abstract at a meeting claiming otherwise does not shake up my concept." He thinks it will be valuable to explore other explanations for the results. ■

LIFE & EVOLUTION

Orb weavers have quick body clocks

Some spider species endure 5- to 7-hour 'jet lag' every day

BY MARIAH QUINTANILLA

If it takes you a while to recover from a few lost hours of sleep, be grateful you aren't an orb-weaving spider. Three species of orb weaver may have the shortest known natural circadian rhythms in animals, scientists reported November 12.

Most animals have natural body clocks that run close to the 24-hour day-night cycle, with light helping to reset the timing each day. But the three orb weavers' body clocks average between 17.4 and 19 hours. Because the spiders still keep time with the natural solar cycle, they must shift their cycle of activity and inactivity — the equivalent of wake and sleep cycles — by five to seven hours each day.

"That's like flying across more than five time zones, and experiencing that much jet lag each day in order to stay synchronized with the typical day-night cycle," said neurobiologist Darrell Moore.

Internal clocks help animals perform recurring daily activities, like eating and sleeping, at the appropriate time of day.

To measure spiders' natural biological clocks without the sun's resetting effect, Moore, of East Tennessee State University in Johnson City, and colleagues put 18 species in constant darkness and monitored them. Three orb weaver species had short cycles of activity and inactivity.

Quick clocks may prevent the spiders from being the proverbial "worm" to the earliest birds, the team says. By becoming more active at dusk and beginning web-spinning three to five hours before dawn, the spiders can avoid day predators. Throughout the day, the spiders remain motionless on their web. By midday, their truncated circadian clocks should have reset, sparking a new round of activity. But in the five to seven hours of daylight left, the orb weavers remain inactive. Light may delay the onset of another short circadian cycle each day, helping the spiders stay synchronized with the 24-hour cycle, the team suspects.

A fast body clock may help this spiny orb weaver (*Gasteracantha cancriformis*) avoid predation.

MKULLEN/WIKIMEDIA COMMONS

"The method or molecular mechanism will be really fascinating to figure out," said Sigrid Veasey, a neuroscientist at the University of Pennsylvania's Perelman School of Medicine. ■

BODY & BRAIN

Blood may contribute to Alzheimer's

Amyloid-beta can travel from body to brain, mouse study finds

BY LAURA SANDERS

An Alzheimer's-related protein can move from the blood to the brain and accumulate there, experiments in mice show for the first time.

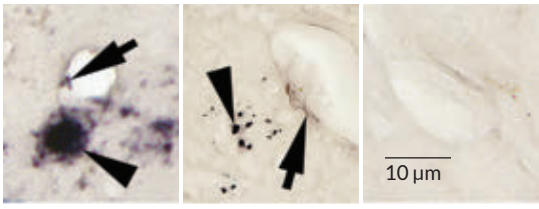
The results, reported October 31 in *Molecular Psychiatry*, suggest that the protein amyloid-beta outside the brain may contribute to the Alzheimer's disease inside it, says neurobiologist Mathias Jucker of the University of Tübingen in Germany. This more expansive view of Alzheimer's may lead to new treatments that target parts of the body that are easier to access than the brain.

The experiments don't suggest that people could contract Alzheimer's from another person's blood. "The bottom line is that this study is thought-provoking but shouldn't cause alarm," says neurologist John Collinge of University College London. "There really isn't any evidence that you can transmit Alzheimer's disease by blood transfusion."

But researchers wondered whether, over time, A-beta might build up in the brain by moving there from the blood, where the protein is found in small quantities. Earlier animal studies showed that A-beta can move into the brain if injected into the bloodstream, but scientists didn't know whether A-beta from the blood can be plentiful enough to form brain plaques.

To test this, researchers used a form of extreme blood-sharing. Six pairs of mice — with one mouse mutated to produce gobs of human A-beta and one normal — were surgically joined for a year, causing blood mingling that's far more extensive than that of a blood transfusion. After a year, the brains of the mice carrying the mutations were full of A-beta plaques, as expected. But these plaques were also inside the brains of the normal mice in the joined pairs.

In those normal mice, A-beta levels weren't as high as they were in the mutated mice, but the fact that plaques existed was notable, says study coauthor Weihong Song of the University of British Columbia in Vancouver. Unjoined control mice without the mutations showed no A-beta accumulation.



Brains of mice mutated to make a lot of amyloid-beta developed plaques (triangle, left) and amyloid along blood vessels (arrow). Mice that shared blood with mutant mice had similar accumulation (middle); control mice did not (right).

Brains of the joined mice showed other signs of deterioration: inflammation, tiny areas of bleeding and the presence of a dangerous version of the protein tau. In people, Alzheimer's is often marked by both A-beta plaques and tangles of tau.

The results don't mean that Alzheimer's is predominantly caused by factors in the blood. But those factors, in some

BODY & BRAIN

Source of cholera epidemics ID'd

Globe-trotting, not local, strains fuel big outbreaks

BY AIMEE CUNNINGHAM

Cholera strains behind worldwide outbreaks of the disease over the last five decades are jet-setters, not homebodies.

It had been proposed that cholera epidemics are homegrown, driven by local strains of *Vibrio cholerae* living in aquatic ecosystems. But DNA fingerprints of the *V. cholerae* strains responsible for recent big outbreaks in Africa and Latin America were more closely related to South Asian strains than local ones, according to two papers in the Nov. 10 *Science*.

This evidence that the guilty strains traveled from abroad could guide public health efforts. "If you don't understand how the bug spreads, then it's very difficult to try to stop the bug," says clinical microbiologist François-Xavier Weill of the Pasteur Institute in Paris, who coauthored both papers.

People are exposed to *V. cholerae* by consuming water or food contaminated by the bacteria (*SN: 8/19/17, p. 4*). A cholera infection can produce mild or no symptoms. But about 1 in 10 people will rapidly develop severe diarrhea and dehydration that, without treatment, can kill within hours.

There have been seven cholera pandemics, or global outbreaks, since the 19th century, when the bacteria spread from their original home on the Indian subcontinent. The seventh one — which began in Indonesia in 1961, reached Africa in 1970 and hit Latin America in 1991 — is still ongoing. That pandemic is attributed to strains that originated near the Bay of Bengal, where cholera is seasonal. But whether the pandemic's large outbreaks were related or had each originated from local strains was unclear.

Weill and colleagues analyzed the genetic information of about 1,700 strains of *V. cholerae*, including those collected during and in between outbreaks over about 40 years from 45 countries in Africa and 14 in Latin America.

In both regions, the strains responsible for the large epidemics were most closely related to South Asian strains, rather than strains existing in the local environment. These "epidemic" strains have been introduced 11 times in Africa since 1970 and have caused large outbreaks that lasted as long as 28 years, the researchers found.

In Latin America, there were three main introductions of the South Asian epidemic strains. One that came through Africa hit Peru in 1991. Another invaded Mexico around the same time, possibly arriving with coca smugglers using an airstrip near Mexico City. The third

X.-L. BU *ET AL/MOLECULAR PSYCHIATRY* 2017

cases, might have the power to nudge the disease along, the results suggest.

A-beta is made by cells in the brain, but also by blood platelets, skin cells, muscles and other parts of the body. Normally, "there is a balance between A-beta inside and outside the brain," Song says. But when this balance is thrown off, such as when the body is chock-full of the protein, or when the blood-brain barrier — the blockade that keeps potential dangers out of the brain — deteriorates with age, the brain may get an extra dose, Song proposes. By tweaking this balance, he says, it's possible that drugs or therapies that reduce A-beta in the body might help slow or prevent Alzheimer's disease.

Evidence has been accumulating that A-beta can behave as a prion, a misfolded protein that can incite normal proteins to go rogue (*SN: 10/17/15, p. 12*). Song says the experiments don't address whether A-beta from blood can prompt already existing A-beta in the brain to form plaques. In the study, normal mice's brain plaques seemed to be built from human A-beta protein; the only source of that was the blood of the mutated partner. ■

introduction, from Nepalese United Nations personnel, devastated Haiti in 2010 (*SN: 2/25/12, p. 16*).

"We now know what cholera is with much more precision," says genome scientist Nicholas Thomson of the Wellcome Trust Sanger Institute in Cambridge, England, who coauthored both papers. "You can find *V. cholerae* in the environment, no doubt about it, but the patterns of spread tell you that that's not the primary route of transmission." Rather, he says, it's transmission between people that allows the bacteria to spread rapidly internationally.

"These studies affirm the primary role that people play in the spread of cholera," says Yonatan Grad, an infectious diseases clinician at the Harvard T.H. Chan School of Public Health in Boston. "The emphasis on infected people as the vectors for spread underscores the importance of vaccination as a strategy to limit cholera." ■

T. TIBBITTS

BODY & BRAIN

Experts redefine hypertension

New guidelines lower cutoff for high blood pressure

BY AIMEE CUNNINGHAM

ANAHEIM, CALIF. – Nearly half of U.S. adults are now considered to have high blood pressure, thanks to a new definition of what is high: 130/80 is the new 140/90. That means 103 million people — up from 72 million under the old definition — need to make diet and exercise changes and, in some cases, take medication to lower their risk of heart attack or stroke.

These new blood pressure guidelines, the first major update since 2003, were announced November 13 at the American Heart Association's annual scientific sessions and published in *Hypertension* and the *Journal of the American College of Cardiology*.

A blood pressure reading measures systolic pressure, or how much force the blood places on the walls of the arteries when the heart beats, and diastolic pressure, the same force but when the heart rests between beats.

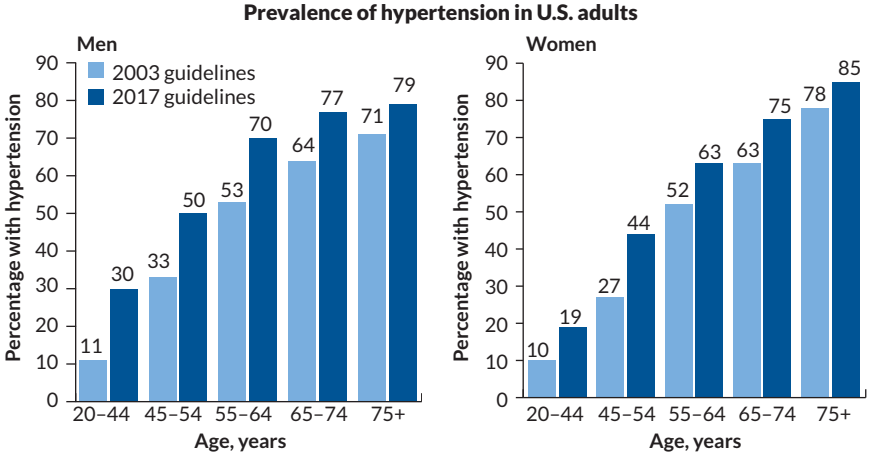
Previously, systolic pressures from 120 to 139 millimeters of mercury and diastolic pressures from 80 to 89 were considered prehypertension, putting a person at risk for high blood pressure. Under the new guidelines, a systolic reading of 120 to 129 along with a diastolic pressure less than 80 is classified as elevated blood pressure. Just over 12 percent of U.S. adults fall in that category.

Hypertension, or high blood pressure, was previously diagnosed once a patient hit 140/90. That definition applied to about 32 percent of U.S. adults. Hypertension as it is now defined, 130/80 or higher, occurs in 46 percent of U.S. adults.

"People with blood pressure levels between 130 and 140 are at about twice the risk of heart attack and stroke as people with normal levels," said David Goff of the National Heart, Lung and Blood Institute in Bethesda, Md., who was not involved with developing the guidelines. The new definition "really highlights the importance of preventing high blood pressure in the first place."

Lifestyle changes are recommended for those with elevated or high blood pressure. For those in the first stage of hypertension — 130 to 139 over 80 to 89 — whether drugs are prescribed will depend on whether patients have had a heart attack or stroke or if their risk is greater than 10 percent in the next 10 years. About 4 million more U.S. adults are projected to require drugs, or 1.9 percent more than was recommended in the old guidelines, said Robert Carey of the University of Virginia School of Medicine in Charlottesville, a coauthor of the new guidelines. ■

Numbers jump More U.S. adults of all ages are considered to have high blood pressure under new guidelines. The change is expected to roughly triple the prevalence of hypertension in men under 45 and double the prevalence for women of that age. SOURCE: P.K. WHELTON *ET AL/HYPERTENSION* 2017



MATTER & ENERGY

How pedestrians send bridges swaying

Threshold number of walkers leads to wobbling, simulation finds

BY EMILY CONOVER

Crowds walking on a bridge can cause it to sway — sometimes dangerously. Using improved simulations to represent how people walk, scientists have now devised a better way to calculate under what conditions this swaying may arise, researchers report November 10 in *Science Advances*.

When a bridge — typically a suspension bridge — is loaded with strolling pedestrians, their gaits can sync, causing the structure to shimmy from side to side. The new study "allows us to better predict the crowd size at which significant wobbling can appear abruptly," says study coauthor Igor Belykh, a mathematician at Georgia State University in Atlanta.

Engineers might eventually use the results to avoid debacles like the one that befell the Millennium Bridge in London. This suspension bridge temporarily shut down just days after it opened in 2000 due to the large wobble that occurred when many people tromped across it at once, necessitating costly repairs.

Pedestrians crossing a bridge can cause slight sideways motion of the bridge as they push with their feet. This swaying may lead to the crowd unintentionally falling into lockstep because it's easier to go with the flow of the swinging bridge than fight it. That synchronization creates larger and larger oscillations.

"It's a dangerous phenomenon that could cause a bridge to collapse if it went unchecked," says Daniel Abrams, an applied mathematician at Northwestern University in Evanston, Ill.

Previous mathematical models of the phenomenon "didn't realistically capture how people exerted force on the bridge," Abrams says, "but this new model is pretty realistic." Whereas earlier simulations focused on the timing of footfalls or the amount of force produced with each step, the new work takes both into account.

Tests of the Millennium Bridge showed that the lurching occurred only after about 165 people entered the bridge. In their simulations, Belykh and colleagues also find that oscillations begin abruptly above a certain threshold number of walkers, depending on the properties of the bridge.

The research challenges some previous assumptions. For instance, in the new simulations, the onset of the wobbling began just before the walkers joined in lockstep. This suggests that the synchrony of the crowd might not be a root cause but instead acts as a feedback effect that amplifies preexisting small-scale wobbles. That insight could be relevant for wobbles that occur in certain bridges without pedestrians syncing, Belykh says. Future work will take a closer look at how the swaying starts. ■

LIFE & EVOLUTION

Sheep recognize human faces

Animals picked out familiar celebrities in photo tests

BY MARIAH QUINTANILLA

Emma Watson, Jake Gyllenhaal and Barack Obama walk into a sheep pen. No, this isn't the beginning of a baaa-d joke.

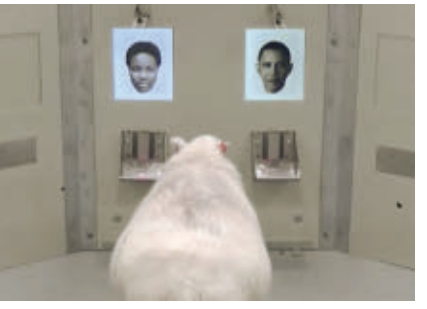
By training sheep using photos of these celebrities, University of Cambridge scientists discovered that the animals can recognize familiar faces from 2-D images. Given a choice, sheep picked a familiar face over an unfamiliar face most of the time, the team reports November 8 in *Royal Society Open Science*.

Even when a celeb's face was tilted rather than face-on, sheep still picked the image more often than not. That means sheep weren't memorizing images, demonstrating for the first time that sheep have advanced face-recognition capabilities similar to humans', say neurobiologist Jennifer Morton and colleagues.

Sheep can pick out pictures of individuals in their flock, and even familiar handlers (*SN: 10/6/12, p. 20*). And like dogs, rhesus monkeys, mockingbirds and some other animals, sheep can distinguish between individuals of other species. But it was unclear whether that skill was recognition or memorization.

Morton's group released eight sheep one by one into a pen with two computer screens. A face would appear on one screen, while a different image appeared on the other. After training sheep to pick out celebrities' faces to earn a food reward, the team paired a celebrity mug with an unfamiliar person. By the end of the experiment, the sheep chose a celebrity's face over a stranger's about 79 percent of the time on average.

To see if sheep were truly recognizing faces, the team repeated the test with pictures in which celebrities' heads were tilted right or left. The sheep didn't do as well but still passed, recognizing celebs about 67 percent of the time on average — a performance drop comparable to that seen in humans doing the same task.



In tests, sheep recognized familiar faces, such as Barack Obama's, over unfamiliar ones — a sign of humanlike face-processing abilities.

J. MORTON/UNIV. OF CAMBRIDGE

Brad Duchaine, a brain scientist at Dartmouth College, isn't surprised by the results. "My guess is that the ability of sheep to recognize human faces is a by-product of selection to discriminate between different sheep faces," he says. "Either the human face is similar enough to the sheep face that [it] activates the sheep face-processing system, or human-face recognition relies on more general-purpose recognition systems." ■

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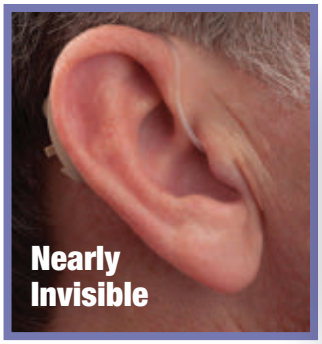

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GENES & CELLS

Current gene drives may be too strong

Scientists muse on how to add brakes before use in the wild

BY SUSAN MILIUS

Gene-editing tools heralded as hope for fighting invader rats, malarial mosquitoes and other scourges may be too powerful to use outside the lab in their current form, two new papers warn.

Standard forms of CRISPR gene drives, as the tools are called because they rely on a gene editor of the same name (*SN: 12/26/15, p. 18*), can make tweaked DNA race through a population so easily that a small number of stray animals or plants could spread it to new territory, predicts a computer simulation released November 16 at bioRxiv.org. Such an event would have unknown, potentially damaging, ramifications, say authors of a paper released the same day in *PLOS Biology*.

"We need to get out of the ivory tower and have this discussion in the open, because ecological engineering will affect everyone living in the area," says Kevin Esvelt of MIT, a coauthor of both papers who studies genetic solutions to ecological problems. What's a pest in one place may be valued in another, so getting consent to use a gene drive could mean consulting people across a species's whole range, be it several nations or continents.

Researchers have constructed this kind of drive in yeast, a fruit fly and some mosquitoes, but none of the tools have been deployed in the wild (*SN: 12/12/15, p. 16*). Meanwhile, some researchers are already working to add brakes or off-switches into a new generation of gene drives.

The major concern is that current gene drives "are probably too powerful for us to seriously consider deploying in conservation," says geneticist Neil Gemmell of the University of Otago in Dunedin, New Zealand. Gemmell is a coauthor of the paper in *PLOS Biology*.

This opinion could prove especially controversial in New Zealand. In 2016, the nation resolved to protect its imperiled biodiversity by exterminating invader rats, stoats and possums. Gene drives might make that possible.

Though warning of perils, the scientists also propose solutions. A weaker system, which Esvelt calls a daisy drive, splits up a component of the drive, such as guide RNAs, across the genome. Guide RNA directs the gene-editing machinery to its DNA target, where the machinery then snips and replaces genetic material. As genes get inherited or not in the chancy jumbling of sexual reproduction, descendants in later generations become less likely to inherit all of the spaced-apart pieces needed to operate the gene drive.

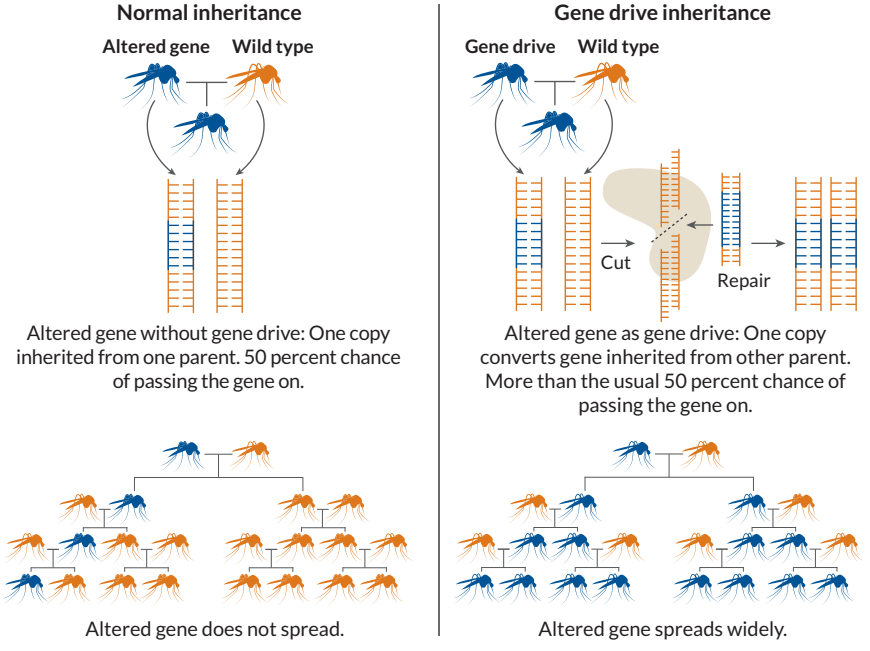
Esvelt's lab is working to create a daisy drive in two kinds of nematode worms and is looking at other species as well.

Anthony A. James of the University of California, Irvine predicts that the gene drives he and colleagues have put in the disease-carrying *Anopheles* mosquito species are already self-limiting. When females end up with two of the genes his team is inserting, the mosquitoes don't "survive very well after they have fed on blood." Researchers are now raising these mosquitoes to see whether the genes spread and then dwindle away. "We don't need our genes to last forever," James says, "only long enough to contribute to getting rid of malaria."

Another lab's disease-fighter mosquitoes already have something like a daisy drive. *Aedes aegypti* mosquitoes engineered with some built-in parts of a gene editor have guide RNA split into two parts and put on different chromosomes, says Omar Akbari of the University of California, San Diego. Pictures of weird mosquitoes created this way — all yellow or with three eyes — show that gene editing works. Akbari's research appears online November 14 in *Proceedings of the National Academy of Sciences*.

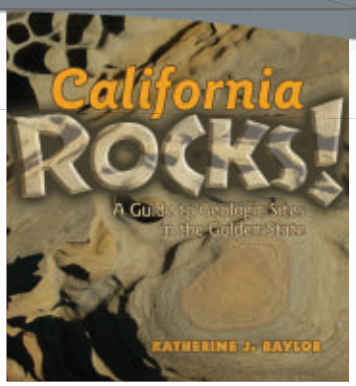
Akbari isn't worried about the risk of accidentally wiping out disease-carrying mosquitoes. It would be unethical not to use a tool that could lessen huge loss of human lives, he says. But he does recognize that the case for caution could be different for other species. "A lot of pet owners would be sad," he says, if a gene drive went wrong and escaped worldwide during some future attempt to rid, say, Australia of its destructive feral cats. ■

Natural vs. driven Organisms that reproduce sexually have a 50 percent chance of passing along a gene to an offspring (left). A gene drive (right) copies and pastes itself into chromosomes from both parents, ensuring it gets passed on more often.



E. OTWELL AND M. TELFER

GEOLOGIC ROAD TRIP OF THE MONTH



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POINT LOBOS STATE NATURAL RESERVE

The Salinian Block and the Carmelo Formation

Today's sea lions were called lobos marinos, or "sea wolves," by early Spanish settlers, giving rise to the name Punta de los Lobos Marinos for the rocky point south of Monterey. Erosion of two different rock types, the Santa Lucia Granodiorite, part of the larger 110- to 78-million-year-old Salinian Block, and the younger Carmelo Formation, a marine sedimentary rock, gives Point Lobos its craggy, rugged landscape. The Salinian Block, bracketed on the east by the San Andreas Fault and on the west by the Sur-Nacimiento Fault, dominates the northern part of the Big Sur coast and extends northward through Point Lobos State Natural Reserve and much of the Monterey Peninsula. The highly weathered granitic rocks of the Salinian Block frequently appear as crumbly outcrops with a salt-and-pepper texture. At Point Lobos, the Santa Lucia Granodiorite has particularly well-developed, white, rectangular crystals of potassium feldspar, generally 1 to 3 inches long. The granodiorite extends across the northern rocky cliffs of the reserve from Point Lobos to Granite Point and also outcrops at Bluefish Cove, Big Dome Cove, Cypress Cove, and Middle Cove. In the southern part of Point Lobos State Natural Reserve, the granodiorite crops out between Hidden and Gibson Beaches.

On the south side of Point Lobos State Natural Reserve, outcrops are mostly sedimentary rocks of the Carmelo Formation, a 1,000-foot-thick sequence of marine conglomerate, sandstone, and shale deposited on top of the older granodiorite. Conglomerate, a sedimentary rock containing rounded pebbles or cobbles, is deposited by fast-moving water. Trace fossils within the Carmelo Formation indicate these rocks were deposited some 60 to 50 million years ago approximately 600 to 1,500 feet below the water surface. This depth, along with the geometry of the deposit, indicates that the sediments were deposited by underwater landslides (turbidity currents) in a submarine canyon. In the intervening quiescent periods between slides, bottom-dwelling organisms burrowed into the sedimentary layers. Over time, the sediments solidified and were tectonically uplifted to their present-day elevation. Within Point Lobos State Natural Reserve, rocks of the Carmelo Formation are most prominent at Sand Hill, Sea Lion, and Headland Coves, all on the south side of Point Lobos, and part of Whalers Cove, on the north side.

The Carmelo Formation closely resembles the Point Reyes Conglomerate. In fact, both may have been deposited in the same submarine canyon but were since separated by more than 100 miles due to offset along the San Gregorio and San Andreas Fault systems.

Carmelo Formation sedimentary rocks on the south side of Point Lobos State Natural Reserve. Waves have eroded the big trough in a weaker layer of the rock.

MATTER & ENERGY

Quantum computing steps forward with 50-qubit prototype

Bit by qubit, scientists are edging closer to the realm where quantum computers will reign supreme.

IBM is testing a prototype quantum processor with 50 quantum bits, or qubits, the company announced November 10. That's about the number needed to meet a sought-after milestone: demonstrating that quantum computers can perform specific tasks that are beyond the reach of traditional computers (*SN: 7/8/17, p. 28*).

Unlike standard bits, which represent either 0 or 1, qubits can indicate a combination of the two, using what's called quantum superposition. This property allows quantum computers to perform certain kinds of calculations more quickly. But because qubits are finicky, scaling up is no easy task. Previously, IBM's largest quantum processor boasted 17 qubits.

IBM also announced a 20-qubit processor that the company plans to make commercially available by the end of the year. — *Emily Conover*

ATOM & COSMOS

Haze may cool down Pluto

Blame Pluto's haze for the dwarf planet's unexpected chilliness. Hydrocarbon clumps in the atmosphere radiate heat back into space, keeping Pluto cool, a new study suggests. The dwarf planet may be the only world in the solar system with atmospheric temperature controlled by solid particles rather than gas, researchers report in the Nov. 16 *Nature*.

Most planets' temperatures are set by the gas content of the atmosphere, since certain gases trap heat from the sun more efficiently than others. Based on the makeup of Pluto's atmosphere, scientists predicted the upper atmosphere would be a brisk −173° Celsius.

But when the New Horizons spacecraft flew past Pluto in 2015, the probe found temperatures closer to −203°. The probe helped reveal another surprising feature: Hydrocarbon particles clump in the atmosphere to form a haze before eventually settling onto the surface (*SN: 4/15/17, p. 14*).

IBM announced two new advances to its quantum-computing hardware (cooling system and other infrastructure shown): a 20-qubit processor that will be available commercially and a 50-qubit prototype undergoing testing.

This haze could keep the atmosphere cool, planetary scientist Xi Zhang of the University of California, Santa Cruz and his colleagues say. The team calculated that the clumps could absorb heat from the sun and from gases in the atmosphere and radiate heat back into space.

That makes Pluto unique in the solar system, the researchers say. Other hazy worlds, such as Saturn's moon Titan, still have temperatures that are mostly determined by gas. — *Lisa Grossman*

ATOM & COSMOS

Sandy core keeps Enceladus' ocean liquid, new simulations suggest

A soft heart keeps Enceladus warm from the inside. Friction within its porous core could help Saturn's icy moon maintain a subsurface liquid ocean for billions of years, scientists report November 6 in *Nature Astronomy*.

Enceladus' icy surface is a shell that's completely detached from its rocky core (*SN: 10/17/15, p. 8*). On other icy moons, a parent planet's gravity flexes the ice and that bending generates enough energy to keep subsurface oceans warm. But ice on Enceladus is too thin to generate enough energy. If that were Enceladus' only heat source, its ocean would have frozen within 30 million years, a fraction of the age of the solar system, which formed roughly 4.6 billion years ago.

Planetary scientist Gaël Choblet of the University of Nantes in France and his colleagues tested whether friction in the sand and gravel thought to make up Enceladus' core could heat things up.

The team made computer simulations of water circulating through the spongy core using data from the Cassini spacecraft and from geoengineering experiments with sand and gravel on Earth. Depending on the core's makeup, the team found, the ocean should get enough heat to stay liquid for up to billions of years. — *Lisa Grossman*

BODY & BRAIN

How dad's stress alters his sperm

Sperm from stressed-out dads can carry that stress from one generation to another. Now a study in mice suggests some answers to the question of *how* a dad's experience changes his sperm.

Neuroendocrinologist Jennifer Chan of the University of Pennsylvania and colleagues focused on the part of the male reproductive tract called the caput epididymis, a place where sperm cells mature. Getting rid of a stress-hormone sensor there called the glucocorticoid receptor stopped the transmission of stress. Normally, when faced with an alarming predator odor, offspring of chronically stressed mice dads overproduce the stress hormone corticosterone. But mice dads that lacked this receptor in the epididymis had offspring with normal hormonal responses, Chan reported November 14 in Washington, D.C., at the annual meeting of the Society for Neuroscience.

Epididymis cells release small packets, or vesicles, filled with RNA that can fuse to sperm and change their genetic payload. Experiments on cells in dishes revealed that chronic exposure to corticosterone changes the RNA in these vesicles. The results offer an explanation of how stress can change sperm: By activating the glucocorticoid receptor, stress tweaks the RNA in epididymis vesicles. Then, those vesicles deliver their altered contents to sperm, passing stress to the next generation.

Chan and colleagues are now testing whether humans carry similar signs of stress in these RNA-loaded vesicles by studying college students' semen samples. — *Laura Sanders*

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Breaking Down Multiple Sclerosis

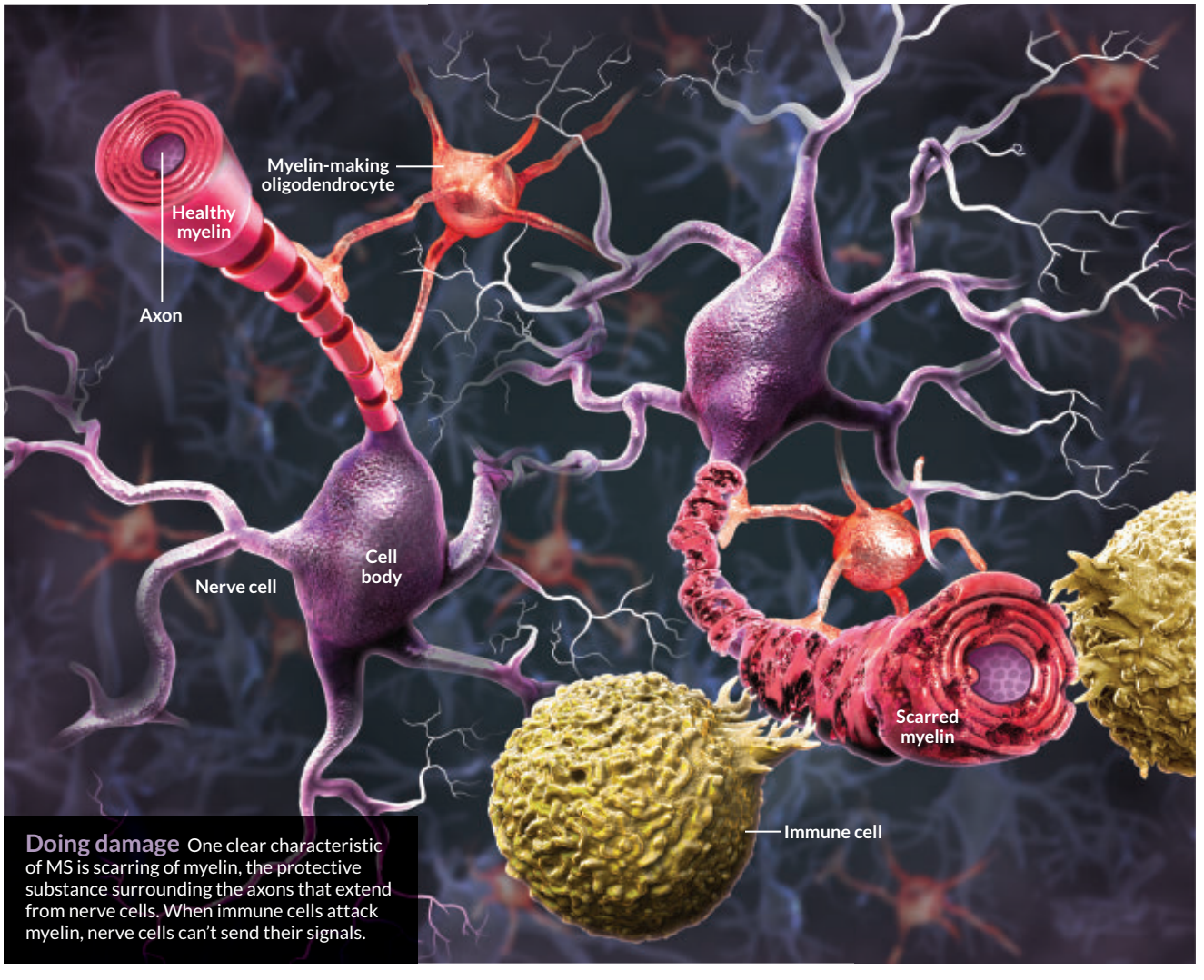
Researchers seek solutions beyond the immune system

By Ashley Yeager

James Davis used to be an avid outdoorsman. He surfed, hiked, skateboarded and rock climbed. Today, the 48-year-old from Albuquerque barely gets out of bed. He has the most severe form of multiple sclerosis, known as primary progressive MS, a worsening disease that destroys the central nervous system. Diagnosed in May 2011, Davis relied on a wheelchair within six months. He can no longer get up to go to the bathroom or grab a snack from the fridge.

Davis hoped life might improve when he was chosen in 2012 to participate in a clinical trial of a drug called ocrelizumab. The drug offered a first sliver of hope for patients waiting for a cure, or at least something to slow down the disease's staggering march. Early research suggested the drug could help some of the roughly 60,000 people in the United States, like Davis, suffering from primary progressive MS. The drug also held promise for patients with the other major form of the disease, relapsing-remitting MS, which afflicts about 340,000 people nationwide.

For some people, ocrelizumab seemed to work. Brain scans of patients with primary progressive MS showed fewer signs of damage and the patients' ability to walk deteriorated more slowly than in individuals who received a placebo, researchers reported in January in the *New England Journal of Medicine*. The drug also helped people with relapsing-remitting MS, which, as the name implies, includes shifts between disability and wellness. Over a year's time, these patients experienced about half as many flare-ups as those taking another commonly prescribed drug, a different research group reported in the same issue of the journal.



Doing damage One clear characteristic of MS is scarring of myelin, the protective substance surrounding the axons that extend from nerve cells. When immune cells attack myelin, nerve cells can't send their signals.

NICOLLE RAGER FULLER

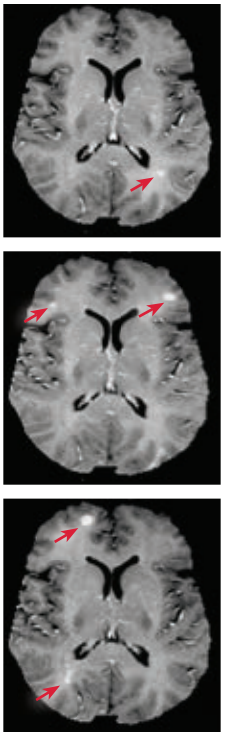
Ocrelizumab was heralded as a breakthrough, and in March, the U.S. Food and Drug Administration approved it as a treatment for primary progressive and relapsing-remitting MS. Genentech now sells the drug as Ocrevus.

"We finally have an approved therapy for primary progressive MS," says Fred Lublin, a neurologist at the Icahn School of Medicine at Mount Sinai in New York City. The first drug to treat relapsing-remitting MS came on the market in 1993, Lublin notes. Now, nearly a quarter century later, there's something that helps some people with the most aggressive form of the disease.

With that hopeful note, though, comes frustration. Ocrevus isn't a cure, and it offers no relief for 30 to 40 percent of patients with primary progressive MS. Davis was in that disappointed group.

In fact, none of the 15 FDA-approved drugs for MS, which all modify or suppress the immune system, actually stop the disease. The drugs only reduce the number and severity of flare-ups and, in some cases, slow the visible marks of brain damage.

"Multiple sclerosis is arguably the most complex disease ever described," says Sergio Baranzini, a geneticist at the University of California, San Francisco. The disease is so complicated, he says, because it involves two of the body's more complex systems — the nervous and immune systems. Scientists don't yet have a good handle on where the damage begins. Does a problem in the nervous system spur an immune response that leads to additional damage to the brain and spinal cord? Or does the immune system attack first, dispatching disease-fighting cells into the brain, where they batter and kill nerve cells? What causes the initial nerve damage or incites the immune attack is still a big question. Scientists aren't even clear whether multiple sclerosis is a single disease or a multitude of maladies.



Time-lapse MRI images show brain lesions (arrows) appearing and disappearing in a person with relapsing-remitting MS.

With so many unanswered questions, researchers have begun looking for potential treatment strategies outside the immune system. Targeting problems in the nervous system, together with the harmful immune reactions, is essential, scientists say. Some researchers have begun scrutinizing the malfunction of specific organelles within nerve cells, or neurons. Others are analyzing the gut's community of microorganisms, its microbiome, which is considered a bridge between the environment and the body. Those researchers are following up on observations that environmental influences play a role in MS. The research is in early stages. But for a challenging disease like MS, attacks on multiple fronts may be exactly what it takes to help Davis and others who are running out of time.

Disrupted signals

More than 2.3 million people worldwide suffer from multiple sclerosis, according to 2013 data, the most recent, from the MS International Federation in London. The disease affects people of all ages, and is more often diagnosed in young adults, women and people who live in northern latitudes. Certain gene variants are associated with an increased risk of disease, but MS is rarely directly inherited. In studies of identical twins, if one has MS, the other's risk increases by about 25 percent. But if a parent has MS, a child's risk of developing the disease is only 2.5 to 5 percent higher than in children of people who don't have MS.

Like Davis, most patients learn they have the disease after experiencing tingling or numbness in the fingers or toes, loss of balance or trouble walking. Vision problems are another early sign. Such symptoms are a result of faulty electrical signals in the central nervous system. Those signals are disrupted because myelin, the insulating sheath around a nerve cell's message-transmitting axons, becomes damaged. Many researchers think that breakdown is driven by the body's immune system: Immune cells flock to the brain, causing inflammation and myelin destruction, which leads to continued nerve cell damage. Depending on the damage and how quickly it accumulates, patients are diagnosed either with the primary progressive form, with a relatively quick decline in health, or the relapsing-remitting form. About half of people with the intermittent form eventually move into the progressive disease, typically within about a decade.

Overworked organelles

Because most if not all people with MS experience nerve cell damage, researchers have begun to focus on two organelles within the neuron's cell body: the power-generating mitochondria and the labyrinth-like endoplasmic reticulum, which makes many of the proteins, lipids and other molecules needed by the cell. These organelles keep a nerve cell running, but in MS, they are pushed into overdrive, working much harder than they should.

Over the last 20 years, researchers have linked malfunctions in mitochondria to neurodegenerative disorders such as Alzheimer's and Parkinson's. More recently, MS has joined the list. In a study published in January in the *Journal of Neuroinflammation*, cellular biologist Thomas Simmen of the University of Alberta in Edmonton, Canada, and colleagues revealed at least one way that mitochondria in nerve cells fail in people with MS.

Brain tissue from people who died with MS had higher levels of a protein called Rab32 compared with tissue from healthy brains, which had very little of the protein. Mice with a condition that mimics MS also had high levels of Rab32. When the endoplasmic reticulum is stressed, inflammation in the brain tissue increases, experiments in nerve cells show, and Rab32 increases too. Its levels rise most often in areas where

scavenger immune cells called macrophages have infiltrated damaged tissue.

In nerve cells, Rab32 is a troublemaker. It gloms on to the surfaces of the endoplasmic reticulum and mitochondria, controlling the organelles' interactions and mitochondrial behavior. In separate experiments, when Rab32 rises in response to stress in the endoplasmic reticulum, or ER, several things happen: The nerve cell fibers (both axons and the message-receiving dendrites) are shorter, mitochondria are bulkier than normal and their numbers spike. These changes indicate that the mitochondria are not working correctly, says Paul Eggleton, an immunologist at the University of Exeter in England and a coauthor of the study. Eventually, the nerve cell commits suicide.

The researchers call Rab32 a marker of MS progression. Other proteins are involved, which Simmen and Eggleton hope to identify. If researchers could develop drugs that target those proteins, perhaps they could stop nerve cell death and prevent MS from progressing, the two scientists say. However, scientists still want to figure out what causes the ER stress that leads to the protein rise. Simmen suggests that it could be a yet-to-be-determined defect with the endoplasmic reticulum itself.

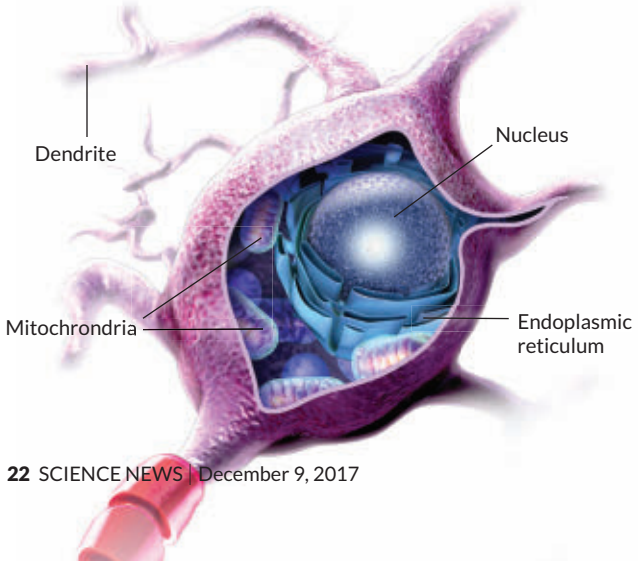
Figuring out how that stress starts is important, as it ties into one of the major characteristics of MS: damage to myelin.

Making myelin

Oligodendrocytes are the central nervous system cells that make myelin. One mature oligodendrocyte can coat about 50 axon segments with myelin. Like spiders, the oligodendrocytes sit alongside nerve cells tending to their myelin webs. When there's damage to myelin, oligodendrocytes repair it, or new oligodendrocytes take over for old ones.

But in people with MS, some oligodendrocytes stop making myelin altogether and young oligodendrocytes have trouble maturing. Most researchers think an immune attack blocks the oligodendrocytes from doing their job. But recent work raises questions about that idea.

Zooming in Inside a nerve cell body are power-generating mitochondria and the protein-constructing endoplasmic reticulum. Malfunctions of these organelles have been associated with MS.



In experiments in mice, researchers used a toxin to destroy myelin and kill oligodendrocytes. The mice had difficulty running on a spinning rod and showed other symptoms of nerve cell deterioration. Five weeks after the toxin injection, the neurological symptoms were at their worst, suggesting the toxin did its job. At 10 weeks, the mice recovered, probably because the brain can heal itself, says study coauthor Brian Popko, a neurologist at the University of Chicago. The brain creates new oligodendrocytes that wrap fresh myelin around the affected axons, Popko and colleagues reported in January 2016 in *Nature Neuroscience*.

At about 30 to 40 weeks, however, the mice went downhill. They lost control of their movements, lost weight and had seizures. By 52 weeks, half were dead. All of the mice had lesions in the brain stem, spinal cord and cerebellum — a region that coordinates and regulates muscle activity. The animals' brains were also inflamed, the team noted. It's as if the initial damage led to the development of a more severe condition down the road, a pattern that appears to mimic what happens in MS.

Popko says myelin-making cells are damaged regularly in healthy individuals and in those with MS. "Our idea is that these hits to the central nervous system in most people do not have long-term detrimental effects," he says. But people with MS have trouble making new oligodendrocytes and so, "central nervous system insults can lead to the development of the disease."

Popko and others are testing an approach to stop disease development: preserve oligodendrocytes' ability to handle stress.

Strengthening nerve cells

Oligodendrocytes have mitochondria and an endoplasmic reticulum too. If oligodendrocytes are making myelin in an environment with a lot of inflammation, as seen in MS patients, then the ER must work extra hard to make proteins to rebuild myelin and respond to surrounding inflammation. The cells eventually can't keep up, so they program their own death.

Preventing the ER from being overwhelmed and signaling cell suicide could be a strategy to treat MS, Popko wrote, along with University of Chicago neurologist Sharon Way, in a 2016 review article in *Lancet Neurology*. That's the aim of two drugs currently in testing. The drugs keep the ER working at a comfortable level, even during stress.

One of the drugs, Guanabenz, is a hypertension medication. In animal studies, the drug kept oligodendrocytes alive, easing MS symptoms and even delaying their onset. But there were side effects, including drowsiness and lethargy, effects sometimes experienced by people with high blood pressure who take the drug. An early trial in MS patients recently ended, but results haven't been reported yet. A second potential drug, Sephin1, which is still in animal testing, keeps oligodendrocytes alive without the side effects of Guanabenz, Popko says. Another drug, salubrinal, also designed to reduce ER stress, wasn't as effective in lab tests as researchers had hoped, he says, so testing has stopped.

NICOLLE RAGER FULLER

Drug options Fifteen medications are available to treat multiple sclerosis, each suppressing the immune system (five are described below). At least a dozen other drugs (two listed) are in various stages of development. SOURCES: NATIONAL MS SOCIETY; S.W. WAY AND B. POPKO/*LANCET NEUROLOGY* 2016

	Drug	Mode of action	Year approved
Available to patients	Betaseron® (interferon beta-1b)	Cell-signaling protein that boosts anti-inflammatory and reduces pro-inflammatory molecules in the brain	1993
	Novantrone® (mitoxantrone)	Chemotherapy drug that blocks creation of immune cells (T cells, B cells and macrophages)	2000
	Tysabri® (natalizumab)	Monoclonal antibody that prevents T cells from crossing into the brain	2006
	Gilenya® (fingolimod)	Small molecule that prevents T cells from crossing into the brain	2010
	Ocrevus™ (ocrelizumab)	Monoclonal antibody that triggers death of B cells	2017
	Drug	**Mode of action**	**Phase of testing**
In development	Guanabenz	Hypertension drug that protects cell from endoplasmic reticulum stress and preserves oligodendrocytes and myelin during inflammation	Early human studies
	Sephin1	A derivative of the drug Guanabenz with the same action but fewer side effects	Animal testing

Cleveland Clinic neuroscientist Ranjan Dutta says it is not clear that an overstressed ER and resulting mitochondrial failure, *causes* MS. There is no question mitochondria are defective in individuals with MS, he says. But the problem may be that the organelles cannot keep up with the energy demand of the cell as it tries to fend off an immune attack. And, if the immune attack comes first, as Dutta and Lublin suspect, what is the trigger?

Going with the gut

The gut is where the immune system learns about what's going on outside the body, says UCSF's Baranzini. Environmental factors are known to increase risk for MS (*SN: 4/16/16, p. 4*). Such factors include smoking and low levels of vitamin D in the blood (which may explain why MS is more common at higher latitudes, since sun exposure boosts vitamin D).

"We see the microbiome as a window into the environment," Baranzini says. With that in mind, he and colleagues compared the gut bacteria of 71 people with relapsing-remitting MS with bacteria from 71 healthy people. Two bacterial groups, *Acinetobacter* and *Akkermansia*, were four times as abundant in MS patients as in healthy individuals. Another bacterial group, *Parabacteroides*, was four times as abundant in people without the disease.

Next, the team exposed immature immune cells from the blood of healthy people to the bacteria found in the guts of MS patients. When the immune cells encountered *Acinetobacter* and *Akkermansia*, they matured into the T helper cells that trigger inflammation. *Acinetobacter* also tamped down regulatory T cells, which keep autoimmune diseases at bay.

When the researchers transferred gut microbes from MS patients or their healthy spouses into mice that had an MS-like condition, the mice given the MS gut microbes got much sicker, Baranzini and colleagues reported in the Oct. 3 *Proceedings of the National Academy of Sciences*.

"It was surprising to see that the differences in bacteria were enough to cause immunological imbalances in the mice," he says. "It implicates the gut microbiome as a cause of MS."

Dutta and other scientists are not yet convinced. Microbiome studies are "encouraging, despite early days," Dutta says. He notes, however, that researchers need to develop more rigorous ways to test the role of the gut microbiome in controlling immune reactions. "This is primarily because of the difference between the mouse and human immune systems." It will also be important to know how the bacteria operate to affect the immune system, he adds.

The results aren't completely unheard of. In the same issue of *PNAS*, researchers found higher levels of *Akkermansia* in twins with MS versus their healthy sibling. The scientists transferred the twins' gut bacteria to mice predisposed to develop a disease that mimics MS. Twelve weeks after the transplant, about 40 percent more mice with gut microbes from a twin with MS developed brain inflammation compared with mice that got gut microbes from a twin without disease.

If researchers can get a handle on the gut microbiome's role, Baranzini can imagine a day when probiotics can be used to shift the composition of microbes in the gut to reduce inflammation. But more work needs to be done, and at least one major question needs answering: Do people with relapsing-remitting MS have a different set of gut bacteria than people with the primary progressive form of the disease?

"We're doing that experiment right now," Baranzini says. He also hopes to expand his analysis to include MS patients from around the world, who eat different diets and may have different gut bacteria, to help pinpoint the microbes that may be contributing to the disease.

MS drugs that target the immune system provide some benefit by reducing relapses, but these drugs can't seem to prevent long-term neurodegeneration, Popko says. Combining current drugs with strategies to protect oligodendrocytes, enhance myelin-making and possibly wrangle the immune system through the gut might be the best strategy for achieving prolonged benefit for people with MS. ■

Explore more

- Alan J. Thompson. "Challenge of progressive multiple sclerosis therapy." *Current Opinion in Neurology*. June 2017.

Three million years ago, Earth's climate was so warm that the High Arctic supported forests (illustrated) in which camels and other animals roamed.

Lessons from the PLIOCENE

A warm period in the past offers a window to the future

By Alexandra Witze

Imagine a world where the polar ice sheets are melting, sea level is rising and the atmosphere is stuffed with about 400 parts per million of carbon dioxide. Sound familiar? It should. We're living it. But the description also matches Earth a little over 3 million years ago, in the middle of the geologic epoch known as the Pliocene.

To understand how our planet might respond as global temperatures rise, scientists are looking to warm periods of the past. These include the steamy worlds of the Cretaceous Period, such as around 90 million years ago, and the boundary of the Paleocene and Eocene epochs, about 56 million years ago.

But to many researchers, the best reference for today's warming is the more recent Pliocene, which lasted from 5.3 million to 2.6 million years ago. The mid-Pliocene was the last time atmospheric CO_2 levels were similar to today's, trapping heat and raising global temperatures to above the levels Earth is experiencing now.

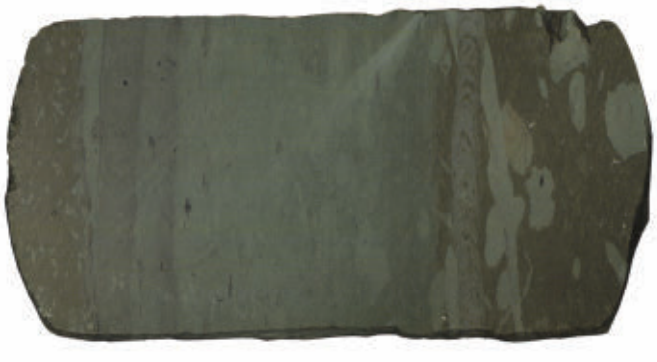
New research is illuminating how the planet responded to Pliocene warmth. One set of scientists has fanned out across the Arctic, gathering geologic clues to how temperatures there may have been as much as 19 degrees Celsius higher than today. The warmth allowed trees to spread far to the north, creating Arctic forests where three-toed horses, giant camels and other animals roamed. When lightning struck, wildfires roared across the landscape, spewing soot into the air and altering the region's climate.

Other researchers are pushing the frontiers of climate modeling, simulating how the oceans, atmosphere and land responded as Pliocene temperatures soared. One new study shows how the warmth may have triggered huge changes in ocean circulation, setting up an enormous overturning current in the Pacific Ocean, similar to the "conveyor belt" in today's Atlantic that drives weather and climate. A second new paper suggests that the Greenland and Antarctic ice sheets might have responded differently to Pliocene heat, melting at different times.

All this research into the last great warm period is helping scientists think more deeply about how the future might play out. It may not be a road map to the next 100 years, but the Pliocene is a rough guide to the high sea levels, vanishing ice and altered weather patterns that might arrive hundreds to thousands of years from now.

"It's a case study for understanding how warm climates function," says Heather Ford, a paleoceanographer at the

Chemical and geologic clues within deep-sea sediment cores, like this one from the Pliocene drilled from the seabed off Alaska's southern coast, can reveal past ocean temperatures. Paleoclimatologists use the data to reconstruct which periods were warm and which were cold.



FROM TOP: JULIUS T. CSOTONYI; JRSO/INTERNATIONAL OCEAN DISCOVERY PROGRAM

University of Cambridge. “It’s our closest analog for future climate change.”

Walk through history

Teasing out the history of the Pliocene is a little like digging through a family’s past. One group of enthusiasts goes through genealogical records, collecting data on who lived where, and when. Another group uses computer software and modeling to look for broad patterns that describe how the family grew and moved over time.

The data detectives begin their work in rocks and sediments dating to the Pliocene that are scattered around the world like family-tree histories in city library archives. In 1988, the U.S. Geological Survey began a project called PRISM, for Pliocene Research, Interpretation and Synoptic Mapping, which aims to gather as many geologic clues as possible about Pliocene environments.

At its start, PRISM focused on a collection of deep-sea cores drilled from the floor of the North Atlantic Ocean. Different types of marine organisms thrive in water of different temperatures. By comparing the relative abundance of species of tiny organisms preserved in the deep-sea cores, PRISM scientists could roughly map how cold-loving organisms gave way to warm ones (and vice versa) at different times in the past. Early results from the project, reported in 1992 by USGS research geologist Harry Dowsett and colleagues, showed that during the Pliocene, warming was amplified at higher latitudes in the North Atlantic.

Scientists continue to add to the PRISM records. One international team drilled a sediment core from beneath a Siberian lake and found that summer air temperatures there, in the mid-Pliocene, were as high as 15° C (about 59° Fahrenheit). That’s 8 degrees warmer than today (*SN: 6/15/13, p. 13*). Other researchers uncovered clues, such as plant fossils from peat bogs, that suggest mean annual temperatures on Canada’s now-frozen Ellesmere Island near Greenland were as much as 18 degrees higher than today (*SN: 4/6/13, p. 9*).

Now, a new group of biologists, geoscientists and other experts in past landscapes have banded together in a project called PoLAR-FIT, for Pliocene Landscape and Arctic Remains — Frozen in Time. The team is focusing on the Arctic because, just as today’s Arctic is warming faster than other parts of the planet, the Pliocene Arctic warmed more than the rest of the globe. “That’s what we call polar amplification,” says Tamara Fletcher, a team member and paleoecologist at the University of Montana in Missoula. “It was even more magnified in the Pliocene than what we’re seeing today.”

PoLAR-FIT scientists travel to the Arctic to collect geologic evidence about how the region responded to rising temperatures in the Pliocene. In the thawing permafrost slopes of Ellesmere Island, for instance, Fletcher and colleagues have been mapping black layers of charcoal in sediments dating from the Pliocene. Each charcoal layer represents a fire that burned through the ancient forest. By tracking the events across

Wildfires swept the Arctic during the warm Pliocene, leaving behind beds of dark charcoal (top). As temperatures rise, Arctic fires, like the one that burned in Greenland in August (bottom), may become more common.

Ellesmere and other nearby islands, Fletcher’s team discovered that fire was widespread across what is now the Canadian Arctic.

Wildfires changed vegetation across the landscape, possibly altering how the Arctic responded to rising temperatures. Soot rising from the fires would have darkened the skies, potentially leading to local or regional weather changes. “How important is that to the warming?” asks Bette Otto-Bliesner, a paleoclimatologist at the National Center for Atmospheric Research in Boulder, Colo. “That’s something we’re still trying to determine.” Fletcher, Otto-Bliesner and colleagues described the charcoal discovery, along with modeling studies of the fires’ effects, in Seattle in October at a meeting of the Geological Society of America.

In 2012, about 283,280 square kilometers of forest burned in Russia. Three years later, more than 20,230 square kilometers burned in Alaska. Last summer, a wildfire broke out in the icy landscape of western Greenland. “We’re already seeing fire in the Arctic, which is unusual today,” Fletcher says. “But it wouldn’t have been unusual in the Pliocene.”

While the work doesn’t predict how much of the Arctic will burn as temperatures rise, the findings do suggest that people need to prepare for more fires in the future.

Trapped ocean heat

Scientists like Fletcher are the genealogists of the Pliocene, collecting records of past environments. Other researchers — the computer modelers — put those old records into broad context, like historians analyzing family trees for patterns of migration and change.

The modelers begin with data on Pliocene temperatures — such as how hot it got on Ellesmere Island or in the North

Atlantic Ocean, as revealed by plant fossils or seafloor sediments. Scientists can also estimate how much CO_2 was in the atmosphere at the time by looking at clues such as the density of holes in fossilized leaves of Pliocene plants, which used those openings to take up CO_2. Estimates vary, but most suggest CO_2 levels were about 350 to 450 ppm in the mid-Pliocene.

It's not clear what caused the gas buildup during the Pliocene; one possibility is it came from long-term changes in the way carbon cycles between the land, ocean and atmosphere. But no matter the source, the high levels of CO_2 caused temperatures to soar by trapping heat in the atmosphere.

The Pliocene isn't a perfect crystal ball for today. For starters, scientists know why CO_2 levels are now increasing — burning of fossil fuels and other human activities (*SN: 5/30/15, p. 15*). As the Industrial Revolution was gaining steam, in the 19th century, atmospheric CO_2 levels were around 280 ppm. Today that level is just above 400 ppm, and rising.

Modeling the Pliocene climate can help reveal how Earth responded in somewhat similar conditions. That means studying changes in the Pliocene atmosphere, the land surface and most of all the oceans, which absorb the bulk of planetary warming. "That's the sort of thing you can understand from studying past warm episodes," Ford says. "What was different about how heat and carbon were moving around in the ocean?"

Ford has begun working with climatologist Natalie Burls of George Mason University in Fairfax, Va., to try to track how the oceans' major water masses shifted during the Pliocene. Today the North Atlantic has a deep, cold, salty layer that is crucial to the ocean's "conveyor belt" circulation. In this pattern, warm waters flow northward from the tropics, then cool and become saltier and denser as they reach higher latitudes. That cool water sinks and travels southward, where it warms and rises and begins the cycle all over again.

Foraminifera, tiny sea organisms, are used to calculate past ocean temperatures, by the chemistry of their shells and the amount of cold-loving versus warm-loving species present in deep-sea cores.

This conveyor belt circulation is important to today's Atlantic climate, because it allows the warm Gulf Stream to moderate temperatures from the U.S. East Coast to Western Europe. Burls and colleagues have now found that a similar pattern might have existed in the Pacific during the Pliocene. They call it the Pacific meridional overturning circulation, or PMOC, just as today's similar Atlantic circulation is known as the AMOC.

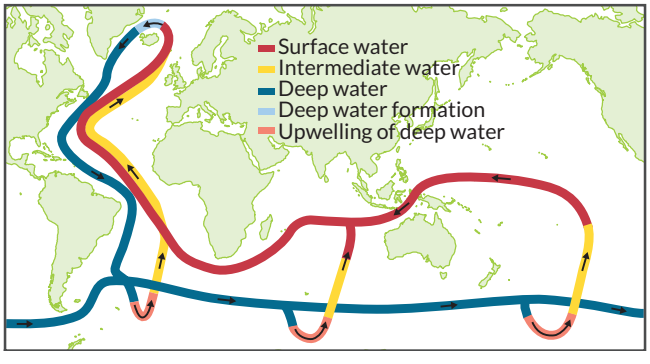
Burls' team discovered this phenomenon by modeling how the Pliocene ocean would have responded to higher temperatures. Because the Arctic was so warm, the temperature difference between the equator and the mid- and high latitudes was not as great as it is today. The weaker temperature gradient would have meant less rainfall and more evaporation in the midlatitude North Pacific. As a result, its uppermost waters would have gotten saltier.

When the North Pacific waters got salty enough, they cooled and sank, setting up an enormous current that dove deep off the coast of northeastern Russia and traveled southward until the water warmed enough to once again rise toward the surface. Real-world data back the claim: Accumulations of calcium carbonate in deep-sea Pacific sediments show that the Pliocene ocean experienced huge shifts at the time, with waters churning all the way from the surface down to about three kilometers deep, as would be expected from a conveyor belt–type circulation. The team reported the finding in *Science Advances* in September.

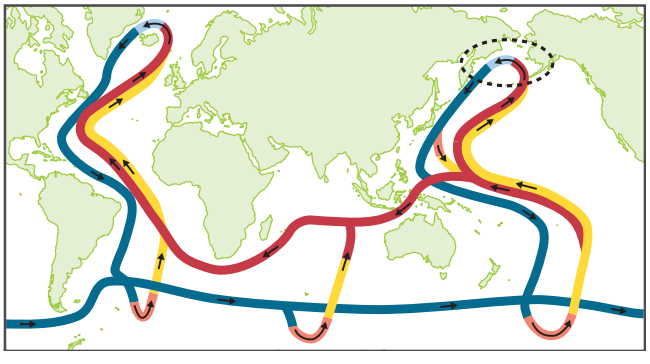
What happened in the Pliocene Pacific may say something about the Pacific of the distant future, Burls says. As temperatures rise today, most of the heat is being taken up by the surface layers of the oceans. Over the short term, that works to prevent changes in deep ocean circulation. "Today we're very quickly turning on the heating, and it will take a while for the deep ocean to adjust," Burls says.

Deep ocean changes Ocean circulation today is driven in part by a deep circulation pattern in the North Atlantic (left). Currents there flow north and then become cooler and saltier, causing the water to sink and return southward in a conveyor belt–like circulation. In the warm Pliocene, some 3 million years ago, a similar conveyor belt may have set up in the Pacific (right) thanks to reduced rainfall in the North Pacific (dotted circle).

Modern day



Pliocene



FROM TOP: USGS; N. BURLS

But in the longer term, thousands of years from now, waters in the North Pacific may eventually become warm and salty enough to establish a PMOC, just as there was in the Pliocene. And that could lead to major changes in weather and climate patterns around the globe.

Land bridges and ice sheets

Other modelers are looking beyond the Pacific to improve their understanding of how different parts of the Pliocene world behaved. About a dozen research groups recently launched a new effort called PlioMIP2, or Pliocene Model Intercomparison Project, Phase 2, to model the climate of a time somewhat similar to today in the mid-Pliocene, about 3.205 million years ago.

"We're working to produce the best picture that we can of what life seemed to be like at the time," says Alan Haywood, a climate modeler at the University of Leeds in England and a leader of the effort.

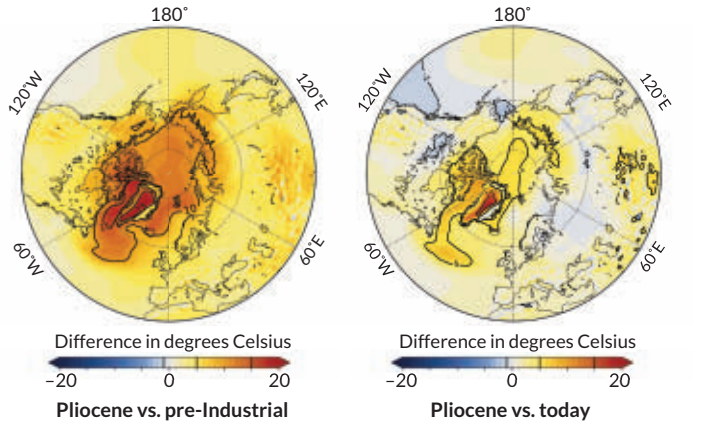
In one discovery, project scientists have found that small changes in the geography of their modeled world make a big improvement in the final results. Early models did not accurately capture how much the polar regions heated up. So PlioMIP2 researchers updated their starting conditions. Instead of assuming that the landmasses of the Pliocene world were identical to today, the group made two small, plausible changes in the Arctic. The researchers made a land bridge between Russia and Alaska by closing the Bering Strait, and they added land to connect a few modern islands in the Canadian Arctic, including Ellesmere.

The change "seems small, but it actually can have a huge impact on climate," says Otto-Bliesner. For instance, closing the Bering Strait cut off a flow in which relatively fresh water from the Pacific travels over the Arctic and into the North Atlantic. With the updated geography, the PlioMIP2 models suddenly did a much better job of simulating heat in the high Arctic.

Otto-Bliesner will describe the team's results in New Orleans this month at a meeting of the American Geophysical Union. Another PlioMIP2 group, Deepak Chandan and Richard Peltier of the University of Toronto, reported similar findings in July in *Climate of the Past*. They too found that closing the Bering Strait allowed their model to better simulate the Arctic heating.

Other Pliocene modelers are trying to figure out how the planet's enormous ice sheets in Greenland and Antarctica might respond to rising temperatures. Geologic evidence, such as ancient beaches from the Pliocene, suggest that global sea levels then were as much as 25 meters higher than today. If all of Greenland's ice were to melt, global sea levels would rise about six meters; if all of Antarctica went, it would contribute about 60 meters. So parts of these ice sheets, but not all, must have melted during the long-ago warm period.

Several of the PlioMIP2 research groups are modeling how the polar ice sheets responded in the heat of the Pliocene. "It will tell us how much we should be worried," Otto-Bliesner says.



Arctic heat-up Simulations of the Arctic during the Pliocene show excess heat (deeper red colors) compared with before the Industrial Revolution (left) and today (right). Today's differences are smaller because levels of heat-trapping carbon dioxide in the atmosphere are closer to Pliocene levels.

One new study suggests that the northern and southern ice sheets may have behaved out of phase with each other. In a simulation of the mid- to late Pliocene, climate modeler Bas de Boer of Utrecht University in the Netherlands and colleagues found that as Greenland's ice melted, Antarctica's ice could have been relatively stable, and vice versa.

"At different points, they could be contributing to the sea level story or against it," says Haywood. He, along with colleagues, reported the results in the Oct. 30 *Geophysical Research Letters*.

That out-of-sync melting suggests the Pliocene was a complicated time. Just because global temperatures were high doesn't mean that all of Earth's ice sheets melted equally. (Today, both Greenland and West Antarctica are losing ice to the oceans as global temperatures rise.)

The Pliocene wound to an end around 2.6 million years ago, as CO_2 levels dropped. Chemical reactions with eroding rocks may have sucked much of the CO_2 out of the atmosphere and tucked it away in the oceans, removing the greenhouse gas. The planet entered a long-term cooling trend. Since the end of the Pliocene, Earth has been in and out of a series of ice ages.

But now, greenhouse gases are once again flooding into the atmosphere. Global temperatures are ticking up inexorably year after year. That makes the lessons of the past all the more relevant for the future. ■

Amount of CO_2 in Earth's atmosphere (parts per million)

Mid-Pliocene
350–450

Pre-Industrial
280

Today
400+

Explore more

- Alan M. Haywood, Harry J. Dowsett and Aisling M. Dolan. "Integrating geological archives and climate models for the mid-Pliocene warm period." *Nature Communications*. February 16, 2016.

D. CHANDAN AND RICHARD PELTIER

Hedy Lamarr longed to be recognized for her inventions.

FILM

Hollywood icon was also an inventor

Once billed as "the most beautiful woman in the world," actress Hedy Lamarr is often remembered for Golden Age Hollywood hits like *Samson and Delilah.* But Lamarr was gifted with more than just a face for film; she had a mind for science.

A new documentary, *Bombshell: The Hedy Lamarr Story*, spotlights Lamarr's lesser-known legacy as an inventor. The film explores how the pretty veneer that Lamarr shrewdly used to advance her acting career ultimately trapped her in a life she found emotionally isolating and intellectually unfulfilling.

Lamarr, born in Vienna in 1914, first earned notoriety for a nude scene in a 1933 Czech-Austrian film. Determined to rise above that cinematic scarlet letter, Lamarr fled her unhappy first marriage and sailed to New York in 1937. En route, she charmed film mogul Louis B. Mayer into signing her. Stateside, she became a Hollywood icon by day and an inventor by night.

Lamarr's interest in gadgetry began in childhood, though she never pursued an engineering education. Her most influential brainchild was a method of covert radio communication called frequency hopping, which involves sending a message over many different frequencies, jumping between channels in an order known only to the sender and receiver. So if an adversary tried to jam the signal on a certain channel, it would be intercepted for only a moment.

During World War II, Lamarr partnered with composer George Antheil to design a frequency-hopping device for steering antisubmarine torpedoes. The pair got a patent, but the U.S. Navy didn't take the invention seriously. "The Navy basically told her, 'You know, you'd be helping the war a lot more, little lady, if you got out and sold war bonds rather than sat around trying to invent,'" biographer Richard Rhodes says in the film. Ultimately, the film suggests, Lamarr's bombshell image and the sexism of the day stifled her inventing ambitions. Yet, frequency hopping paved the way for some of today's wireless technologies.

Bombshell
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Throughout *Bombshell,* animated sketches illustrate Lamarr's inventions, but the film doesn't dig deep into the science. The primary focus is the tension between Lamarr's love of invention and her Hollywood image. With commentary from family and historians, as well as old interviews with Lamarr, *Bombshell* paints a sympathetic portrait of a woman troubled by her superficial reputation and yearning for recognition of her scientific intellect. —*Maria Temming*

A Cold Welcome
Sam White
HARVARD UNIV., $29.95

BOOKSHELF

Climate foiled European explorers

Many people may be fuzzy on the details of North America's colonial history between Columbus' arrival in 1492 and the Pilgrims' landing on Plymouth Rock in 1620. But Europeans were actively attempting to colonize North America from the early 16th century onward, even though few colonies survived.

As historian Sam White explains in *A Cold Welcome*, most early attempts were doomed by fatally incorrect assumptions about geography and climate, poor planning and bad timing.

White weaves together evidence of past climates and written historical records in a comprehensive narrative of these failures. One contributing factor: Explorers assumed climates at the same latitude were the same worldwide. But in fact, ocean currents play a huge role in moderating land temperatures, which means Western Europe is warmer and less variable in temperature from season to season than eastern North America at the same latitude.

On top of that, explorations occurred during a time of global cooling known as the Little Ice Age, which stretched from the 13th to early 20th centuries. The height of exploration may have occurred at the peak of cooling: Starting in the late 16th century, a series of volcanic eruptions likely chilled the Northern Hemisphere by as much as 1.8 degrees Celsius below the long-term average, White says.

This cooling gave Europeans an especially distorted impression of their new lands. For instance, not long after Spanish explorer Sebastián Vizcaíno landed in California's Monterey Bay in December 1602, men's water jugs froze overnight — an unlikely scenario today. Weather dissuaded Spain from further attempts at colonizing California for over a century.

Harsh weather also heightened conflict when underprepared Europeans met Native Americans, whose own resources were stretched thin by unexpectedly bad growing seasons.

A Cold Welcome is organized largely by colonial power, which means findings on climate are repeated in each chapter. But White's synthesis of climate and history is novel, and readers will see echoes of today's ignorance about the local consequences of climate change. "Human psychology may be both too quick to grasp at false patterns and yet too slow to let go of familiar expectations," White writes. —*Diana Steele*

ADRIAN CURRY/ZEITGEIST FILMS

To see where *Bombshell* is playing, visit **bit.ly/Bombshell_film**

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2017 Broadcom MASTERS awards $100,000 in prizes

Congratulations to the winners of the 2017 Broadcom MASTERS, who took home a total of $100,000 in awards. These middle school students were recognized in October for their achievements in science, technology, engineering and mathematics (STEM), critical thinking, communication, creativity and collaboration during the seventh annual Broadcom MASTERS competition.

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Santa Fe, N.M.

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Emily Tianshi
San Diego, Calif.

ROBERT WOOD JOHNSON FOUNDATION AWARD FOR HEALTH ADVANCEMENT: **$20,000**

Meghna Behari
Sewickley, Pa.

MARCONI/SAMUELI AWARD FOR INNOVATION: **$10,000**

Helen Lyons
New York, N.Y.

LEMELSON AWARD FOR INVENTION: **$7,500**

Science Award

Pujita Tangirala
Los Gatos, Calif.
FIRST PLACE

Jessie Gan
San Diego, Calif.
SECOND PLACE

Engineering Award

Andrew Chiang
Saratoga, Calif.
FIRST PLACE

Spencer Green
Huntington Beach, Calif.
SECOND PLACE

Rising Stars Award

Mithra Karamchedu
Portland, Ore.

Regan Williams
Wilmington, N.C.

Technology Award

Herin Kang
Los Gatos, Calif.
FIRST PLACE

Anthony Hill
Holladay, Utah
SECOND PLACE

Mathematics Award

Zoe Gotthold
Richland, Wash.
FIRST PLACE

Sara L. Kaufman
Cooper City, Fla.
SECOND PLACE

Scott A. McGregor Leadership Award

Arjun Moorthy
Scottsdale, Ariz.

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Culture club

The book Rise of the Necrofauna *tackles the challenges of using gene-editing tools to bring woolly mammoths and other long-gone species back from the dead. These "de-extincted" creatures would have to contend with a radically changed world that includes new habitats and diseases,* ***Tina Hesman Saey*** *wrote in her review "Species resurrection raises ethical questions"* (SN: 10/28/17, p. 28). Readers online had a lot to say.

"You can bring these animals back physically, but many animal species also have culture and cultural evolution," reader **Greg** wrote. "How are we to resurrect that?"

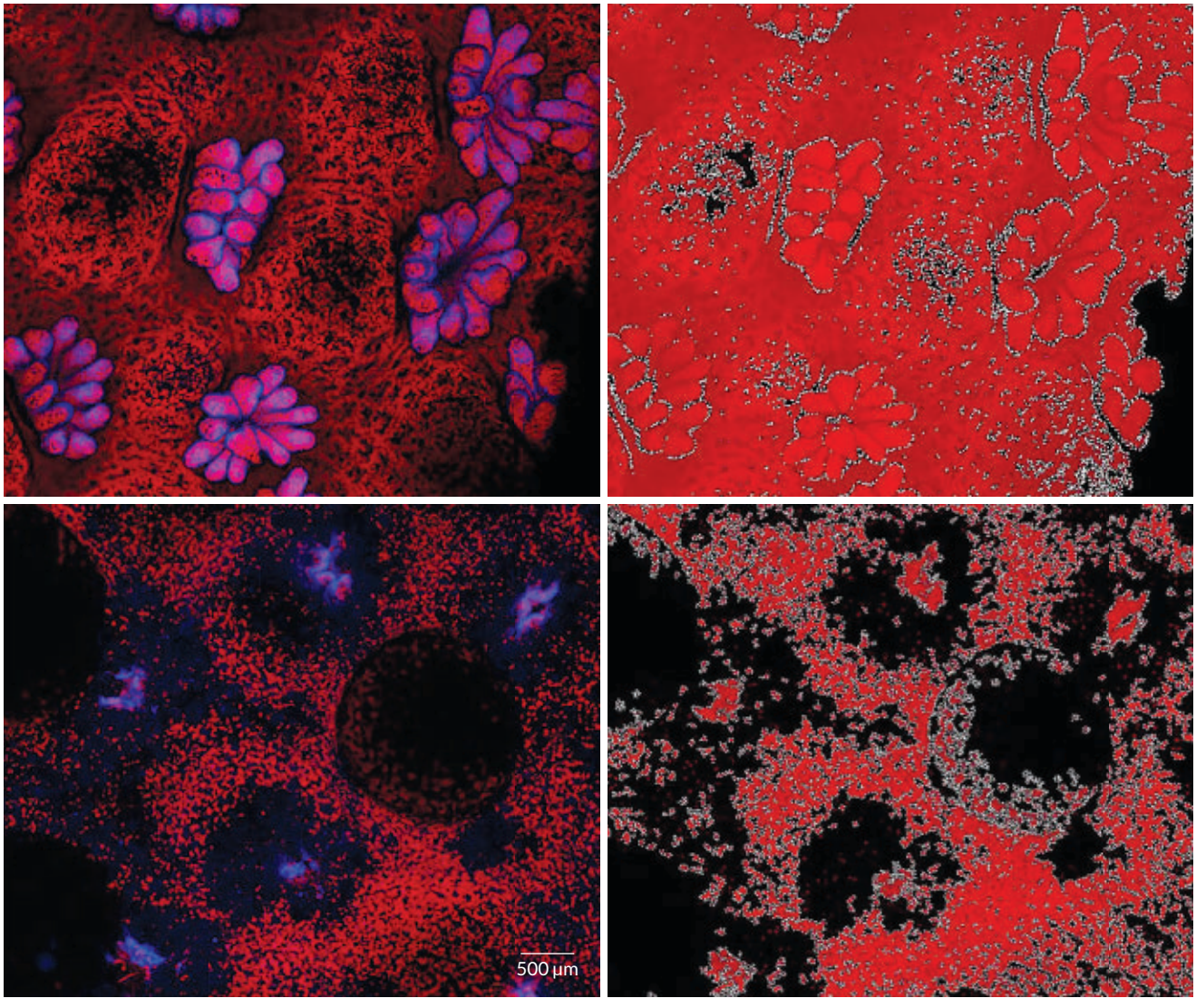
Whether some animals have culture is hotly debated. Another reader agreed that resurrected species' behaviors should be considered. "But that could be something that emerges naturally as they interact with the environment," **MJF Images** wrote. "These animals would not be the same as those that went extinct," and may have different behaviors.

Reader **John Turner** noted that some breeding programs for endangered species already account for environment. For example, conservationists have reared endangered California condors using condorlike hand puppets and minimal human contact, **Turner** wrote. To resurrect the woolly mammoth, "we shall require numerous copies of the Mr. Snuffleupagus costume," he joked.

Corrections

In "Net to halt runaway airliners" (*SN: 10/28/17, p. 4*), the description of the nets used in airplane arresting systems in 1931 is incorrect. The nets were not made of nylon until after the material was invented in 1935. Instead, the nets were most likely made of canvas.

The photo of the whiptail lizard featured in "The fuzzy art of defining species" (*SN: 11/11/17, p. 22*) should have been credited to Roger Shaw.



How to spot sick corals early

Sickness makes some corals lose their glow. Disease reduces a coral's overall fluorescence even before any sign of the infection is visible to the naked eye, a new study finds. An imaging technique that illuminates the change could help with efforts to better monitor coral health, researchers say November 6 in *Scientific Reports*.

Many corals naturally produce fluorescent proteins that glow in a wavelength that human eyes can't see in natural light. Heat stress and wounding, among other stressors, are known to affect coral fluorescence, but the new study is the first to look at fluorescence and infectious disease.

Disease ecologist Jamie Caldwell of Stanford University and colleagues used a technique called live-imaging laser scanning confocal microscopy to compare living fragments of healthy (top left) and diseased (bottom left) *Montipora capitata* coral. The reef coral, common in Hawaii, fluoresces in red and cyan and can contract a bacterial infection called *Montipora* white syndrome, which causes lesions and tissue loss.

The diseased bits looked healthy, but under the researchers' microscope, the sick coral's pallid complexion was pronounced. Computer analyses of the microscopy images (right column) quantified the lost glow (red is the total area of fluorescence and black is where fluorescence was lost). Among the samples studied, healthy coral had on average 1.2 times as much fluorescence area as diseased samples. Diseased coral had fragmented patterns of fluorescence. The coral appeared patchy, similar to a forest that has been logged extensively, the researchers found.

Many coral diseases appear to be increasing around the world, Caldwell says. Along with heat stress and pollution, which can lead to bleaching events, disease is considered one of the major contributors to reef declines globally. The new technique could be used for other coral species and diseases, she says. — *Emily DeMarco*

ALL: J.M. CALDWELL *ET AL/SCIENTIFIC REPORTS* 2017

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