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ScienceNews

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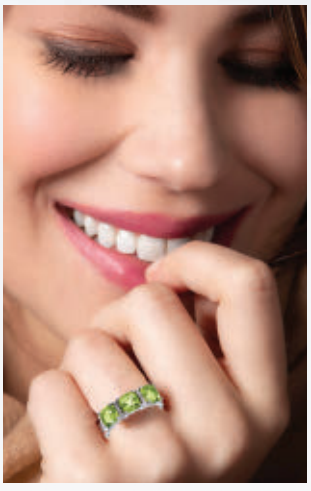

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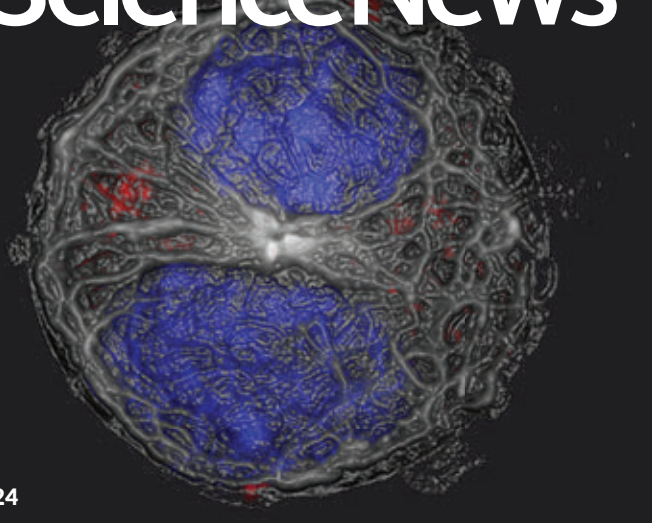

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COVER Claims made by commercial stem cell clinics go way beyond what science says stem cells can do. *Kat Goodloe*

FROM TOP: MARK KIEL AND SEAN MORRISON/CHILDREN'S MEDICAL CENTER RESEARCH INST./UT SOUTHWESTERN; A.L. FAYET; FREDRIK JUTFELT/NORWEGIAN UNIV. OF SCIENCE AND TECHNOLOGY

Pain opens the door for dubious stem cell therapies

About 50 million adults in the United States have chronic pain, which can make daily life a struggle, spark anxiety and depression, and lead to a cascade of problems for individuals, families and society. We've learned the hard way that opioids are not the answer. Wouldn't it be great if there was a solution that was safe, effective and cutting-edge?

Enter stem cells. These starter kits for the human body have been used for years to treat disease, as in the case of bone marrow transplants to replace immune cells destroyed by chemotherapy. But the usefulness of stem cells so far has been limited; they're tricky beasts that don't easily bend to our will. Scientists are experimenting to see if they can re-engineer the cells to treat a range of diseases, including heart failure and Parkinson's. But the purveyors of promise at stem cell clinics aren't waiting for the scientists' results and are enthusiastically marketing stem cell treatments to ease pain, including from arthritis, which affects 23 percent of adults in the United States, among other problems.

This issue's cover story, "Stem cell hard sell" (Page 24), details how people are sometimes being misled or injured by practitioners selling stem cells. Ads proliferating on billboards and online tell us just what we'd like to hear: all natural, no surgery, regenerative. Globally, it's a $2 billion business — and growing, reports our contributing correspondent Laura Beil. None of the treatments in the ads have been approved by the U.S. Food and Drug Administration. And there's scant evidence of benefit.

Researchers are trying to figure out if stem cell treatments can help with conditions such as knee arthritis. Over decades of use, the cartilage in knees can break down, causing pain, stiffness and swelling. The goal is to have stem cells help restore cartilage, or at least reduce pain. Doing a controlled trial requires using a sham treatment, for example, an injection of saline, and some studies have found that people improved just as much with saline injections as with stem cells. Scientists don't yet know if the saline really helped, or if it had more of a placebo effect. Given the lack of clarity, it's easy to imagine talking oneself into giving stem cells a try.

That's especially so since evidence-based treatments for knee arthritis lack the sheen of cutting-edge technology. The U.S. Centers for Disease Control and Prevention recommends 150 minutes of moderate physical activity each week, which is much cheaper than stem cell treatments and proven to reduce pain and lift mood.

I'm dismayed that people in pain are being exploited by some bad actors, and I hope Beil's excellent article (and her new podcast on stem cell scams, "Bad Batch") will help more people learn that very few of these treatments have been tested for safety and effectiveness. Too often early research results in petri dishes and lab animals are described as breakthroughs, only to not pan out later. Humans, being who we are, remember the exciting potential, not the cautions that any possible benefit remains very far away.

— Nancy Shute, Editor in Chief

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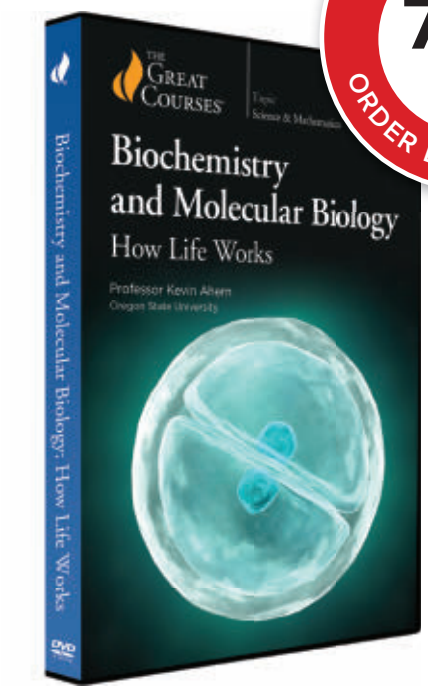

Understand the Science of Us

Biochemistry and its allied field of molecular biology are the fundamental sciences of life and the cornerstones of today's biotechnology revolution. But despite being about a subject that concerns us all—life—they are considered almost unapproachably difficult by non-scientists. Not anymore.

Biochemistry and Molecular Biology: How Life Works is an accessible and entertaining introduction to this vital discipline in 36 half-hour lectures. With lavish visuals, it is tailored to viewers with no more of a science background than high school chemistry. Using innovative teaching methods, Professor Kevin Ahern of Oregon State University introduces viewers to amino acids, proteins, enzymes, genes, and dealing with the intricate workings of living cells. Together, biochemistry and molecular biology have sparked a scientific revolution every bit as momentous as Einstein's relativity or Hubble's discovery of the expanding universe, but at the core, this remarkable field of study, says Professor Ahern in **Biochemistry and Molecular Biology**, is truly just"the science of us."

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Excerpt from the January 31, 1970 issue of *Science News*

50 YEARS AGO

Vaccine controversy

In the early 1950s, when smallpox was declared officially to have been wiped out in the United States, specialists in infectious diseases began to argue whether the risks of [smallpox] vaccination finally outweighed the benefits.... Today, many specialists hold that vaccination against smallpox is no longer justified in this country.

UPDATE: Routine smallpox vaccinations in the United States ended by 1972 and globally by 1980, when the disease was declared eradicated. But the world isn't totally safe from smallpox. Russian and U.S. labs keep samples of the disease-causing virus for research, and weaponized versions may exist. In 2003, the U.S. government pushed to restart vaccinations for health workers and first responders as a precaution against potential bioterrorist attacks. That effort failed, partly because people feared the possibility of health complications from the vaccine. But U.S. officials have stockpiled smallpox vaccines, along with the first smallpox treatment, which was approved for use in 2018 (*SN: 5/26/18, p. 10*).

SOAPBOX

Clinical trial volunteers merit scientists' support

Some see clinical trials as a chance for a miracle cure. In reality, these experimental drug tests and medical interventions often fail. With researchers in the United States testing the gene editor CRISPR/Cas9 for the first time in people (*SN: 8/31/19, p. 6*), one bioethicist says the scientists who are running these trials must remember they are responsible for their study volunteers.

That's not to say scientists conducting clinical trials are doing anything wrong, or that such studies should stop, says Laurie Zoloth of the University of Chicago. Her role as a bioethicist, she explains, is "to make sure that human progress goes forward in a way that's safe and ethical."

Clinical trials — whether testing new drugs, devices, surgical methods or CRISPR technology — must meet higher ethical standards than work conducted in a lab, she says. "Having a human being as a subject means you have different obligations than you would to an animal or a petri dish," Zoloth says. A scientist conducting a clinical trial on humans should be "responsible for them, in my opinion, forever."

That means researchers should pay not just for the experimental treatment, but also for treating side effects — including those that show up later — or for anything that goes wrong during the trial. Insurance policies often don't cover experimental procedures, yet informed consent forms that must be signed by participants in clinical trials sometimes contain clauses claiming a participant's insurance will cover side effects. When Zoloth and others who review research applications point this out to scientists, "sometimes they're surprised that they haven't thought of it," she says. "Sometimes they hope that, by putting that [clause] in there, it will release them from some burden."

Zoloth, a religion professor, understands why some view clinical trials as godsends. Many who sign up are struggling with untreatable conditions or poor prognoses. But the experiments often don't work. For instance, stem cells for spinal cord injuries didn't work as well in people as in rats, Zoloth says. "That's very, very frustrating for scientists and certainly for patients," she says.

But that's why researchers need to do these tests in people, whom Zoloth calls research subjects instead of patients. Research subjects play a key role in making sure treatments that show promise in animal tests are safe and effective in humans.

"It's very brave and very noble of people with these diseases to take on the additional risk of being a subject," Zoloth says. "Without this act of nobility, clinical research would collapse."

In most cases, "the intervention will not help," nor will subjects benefit financially from the trial, she says. "If you don't benefit at all, at least you shouldn't be harmed at all. You shouldn't alone have to bear the burden of a severe adverse event."

"It's very brave and very noble of people with these diseases to take on the additional risk of being a subject. Without this act of nobility, clinical research would collapse."

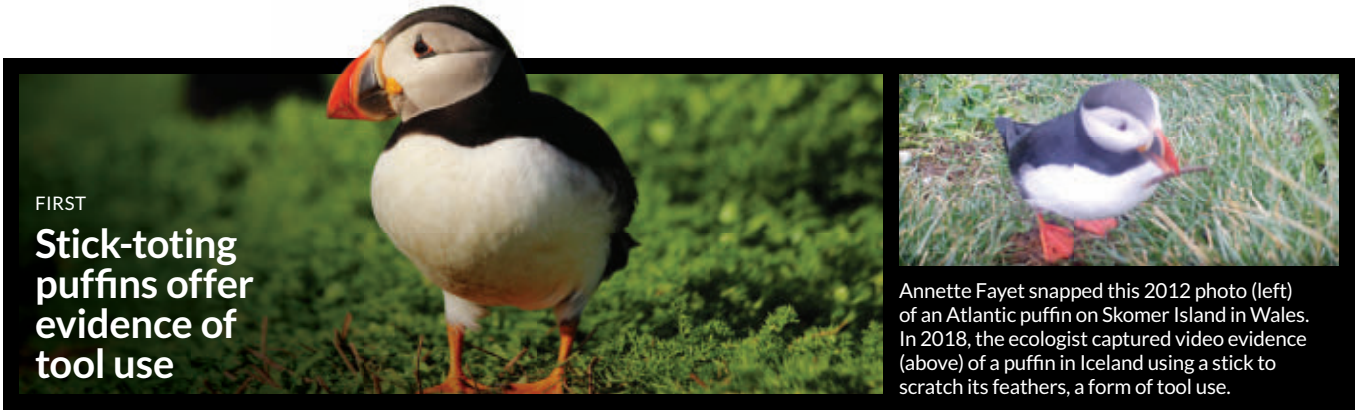
LAURIE ZOLOTH

U.S. researchers generally do follow guidelines set by institutional funders and review boards. For instance, a trial at the University of Pennsylvania testing the safety of CRISPR-edited immune cells against certain types of cancer made clear it was an experiment. Early results showed the edited cells were safe, but didn't stop the volunteers' cancer (*SN: 12/21/19 & 1/4/20, p. 28*).

Zoloth plans to continue reminding scientists of their responsibility and the risks subjects face. That "sobers the scientists," she says. "It makes [scientists] understand the gravitas of their interactions with a human subject."

— Tina Hesman Saey

SETH JOEL, UNIV. OF CHICAGO



FIRST

Stick-toting puffins offer evidence of tool use

Annette Fayet snapped this 2012 photo (left) of an Atlantic puffin on Skomer Island in Wales. In 2018, the ecologist captured video evidence (above) of a puffin in Iceland using a stick to scratch its feathers, a form of tool use.

Scanning a large colony of Atlantic puffins off Wales in 2014, ecologist Annette Fayet saw something unusual: A puffin bobbing on the water held a stick in its orange-black bill, and then used the stick to scratch its own back.

Puffins hadn't ever been seen using tools. In fact, no seabird had.

But it would take four more years before Fayet, of the University of Oxford, had photographic evidence to help persuade other scientists. In 2018, on Iceland's Grimsey Island, one of her motion-sensitive cameras captured a puffin snatching a stick from the ground and using the stick to scratch its chest feathers.

Those observations, described online December 30 in *Proceedings of the National Academy of Sciences*, represent the only known examples of a wild bird using a tool to scratch itself.

Scientists have long known that some birds use tools, mostly to extract food. Stick-wielding crows wow biologists with their ingenuity. Some parrots grind seashells with pebbles. Egyptian vultures crack ostrich eggs with rocks. But seabirds, which tend to have smaller brains than other birds, had been written off as prospective tool users.

The fact that this behavior was seen in two puffin populations four years and 1,700 kilometers apart bolsters the argument that the observations weren't a fluke, and suggests that tool use may be more widespread and varied in birds than thought.

Atlantic puffins (*Fratercula arctica*) might use sticks to flick ticks from their plumage, Fayet and colleagues suggest. Summer 2018 in Iceland, when the scratching was caught on video, was an especially bad tick season. —*Jonathan Lambert*

PUFFINS: A.L. FAYET; KNOT: JOSEPH SANDT; PULSAR: NASA GODDARD SPACE FLIGHT CENTER

MYSTERY SOLVED

Some knots hold faster than others. Here's why

Knowing if a knot is fit to be tied just got more scientific. Using color-changing fibers, researchers developed mathematical rules to gauge which knots may be stronger than others based on how the knot is tied.

Hues in the fibers revealed areas of strain (yellow and green above). The strain in fibers tied into knots agreed with the strain calculated in computer simulations, the team reports in the Jan. 3 *Science*. That simulation technique was used to predict the relative strength of bends, more complex knots that connect two pieces of rope.

Three characteristics explained a knot's strength: First, the more times the strands cross, the stronger the knot. The twisting of crossing strands also matters: If strands are twisted in opposite directions, the twist balances out, locking the knot into place. Finally, if adjacent strands slide in opposing directions as a knot is tightened, that also strengthens the knot. The rules don't predict a knot's overall strength, though. For that, researchers would need to look at details like the type of rope used.

Still, the results explain why a square knot is stronger than a granny knot, notorious for causing loose shoelaces. Unlike a square knot, the granny knot has an unbalanced twist — and that could really trip you up. —*Emily Conover*

Watch a video of a puffin using a stick as a tool at **bit.ly/SN_PuffinTool**

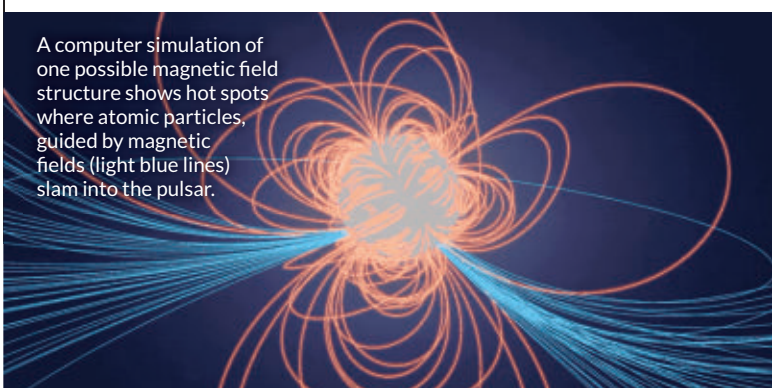
RETHINK

Glimpses of a pulsar suggest the star has wonky magnetic fields

A pulsar in the Milky Way is upending what we thought we knew about such a star's magnetic fields.

Two studies in the Dec. 10 *Astrophysical Journal Letters* report the first views of a pulsar's surface. These fast-spinning neutron stars, left behind by supernova explosions, pack roughly the mass of the sun into a city-sized orb. Scientists don't really know what happens to matter when it's squeezed so tight. But the new data could shed some light.

Fluctuations in X-rays coming off pulsar PSR J0030+0451, just over 1,000 light-years from Earth, revealed bright spots where atomic particles, guided by magnetic fields, slam into the star. These spots were clustered in the southern hemisphere, rather than positioned at the north and south poles. That suggests the pulsar has magnetic fields far more complex that what's typically imagined. —*Christopher Crockett*



A computer simulation of one possible magnetic field structure shows hot spots where atomic particles, guided by magnetic fields (light blue lines) slam into the pulsar.

EARTH & ENVIRONMENT

Wild bees are in decline globally

Diversity began to drop in the 1990s, a study finds

This ground nesting bee (*Megachile bicolor*) found in Thailand is among the species losing habitat to urbanization and agriculture.

BY YAO-HUA LAW

Far fewer bee species are buzzing across Earth today, following a steep decline in bee diversity during the last three decades, according to an analysis of bee collections and observations going back a century.

About half as many bee species are turning up in current collecting efforts for museums and other collections compared with in the 1950s, when surveys counted around 1,900 species a year, scientists report December 10 at bioRxiv.org. That high diversity in collections endured for several decades, but then began to plummet around the 1990s, likely reflecting a real drop in global bee diversity, according to the study, which is under peer review.

"This is the first study suggesting that bee decline is a global process, and that the most significant changes have occurred in recent years," says Margarita López-Uribe, a bee evolutionary ecologist at Penn State who was not part of the research.

The work evaluates global trends in bee diversity since the 1920s by tapping the database of the Global Biodiversity Information Facility. This international data-sharing network holds what López-Uribe calls "the most comprehensive dataset of insect collection records worldwide," including photos of bees in the field and of museum specimens dating back to the 18th century. Previous bee studies have reported falling populations, but evidence has often been limited to Europe and North America. Numbers of western honeybees (*Apis mellifera*) have been decreasing in North America and Europe, for example, but have increased in Asia, Africa and South America. For bees overall, though, the global situation was unclear.

Eduardo Zattara and Marcelo Aizen of the Pollination Ecology Group at the Biodiversity and Environment Research Institute in Rio Negro, Argentina, found that the number of instances of observing bees has climbed in recent decades, probably due to more researchers going into the field to document and study bees.

But the number of observed species fell. In the 1950s, collectors added about 22,000 bee records per year, of about 1,900 species each year, for a global total of 5,600 species collected over the whole decade. After correcting for sampling effort, the researchers estimate that about 6,700 species would have been found in the wild. In the 2010s, collectors tallied an average of 860 species per year from an average of more than 37,000 observations per year, leading to an estimate of only about 3,400 species to be found worldwide in the wild.

6,700
Estimated number of wild bee species worldwide in the 1950s

3,400
Estimated number of wild bee species worldwide in the 2010s

Declines in the number of species occurred on nearly every continent, starting at various points in the last four decades but largely in the 1990s on most continents. One exception was Australia and nearby islands, where the number of bee species estimated from observations spiked from about 300 to 500 in the 2000s. But species numbers in that region dropped back to 300 in the 2010s. Globally, thousands of bee species have become so rare that they are difficult to find or have gone extinct.

These results should convince people that bee diversity losses are not confined to specific regions, but are part of a global trend, says Zattara, who is now a visiting scientist at the Smithsonian National Museum of Natural History in Washington, D.C.

While the study provides an "invaluable" overview, the dataset is surely missing important insect collections from Asia, says bee biologist Natapot Warrit of Chulalongkorn University in Bangkok, Thailand. He hopes the study encourages researchers in the region to study and share data on pollinators.

Bees and other insects such as butterflies and flies pollinate more than 75 percent of the most important crop types grown, scientists say. But these pollinators face multiple threats including the expansion of monoculture crop cultivation, pesticides (*SN: 2/3/18, p. 14*), climate change and pathogens that can spread with the international bee trade (*SN: 2/16/19, p. 32*). Researchers also have suggested that bees may be at risk from power lines (*SN: 12/7/19, p. 8*).

While the study reveals an ongoing drop in bee diversity, the team isn't trying to sound "gloomy or apocalyptic," Zattara says. Rather, the researchers hope the study prompts other scientists, policy makers and business leaders to take action to reverse the decline. ■

NONTHAWAT CHATTHANABUN

LIFE & EVOLUTION

Trove of new bird species is found

Discoveries come from a group of remote Indonesian islands

BY JAKE BUEHLER

It's a veritable bevy of birds: Ten songbirds hailing from a cluster of small Indonesian islands near Sulawesi have officially joined the scientific record.

Typically, only five or six new bird species are described each year across the globe. So the discovery of five new species and five new subspecies, described in the Jan. 10 *Science*, marks a remarkable expansion of bird biodiversity, considering that birds are among the most comprehensively categorized animal groups.

Evolutionary biologist Frank Rheindt of the National University of Singapore and colleagues had an inkling these remote, forested islands with mountain highlands held an unrecognized wealth of bird life. The islands — Taliabu, Peleng and the Togian group — sit in the middle of Wallacea, a geologically and biologically complex region of Southeast Asia. With deep waters separating these islands from the nearest large landmass of Sulawesi, the islands have remained isolated even during periods of low sea level, limiting the opportunities for many animals to intermingle. This includes tropical forest birds, which rarely venture out from the shady cover of the forest, let alone fly kilometers over open ocean.

In searching for new species, "it's very important to pick deep-sea islands," Rheindt says. "Those are the ones that are likely to have endemic species that are not shared with other landmasses." Even more encouraging, the islands' interior highlands hadn't received much attention from 19th century European explorers or naturalists, who instead had focused on the coasts, Rheindt says.

Other researchers in the 1990s had reported what appeared to be distinct songbird species on the islands. But the researchers hadn't collected specimens, nor formally described any findings.

So in 2013 Rheindt and colleagues teamed with Dewi Prawiradilaga's group at the Indonesian Institute of Sciences in Jakarta for an expedition to investigate the islands' bird life and collect specimens for study. Most of the birds were found on Taliabu, the largest of the islands with the highest elevation.

Based on the birds' physical features, DNA and their songs, the researchers identified the five new species and five new subspecies. Some were visually striking, such as the fiery red-orange male Taliabu myzomela honeyeater (*Myzomela wahe*) and the Togian jungle-flycatcher (*Cyornis omissus omississimus*) with a yellow belly and a cap of iridescent blue feathers.

While the researchers expected to find some new wildlife on the islands, "we weren't aware that this was going to be a bonanza of new species and subspecies," Rheindt says.

Rheindt's favorite of the new finds is the Taliabu grasshopper-warbler (*Locustella portenta*), part of a group of inconspicuous brown birds with cricket-like songs that vary wildly between species. The Taliabu species was particularly shy and elusive, Rheindt says, and only after multiple mountain ascents did he find one to match the songs he had been hearing. He saw immediately that it was a darker hue than the known grasshopper-warbler in the region.

"That one is the one that caught my imagination," Rheindt says.

The cache of new birds is impressive, says Pamela Rasmussen, an ornithologist at Michigan State University in East Lansing. In recent decades, most new bird species have been found in Peru and Brazil, she says. And while it's not necessarily surprising that there are places in Indonesia that haven't been well surveyed, the find is "unusual in the fact that these birds have existed so long without being documented." But more such finds aren't so likely, she says. "There are very few places left that are likely to have so many [birds]."

Many of the newly described birds are threatened by habitat loss, driven by logging and increasingly frequent and severe forest fires. Of particular concern is the Taliabu grasshopper-warbler, which has been squeezed into tiny vestiges of highland habitat. The species "might not survive beyond a few decades," Rheindt says.

But conserving species requires first knowing what's out there, so studies like these are important, he says. "Time is limited, and biodiversity is going down the drain." ■

On a remote cluster of Indonesian islands, researchers discovered five new songbird species and five subspecies. These birds include (clockwise from left) the Togian jungle-flycatcher (*Cyornis omissus omississimus*), the Taliabu grasshopper-warbler (*Locustella portenta*), the Taliabu leaf-warbler (*Phylloscopus emilsalimi*) and the Taliabu myzomela honeyeater (*Myzomela wahe*).

ALL: JAMES EATON/BIRDTOUR ASIA

A fire burns on January 4 in Lake Tabourie, about 200 kilometers south of Sydney.

EARTH & ENVIRONMENT

Why Australia's fires are so intense

A scientist explains a weather pattern's role in the blazes

BY CAROLYN GRAMLING

Australia's fire season normally peaks in late January. But by January 2020, wildfires had already been raging for four months, especially in eastern Australia. As of mid-January, the fires had ruined over 2,000 homes, burned over 11 million hectares and killed at least 28 people.

The wildfires are being fueled by a deadly combination: record high temperatures, long-term drought, very low air and soil moisture, and human negligence. But climate change, scientists say, could make such extreme blazes three times as common by the end of the century.

It's hard to directly identify the fingerprints of climate change in the fires. But for years, fire managers have kept an eye on one culprit behind eastern Australia's very hot, dry years that may be affected by global warming: an oscillating El Niño–like ocean-atmosphere weather pattern that begins in the Indian Ocean.

Like El Niño, this "Indian Ocean dipole" pattern has positive, negative and neutral phases, depending on temperatures in the eastern and western Indian Ocean. The more extreme the temperature difference between the ocean's eastern and western regions, the stronger the phase. When the Indian Ocean dipole is in a particularly strong positive phase — as it was in 2019 — it correlates with some of Australia's worst fire seasons, says Wenju Cai, a climate scientist at the Commonwealth Scientific and Industrial Research Organization who is based in Melbourne, Australia.

Global warming is likely to make such extreme positive phases more common, Cai says. In a 2014 study in *Nature*, he and colleagues simulated future sea-surface temperature changes in the Indian Ocean in a world where greenhouse gas emissions continue on a "business-as-usual" track. The frequency of extreme positive phase events could increase from about once every 17 years to once every six.

Science News talked with Cai about the link between the Indian Ocean dipole and Australia's fires. His responses are edited for brevity and clarity.

What exactly is a positive phase of the Indian Ocean dipole?

It's when the eastern Indian Ocean is cooler than normal, and the west is warmer than normal. When you have [that], the rain moves toward the west. That's why we're seeing huge impacts in East Africa. [In 2019,] rains, flooding and landslides killed over 300 people and affected [millions].

On the eastern side, we tend to have drought and wildfires in Indonesia and Australia. In southeastern Australia, the rainy season is normally June through November. That's also when the Indian Ocean dipole normally develops.

If there isn't rain during those months, it will build up these dry conditions. Trees and vegetation die, and [wildfire] fuel builds up. The summer is the dry season anyway, and we often have bushfires. But [without spring rains], they become much more severe and damaging because it's so much easier for vegetation to burn.

Meanwhile, one of the worst droughts on record is occurring in southeastern Australia.

Yes, we are in a dry season that is already suffering from the impacts of two previous dry seasons. We have this kind of three-year drought regularly in Australia. It's a very dry continent anyway, particularly in the south. It's one of the driest regions in the world.

But you have a cumulative effect now, because the soil moisture is very low, and then this dipole comes about, and we also accumulate a lot of [wildfire] fuel.

Is there a link between the positive phase dipole and wildfires in Australia?

There's a very good correlation: All the major bushfires in southeastern Australia are preceded by [a positive] Indian Ocean dipole. For example, in 2009, [such a dipole preceded] a bushfire called Black Saturday that killed 173 people in Melbourne in just a few hours, and destroyed more than 2,000 houses.

There was also a [positive] dipole before the 1997 bushfire in Indonesia, which lasted many months and created haze affecting tens of millions of people and really hit the economy there. The 2019 dipole is second [in strength] only to 1997 in the record going back to 1870.

So what's the link to climate change?

The big message of our 2014 paper is that, under global warming, that kind of huge Indian Ocean dipole will increase threefold [in frequency] by 2100.

In a subsequent paper in *Nature Communications*, we looked at how the frequency of the dipole would change if temperatures are stabilized [at] 1.5 to 2 degrees Celsius [above preindustrial times]. The good news is that really cutting greenhouse gas emissions is actually very effective! Once you cut them, it stabilizes [the dipole]. ■

BRETT HEMMINGS/GETTY IMAGES PLUS

BODY & BRAIN

Zika linked to developmental delays

Even babies born healthy after in utero exposure need follow-up

BY AIMEE CUNNINGHAM

Babies from Colombia who were born healthy after being exposed to Zika virus in the womb showed signs of developmental delays by 18 months of age, a small study finds. The work suggests the need for long-term follow-up of babies whose mothers had the viral infection during pregnancy, researchers say.

As a group, the 70 babies exposed to Zika didn't hit certain developmental milestones for movement and social interaction around the times expected for healthy, nonexposed babies of the same age, researchers report online January 6 in *JAMA Pediatrics*.

Overall, the children lagged in mobility skills such as rolling over or sitting up, and in play skills like peekaboo and searching for an object that has dropped out of sight, says fetal neonatal neurologist Sarah Mulkey of Children's National Hospital in Washington, D.C. Within the group, some babies developed as expected, some showed obvious delays, and some showed subtle delays that caregivers might not have noticed.

Because there was variability between individuals, "looking at a population enables one to see overall trends," says neurologist Ken Tyler of the University of Colorado School of Medicine in Aurora, who was not involved in the research. "We need to aggressively follow all children whose mothers were exposed to Zika during pregnancy to understand the nature of their neurological delays."

Mulkey's team, including researchers in Colombia, assessed babies born between August 1, 2016, and November 30, 2017 — during and after the Zika epidemic that gripped Brazil, Colombia and other countries in the Americas (*SN: 11/11/17, p. 12*). In a study in the United States and U.S. territories, about 5 to 10 percent of babies born to Zika-infected mothers had severe birth defects, including an abnormally small head and brain damage. But the large majority weren't born with these defects.

The new study followed the 70 babies from Colombia for a year and a half, assessing them at least once between the ages of 4 months and 18 months old. The babies had normal fetal development and head circumference. But results from a questionnaire for parents and an observational exam of the babies suggest that it's possible that problems may not arise until later.

"We are still learning exactly how Zika exposure can affect a developing fetus and beyond," says Nassim Zecavati, a neurologist at Georgetown University School of Medicine in Washington, D.C., who was not involved in the research. More work is needed to determine the reasons for the delays in this group of children and whether the problems are temporary, she says.

The neurodevelopmental differences found in the children can be addressed with physical and occupational therapy, Mulkey says. Her team will follow this group until 5 years of age. "We don't yet know the future of how these children are going to develop long-term," she says. ■

SHRIHARSH TENDULKAR/GEMINI OBSERVATORY

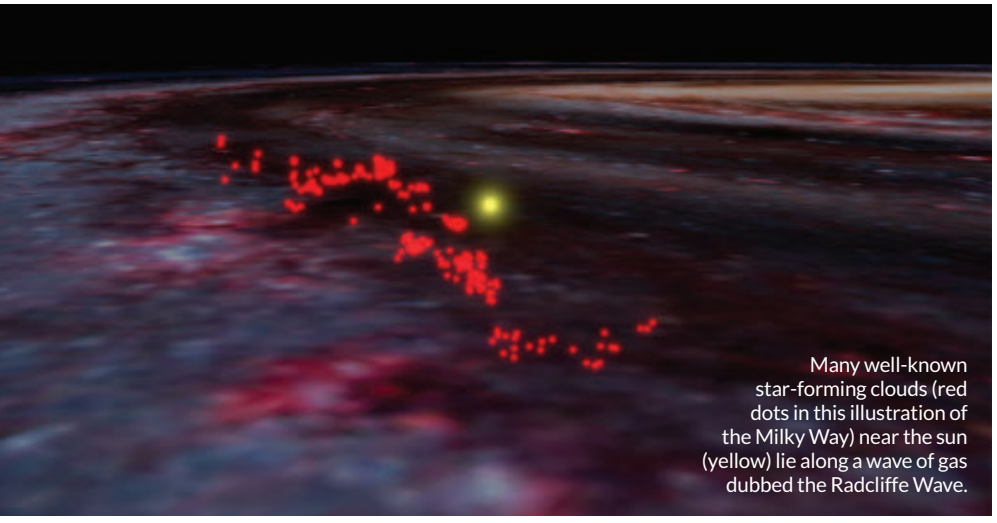
ATOM & COSMOS

Another fast radio burst puzzle

Brief, brilliant flashes of radio waves have been traced to a galaxy that looks radically different than the galaxies where scientists have seen the radio flares before. Until now, the only source known for a recurrent fast radio burst like this was a tiny, star-forming dwarf galaxy, while nonrepeating bursts have been tracked to more massive, mellow galaxies. That implied that the two types of fast radio bursts, or FRBs, might have different sources (*SN: 8/3/19, p. 10*).

But researchers have pinned a second repeating FRB to a different kind of host galaxy: a star-forming spiral (shown, arrow points to the FRB's location) similar in size to the Milky Way, scientists report in the Jan. 9 *Nature*.

The second FRB may have a different engine than the repeater in the dwarf galaxy. Unlike a spiral galaxy, dwarf galaxies are thought to have the conditions necessary to form highly magnetized neutron stars that could power repeated bursts, says astrophysicist Jason Hessels of the University of Amsterdam. He thinks a black hole gobbling up material could account for the spiral galaxy's FRB. — *Maria Temming*



Many well-known star-forming clouds (red dots in this illustration of the Milky Way) near the sun (yellow) lie along a wave of gas dubbed the Radcliffe Wave.

ATOM & COSMOS

Giant gas wave lurks near solar system

Stellar nurseries are strung along the newfound 'Radcliffe Wave'

BY CHRISTOPHER CROCKETT

The Earth and sun are right next to a wavy rope of star-forming gas, but astronomers only just noticed it.

Many of the most well-known nearby stellar nurseries — places like the Orion Nebula — are strung along a thread of gas that stretches roughly 9,000 light-years, researchers say. The thread resembles a sine wave, soaring above and below the disk of the Milky Way by about 500 light-years, and at one point, coming within 1,000 light-years of our solar system.

"Perhaps the oddest feature is how close it is to the sun, and we didn't know about it before," said Alyssa Goodman, a Harvard University astrophysicist who presented the results January 7 during a news conference at the American Astronomical Society meeting. The study was published the same day in *Nature*.

The team dubbed the structure the Radcliffe Wave, Goodman said, in honor of both the institute where much of the work was done and early 20th century female astronomers from Radcliffe College, which became part of Harvard.

Despite its proximity to us, astronomers noticed the wave only now because of recent advances in the ability to pinpoint distances to known star-forming gas clouds. To nail down the distances, Goodman and colleagues looked at stars behind the clouds and deduced how dust within the clouds altered the colors of the stars. Combining those measurements with distances to those stars allowed the team to map the 3-D locations of the clouds with newfound precision, revealing they line up along the wave.

"These kinds of waves have been seen in external galaxies," says astrophysicist Lynn Matthews of the MIT Haystack Observatory in Westford, Mass. "This gives us an opportunity to tie together phenomena that have been observed in several galaxies and develop a unifying picture of what might cause these sorts of features."

Goodman suggested that the wave "could have been from a collision, something falling down on the Milky Way." Matthews, along with colleagues, saw something similar in a spiral galaxy dubbed IC 2233 and thinks such gas waves arise from gravitational disturbances from various interacting components of the galaxy itself.

Regardless of how the newfound wave formed, tracing the motion of the sun through space backward in time reveals that the solar system passed through the Radcliffe Wave roughly 13 million years ago, Goodman said. ■

ATOM & COSMOS

LIGO finds second neutron star duo

With no flash of light seen, scientists have few details

BY CHRISTOPHER CROCKETT

For the second time, a collision between two neutron stars in another galaxy has rattled a gravitational wave detector on Earth. But this duo is being more coy than the first.

In 2017, astronomers announced with much fanfare the detection of ripples in spacetime from the merging of two neutron stars, the ultradense remains of massive stars (*SN: 11/11/17, p. 6*). Observatories around the world and in space witnessed a simultaneous flash of

ATOM & COSMOS

Reionization clues seen in bubbles

Old galaxies highlight potential step in universe's makeover

BY CHRISTOPHER CROCKETT

A trio of bubble-blowing galaxies may offer clues about one of the greatest makeovers in the history of the universe.

Sometime during the universe's first billion or so years, most of the hydrogen atoms in the cosmos became ionized when their electrons were torn away (*SN: 12/7/19, p. 8*). Astronomers suspect that this reionization — so called because all hydrogen had been previously ionized for the first few hundred thousand years — was triggered by harsh ultraviolet light from the first generations of stars.

Now, researchers say they've caught a few galaxies blasting out ionizing light and stripping electrons from surrounding hydrogen just 680 million years after the Big Bang. If so, this would be the first direct evidence of galaxies working together to ionize the early cosmos.

James Rhoads, an astrophysicist at the NASA Goddard Space Flight Center

WORLDWIDE TELESCOPE, COURTESY OF A. GOODMAN

radiant energy, light from all across the electromagnetic spectrum.

Now, gravitational waves from a second neutron star smashup have been detected. But unlike with the first detection, researchers were not able to pinpoint the collision's location on the sky and did not see an accompanying burst of light. Astrophysicist Katerina Chatziioannou of the Flatiron Institute in New York City presented the results January 5 at the American Astronomical Society meeting.

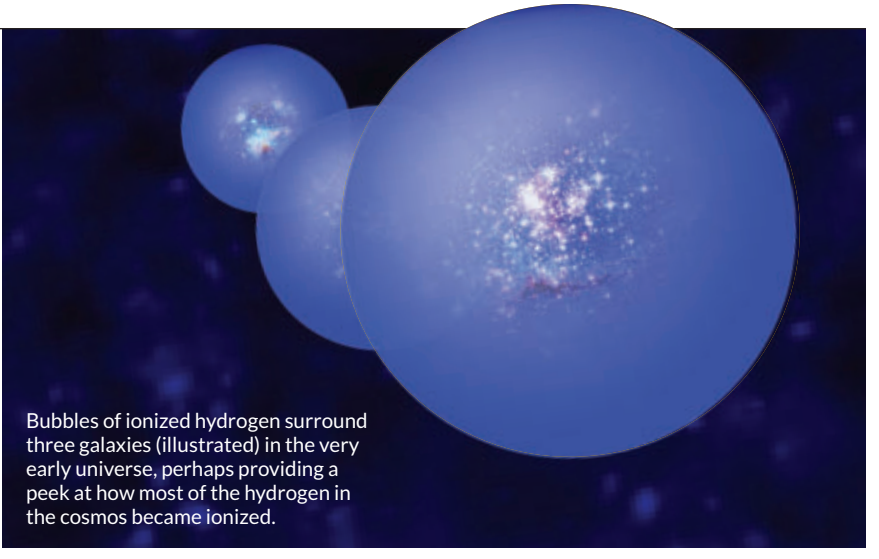
The event was picked up on April 25, 2019. Only one of LIGO's two detectors registered the collision — the one in Livingston, La. The other LIGO facility, in Hanford, Wash., was offline at the time — and the event was too weak for the Virgo observatory in Italy to definitively detect.

Nevertheless, researchers deduced that the most likely source of the gravitational waves was a collision between a pair of neutron stars with a combined mass 3.4 times as great as the sun. The smashup occurred between 290 million and 720 million light-years from Earth, Chatziioannou said.

While the lack of an electromagnetic counterpart is disappointing, it's not too surprising. "We do not expect a detectable counterpart from most mergers," says astrophysicist Avi Loeb of Harvard University, who is not part of the LIGO-Virgo collaboration. The light from a neutron star collision, he says, comes from jets of gas that spew out from the crash. Those jets are so narrow that a fortuitous alignment is needed to see the light from Earth.

It is possible that there was a flash and astronomers missed it. With a gravitational wave detection at only one facility, researchers weren't able to narrow down where on the sky to look. "It was very poorly localized, to about one quarter of the entire sky," says Edo Berger, a Harvard astrophysicist who participated in one search for visible light from the collision. "No electromagnetic search could have covered the entire region of interest.... The bottom line is that we can't actually state that this event had no [electromagnetic] counterpart."

However, even if a telescope had been pointed in the right direction, there still might have been no light. The relatively high combined mass of the neutron stars means the final product likely collapsed immediately into a black hole, Chatziioannou says. If that's the case, then little material would have escaped to be seen. ■



Bubbles of ionized hydrogen surround three galaxies (illustrated) in the very early universe, perhaps providing a peek at how most of the hydrogen in the cosmos became ionized.

V. TILVI ET AL./ARXIV.ORG 2020, NSF'S OPTICAL-INFRARED ASTRONOMY RESEARCH LAB, KPNO, AURA

in Greenbelt, Md., presented the results January 5 during a news conference at the American Astronomical Society meeting.

To look for ionizing galaxies, the researchers sought out remote galaxies emitting a specific wavelength of UV light. Neutral hydrogen absorbs this wavelength, preventing it from reaching Earth, but ionized hydrogen lets it slip by. Using the Mayall 4-meter Telescope in Arizona, Rhoads and colleagues went hunting for this light in a well-studied strip of the sky. They found three galaxies huddled together shining with the light, which took over 13 billion years to reach Earth.

During that long-ago epoch, much of the universe's hydrogen was still neutral. But the team argues that these three galaxies have created overlapping bubbles of ionized hydrogen in a sea of neutral hydrogen, allowing the UV light to escape the galaxies unimpeded. The largest of these bubbles is calculated to be over 6 million light-years across, an estimate based on how much ionizing light the brightest galaxy likely pumped out over its lifetime. That's large enough for the ongoing expansion of the universe to stretch the light out to a longer wavelength during its travel time, so that by the time it reaches the edge of the bubble, it can pass through the enveloping neutral hydrogen.

While the brightest of these galaxies was known to emit ionizing light, no one had noticed that its neighbors did as well, says Brant Robertson, an astrophysicist at the University of California, Santa Cruz who wasn't involved with the work.

"What's interesting about the galaxies being together is they can work together as a team," Robertson says. "Once the bubbles around them overlap, then it becomes easier for them to start ionizing a larger region around them than if they each had to work on their own in separate little bubbles."

These galaxies are so far away that it's tough to measure more than a few properties about them, Rhoads says. So it's hard to say exactly what lets the galaxies send out so much ionizing radiation.

To grapple with that question, Rhoads and others are looking closer to home. "We're studying nearby galaxies that are similar in nature to these," he says. By investigating those closer star systems, "we are able to look for trends in what galaxy properties allow ionizing photons to escape." ■

BODY & BRAIN

Old tuberculosis vaccine gets a boost

Changing the delivery method improved protection in monkeys

BY TARA HAELLE

Delivering a high dose of a vaccine against tuberculosis intravenously, instead of under the skin, greatly improves the vaccine's ability to protect against the deadly disease, a new study finds.

Changing the typical dose and method of administration of the bacille Calmette-Guérin, or BCG, vaccine prevented TB in 90 percent of tested rhesus macaques, researchers report in the Jan. 2 *Nature*.

Most "astonishing" is that six of the 10 monkeys that got the IV vaccine never even developed an initial infection when exposed to TB, says Joel Ernst, an immunologist at the University of California, San Francisco. Preventing infection, not just disease — called sterilizing immunity — is extremely rare with any TB vaccine, Ernst says. Thwarting that infection means that no TB bacteria can reactivate to cause an active infection or a latent one, an infection that induces an immune response but hasn't progressed to active TB.

The BCG vaccine, around for nearly a century, is the only currently licensed TB vaccine. More than 150 countries, but not the United States, where TB risk is low, regularly use BCG to protect infants against some forms of TB. But the vaccine often fails to prevent the most common type of infection, in the lungs, in adolescents and adults.

Globally, TB sickened about 10 million people in 2018. It kills about 1.5 million a year, making it the most lethal infectious disease. Up to 13 million people in the United States have a latent infection.

Creating an effective TB vaccine has been difficult because the bacteria that cause the disease, *Mycobacterium tuberculosis*, hide within cells, where they're more protected from antibodies, which primarily attack outside cells. Fighting most intracellular infections requires immune cells called T cells to attack the infected cells, says immunologist Robert Seder of the National Institute of Allergy and Infectious Diseases' Vaccine Research Center in Bethesda, Md.

When the BCG vaccine is delivered just under the skin, the body makes some T cells to fight TB, but not enough to get to where they need to be and stay there — the lungs, for example — limiting the vaccine's effectiveness, says microbiologist and immunologist JoAnne Flynn of the University of Pittsburgh's Center for Vaccine Research.

A malaria infection similarly requires T cells to fight the malaria parasite inside cells, Seder says. After success with an intravenous malaria vaccine in another trial, researchers wondered if BCG vaccine injected directly into the blood, where it could travel throughout the body, would likewise be successful.

Flynn, Seder and colleagues tested five BCG formulations in monkeys: a standard under-the-skin, or intradermal, dose; a high dose given under the skin; a high aerosol dose administered with a mask; a high intravenous dose; and a combination of high-dose aerosol and standard-dose intradermal. Six months later, the researchers exposed the five vaccinated groups of monkeys and a sixth unvaccinated control group to TB.

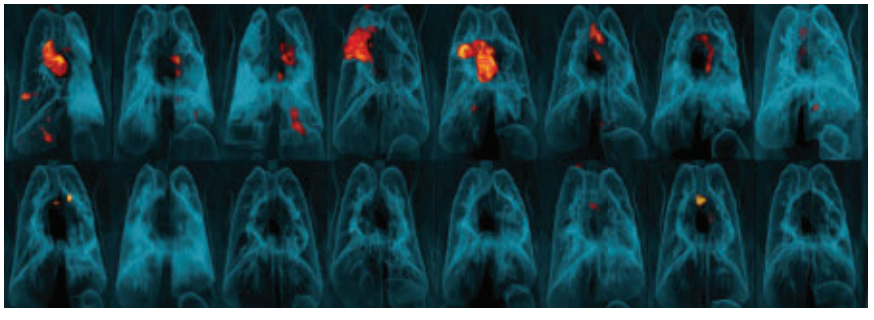
All of the unvaccinated, standard-dose intradermal and aerosol-vaccinated monkeys developed a TB infection. All but one of the eight monkeys that got the high intradermal dose developed infection, though two monkeys cleared it several weeks later. In contrast, six of 10 IV-vaccinated monkeys never developed a TB infection; three that were infected had 45 or fewer individual TB bacteria in the lungs, a very low amount, and cleared the infection.

One possible reason that the intravenous vaccine worked best is the high number of T cells induced in monkeys' airways — 100 times as many as in the intradermal and aerosol groups. The vaccine also induced production of tissue-resident memory T cells, primed T cells in the tissue itself not just the blood.

Preventing TB in adolescents and adults is crucial, Flynn says, so the major question is whether this approach would be safe and effective in that population. The only adverse effect seen in the monkeys was a temporary, modest increase in inflammation.

Ernst says one safety concern is whether intravenous BCG could induce a harmful inflammatory response in people with a latent TB infection — about a quarter of the planet's population. The researchers plan to test the IV vaccine in monkeys with latent infections. If it could cause harm, screening before vaccination would be necessary.

For now, the next step is to test how low a dose still offers protection, Flynn says. "This study really provides us hope that a truly effective vaccine against TB is on the horizon," she says. "I've been in the field for 30 years, and I feel we are making progress in really starting to understand the disease and vaccines that can prevent infection." ■



PET-CT scans of rhesus macaque lungs show spots of tuberculosis infection and tissue inflammation (red and orange). Monkeys that received a TB vaccine intravenously (bottom row) were better protected against infection than monkeys that got vaccinated under the skin (top).

UNIV. OF PITTSBURGH SCHOOL OF MEDICINE

Coating provides infrared camouflage

Material breaks rule that hotter objects radiate more light

When heated, a material radiates more brightly, and an infrared camera registers a higher temperature (brighter colors, top). A coating fools the camera into seeing little change (bottom).

BY EMILY CONOVER

Hotter objects typically glow brighter than cooler ones, making them stand out in infrared images. But a new coating bucks that rule. For certain wavelengths of infrared light, the material's brightness doesn't change as it warms, researchers report in the Dec. 26 *Proceedings of the National Academy of Sciences*.

The thin coating — a compound of nickel, oxygen and samarium, a rare-earth metal — "hides temperature information of surfaces from infrared cameras" and could be used as a privacy shield, says applied physicist Mikhail Kats of the University of Wisconsin–Madison.

A basic rule of physics states that the brightness of thermal radiation emitted by an object grows rapidly with increasing temperature. Infrared cameras measure how much thermal radiation objects emit in infrared to estimate temperatures. If the link between temperature and radiation is broken, the camera can be fooled.

For the material samarium nickel oxide, increasing brightness at higher temperatures is counteracted by a decrease in its tendency to emit thermal radiation. That decrease occurs because the material switches from an insulator to a metal. The two effects balance out so that, for certain infrared wavelengths, brightness remains constant as temperature changes, Kats and colleagues found.

For a heated sapphire sample coated with the material, its temperature in infrared images looked mostly unchanged from about 105° to 135° Celsius.

Physicist Karl Joulain of the University of Poitiers in France says current applications are "quite limited." Because the effect applies only to certain infrared wavelengths, detectors that look at other wavelengths would see a change.

Still, with infrared devices becoming cheaper and more common, the finding "comes with quite a bit of privacy implications," Kats says. Infrared cameras can be used to detect people or other sources of heat. For now, the high temperatures at which the camouflage effect occurs mean the coating wouldn't be useful for hiding people. But Kats thinks the temperature range can be changed by working with alloys of samarium nickel oxide, which may have different properties. ■

COURTESY OF PATRICK RONEY, ALIREZA SHAHSAFI AND M. KATS

EARTH & ENVIRONMENT

Early spring dries summer soils

Longer growing seasons may bring more heat waves

BY JONATHAN LAMBERT

Early arrival of spring is often cause for celebration in northern climates. But it may come at the cost of drier, hotter days in summer.

As winter wanes and leaves start to peek out from branches, trees draw more water from the soil and move it into the sky — a process called evapotranspiration. When this greening starts earlier in the year, scientists worry, more moisture could be sucked from the soil than if the season starts later.

Now, analyses of satellite data and climate simulations show that earlier spring greening can leave soils drier in summer across much of the Northern Hemisphere. That, in turn, could lead to more frequent and intense summer heat waves, researchers report January 3 in *Science Advances*.

As the climate warms, scientists expect to see earlier springs and longer growing seasons. "More green on the ground causes evapotranspiration to go up," says Chris Huntingford, a climate modeler at the U.K. Centre for Ecology and Hydrology in Wallingford, England. But it wasn't clear if a local increase in water being pumped into the atmosphere due to evapotranspiration would be offset by rain falling back to Earth, or whether certain geographic areas might be more affected than others.

So Huntingford, climate researcher Xu Lian of Peking University in Beijing and colleagues analyzed satellite data of vegetation cover and soil dryness across the Northern Hemisphere from 1982–2011. Across much, but not all, of the top half of the globe, earlier spring greening was associated with drier summer soils than in years when spring arrived later.

The association did not hold in places dominated by croplands, such as parts of central Europe and the U.S. Great Plains, that are under intense irrigation, the researchers say. Weather also may swamp the effects of an earlier spring in certain regions. Siberia, for instance, experienced many early springs without summer dryness, perhaps because weather patterns there consistently bring in excess moisture from Europe.

Soil drying can have myriad consequences, including raising local air temperatures and triggering heat waves or making them worse. The team estimates, based on simulations, that soil dryness due to earlier greening could increase the number of very hot summer

HUMANS & SOCIETY

Later arrival on Java for *Homo erectus*

Timing hints the hominid made several migrations into Asia

BY BRUCE BOWER

Homo erectus reached the Indonesian island of Java some 300,000 years later than many researchers have thought.

New analyses of sediment that yielded *H. erectus* fossils at a site called Sangiran show that the hominids arrived on Java about 1.3 million years ago, scientists report in the Jan. 10 *Science*.

More than 100 hominid fossils have been found at Sangiran since 1936. Many researchers accept that Sangiran sediment dates — based on the rate of decay of radioactive argon in volcanic rocks — put *H. erectus* on the island from roughly 1.7 million until 1 million years ago. Others say the best evidence points to an *H. erectus* presence at Sangiran from between 1.3 million and 1.1 million years ago until roughly 600,000 years ago.

The new work supports that younger timeline. A team led by paleoanthropologist Shuji Matsu'ura of the National Museum of Nature and Science in Tsukuba City, Japan, analyzed mineral grains called zircons from above, below and within sediment layers where *H. erectus* fossils had been found. One approach gauged the time since zircons had crystallized and the other estimated the time since a volcanic eruption deposited zircons.

Using two methods not tried before on volcanic material in Sangiran sediment makes the study "a vast improvement" on efforts to gauge *H. erectus*' arrival, says Kira Westaway, a geochronologist at Macquarie University in Sydney who wasn't involved in the study.

Understanding when *H. erectus* arrived in Indonesia could give insight into early hominid migrations into Asia. *H. erectus* fossils have been found throughout Asia and Africa. Given the age of some of those fossils, some scientists had thought that *H. erectus* dispersed in a single big push from Africa to Asia starting more than 2 million years ago. But the new age estimates indicate that *H. erectus* moved eastward several different times, Matsu'ura and colleagues say.

That's the best explanation for the hominid's arrival in different corners of Asia at different times, the team contends. For instance, *H. erectus* may have reached central China about 2.1 million years ago (*SN: 8/4/18, p. 7*). Another *H. erectus* migration may have reached southwestern Asia, closer to the African homeland, about 1.8 million years ago.

A new study is shedding light on when *Homo erectus* reached Indonesia's Sangiran site. This Sangiran skull is about 820,000 years old.

HISAO BABA/NATIONAL MUSEUM OF NATURE AND SCIENCE

days for a region by nearly one day per decade and raise maximum temperature by 0.07 degrees Celsius per decade.

"That doesn't seem like a lot, but in four or five decades, heat waves could be so strong that minor increases like this could matter," says Sebastian Sippel, a climate scientist at ETH Zurich who was not involved in the study. The new study shows that "for almost the entire Northern Hemisphere, earlier springtime greening can significantly alter summer water content" of soils, he says.

However, the study can't weigh the relative impact of springtime greening on soil dryness compared with other factors like an especially hot or dry season, Sippel says.

The team plans to repeat the study for the Southern Hemisphere to see whether earlier greening and soil dryness might be increasing the risk of heat-associated disasters, such as wildfires. ■

Java arrivals about 1.3 million years ago probably resulted from a separate trek eastward through South Asia or along its coast to Indonesia, the scientists say. Or those travelers may have descended from an earlier *H. erectus* group in China.

Researchers have long observed that *H. erectus* from older Sangiran sediments look similar to African *H. erectus* finds from as early as 1.7 million years ago. Younger Sangiran *H. erectus* fossils, however, resemble Chinese *H. erectus* fossils from about 780,000 years ago.

Younger Sangiran *H. erectus* fossils appeared after roughly 900,000 years ago, the new study estimates. Geologic studies indicate that global cooling around that time caused dramatic sea level declines, creating a land bridge from Java to mainland Southeast Asia. An *H. erectus* migration to Java across the land bridge could explain why younger Sangiran fossils differ from older ones, Matsu'ura says.

Still, uncertainties about the original positions of some Sangiran specimens suggest that some might date to as early as 1.5 million years ago, the team says. ■

JULIUS T. CSOTONYI

A dinosaur called *Nanotyrannus* (left), once thought to be a smaller cousin of *Tyrannosaurus rex* (right), was probably a juvenile *T. rex*.

LIFE & EVOLUTION

Small dinosaur may be a *T. rex* teenager

Young tyrannosaurs may have dined differently than their elders

BY SID PERKINS

Small but fearsome dinosaurs once thought to be pygmy kin of *Tyrannosaurus rex* may instead have been mere juveniles of the species, researchers report January 1 in *Science Advances*.

Paleontologists estimate that the largest *T. rex* individuals measured more than 12 meters from snout to tail. The dinosaurs had teeth about the size and shape of bananas, likely tipped the scales at more than 8,000 kilograms and may have lived to be 30 years or older.

In the 1940s, paleontologists unearthed a skull that, although similar to that of a *T. rex* and from the same time period, was about half the size and had teeth shaped more like daggers than bananas. Researchers eventually dubbed the dinosaur *Nanotyrannus*.

But for the last 15 years or so, debate has raged about whether *Nanotyrannus* was indeed separate from *T. rex*, says Holly Woodward, a paleohistologist at the Oklahoma State University Center for Health Sciences in Tulsa. For instance, some features once thought to be unique to *Nanotyrannus* have now been found in other tyrannosaurs, including *T. rex*.

Woodward and colleagues decided to slice open the leg bones of the two most recently described *Nanotyrannus* specimens, nicknamed Jane and Petey, and investigate the microstructure.

Cross sections revealed features similar to growth rings that suggest that Jane was at least 13 years old at death. The slightly larger Petey was at least 15. The microscopic structure of the bones — and especially the number and orientation of blood vessels — hints that the tissues were still growing vigorously, as they would in individuals that weren't fully mature.

"It's clear that these creatures were not adults," says Thomas Holtz Jr., a vertebrate paleontologist at the University of Maryland in College Park who wasn't involved in the study.

Scientists have yet to agree on whether the first known example of *Nanotyrannus* — the 1940s skull — was an adult or a juvenile. Some paleontologists claim that the bones in the skull were fused, a sign of adulthood, but others aren't convinced.

Other analyses have found that fossils first thought to be anatomically distinct species were actually different life stages of the same type of dinosaur.

The new study implies young and old *T. rex* behaved much differently, Woodward says. A juvenile's teeth were strong enough to puncture the bones of prey but couldn't crush them like adult teeth could. So youngsters and adults probably chased and consumed different prey, Woodward notes. ■

EARTH & ENVIRONMENT

Will acidification alter fish behavior?

Scientists rethink proposed effect of climate change

BY JONATHAN LAMBERT

Several species of damselfish swim on the Great Barrier Reef off Australia. New research suggests that expected ocean acidification won't impair these fishes' behavior, in contrast to earlier studies.

FREDRIK JUTFELT/NORWEGIAN UNIV. OF SCIENCE AND TECHNOLOGY

Climate change threatens coral reef fish in myriad ways, but maybe not in all the ways we thought. Some studies have suggested that ocean acidification might warp fish behavior. But new research shows that fish may be far more resilient.

Scientists predict that as atmospheric carbon dioxide levels continue to rise, and oceans absorb even more, the waters will increasingly acidify to a pH of about 7.8 by 2100. About a decade ago, a series of high-profile studies alarmed biologists with reports of severe behavioral impairments in coral reef fish exposed to mildly acidified water. Larval fish lost the ability to smell predators and became dangerously hyperactive and confused when exposed to acidification levels projected for 2100 if fossil fuel use continues at current rates. Research into the effects of ocean acidification ballooned, becoming one of the most studied subjects in marine science.

But a three-year attempt to replicate and improve upon some of those earlier studies paints a starkly different picture. Tests of more than 900 coral reef fish from six species showed that exposure to acidified waters had no worrying adverse effects on fish activity or predator avoidance, researchers report online January 8 in *Nature*.

"Climate change is a huge threat to reef fishes," says Timothy Clark, a comparative physiologist at Deakin University in Geelong, Australia. But "the acidification levels we'll see by the end of this century aren't going to have any real impact on fish, even beyond just coral reef fish."

Other biologists question such strong statements. The study is the most robust yet to find no effect from acidification, and the "data is unimpeachable," says Andrew Esbaugh, a comparative physiologist at the University of Texas at Austin who was not involved in any of the research that the new study sought to replicate. But because the study looked only at six reef fish species, the "conclusion that acidification won't affect fish behavior is a bit of an overreach," he says.

Alarming studies

Early research linking behavioral impairments to ocean acidification was dramatic. "These papers were, without a doubt, some of the most incredible things I've ever seen in biology," Clark says.

One 2010 paper found a near complete reversal of chemical cue preferences after exposure to slightly acidified water with a pH of 7.8. After just days, lab-reared larval clownfish became attracted to their predators' scent, preferring it 90 percent of the time.

Another 2010 study found similar impairments in clownfish and damselfish. Up to 90 percent of exposed fish were gobbled up when transplanted to a live reef, compared with 10 percent mortality for fish never exposed to acidified water (*SN Online: 7/6/10*).

"These are just mind-blowing results," Clark says. Studies since have reported more variability, including many seeing no effects. But Clark and colleagues wanted to replicate the earlier studies, he says, partly to understand the physiology behind these aberrant behaviors.

Differing conclusions

From 2014 to 2016, Clark's team collected over 900 individuals from six species of coral reef fish in Townsville, Australia, where much of the previous work had been done. Most fish were wild-caught adults or juveniles, while a subset came from a local aquarium.

After a first round of tests on five species in 2014, the researchers did not see any effects. "At first I was quite skeptical of our results," says behavioral ecologist Josefin Sundin of the Swedish University of Agricultural Sciences in Stockholm. If the effects are truly as severe as earlier studies found, she thought, the results should be easy to replicate.

Over the next two field seasons, the team tested more fish, and at different life stages. Each experimental trial was recorded on video; automated tracking software was used to measure behaviors in an effort to improve upon previous, more subjective methods of tracking, the researchers say.

All told, the study found little evidence that acidification impairs fish behavior. Juvenile spiny chromis damselfish showed a slightly impaired ability to discriminate between predator and nonpredator chemical cues. But other fish exposed to acidified waters avoided the scent of predators, like normal fish. The team also found little evidence of hyperactivity or other behavioral impairments.

"After three years of experimentation, we're confident in our data," Sundin says. The papers showing large effects from acidification are still being cited as the foundation of this field, she says. "If they're incorrect, perhaps we should

shift our focus to studying other climate change–related stressors besides ocean acidification," such as heat waves.

Apples to oranges?

Danielle Dixson, a behavioral ecologist at the University of Delaware in Newark, conducted some of the 2010 research while a Ph.D. student in the lab of Philip Munday of James Cook University in Townsville. She says the new study is not a fair replication. "It's comparing apples to oranges," she says.

Much of her team's early work included clownfish; the new study did not. And the early studies used larval fish raised in the lab, while the new study used mostly wild-caught adults or juveniles. "Those two groups have very different life experiences," Dixson says. "My fish were naïve to different acidification levels or predator cues, while wild-caught fish are not."

She notes that the field of ocean acidification's effect on fish behavior has advanced significantly since 2010. "We've improved a lot on those early studies, and subsequent research suggests that the effects of ocean acidification may not be as drastic as initially reported," she says. "That doesn't negate the earlier work."

But Clark and Sundin contend that their work replicated the crucial elements of the earlier studies. The team used four of the same species and tested some of the same life stages. While some earlier studies looked only at lab-reared fish, Sundin says Munday's research group and others also reported impairments from acidification in wild-caught individuals in over a dozen studies. "It's certainly a comparison of apples and apples," Clark says.

Howard Browman, a marine scientist at the Institute of Marine Research in Bergen, Norway, who wasn't involved in any of these studies, calls the new research "a healthy step forward in rebalancing the picture" of the effects of ocean acidification on fish behavior.

The field already is shifting toward the view that ocean acidification is one of many challenges fish face from climate change, he says, and in many cases it's probably less important than factors like ocean heat waves. ■

HUMANS & SOCIETY

Scientists map childhood malnutrition

Global progress on the problem masks some disparities

BY SUJATA GUPTA

The percentage of children with serious malnutrition decreased globally from 2000 to 2017. But the problem stayed the same or even worsened in some countries, including in Nigeria, Congo, Pakistan, Afghanistan and Guatemala. Trouble spots remained in even relatively well-off places such as China and Peru, researchers report in the Jan. 9 *Nature*.

"There are areas that have been left behind," says public health expert Damaris Kinyoki of the University of Washington in Seattle. The results were especially disappointing for middle-income countries, she says. "We expected them to have better progress."

Prolonged childhood malnutrition — associated with lifelong cognitive and physical impairments, and even death — is difficult to measure. So scientists use a proxy called childhood growth failure, defined as insufficient height and weight for children under age 5. Typically, growth failure rates are assessed at the state or national level. Because that broad geographic scale can obscure localized health disparities, municipal leaders can struggle to make targeted programs.

Building on earlier work in Africa, Kinyoki and colleagues have zoomed in on almost 3.7 million 25-square-kilometer "pixels" across the 105 low- and middle-income countries where 99 percent of all children suffering from malnutrition live. The team estimated annual childhood growth failure rates in each pixel from 2000 to 2017 using information from household surveys representing 4.6 million children. Those surveys were scaled up to the broader population using information from another study that included national-level estimates of malnutrition.

The team calculated three aspects of growth failure: stunting (short stature for age), wasting (low weight for height) and underweight (low weight for age). Stunting arises from chronic malnutrition; wasting arises from acute events, such as a famine, and is often lethal.

Malnutrition has been declining, the team found. For instance, stunting — the most prevalent and widespread indicator of growth failure — dropped from 36.9 percent in 2000 to 26.6 percent in 2017, although 176.1 million children were still stunted in 2017. Areas that saw the most progress include Central America, the Caribbean, the Andean region of South America, North Africa, East Asia and some parts of sub-Saharan Africa.

But only 28 of the 105 countries are on track to meet the World Health Organization's national-level malnutrition reduction targets for 2025. Targets include reducing the number of children who suffer from stunting to about 105 million and reducing wasting levels from about 7.8 percent in 2012 when the targets were set to less than 5 percent. (Some 58.3 million children, or 6.4 percent, suffered from wasting in 2017, the study found.)

Zooming in further, only five countries are set to meet both of those targets in every district: Palestine (counted as a country in this analysis), Paraguay, Peru, São Tomé and Príncipe, and Turkmenistan. A district is the equivalent of a town or suburb.

176.1
million
Number of children with stunted growth in 2017

The data revealed disparities within countries. For instance, Peru has achieved the WHO's national-level target for stunting, but almost a third of children in two provinces are affected.

These sorts of mapping projects should be approached with caution, says geographer Andrew Tatem of the University of Southampton in England. Surveys can vary widely in quality, and population counts for many countries may be out of date. But, Tatem says, such maps are still valuable. "It's the best estimate [of childhood malnutrition] given existing data." ■

LIFE & EVOLUTION

Tame fox study questioned

Researchers cast doubt on 'domestication syndrome'

BY JAKE BUEHLER

For the last 60 years, scientists in Siberia have bred silver foxes to be increasingly tame, with the goal of revealing the evolutionary and genetic underpinnings of domestication (*SN: 9/15/18, p. 12*). This research also famously showed a link between tameness and a collection of physical traits, including curled tails and spotted coats, known as "domestication syndrome."

But that story is flawed, some scientists claim. The foxes weren't totally wild to begin with, and some of the traits attributed to domestication existed long before the experiment, Elinor Karlsson, a biologist at the University of Massachusetts Medical School in Worcester, and colleagues argue online December 3 in *Trends in Ecology & Evolution*.

Fur farmer Leo Frank holds an apparently tame silver fox on Prince Edward Island in Canada in 1922.

The silver fox experiment, at the Russian Academy of Sciences' Institute of Cytology and Genetics in Novosibirsk, didn't seek to breed foxes that looked different from wild ones. But several generations after geneticist Dmitry Belyaev took 130 silver foxes (*Vulpes vulpes*) from Soviet fur farms and began selecting for friendliness toward humans, physical changes emerged. Floppy ears, spotted coats and other traits were known in other domestic mammals, so the changes have since been thought of as linked to the domestication process.

It's no secret that the foxes weren't truly "wild," Karlsson says. The Soviet foxes originally came from fur farms on Prince Edward Island in Canada, with selective breeding dating back to at least the 1880s. One of Karlsson's colleagues stumbled across fur farm photographs from the 1920s during a visit to a museum on the island. The foxes appeared tame with spotted coats — a trait that supposedly took generations to emerge in the Russian experiment.

The timeline undermines the narrative that the domestication syndrome traits sprang entirely out of the selection for tameness, Karlsson says. "These traits didn't get created within 10 generations. They were actually preexisting in the population."

The photos also led Karlsson's group to reconsider a bigger question: What's the evidence supporting domestication syndrome? The team soon found that not only was domestication syndrome loosely defined, so too was domestication itself.

So the team developed its own criteria for the syndrome. For instance, the traits should appear shortly after the onset of breeding for tameness, and grow in frequency and degree with increasing tameness. When these criteria were applied to domestication syndrome traits reported in foxes and other domesticated animals, no single species met all criteria, undermining the validity of a shared syndrome between domesticated mammals, the team claims.

Behavioral ecologist Christina Hansen Wheat of Stockholm University agrees that domestication syndrome isn't well-supported. "I find it problematic that we continue to conduct research on domestication based on too broad and unclear definitions and untested hypotheses," she says. "We need to reevaluate our expectations of the consequences of domestication."

But other scientists have doubts about the takedown. Adam Wilkins, an evolutionary biologist at Humboldt University of Berlin, has investigated the developmental underpinnings of domestication

Dark-furred silver foxes similar to this one have been bred for tameness in a long-running experiment in Russia.

syndrome. He says the new study misrepresents the idea. It's treated as a specific and constant set of characteristics across domestic mammals. But domestication syndrome has been envisioned as differing from species to species, he says. For instance, it may result in floppy ears in domesticated rabbits, pigs and sheep, but in smaller but similarly shaped ears in cats, ferrets and camels.

Lyudmila Trut, who has been involved in the silver fox experiment from the start, also disputes Karlsson's arguments. Trut admits that a small percentage of fur farm foxes were not very fearful or aggressive toward humans. But "we repeatedly visited those large fur farms," and none of the other traits associated with domestication syndrome were present, she claims. The allegation that tameness and spotting were imported into the experiment along with the Canadian foxes is "a misguided contention," Trut says.

Lee Alan Dugatkin, a behavioral ecologist at the University of Louisville in Kentucky who wrote a book on the Russian foxes with Trut (*SN: 5/13/17, p. 29*), agrees. He notes that the physical traits didn't crop up until six to 10 generations in. "It's extraordinarily unlikely that there was ... hidden genetic variation for these traits," he says.

And the fur farm images from the 1920s "could easily have [shown] animals that had been trained or learned how to be friendly with the person in the picture," Dugatkin says. "That's very different than suggesting that the animals are inherently friendly." ■

FROM TOP: MONTY SLOAN; KEYSTONE-MAST COLLECTION/CALIFORNIA MUSEUM OF PHOTOGRAPHY/UNIV. OF CALIFORNIA, RIVERSIDE

Outrunning Alzheimer's

An exceptional case within a large family prone to early dementia points to new treatment ideas

By Laura Sanders

A cruel twist of genetic fate brought Alzheimer's disease to a sprawling Colombian family. But thanks to a second twist, one member of the clan, a woman, managed to evade the symptoms for decades. Her escape may hold the key to halting, or even preventing, Alzheimer's.

The inherited version of Alzheimer's disease erodes people's memories early, starting around age 40. In this family and others, a mutation in a gene called *presenilin 1* eventually leaves its carriers profoundly confused and unable to care for themselves. Locals around the Colombian city of Medellín have a name for the condition: *la bobera*, or "the foolishness."

The woman in the afflicted family who somehow fended off the disease carried the same mutation that usually guarantees dementia. And her brain was filled with plaques formed by a sticky protein called amyloid. Many scientists view that accumulation as one of the earliest signs of the disease. Yet she stayed sharp until her 70s.

Researchers were stumped, until they discovered that the woman also carried another, extremely rare genetic mutation that seemed to be protecting her from the effects of the first one. This second mutation, in a different Alzheimer's-related gene called *APOE*, seemed to slow the disease down by decades, says Joseph Arboleda-Velasquez, a cell biologist at Harvard Medical School.

"There was this idea of inevitability," he says. But the woman's circumstances bring "a different perspective" — one in which amyloid buildup no longer guarantees problems. Arboleda-Velasquez and colleagues reported the details of the woman's exceptional case November 4 in *Nature Medicine*, omitting the woman's name and precise age to protect her privacy.

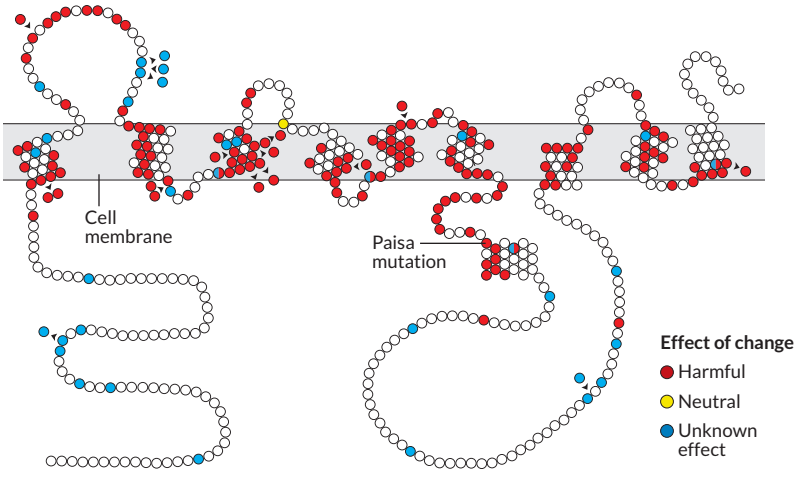
Although the discovery is based on one person,

In Colombia, Oderis Villegas (center), who is showing signs of Alzheimer's, and his sister Maria Elsy (left), whose disease is more advanced, meet with neurologist Francisco Lopera (right) in this 2010 photo.

TODD HEISLER/*THE NEW YORK TIMES*/REDUX PICTURES

it points to a biological weak spot in the degenerative disease that affects an estimated 5.8 million people in the United States alone. So far, nearly every clinical trial designed to slow or stop the disease has failed. Those heartbreaking disappointments have prompted scientists to expand their search for treatments (see "Casting a wide net," Page 22).

Perhaps this unusually resilient woman in Colombia shows a way to halt the disease, or at least slow it down. "Can we come up with a drug that does this to people who don't have a mutation?" asks Arboleda-Velasquez. "The potential for that is tremendous."



Small change, big effect The presenilin 1 protein spans cell membranes and helps chop a long protein into amyloid-beta and other bits. Researchers know of about 300 genetic mutations that alter the protein's amino acids (circles; colors show changes). The Paisa mutation leads to early onset Alzheimer's. SOURCE: ALZFORUM.ORG

Family tree

The vast majority of people with Alzheimer's have a sporadic form of the disease with no clear genetic culprit. These people often reach their 70s or 80s before signs of dementia appear. Mutations that cause trouble much earlier, such as the Paisa mutation found in the Colombian family, are unusual. But despite their different origins and different timelines, these two versions of Alzheimer's are thought to progress in somewhat similar ways.

Normally, *presenilin 1* makes a protein that helps chop up the long, sticky amyloid precursor protein. One of the resulting small bits is called amyloid-beta. Those smaller pieces are harmlessly washed out of the brain. The mutated *presenilin 1* gene found in the Colombian family, however, creates a kink in the chopping process that leads to an abundance of a version of amyloid that knits itself into plaques between brain cells.

This pileup is already visible in brain scans of people in their 20s who carry the mutation. By their mid-40s, many of these people have trouble remembering; they typically develop full-blown dementia by age 50.

Inheriting just one copy of the mutation is enough to lead to excess amyloid, and ultimately dementia. The mutation's powerful effect in this family is "one of the strongest arguments for the fact that amyloid plays a critical role" in Alzheimer's, says immunologist and aging expert Richard J. Hodes, director of the National Institute on Aging in Bethesda, Md. Since taking on the role in 1993, Hodes has helped set the course for U.S.-funded Alzheimer's research, allocating support for promising projects, including studies happening in Colombia.

The Colombian family, 5,000 members strong, includes an estimated 1,000 or so people who carry the Paisa mutation in the *presenilin 1* gene. Their involvement in the research has been invaluable. Access to hundreds of people known to be at high risk for the disease allows scientists to study how Alzheimer's unfolds, particularly at its earliest stages, and has led to reports of early signs of Alzheimer's, both in the brain and the blood. Family members have gone to great lengths to help, "walking or taking a bicycle to the nearest bus stop, and then taking a bus to a train, for many hours, to come to the clinic," Hodes says.

During Hodes' recent visit to the Medellín area, a resident told him how the disease is just a part of their lives: "If I have the disease, I know that my family, my brother and my sister, will take care of me. And if I don't, I will take care of them."

A unique brain

When Colombian researchers learned of the woman who stayed sharp until her 70s, they arranged for her to travel to Boston in the summer of 2016, accompanied by family members and a research assistant. There, neuroimaging researcher Yakeel T. Quiroz and her colleagues used brain scans to measure levels of amyloid and other markers of brain health, including another Alzheimer's-related protein called tau, which can tangle up inside nerve cells.

Those scans revealed a brain loaded with amyloid, says Quiroz, of Harvard Medical School. This woman had most likely been accumulating amyloid for decades. On a scale commonly used to quantify amyloid in the brain, she scored 1.96, well above the threshold of 1.2 that signifies extensive amyloid buildup. Her score was, "pretty much the highest that we have seen in anybody we have scanned so far," Quiroz says.

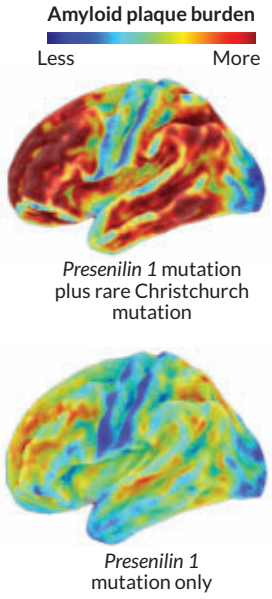
Genetic analyses revealed that the woman had

5.8 million
Estimated number of people in the United States with Alzheimer's disease

13.8 million
People expected to have Alzheimer's disease in the United States by 2050

SOURCE: ALZHEIMER'S ASSOCIATION

E. OTWELL



Brain aflame These brain scans are from two people with the Paisa mutation linked to early Alzheimer's. The top scan is from a woman with a rare second mutation that seemed to protect her until her 70s, even though she had lots of amyloid (red). At bottom, is a person with amyloid buildup whose memory problems began closer to age 40.

Presenilin 1 mutation plus rare Christchurch mutation

Presenilin 1 mutation only

what's called the Christchurch mutation in both copies of her *APOE* gene. Further tests suggested that this mutation, named for the New Zealand city where it was first found, was shielding her from the disease. The fact that the woman had huge amounts of amyloid in her brain, yet didn't seem impaired until her 70s, is "extremely surprising, interesting, provocative and potentially very, very informative," Hodes says.

Scientists need to do more work to confirm that the *APOE* Christchurch mutation protected her brain. Still, the results reveal a simple truth, Hodes says. "Amyloid itself is not necessarily sufficient to cause dementia."

Studies outside of the Colombian family also make clear that amyloid isn't the whole story. Other cellular actors contribute to the death of nerve cells and memory loss that Alzheimer's brings. Nerve cell–clogging tangles of tau and other signs of brain illness are tightly linked to brain decline, research from many studies has shown. That's reflected in observations from a study of 480 people age 60 and older who live around Rochester, Minn.

These people, none of whom showed signs of dementia, were randomly chosen to be invited into the study, an unbiased selection that offered researchers a glimpse of brain health in the wider population.

To find out which brain changes best predict future memory loss, neuroradiologist Clifford R. Jack Jr. of the Mayo Clinic in Rochester and colleagues tested volunteers' memory performance while measuring their amyloid levels and other brain signals. Amyloid seemed to be closely involved in memory decline over about five years — but only in the right context, the team reported in June 2019 in *JAMA*.

Without either of two other troublesome markers — tau tangles or brain shrinkage — amyloid didn't predict memory loss. In other words, amyloid might be setting up the shot, but then it passes the ball.

Stretching the lag

"Amyloid in the head is the first stage of what will ultimately lead to full-blown Alzheimer's disease," Jack says. But there can be a lot of time between that early stage of amyloid accumulation and the development of symptoms.

Among the Colombian family members, that interval lasts around 10 to 15 years. The same is roughly true for people with the sporadic form of Alzheimer's. But for the woman described in the report in *Nature Medicine*, that lag seemed twice as long.

"That suggests that at least it's possible to live with amyloid not just for 15 years, but for many decades," says Paul Aisen, director of the University of Southern California's Alzheimer's Therapeutic Research Institute in San Diego. Living healthy longer: "That's very exciting."

The protective effect of the woman's mutation seems to come from an extremely specific change. In the Christchurch variant, a single spot in the *APOE* gene is tweaked. The resulting

Casting a wide net

The search for drugs that slow or stop Alzheimer's disease has largely fizzled. Between 2002 and 2012, 413 clinical trials were under way. Of those, only one drug crossed the finish line and got approved as safe and effective for patients. But like the four other drugs approved before then, it only treats symptoms; it doesn't stop the disease.

"There have been a lot of ups and downs in recent months and years, unfortunately more downs than ups," said neurologist Ronald Petersen of the Mayo Clinic in Rochester, Minn. Petersen's comments, made December 5 at the Clinical Trials on Alzheimer's Disease meeting in San Diego, preceded news of study results that resurrected the amyloid-targeting drug aducanumab (*SN: 1/18/20, p. 8*). Scrapped earlier in 2019, the drug now seems to have helped some people who took the highest dose. In other tests, drugs aimed at amyloid have failed, leading scientists to look for other ways to slow or stop the disease:

Tau and **APOE** proteins are the targets in several clinical trials. Some of the studies are testing antibodies that bind to tau and lead to its destruction. Scientists are recruiting volunteers for a gene therapy trial that will attempt to swamp the brains of people who carry high-risk APOE4 proteins with low-risk APOE2 versions.

Inflammation is thought to lead to nerve cell death, a hallmark of Alzheimer's, in many ways. Several clinical trials are tweaking the behavior of immune cells called microglia to either quiet the dangerous inflammation or eat up amyloid.

As part of a two-year study called U.S. POINTER, 2,000 senior volunteers in the United States are improving their **food intake**, **exercise** and **intellectual and social stimulation**, to try to stave off decline. A study in Finland found that older people who put in effort to make such lifestyle changes had a slower rate of mental decline.

— Laura Sanders

J.F. ARBOLEDA-VELASQUEZ *ET AL/NATURE MEDICINE* 2019

protein has a serine amino acid swapped in for the standard arginine.

The swap prevents the APOE protein from binding to some sugar-dotted proteins called heparan sulfate proteoglycans, or HSPGs, experiments on the isolated proteins revealed. Earlier studies showed that HSPGs may promote amyloid accumulation and nudge nerve cells to slurp up more toxic tau.

But to misbehave, HSPGs might need to partner with the APOE protein. The Christchurch mutation could have protected the woman's brain by scrambling that nefarious relationship, the researchers suspect. Without that specific connection between APOE and HSPGs, "the disease process gets stalled," Arboleda-Velasquez says. "This really puts a block on the cascade of events."

Fleshing out the APOE protein's normal biological cascade, and how that changes with the Christchurch mutation, is "going to allow for much more finely targeted drug development," says Aisen, who also works as a consultant for Biogen, a biotechnology company in Cambridge, Mass. The company is developing an amyloid-targeting drug called aducanumab and is expected to apply for approval from the U.S. Food and Drug Administration this year (*SN: 1/18/20, p. 8*).

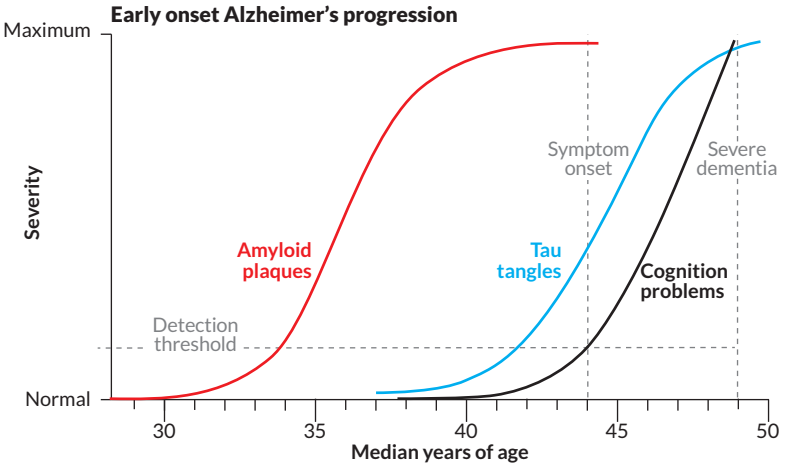
As one of the strongest genetic risk factors for dementia, the *APOE* gene has long been scrutinized as a possible target for Alzheimer's drugs. People who carry a version of the gene called *APOE4* have a higher risk of Alzheimer's.

The *APOE2* version dramatically lowers the risk, Quiroz, Arboleda-Velasquez and colleagues report in preliminary research posted online November 2 at medRxiv.org. *APOE3* usually brings an average risk of Alzheimer's, with the notable exception of the version with the Christchurch mutation carried by the Colombian woman.

Hope for the future

In the general population, old age is the biggest risk factor for Alzheimer's. As the number of older people balloons, so too will the number of people with dementia. By 2050, an estimated 13.8 million people in the United States will have Alzheimer's. Worldwide, an estimated 50 million people have dementia; Alzheimer's accounts for the bulk of those cases.

The family in Colombia continues to help. A clinical trial testing a drug that is designed to lower amyloid is under way in Colombia. People who have the Paisa mutation but have not shown Alzheimer's symptoms, as well as people without the mutation, are receiving the drug. The drug, crenezumab, is an antibody that's thought to mark amyloid for destruction by immune cells. It's being developed by Roche/Genentech.



Push ahead People with the Paisa and other *presenilin 1* mutations begin building amyloid in their brains in their 20s, followed by tau tangles and dementia symptoms, as in this approximation. Researchers aim to stretch the time between early disease signs and symptoms in people with early onset Alzheimer's and those with the more common form of the disease. SOURCE: E. MCDADE AND R.J. BATEMAN/*JAMA NEUROL.* 2018

Quiroz and her colleagues also plan to follow the Colombian woman and other members of the family over time, as part of a research exchange between Fundación Universidad de Antioquia in Medellín, which has led the studies on this family, and Massachusetts General Hospital in Boston.

Each month, the project, called COLBOS, for Colombia-Boston, flies a new group of about five adult participants to Boston for extensive evaluation, including thinking and memory tests, brain scans and measurements of smelling ability, fitness and music perception. Participants being studied in Colombia are as young as 9 years old.

The project may yield insights about how Alzheimer's takes hold early on. But in a way, the initial trigger might not even matter. It could be that the cause — or more likely, causes — of Alzheimer's might ultimately be poor targets for drugs, Arboleda-Velasquez says.

People with loved ones suffering from Alzheimer's, including the Colombian family, don't necessarily care what causes the disease, Quiroz says. "They are more interested in seeing if there is anything that can help them to get better. That's what the patients and families are waiting for." ■

Explore more

- National Institute on Aging. Alzheimer's Disease Genetics Fact Sheet: bit.ly/AlzGenetics

E. OTWELL

STEM CELL HARD SELL

Research on 'regenerative' treatments don't support clinics' claims

By Laura Beil

Joanna had just turned 62 when she noticed that she couldn't stand very long before her right leg would hurt. She thought it was from an old injury, when her dog had slammed into her thigh. When the ache moved to her wrist, she went to a doctor who said she might be getting arthritis.

The pain quickly intensified. "It just happened so rapidly, and I couldn't figure out why," says Joanna, who lives in a Houston suburb. Her doctors chalked it up to wear and tear. "'You're getting older,'" she remembers them telling her. This was in early 2018.

1,000
Estimated number of U.S. clinics offering stem cell treatments

Then she got an e-mail with a link to a video about stem cells and the conditions they could cure, including arthritis. "I started watching it and then I just turned it off for a while because I thought, 'I'm afraid I'm going to get my hopes up too high,'" says Joanna, who asked that her last name not be used to protect her medical privacy.

She started seeing full-page ads for stem cell seminars in the newspaper. She attended one at a local hotel, and the presenter announced that thousands of patients had benefited from stem cell injections. It was natural, the woman said. No one had ever been harmed. The idea that the treatment wasn't a drug reassured Joanna.

She made an appointment for the next day. "It sounded too good to be true, but I was desperate," she says. She received injections into her back, neck and shoulder of stem cells from donated umbilical cord blood followed by an IV of the product the next day. The cost was $30,000, siphoned from her husband's pension. She knew she was taking a risk, but she felt hopeful.

Two days later, her face began to burn and itch. Then her feet. She had pain in places that had never hurt before, like the joints of her fingers. Her hair started falling out, and she descended into a deep depression. "I'm totally miserable," she says, months later. "I'm just agonizing in pain.... Now I don't see any hope."

Stem cells sold at clinics are driving what's thought to be a $2 billion global industry. Facebook pages announce seminars.

C. CHANG

Local newspapers are wrapped in ads vowing "relief without surgery." Stem cells are billed as treatments for everything from autism to multiple sclerosis to baldness. Most commonly, the ads focus on orthopedic issues, especially aching knees.

An important point gets left out of the cheery ads: There's not enough science to justify using stem cells for any of the advertised conditions, including joint pain. None of the treatments advertised have been approved by the U.S. Food and Drug Administration. (The only approved stem cell treatments are for certain cancers and blood disorders.) Very few of the orthopedic studies in humans have been scientifically rigorous, and none have shown stem cells regrowing cartilage.

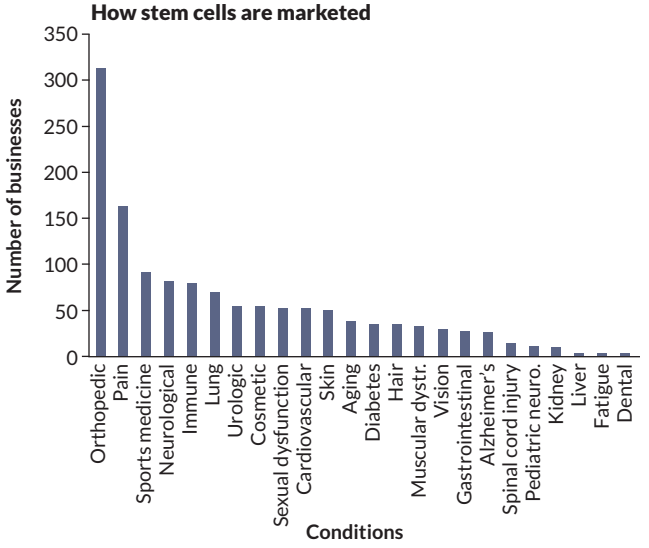
It's not even clear whether treatments being touted as "stem cells" contain viable stem cells or whether the contents should be defined as stem cells at all. As the stem cell industry grows rapidly, many researchers who are studying stem cells for their potential to regenerate tissues worry that the booming marketplace, which conflates hype with reality, might ultimately damage research progress.

"The scientific and medical community is having to play defense," says Shane Shapiro of the Mayo Clinic Jacksonville in Florida, who has conducted one of the very few published trials that compared stem cells with a placebo for people with osteoarthritis. "Misinformation and misunderstanding about how cells are used to treat disease is proliferating."

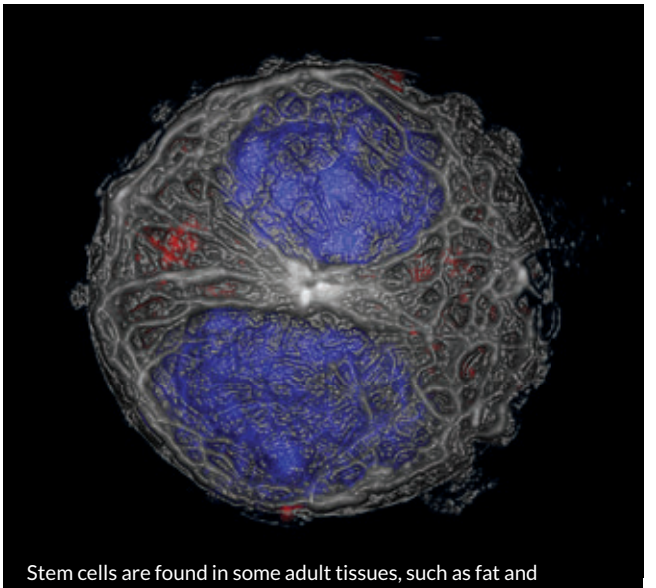
Early infamy

The explosion of advertising reflects a dramatic turnaround from the controversy over stem cells that occupied the public in the early 2000s. At the time, scientists had learned how to generate lines of cells from embryos that were left over from in vitro fertilization and donated for research. In theory, the embryonic cells had the potential to treat disease by becoming

Seeking relief U.S. clinics advertise stem cells to treat a variety of diseases (top ones shown below), but most treatments are for orthopedic conditions and pain. SOURCE: L. TURNER AND P. KNOEPFLER/*CELL STEM CELL* 2016



FROM TOP: MARK KIEL AND SEAN MORRISON/CHILDREN'S MEDICAL CENTER RESEARCH INST./UT SOUTHWESTERN; C. CHANG



Stem cells are found in some adult tissues, such as fat and bone marrow (one bone marrow stem cell shown dividing), as well as umbilical cord blood and placentas. But the cells' ability to cure osteoarthritis has not been proven.

a slew of different tissues, but their use became entangled with the politics of abortion. Then in 2001, President George W. Bush banned federal funding for research on embryonic stem cell lines not already in labs. Embryonic stem cell research has ridden the political tides since then: Restrictions were eased under Barack Obama's presidency, then Donald Trump's administration added restrictions on fetal tissue use.

Scientists tried to persuade the public to support the research by focusing on the great promise. They argued that stem cells might one day cure diseases by naturally repairing lost or damaged tissue. Actor Michael J. Fox, the most famous Parkinson's patient of his generation, testified to Congress in 1999 that stem cells could one day cure degenerative brain diseases. Joanna remembers Fox's passion. "That's what kind of made us aware of what stem cells were at the time," she says.

Broadly speaking, stem cells are cells capable of renewing themselves and taking on the identity of the tissue around them (*SN: 3/19/16, p. 22*). The early controversy about using embryonic cells has largely quieted down. The stem cells being marketed today are not embryonic; they come from bone marrow, fat tissue or birth products such as umbilical cord blood or amniotic fluid, all advertised as being able to regenerate cartilage. Clinic websites usually feature earnest testimonials with no hint of any possible negative side effects.

Free market

For the first part of the 2000s, stem cell treatments were largely sought through medical tourism. U.S. patients would travel to other countries for experimental treatments to cure diseases such as multiple sclerosis or spinal cord injury.

Bioethicist Leigh Turner of the University of Minnesota in Minneapolis noticed a shift around 2012. "One of the businesses

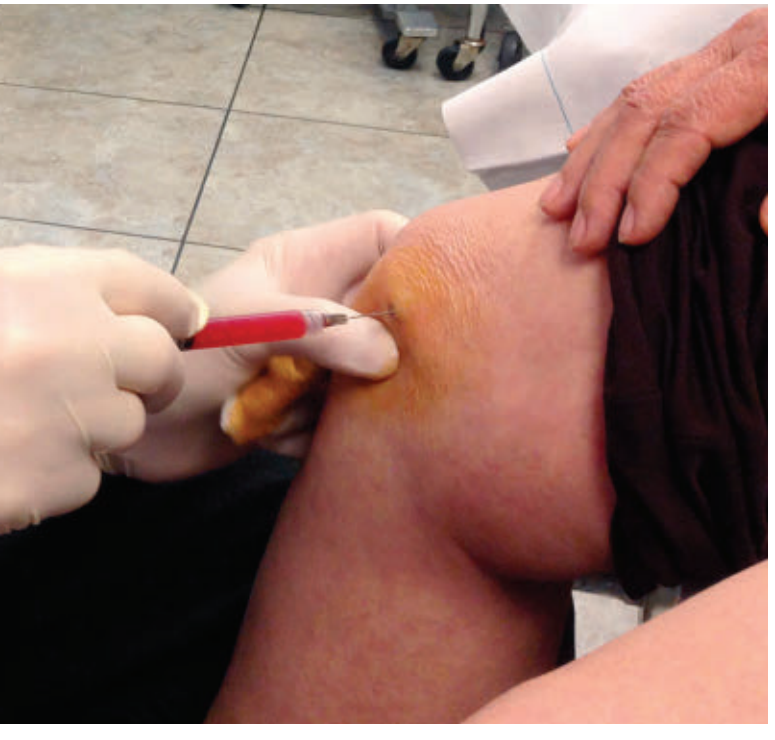
that ... was part of that marketplace, a company in South Korea, ended up popping up in the United States," he says. The company, operating under the name Celltex, offered to remove stem cells from a patient's own fat tissue, grow the cells in a lab and then reinject them.

When the FDA sent a warning letter to the company in September 2012 stating that its products must be approved before use in patients, the company moved its treatments to Mexico. Regulations there are less stringent, though the business remains headquartered in Houston.

Today, many clinics have learned how to operate just inside the margins of federal regulations, or simply ignore them. Turner and Paul Knoepfler of the University of California, Davis mapped the rise in U.S. stem cell clinics that market unapproved therapies, reporting in 2016 in *Cell Stem Cell*. The first few clinics emerged about 10 years ago, Turner says. "By 2014, 2015, companies are pouring into the marketplace at a very rapid rate."

> "We weren't able to conclude that the stem cell product was any superior in pain relief to anything else."
>
> **SHANE SHAPIRO**

Knoepfler estimates that today more than 1,000 clinics across the country offer stem cells, though there are probably more because many doctors and chiropractors have simply added stem cells as a sideline to their main services. For some, stem cells are lucrative enough to support a business on its own. One in 4 stem cell providers in the Southwest offers the treatment exclusively, researchers at Arizona State University reported in August in *Stem Cell Reports*.



Thousands of patients are paying cash for stem cell injections into their joints to cure their arthritis. But proof of effectiveness is lacking, and there's no evidence that the injections can regrow cartilage.

RAQUEL MARIA DILLON/ASSOCIATED PRESS

The business is extremely profitable, and the treatments are rarely covered by insurance. Patients pay cash — sometimes draining their life savings, taking out loans or drawing down retirement funds like Joanna did.

"Often, when you go to business websites, there's not this kind of sober, frank, judicious accounting of risks and benefits — or the possibility that there might be no benefit, that someone could be harmed," Turner says. The websites "tend to frame risks and benefits in a very misleading kind of way."

And because each year tens of thousands of U.S. patients — by Knoepfler's estimate — are getting cells in clinics outside of clinical trials, it's difficult to know exactly what the risks of the direct-to-consumer marketplace are. No one is keeping track.

In 2018, researchers writing in *Stem Cells Translational Medicine* resorted to searching Google and the scientific literature, where they found 35 reports of serious consequences. Some patients in Florida lost their sight after getting stem cell injections into their eyes. In December, the FDA warned of "serious illnesses" in Nebraska linked to treatments with "exosomes," products taken from placentas that are offered by some stem cell clinics.

A recent survey of neurologists, presented in March 2019 in Dallas at a meeting of the Americas Committee for Treatment and Research in Multiple Sclerosis, asked doctors how their patients had fared after receiving unapproved stem cell treatments. About 25 percent of the 204 neurologists who responded said patients had suffered serious consequences such as strokes and seizures. Three doctors reported that patients had died. Without study, it's impossible to know why.

The evidence on knees

It makes sense that treatments for knee pain appear to dominate the industry — the potential customer base is large and growing. More than 600,000 people in the United States had knee replacement operations in 2014, according to data released in 2018 by the American Academy of Orthopaedic Surgeons. That number is expected to rise as baby boomers age and obesity rates climb. As the body ages, shock-absorbing cartilage in the joints wears away, which can lead to painful bone-on-bone rubbing. Stem cells are advertised as an easy way to avoid surgery.

So far, though, it's not clear that's true. Recently, Maarten Moen, a sports medicine physician at Bergman Clinics in Naarden, Netherlands, and his colleagues reviewed every clinical trial he could find that examined using stem cells for knee osteoarthritis. "Stem cells are prohibited for use in Holland," says Moen, a member of the medical staff of the Dutch Olympic Committee. "That's why we were conducting this: to see if we could convince people in our country to possibly start using this therapy. But only if we answer these two questions: Is it helpful? And also, is it safe?"

The group's results appeared in 2017 in the *British Journal of Sports Medicine*. The team found only six human studies testing stem cells for knees, and none were large trials that included a placebo comparison. While the treatment appeared safe, effectiveness couldn't be determined. Every study had methodological problems. As a result, the authors didn't recommend stem cells for knee osteoarthritis. Moen has recently updated his review, but those results have not yet been published. He offers a preview: "The evidence didn't get any stronger." He found only two clinical reports that had compared the treatment with a placebo.

Both were from Shapiro, of the Mayo Clinic. "It's been nearly three years since we first published our early results," Shapiro says. With his trial as "the first piece of the puzzle," he expected that, "like anything else in science, we would be followed by a bunch of other trials." So far, other published results have not been pouring in.

Shapiro and colleagues from the Mayo Clinic and Yale University School of Medicine had tested 25 patients with mild knee osteoarthritis in both legs. The researchers took about 50 milliliters of cells from each patient's bone marrow, concentrated the cells in the laboratory and then injected them back into one knee of each patient. The other knee got an injection of saline as a placebo. The patients did not know which knee got the experimental treatment.

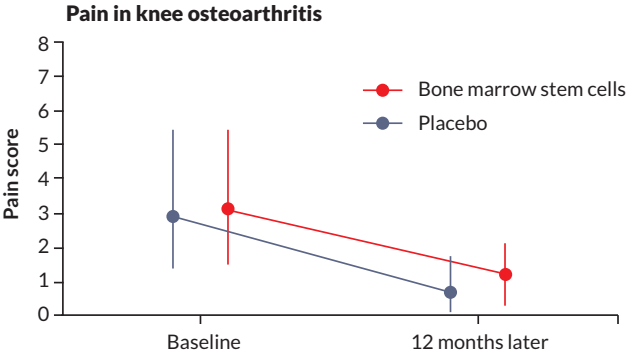
Both the stem cell knee and the placebo knee improved by about an equal degree — about 50 to 75 percent on a pain scale, the team reported in 2017 in the *American Journal of Sports Medicine*. "We weren't able to conclude that the stem cell product was any superior in pain relief to anything else," he says. "Additionally, we did not see any cartilage regrowth." A 12-month follow-up published in October in *Cartilage* found similar results.

The fact that the saline alone helped patients feel better was not surprising. A meta-analysis published in 2017 in the *American Journal of Sports Medicine* examined whether simple saline injections helped knee pain. That review, by researchers at Rush University Medical Center in Chicago and the University of Toronto, found that saltwater alone provided relief on the level of some drugs. But why the knees that got the bone marrow cells in Shapiro's study improved to an equal degree is still unclear, he says. So he's not yet ready to say the stem cells don't work.

"What I think actually happened is that we injected a therapeutic substance in one of their knees, and we injected a nonharmful substance, which is the saline, in the other knee, and the patients were able to get back to their life for a period of time that overall made them feel better," he says.

The Mayo study tested stem cells from the patients' own bone marrow. But clinics also offer cells from the body's fat tissue, extracted by liposuction. Doctors can use enzymes to strip away extraneous cells from the fat, leaving only the regenerative cells. But that therapy is also unproven. One study, conducted by researchers in Australia and published in February 2019 in *Regenerative Medicine*, involved 30 patients

No difference In one of the only placebo-controlled studies of stem cells for knee osteoarthritis, a placebo shot (blue) was as effective in reducing pain as an injection of stem cells from the patients' own bone marrow (red). SOURCE: S.A. SHAPIRO *ET AL*/*CARTILAGE* 2019



with knee osteoarthritis. Patients who received stem cells from fat reported a 69 percent improvement in their pain, compared with no change in a comparison group that did not receive the treatment. But that study offered no placebo injection for comparison.

A second study, by a South Korean team, had a placebo, but a small number of patients. Twelve patients who received stem cells from their own fat had a 55 percent improvement in pain (based on their responses to a questionnaire) compared with no substantial improvement among 12 patients who got a placebo shot, the researchers reported in March 2019 in *Stem Cells Translational Medicine*. Cartilage didn't regrow with stem cells, but it didn't shrink, which it did in the placebo group.

But the treatments in those studies differ from what is actually offered at stem cell clinics. In both of those studies, the researchers expanded or concentrated the cells in the laboratory before injecting them into patients — a practice that is allowed in the United States only in a scientific study. Under FDA rules, U.S. stem cell centers are only allowed to move a patient's own tissue from one place to another, with little manipulation of the cells, otherwise the treatment may be considered an unapproved drug.

Sports medicine physician Kenneth Mautner and colleagues at Emory University in Atlanta compared outcomes for 76 patients with arthritis who received a treatment that was closer to what doctors can do in their offices. Each patient received either cells taken from their own bone marrow or from fat tissue. In both cases, the cells were simply moved to another place within the patient's own body. After six months, both groups showed pain reductions and neither treatment was better than the other, the researchers reported in the November 2019 *Stem Cells Translational Medicine*. "There was about 70 to 75 percent improvement for those who actually did improve," Mautner says. About one-quarter of the patients did not get better. Patients with more advanced arthritis were less likely to benefit.

But his study had that common shortcoming: no placebo comparison. "When you're paying a lot of money, there's obviously a

C. CHANG

placebo effect," Mautner says. "It's not just your mind convincing you that you feel better. The placebo effect can actually be chemicals and cytokines that then produce anti-inflammatory effects in your joints."

In addition to bone marrow and fat tissue, a growing number of clinics are offering products made from donated umbilical cord blood or other birth products, Knoepfler says. Those cells are easy to administer and don't require the expertise to extract cells from the body.

But if there is little evidence for the effectiveness of stem cells from fat and bone marrow, Shapiro says, "there is zero support" for umbilical products in human studies. "I'm not even studying them yet," he says.

No regrowth

Hardly any evidence supports the idea that treatments marketed as "stem cells" can regenerate worn tissue, which is what many patients think they are buying. "There's very little evidence that it will regrow your cartilage," says orthopedic surgeon Jason Dragoo of the University of Colorado Denver.

His research team is conducting a study to see whether there are treatments that might increase cartilage thickness. One study pairs the cellular treatment with surgery. The existing tissue may be more receptive to regrowth, he says, "if you clear away the debris and all the other things, get it as 'cleaned up' as possible, then give the cells." He's also conducting a study comparing the ability of cells from fat to repair tiny tears in cartilage that is otherwise mostly healthy, a process he compares with filling potholes.

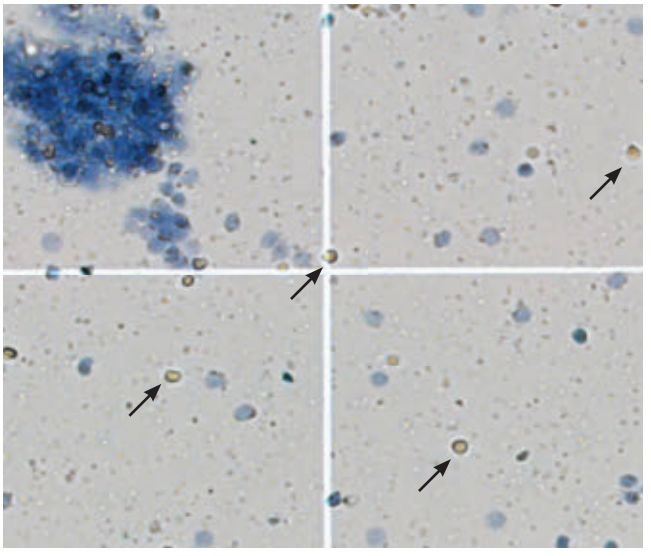
But even if cartilage won't regrow, he and others say, the procedure may still reduce inflammation, which could quiet a painful knee. There's also early evidence from animal studies that cells from bone marrow or fat might send chemical signals that jump-start a person's own healing.

Biomaterials scientist Sowmya Viswanathan of the University of Toronto and colleagues reported a study of 12 patients receiving bone marrow cells in August in *Stem Cells Translational Medicine*. The study had no control group. "We saw improvement in symptoms, in pain, in quality of life and in joint stiffness for all the patients. These are the things that the patients care about. The fact that it doesn't regenerate cartilage doesn't disprove its ability to still be a functional, useful cell therapy," she says. It might work, but maybe not in the way that patients expect.

All in a name

Viswanathan worries that the current stem cell market is exploiting the work of scientists, piggybacking off of the legitimate — but early — studies for immediate commercial gain, she says.

"Everything gets called stem cells. Nomenclature is still very important because if you can't name it properly, then you don't even know that you're talking about two different or three or four different things," she adds.



Some experts question the viability of stem cells taken from cord blood or placentas. In this analysis of one product, 70 percent of the cells were dead, as shown by the blue stain. The arrows point to live cells.

COURTESY OF LISA A. FORTIER

Many clinics call the cells in their products "mesenchymal stem cells," a term taken from a 1991 paper by biologist Arnold Caplan of Case Western Reserve University in Cleveland. Yet in 2017 in *Stem Cells Translational Medicine*, Caplan advocated for a name change: "Stem cell misconceptions have led some practitioners in the United States and worldwide to advertise the availability of stem cell treatments (i.e., MSCs can cure the blind, make the lame walk and make old tissue young again)."

Viswanathan and other members of the International Society for Cellular Therapy published a position statement in October in *Cytotherapy* that cells commonly identified as "mesenchymal stem cells" should more precisely be called "mesenchymal stromal cells" in the scientific literature to reflect the lack of evidence that, when used as a medical treatment, those cells can renew themselves and form different tissues. (Stromal cells form the body's connective tissue.) As long as everything is called "stem cells," she says, clinics focused on profits will be able to exploit legitimate research for marketing purposes.

And there are so many questions left to answer. She worries about what happens when people have bad reactions, like Joanna did. "We don't fully understand repeat injections. We don't know the dosing. If there is an adverse event, then what?" she says. "Then it sets back the kind of legitimate work that's being done because the difference isn't apparent to the funders and to the lay public because everyone's calling it exactly the same thing." ■

Explore more

- International Society for Stem Cell Research. "Nine things to know about stem cell treatments." bit.ly/9thingsonstemcells

Laura Beil is a contributing writer based in Texas. Her most recent podcast, "Bad Batch," is about the stem cell industry.

The Crowd and the Cosmos
Chris Lintott
OXFORD UNIV., $24.95

BOOKSHELF

An astrophysicist pays tribute to citizen science

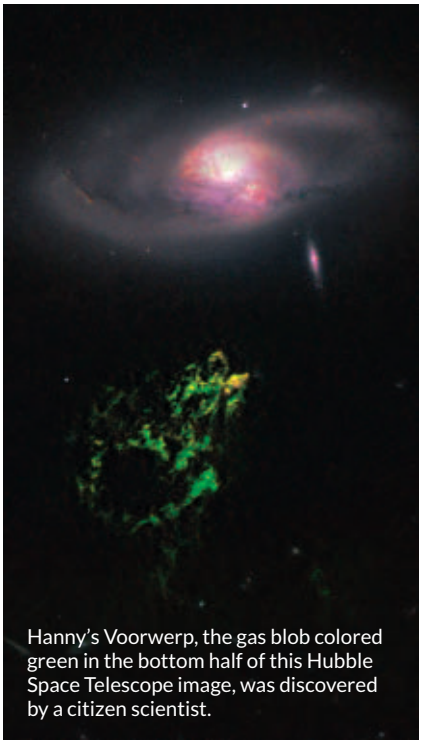
Astrophysicist Chris Lintott had a problem back in the mid-2000s. He wanted to know if the chemistry of star formation varies in different types of galaxies. But first he needed to sort through images of hundreds of thousands of galaxies to gather an appropriate sample to study. The task would take many months if not longer for one person, and computers at the time weren't up to the challenge. So Lintott and colleagues turned to the public for help.

The group launched Galaxy Zoo in 2007. The website asked volunteers to classify galaxies by shape — spiral or elliptical. Interest in the project was overwhelming. On the first day, so many people logged on that the server hosting the images crashed. Once the technical difficulties were resolved, more than 70,000 image classifications soon came in every hour. And as Lintott would learn, amateurs were just as good as professionals at categorizing galaxies.

Galaxy Zoo's success helped awaken other scientists to the potential of recruiting citizen scientists online to sift through large volumes of all sorts of data. That led to the birth of the Zooniverse, an online platform that lets anyone participate in real science. Projects on the platform ask volunteers to do everything from digitizing handwritten records from research ships to identifying animals caught on camera to sorting through telescope data to find signs of exoplanets.

In *The Crowd and the Cosmos*, Lintott, who cofounded the Zooniverse, shares his experiences with citizen science. The book is not a recounting of the history of Galaxy Zoo and the Zooniverse. It's more of an ode to citizen science. Lintott celebrates the successes, exploring the ways amateurs can contribute to science and how that contribution might change as artificial intelligence catches up with some kinds of human smarts.

By no means was Galaxy Zoo the first citizen science project. As Lintott explains, the roots of citizen science go back to at least the 18th century. Even Charles Darwin benefited from observations contributed by a wide network of people. *The Crowd and the Cosmos* focuses on the importance of citizen scientists in the age of big data and largely sticks to what Lintott knows best: astrophysics and astronomy.



Hanny's Voorwerp, the gas blob colored green in the bottom half of this Hubble Space Telescope image, was discovered by a citizen scientist.

NASA, ESA, W. KEEL/UNIV. OF ALABAMA, THE GALAXY ZOO TEAM

The book peruses a range of space topics, offering up-to-date, accessible overviews of exoplanets, supernovas, galaxies and dark energy, the mysterious force that is causing the universe's expansion to accelerate. Lintott is a knowledgeable and witty guide. His humor helps drive the story and even pops up in numerous footnotes. After describing how he often ends talks with the idea that, far into the future, the universe will likely "become a nearly empty void, a vast sea of space expanding forever into yet more nothingness," he quips: "I do like to send an audience home happy."

Just as the universe's future may seem bleak, so too might the future of some forms of citizen science. When Lintott first enlisted volunteers to help wade through a deluge of galaxy images, computers were terrible at tasks that required pattern-recognition skills. But times have changed. Machine-learning algorithms' abilities on visual tasks are improving, and researchers are on the verge of automating many time-intensive, often tedious jobs. In fact, some Zooniverse projects today ask citizen scientists to classify data as a way to amass large datasets to help train machine-leaning algorithms. As artificial intelligence continues to get better, will there come a time when citizen scientists' services are no longer needed?

Lintott doesn't think so. He predicts humans and machines will keep working side by side, and at least for the foreseeable future, citizen scientists will still be needed to help train machine-learning algorithms. But he also envisions these volunteers making other important contributions. For instance, he argues that when looking through seemingly endless piles of images or historical records or even graphs of data, these amateurs are in the best position to notice something rare or unusual; experts tend to be too focused on the task at hand, and computers might not be trained to identify something out of the ordinary.

That was the case in 2007 when a volunteer in the Netherlands named Hanny van Arkel found a strange blob in an image and implored scientists to investigate. Dubbed Hanny's Voorwerp (Dutch for "object"), the blob is now known to be a large gas cloud still glowing after being hit by a jet of radiation from a nearby galaxy's black hole (*SN: 12/23/17 & 1/6/18, p. 5*). Researchers have learned that such gas clouds can be indicators that a now-quiet galaxy was active not too long ago (*SN Online: 4/24/15*).

Lintott's enthusiasm for citizen science — and his admiration of the talents and tenacity of citizen scientists — is inspiring. By the end of the book, I was ready to sign up for some projects in the Zooniverse. — *Erin Wayman*

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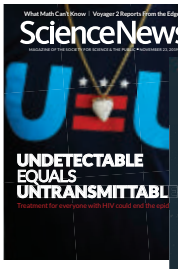


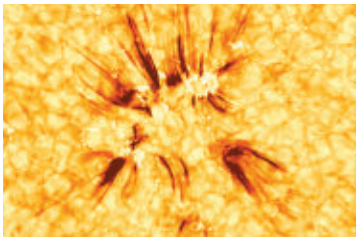
NOVEMBER 23, 2019

DECEMBER 7, 2019

SOCIAL MEDIA

Amber waves of plasma

The realignment of the sun's magnetic fields may form plasma jets (dark structures in the image below) that "undulate like a wind-whipped field of wheat" in a layer of the solar atmosphere called the chromosphere, **Christopher Crockett** reported in "Corona may catch heat from solar jets" (*SN: 12/7/19, p. 14*). Twitter user **@RadioPirate99** found the description of the jets enchanting, adding: "A 'wind-whipped field of wheat' is commonly known as 'wind wolves.'" The idea of a pack of wolves "rambling through the chromosphere is fantastically picturesque."



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T. SAMANTA, GST, SDO

Math whiz

*In "Known for the unknowable" (SN: 11/23/19, p. 22), **Evelyn Lamb** examined the life and career of mathematician Julia Robinson, who helped redefine the limits of mathematical knowledge.* Readers had a lot to say about the story.

"I found your article inspiring; it was really well written. I particularly liked the graphics, which helped my understanding a lot," reader **Richard Purser** wrote. "It has been my (unfortunate) experience that young women are socialized that they are 'less than' with regards to math. This story shows that to be a lie." Reader **Luis Villarruel** echoed **Purser's** comment and requested more profiles of women who contributed to STEM breakthroughs but were not widely acknowledged.

Reader **Lenore Blum** was pleased by an illustration of Robinson featured in the story. "I knew Julia from the time I went to Berkeley as a postdoc in 1968 until her death in 1985," **Blum** wrote. The illustration by Anna and Elena Balbusso "captures perfectly her impish smile and her sense of herself," **Blum** wrote. She believes Robinson would have been "honored and delighted" by the portrait.

Reader **Mike Dudzik** found a particular portion of the story humorous. "When I got to the part describing that earlier this fall, two mathematicians had 'used a mix of clever algorithms and a powerful supercomputer' to find a solution to the Diophantine equation involving 42 ... a hearty laugh welled up inside me," **Dudzik** wrote. "They had found the answer to the ultimate question of life, the universe and everything (42)! Was the supercomputer named 'Deep Thought'?" he wrote, referring to the science fiction series *The Hitchhiker's Guide to the Galaxy*. "Douglas Adams would be pleased."

Choice words

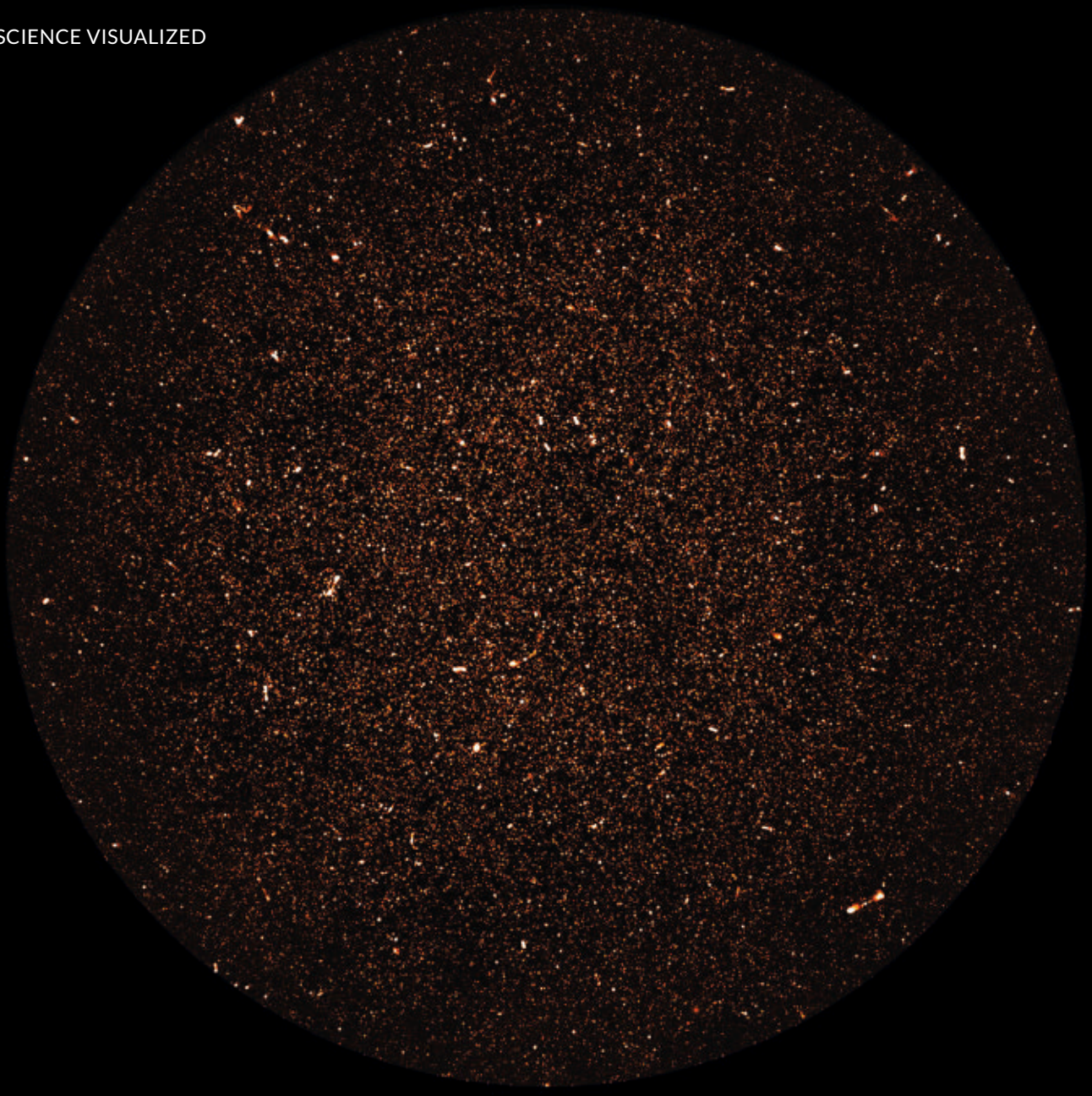
A Polar Affair *recounts a nearly century-long cover-up of Adélie penguins' "dirty" sexual behaviors, **Carolyn Gramling** reported in "The sex lives of penguins shocked early explorer" (SN: 12/7/19, p. 26).*

"I must object to using the word 'dirty' when describing the sexual behaviors of these fascinating animals," reader **Michelle Ashkin** wrote. "This is not dirty behavior. It is simply a natural behavior, and we need not ascribe our values to it," **Ashkin** wrote. "Please be aware that words have a lot of impact. And how we use them shapes readers' thoughts and opinions."

It was the book's author, penguin biologist Lloyd Spencer Davis, who used the word "dirty" in reference to the penguins' promiscuity and infidelity, **Gramling** says. "Davis was not himself intending to place a moral judgment on those observations," she says. He described the sexual behaviors sans judgment in scientific papers several decades earlier. "In *A Polar Affair*, he used that language ironically to highlight a major theme of the book, which is at its core a meditation on a strange case of scientific information being suppressed by misplaced moral judgment," **Gramling** says. "It should be noted that this particular quote was one of the milder ways in which Davis mocks that sort of judgment, so *caveat emptor*!"

Correction

"First black hole image made its debut" (*SN: 12/21/19 & 1/4/20, p. 22*) incorrectly stated that the black hole at the center of galaxy M87 is 1,000 times as massive as Sagittarius A*, the black hole at the center of the Milky Way. M87's black hole is about 1,600 times as massive as Sgr A*.



SARAO, NRAO/AUI AND NSF

Radio galaxies finally come into the light

Never-before-seen radio waves from tens of thousands of galaxies have a secret to share: The height of star formation in the cosmos may have been more prolific than imagined.

Radio telescopes are good probes of star formation. But until now, they haven't been sensitive enough to see radio waves coming from the vast majority of galaxies that produced stars during the peak of star production, an epoch roughly 10 billion years ago known as cosmic noon.

Now, an image (above) from the MeerKAT telescope in South Africa has lifted the radio veil on those unsung galaxies. In the image are more than 17,000 pinpoints of radio energy — nearly every one a star-forming galaxy. (A handful of the brightest blobs are gas spewing from supermassive black holes in some galactic centers.)

Astrophysicist James Condon and colleagues found that the radio waves come from star-forming galaxies at cosmic noon that churned out stars at about 10 times the rate of modern galaxies. What's more, there are nearly twice as many of these sources as expected, says Condon, of the National Radio Astronomy Observatory in Charlottesville, Va. That suggests that star formation was much higher around cosmic noon than previously predicted, the scientists report December 15 at arXiv.org.

— *Christopher Crockett*

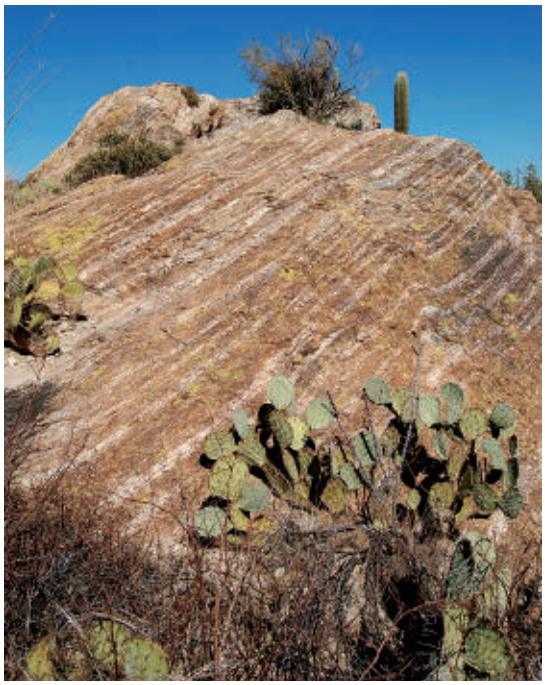
GEOLOGIC ROAD TRIP OF THE MONTH

SAGUARO NATIONAL PARK, EAST

Catalina Detachment Fault

Rarely can you see the lower plate, the upper plate, and the detachment fault of a metamorphic core complex all in one place. That's part of the reason they are so difficult to envision. But you can see all three parts in the east unit of Saguaro National Park. This example is so good, in fact, that the Geological Society of America included it in a volume about the best geologic sites in the western United States. Here, a huge amount of intact rock of the upper plate slid west along the Catalina Detachment Fault.

Cactus Forest Drive is a one-way loop road that begins at the park's visitor center. The first few miles cross rocks of the upper plate. About 0.6 mile from the drive's entrance is a small canyon where metamorphic rock called Pinal Schist is nicely exposed. Although this is some of the oldest rock in Arizona, dating to around 1.7 billion years ago, it lies above the detachment fault that exposed the igneous intrusions of the lower plate.



The Javelina Rocks are composed of Catalina Gneiss in the lower plate of the Catalina Detachment Fault.

The road passes from the upper plate onto the lower plate when it crosses the Catalina Detachment Fault (buried and not visible at this point) at the Loma Verde Trail. During the climb toward the Tucson Basin Overlook, look east at the Rincon Mountains. They are the lower plate, rock that once was far below the surface and covered by thousands of feet of upper plate rock. Now look to the west. Most of that upper plate material now lies buried under young sand and gravel beneath Tucson, with one large block forming the Tucson Mountains west of the city (Site 31).

About 5.5 miles from the visitor center, you can look down to the west and see the path of the Catalina Detachment Fault crossing low ridges, where it separates light-colored limestones of late Paleozoic age in the upper plate from darker, brownish rocks in the lower plate.

Javelina Rocks, at mile 6.1, are an outstanding example of the lower plate's Catalina Gneiss, a rock whose origin puzzles geologists. Before being metamorphosed during the extreme stretching that produced the Basin and Range Province, this rock might have been Oracle Granite. Others feel Precambrian sediments called the Apache Group are more likely. Both rock units are about 1.4 billion years old and are exposed in the Santa Catalina Mountains to the northwest. In either case, metamorphism at an estimated depth of 7 miles stretched, sheared, and recrystallized the rock into this distinctive banded gneiss, the core of the metamorphic core complex.

Returning back toward the visitor center, the road again crosses the detachment fault just west of the turn off to Javelina Picnic Area. Although the fault is not visible, the shallow valley north of the road marks its trace.

The Catalina Detachment Fault is visible where it separates light gray limestones in the upper plate (right) from darker metamorphic rocks in the lower plate (left).

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