JANUARY 2021

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How COVID-19 Wrecks the Immune System

Ancient Galaxy Clusters

Understanding Mountain Ice

DINO STAR

Scientists reveal the real *Dilophosaurus*, a *Jurassic Park* icon

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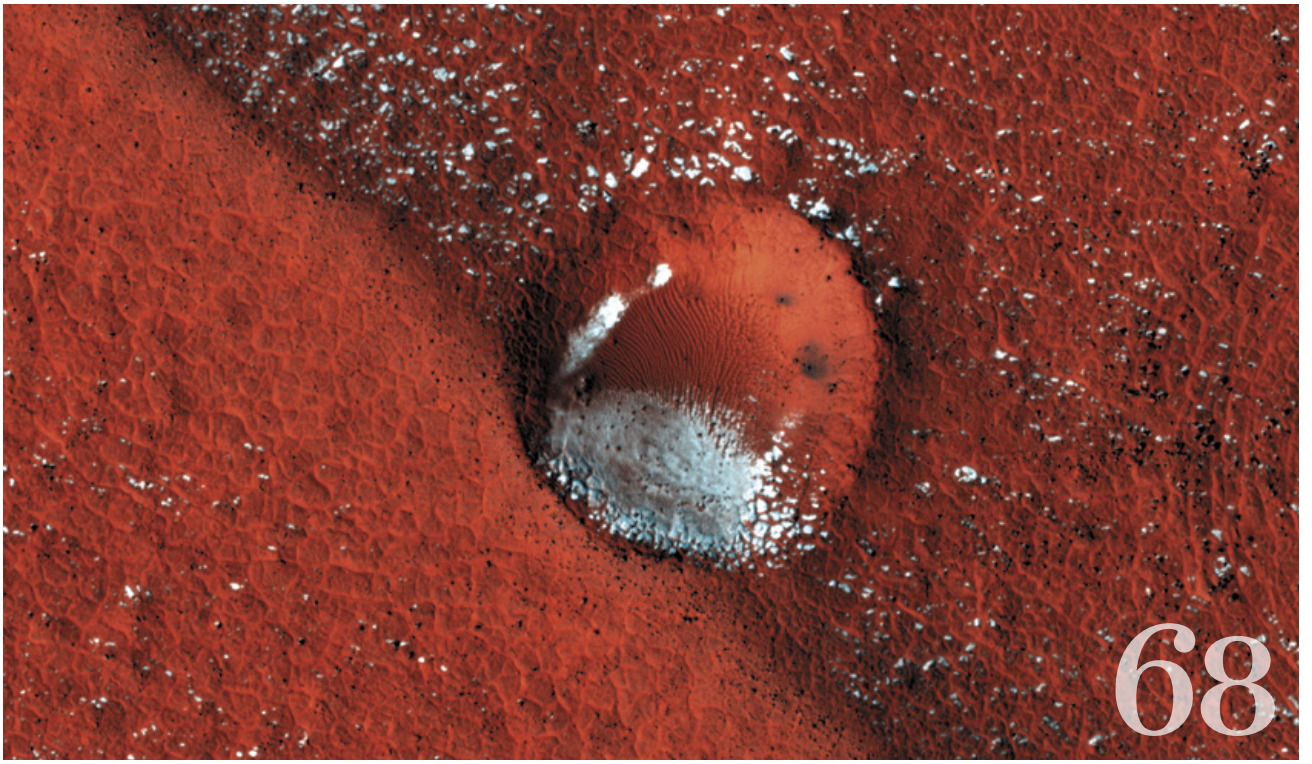
VOLUME 324, NUMBER 1

SCIENTIFIC AMERICAN



NASA, JPL AND UNIVERSITY OF ARIZONA

ON THE COVER

Jurassic Park made *Dilophosaurus* famous before scientists had a thorough understanding of this dinosaur. A new analysis of *Dilophosaurus* remains has provided the most detailed picture yet of a dinosaur of its vintage and revealed the creature as it truly was: a large-bodied, nimble predator that hunted other dinosaurs. **Illustration by Chase Stone.**

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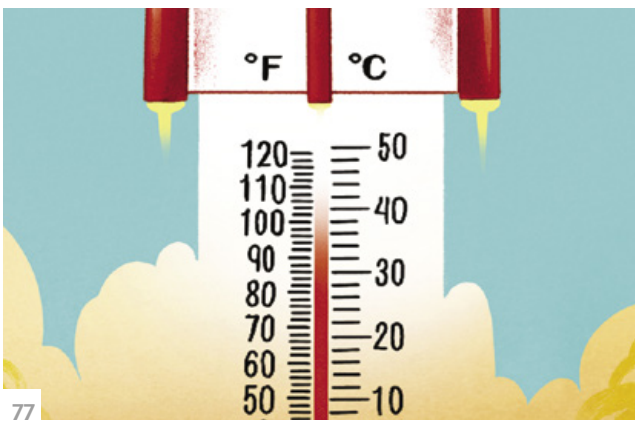

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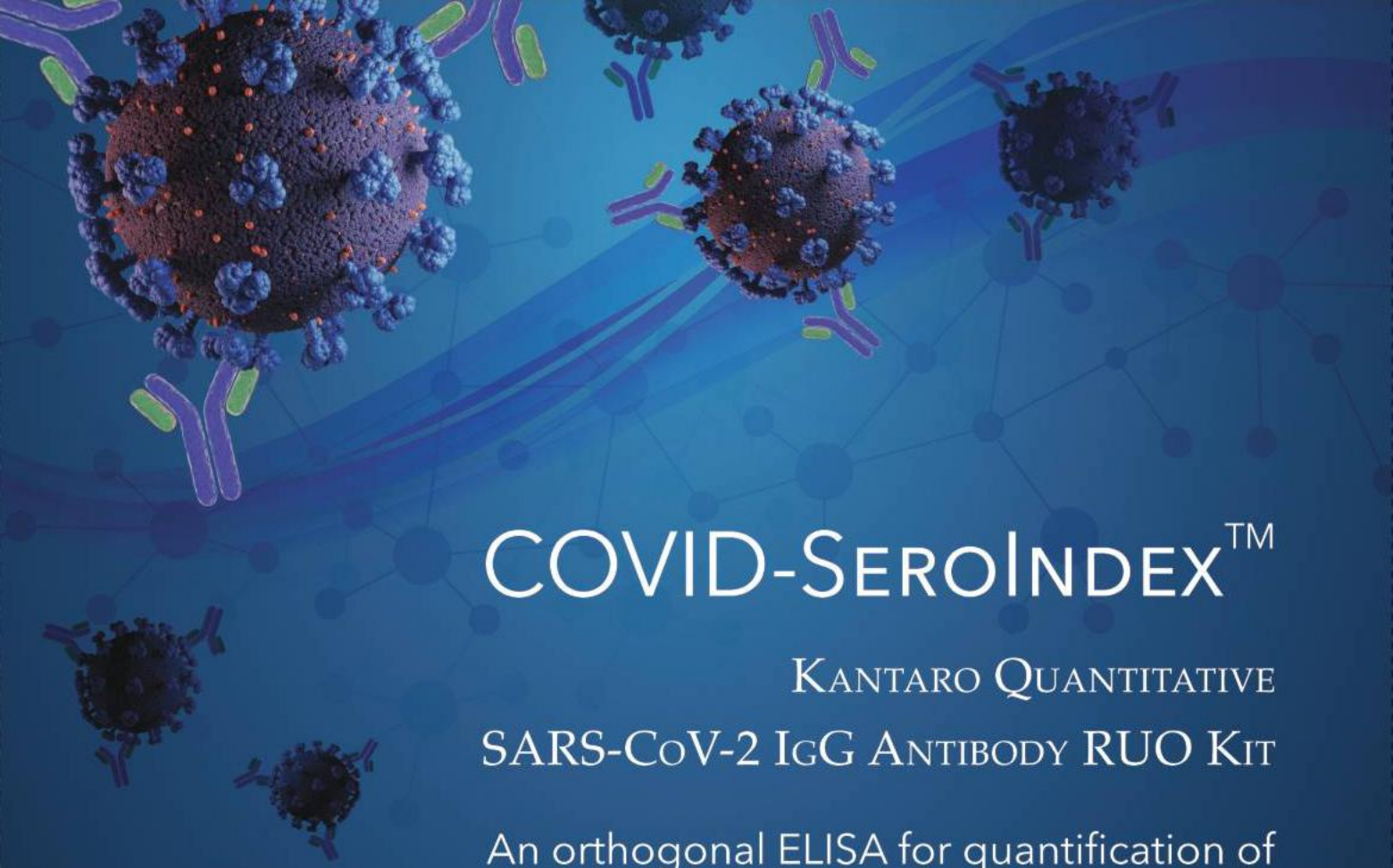
Scientific American (ISSN 0036-8733), Volume 324, Number 1, January 2021, published monthly by Scientific American, a division of Springer Nature America, Inc., 1 New York Plaza, Suite 4600, New York, N.Y. 10004-1562. Periodicals postage paid at New York, N.Y., and at additional mailing offices. Canada Post International Publications Mail (Canadian Distribution) Sales Agreement No. 40012504. Canadian BN No. 127387652RT; TVQ1218059275 TQ0001. Publication Mail Agreement #40012504. Return undeliverable mail to Scientific American, P.O. Box 819, Stn Main, Markham, ON L3P 8A2. **Individual Subscription rates:** 1 year $49.99 (USD), Canada $59.99 (USD), International $69.99 (USD). **Institutional Subscription rates:** Schools and Public Libraries: 1 year $84 (USD), Canada $89 (USD), International $96 (USD). Businesses and Colleges/Universities: 1 year $399 (USD), Canada $405 (USD), International $411 (USD). Postmaster: Send address changes to Scientific American, Box 3187, Harlan, Iowa 51537. **Reprints inquiries: RandP@sciam.com. To request single copies or back issues, call (800) 333-1199. Subscription inquiries: U.S. and Canada (800) 333-1199; other (515) 248-7684. Send e-mail to scacustserv@cdsfulfillment.com.**



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AD_ScientificAmericaJan2021_STRY0050466

Laura Helmuth is editor in chief of *Scientific American*.
Follow her on Twitter @laurahelmuth

How Science Works

The heroes of the COVID-19 pandemic are legion: nurses, doctors and others who care for the sick; epidemiologists and public health experts who track the disease and offer clear lifesaving guidance; and everyone who masks up and avoids crowds and protects their own health and the health of their communities. And around the world many scientists are working practically nonstop to understand the virus, how it spreads and what it does to the body.

We learned more about the immune system in 2020 than in any year in history. Akiko Iwasaki heads one of the labs leading the global effort to save people from COVID-19. Starting on page 34, she and grad student Patrick Wong explain how the immune system reacts to the new virus and how that knowledge might lead to new treatments. They describe how their team took on the urgent challenge and how the process of science changed in 2020 (*page 40*).

Understanding the process of science can protect people against misinformation—or at least we hope so. One of the outrageous myths about the pandemic is that the death toll is exaggerated. It's not. More than a quarter of a million people in the U.S. have died of COVID-19 as of November. Beginning on page 42, journalist Christie Aschwanden details how we know the disease has become the third leading cause of death in the U.S.

One of the most intriguing stages in the process of science is noticing when something is … weird. Astronomers using new tools to see parts of space that had been shrouded by dust have observed that galaxy clusters formed much faster than anyone expected and that they seem to be too big for our universe. Grad student Arianna S. Long recounts the excitement of rethinking the time line of the early universe and how any unusual discovery is first assumed to be a software bug before it is accepted. Turn to page 26.

We're in a great age of dinosaur discoveries. Starting on page 46, fossil experts Matthew A. Brown and Adam D. Marsh show how much has been learned about *Dilophosaurus,* our cover Dino Star, since it appeared in the film *Jurassic Park* in 1993. They point out that paleontology is more tedious and less glamorous than how it's depicted in movies, but understanding the bodies and habitats and behaviors of a 183-million-year-old dinosaur is the next best thing to bringing it back to life.

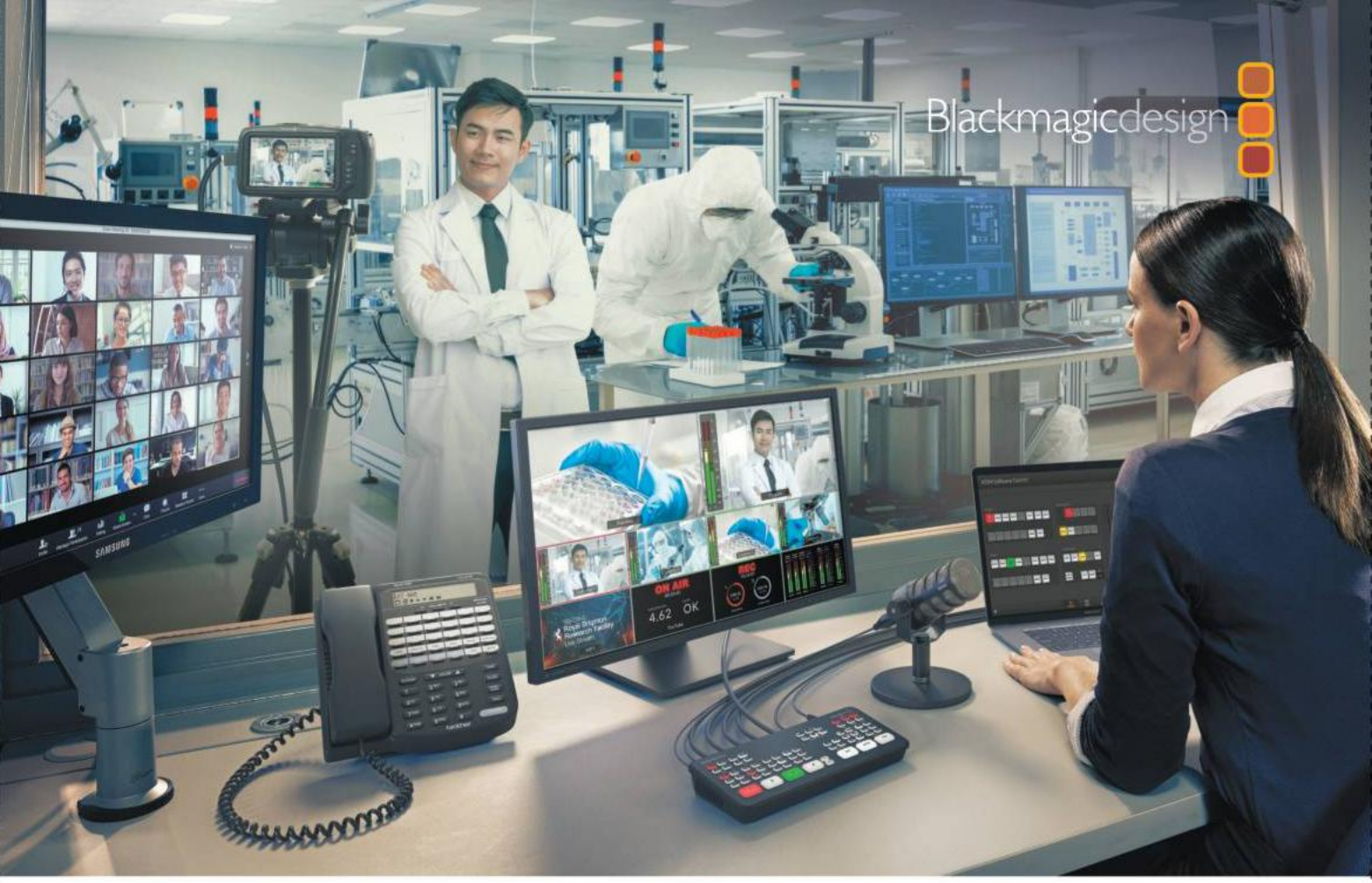
Some data are harder to gather than others. To understand the water cycle that sustains billions of people, mountain hydrologist Walter Immerzeel and his colleagues camp at 5,300 meters elevation (about 17,400 feet) and go higher to set up monitoring stations that have been twisted by winds and knocked over by avalanches. As he reports on page 54, climate change is disrupting ice melt, monsoons and river flows, and the consequences could be catastrophic.

Evolutionarily, we are all well-functioning cellular civilizations, according to psychologist and evolutionary biologist Athena Aktipis (*page 62*). Multicellularity has a lot of advantages, and it has led to exquisite cooperation. But when some cells cheat, they can threaten the entire organism. Thinking of cancer cells as cheaters has led to new approaches to treatment.

When things get tumultuous on our planet, it's a nice escape to look at another one. The Mars Reconnaissance Orbiter has been photographing the Red Planet for 15 years now, and on page 68 senior editor Clara Moskowitz shares some of the most gorgeous views. They reveal that Mars has dust devils, landslides and asteroid impacts just like Earth does.

All of us at *Scientific American* wish you a Happy New Year. We hope your 2021 is healthy and full of pleasant discoveries. And may science keep showing us more ways to save lives. SA

Illustration by Nick Higgins



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The compact television studio that lets you create training videos and live streams!

Blackmagic Design is a leader in video for the medical industry, and now you can create your own streaming videos with ATEM Mini. Simply connect up to 4 HDMI cameras, computers or even technical equipment. Then push the buttons on the panel to switch video sources just like a professional broadcaster! You can even add titles, picture in picture overlays and mix audio! Then live stream to Zoom, Skype or YouTube!

Create Training and Educational Videos

ATEM Mini's includes everything you need. All the buttons are positioned on the front panel so it's very easy to learn. There are 4 HDMI video inputs for connecting cameras and computers, plus a USB output that looks like a webcam so you can connect to Zoom or Skype. ATEM Software Control for Mac and PC is also included, which allows access to more advanced "broadcast" features!

Use Professional Video Effects

ATEM Mini is really a professional broadcast switcher used by television stations. This means it has professional effects such as a DVE for picture in picture effects commonly used for commentating over a computer slide show. There are titles for presenter names, wipe effects for transitioning between sources and a green screen keyer for replacing backgrounds with graphics!

Live Stream Training and Conferences

The ATEM Mini Pro model has a built in hardware streaming engine for live streaming via its ethernet connection. This means you can live stream to YouTube, Facebook and Twitch in much better quality and with perfectly smooth motion. You can even connect a hard disk or flash storage to the USB connection and record your stream for upload later!

Monitor all Video Inputs!

With so many cameras, computers and effects, things can get busy fast! The ATEM Mini Pro model features a "multiview" that lets you see all cameras, titles and program, plus streaming and recording status all on a single TV or monitor. There are even tally indicators to show when a camera is on air! Only ATEM Mini is a true professional television studio in a small compact design!

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*Price subject to change. ATEM Mini for use in training, conferencing and teaching purposes only.

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September 2020

"A great sadness has overtaken me as I parse the odds of life on this planet making it through. Let's keep our fingers crossed."

SUSAN WILLIAMS *LAKEWOOD, COLO.*

NATURAL HISTORY REPEATS

"The Worst Times on Earth," by Peter Brannen, describes past mass extinctions and what they could mean for our future. Brannen has written one of the most beautiful and poignant pieces I've ever read here, all the more so because a great sadness has overtaken me as I parse the odds of life on this planet making it through. Let's keep our fingers crossed.

SUSAN WILLIAMS *Lakewood, Colo.*

SCIENCE VS. ANTISCIENCE

In their article, "Reckoning with Our Mistakes," Jen Schwartz and Dan Schlenoff state, "Americans who are willing to sacrifice the lives of people who are disabled, poor, elderly or from historically oppressed groups so that the U.S. economy can 'go back to normal' sound like modern-day eugenicists."

I am supervisor of a day program for adults with intellectual and physical disabilities. After we were required to close our program in late March, I received continuous calls pleading for information about when we would reopen. These calls came from the individuals we serve, as well as their families. Our clients missed their friends and our structured program of vocational and social-skills classes, the volunteer jobs we facilitate for them around the community daily, our healthy lifestyle activities, and more.

Parents' constant concern was that our clients were losing the abilities we had helped them develop to integrate into the larger community, to pursue lives of meaning and purpose.

Of course, we created a daily schedule of Zoom classes, but not every client is able to participate or benefit from those. And without our structure, some of our clients engaged in behaviors at home that endangered them and sometimes their family members.

I beg the authors and your readers not to write off those whose opinions are different from yours as oppressors or worse. Schwartz and Schlenoff ask "how else to explain" some people's advocacy for "going back to normal." But there are other ways to explain it. Rather than assuming those advocates believe "some of us are inherently more worthy of life than others," put yourselves in the shoes of our clients and their families. They want the best for their loved ones, and that may mean masks and social distancing rather than lockdowns.

RENEE KAMEAH
Rockland County, New York State

It is laudable that *Scientific American* acknowledges, and endeavors not to repeat, its role in disseminating and legitimizing scientific racism, sexism and imperialism. Human fallibility aside, Schlenoff and Schwartz mention several sources of scientific error, but they do not mention the potential for systematic error deriving from scientific methodology itself.

Because we can only gauge the likely truth of new hypotheses by drawing on existing beliefs, insofar as histories of racism, sexism and imperialism shape our current corpus of scientific belief, these legacies will continue to distort scientific inquiry. Science is a social enterprise, and it is shaped not only by theories and data but also by personal experience, common sense and the social uses to which it is put. Research may gain currency not from the weight of evidence but because it serves the political and economic interests of those with the power to promulgate it (for example, by justifying economic and racial inequality). When that happens, it has an enduring, distorting effect on science. Once absorbed into received knowledge, such research misinforms subsequent scientific judgments.

Thus, to foster accuracy in the field, we must do more than weigh the existing evidence. We must evaluate how relevant evidence may have been shaped by science's social uses and actively investigate and correct resulting errors. That is, acting with integrity as scientists requires applying sociopolitical theories about how our political economy shapes scientific belief and organizing to overturn distorting forces.

Contrary to many scientists' demands for a "politics-free science," merely using sociopolitical theories to assess evidence is not "bias." The reverse is true: failing to consider how our political economy shapes scientific evidence heightens the risk of error.

JERZY EISENBERG-GUYOT AND NADJA EISENBERG-GUYOT *New York City*

Schwartz and Schlenoff note that only half of Americans responded to a poll that they would get a coronavirus vaccine when it is available, which they called an "anti-science" stance.

The authors should be very careful of the context of the poll and answers to it. I am not in any way an anti-vaxxer. My wife and I get flu shots annually and were diligent in keeping our children up-to-date on their inoculations when they were young. But if I were asked whether I would get the hypothetical coronavirus vaccine, I'm not sure how I would answer.

After watching the Food and Drug Administration's and Centers for Disease Control and Prevention's responses to the pandemic, I fear that the basis for far too many of their decisions concerns politics, not science. These government agencies

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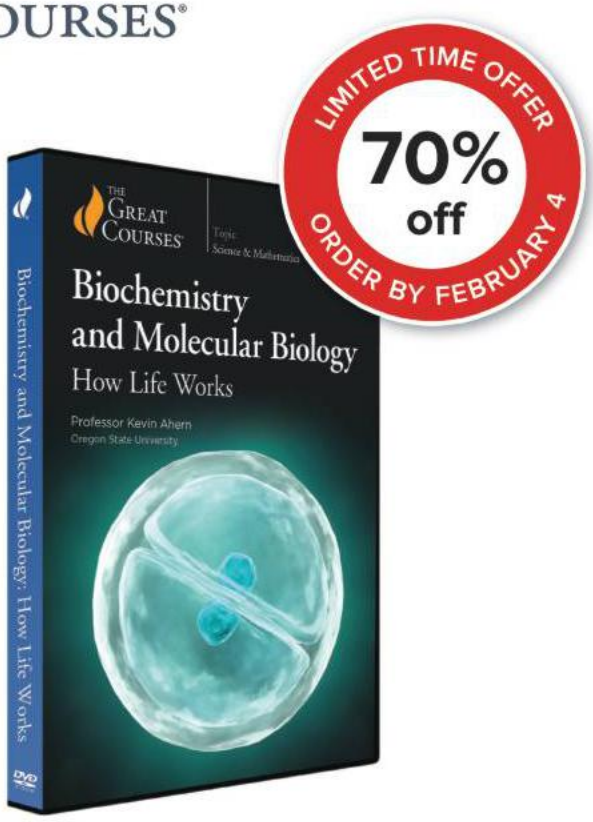

Understand the Science of Us

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.

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seemed to have become a wing of the committee to reelect President Donald Trump.

JOHN MELQUIST *Caledonia, Ill.*

THE AUTHORS REPLY: Melquist's point is well taken. In criticizing individuals' unwillingness to receive a potential vaccine against COVID-19 in our article, we indeed meant one that would be well tested, well studied, well prepared, and recommended on a sound scientific and statistical basis.

MITIGATING LETHAL FORCE

In "How to Reinvent Policing" [Science Agenda], the editors make a number of good points about bettering policing by improving police accountability and communities' perception of officers. They do not mention, however, that improvements can also be made to hiring practices.

Police departments should recruit candidates who have good problem-solving, negotiation, communication and interpersonal skills, as well as empathy and sensitivity. They can come close to achieving that goal by expanding their pool to include more women, minorities and college graduates. Doing so will create a workforce that understands that all people should be treated with dignity, respect and fairness.

VASILIOS VASILOUNIS *Brooklyn, N.Y.*

SCIENTIFIC PROGRESS

I have been an avid reader of *Scientific American* since my college and university days in the 1960s, and the magazine has, to me, largely represented a specified direction for American scientific and economic culture. It is unsurprising that there has been a significant change of emphasis during the term of the most recent presidential incumbent. Like many overseas readers, I find this change welcome.

A particular, though not singular, example of it is a phrase found in "Return of the Germs," Maryn McKenna's useful and thought-consuming piece on the need for social interventions to fight diseases in the light of the current COVID-19 pandemic. On this subject, the article quotes physician and vaccine developer Peter J. Hotez as saying, "Poverty has more impact than any of our technical interventions."

ALAN LAFFERTY *London*

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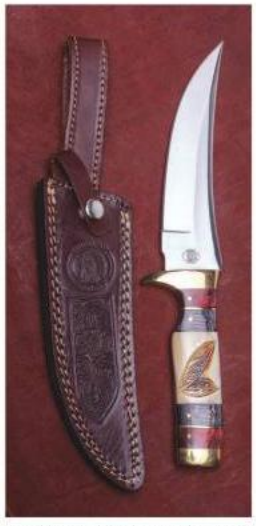

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By the Editors

If the *New York Times* or the *Las Vegas Review-Journal* or *Scientific American* publishes a false statement that hurts someone's reputation, that person can sue the publication. If such defamation appears on Facebook or Twitter, however, they can't. The reason: Section 230 of the federal Communications Decency Act. Signed into law in 1996, it states that online platforms—a category that includes enormously rich and powerful tech companies such as Facebook and Google, as well as smaller and less influential blog networks, forums and social media start-ups—are not considered "publishers." You can sue the person who created the video or post or tweet but not the company that hosted it.

The law was designed to protect Internet companies, many in their infancy at the time, from legal actions that could have held back their ability to innovate and grow. But today immunity from consequences has also allowed hate speech, harassment and misinformation to flourish. In a belated effort to deal with those problems, the biggest platforms now attempt to flag or ban what they feel are objectionable content generated by users. This often infuriates those whose posts or tweets have been singled out, who complain their freedom of speech is being suppressed.

Last October the U.S. Senate Committee on Commerce, Science and Transportation held a hearing about how to modify the law. But high-handed changes often don't consider all consequences—and that can lead to real danger. Prime examples are 2018's Fight Online Sex Trafficking and Stop Enabling Sex Traffickers Acts (FOSTA-SESTA). These laws removed Section 230's protections for content that advertises prostitution, in an effort to stop victims of sex trafficking from being bought and sold online. The idea was to use potential legal liability to force platforms to remove content that encouraged such crimes. In practice, sites that lacked the resources to patrol users' activity ended up banning legitimate pages where illegal content had appeared in the past, deleting large swaths of material or shutting down entirely. The purge kicked consensual sex workers out of online spaces they had used to find clients and assess any risk of harm before agreeing to in-person meetings. Without the ability to screen clients online, prostitution can be extremely dangerous; one 2017 paper, updated in 2019, suggests that in cities where the online classified ad service Craigslist allowed erotic listings, the *overall* female homicide rate dropped by 10 to 17 percent. Although other researchers have contested the link between online advertising and greater safety, consensual sex workers have reported negative effects as a result of FOSTA-SESTA.

Joe Biden and Donald Trump have both called for outright repeal of Section 230. Others in Congress are proposing less radical changes, offering bills such as the Platform Accountability and Consumer Transparency (PACT) Act, which would require social media companies to disclose their moderation practices to show they are not arbitrary and to promptly take down content that a court deems illegal. The stricter takedown standard would favor wealthy companies such as Facebook, which can afford to employ armies of moderators and lawyers, and disfavor start-ups—just the problem Section 230 was meant to prevent. In addition, as they did in response to the laws intended to curtail sex trafficking, smaller platforms are likely to increase overly broad censorship of users to avoid legal challenges.

As digital-rights group the Electronic Frontier Foundation (EFF) points out, hobbling Section 230 could have a chilling effect on free speech online and make it much more difficult for new competitors to challenge the dominance of big tech. The EFF is not the only voice picking holes in legislation like the PACT Act: academics and other technology advocacy groups have offered measured critiques of the bill and proposed their own solutions for strategically modifying Section 230. One of their suggestions is to ensure the bill would apply only to platforms that host users directly—not to the companies providing background support for functions such as Internet access and payment processing—to protect the larger infrastructure of the Internet from legal liability. Another idea is to improve users' ability to flag problematic content by working with legal authorities to develop a standardized reporting process that any platform could apply.

Hobbling [the law] could have a chilling effect on free speech and make it harder for competitors to challenge big tech.

Input from experts like these—not just from billionaire CEOs such as Facebook's Mark Zuckerberg and Twitter's Jack Dorsey, the usual suspects when hearings are convened on Capitol Hill—is crucial to craft nuanced legislation that will give online platforms incentives to protect users from harassment and to suppress malicious content without unduly compromising free speech. If that happens, we might get Internet regulation right. SA

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Jordan Salama is a writer and journalist. His first book, *Every Day the River Changes*, a book of travels along the greatest river in Colombia, will be published by Catapult in 2021.

Earth Is on Fire

COVID is no excuse to ignore environmental flames

By Jordan Salama

I've never known an Earth that wasn't on fire.

I'm 23 years old, and my entire generation has come of age in a world so defined by climate change and other forms of environmental degradation that it's sometimes been hard to fathom what an even more dismal future might look like. It has, that is, until the pandemic reared its ugly head. The fate of nature, like so much else, has temporarily become an agonizing side story to COVID—and now the environment is a real-time plot followed mostly, I think, by those of us young enough to one day see the worst of it.

At first, things seemed hopeful. Struggling to adjust to the new normal of life in quarantine in March and April, we were relieved to read that emissions levels had dipped, even if only temporarily, and that the skies over New Delhi and Los Angeles and Buenos Aires had cleared of smog. I smiled, as we all did, to notice that animals were roaming free through quiet, traffic-free cities. Nature seemed to be reclaiming spaces humans had abandoned. In the midst of so much present grief, these story lines gave us faith in the planet's resilience. Maybe, some optimists speculated, it would even inspire us to be better stewards of our world when this was over. This "anthropause" was a once-in-a-lifetime opportunity for humans to understand our impact on wildlife in a crowded world that seemed, for a moment, a little less crowded.

But only for a moment. Pandemics like this happen and will keep happening because we humans have long encroached on wild spaces, increasing the chances of spillovers of disease from animals to people. In the temporary absence of international watchdogs and local enforcement, South America's Pantanal, the world's largest tropical wetland, has burned like never before. In May there was a major oil spill in the Russian Arctic, followed by others in places such as Mauritius and Venezuela—terrible ecological catastrophes that are buried underneath headlines of case numbers and mortality rates. Poaching is on the rise in Africa. The list goes on.

And in the U.S., we've somehow become *less* thoughtful in our daily choices—accepting that extra plastic bag at the supermarket, ordering takeout despite all the single-use containers and, if we're privileged enough, driving instead of taking public transportation—because, well, "it's a global pandemic." Take a walk outside, and you'll find masks and latex gloves littering our streets and beaches and parks that will eventually fill rivers, lakes and seas.

It's as if the pandemic has suddenly given people everywhere even more of a license to dirty the world—if that's even possible—with carelessness, if not outright contempt. I fear that for every day it continues, today's young people will be paying the ecological price for the rest of our lives. I'm not just talking about those of us living in developed nations. I'm talking about children from impoverished families worldwide whose health and food security have plunged into even more uncertainty because of the devastating one-two punch of climate change and the coronavirus—both of which have laid bare systemic racism and socioeconomic inequities. I'm talking about young climate organizers across the globe who have been calling out people's ignorance of science for years and feel now more than ever that they're screaming into a void. And, perhaps most brutally, I'm talking about young Indigenous people in Latin America, whose entire cultures (many of them predicated on harmony with nature) are being erased as their elders die of infection and as ranchers and miners violently and illegally drive them from their ancestral lands.

In today's pandemic moment, nature's story line has reached a low point. It's unfathomable to me that some people can still so easily shrug it off—especially if they have kids or love anyone who is younger than they are—while for so many in my generation, it is such a constant, excruciating worry. Apathy, let alone denial, is no longer an acceptable option, because we know that if we stay on this course, the destruction will inevitably come for us, too.

But I like to think that the anthropause still gives some hope—that perhaps if we all live a little lighter, if we listen to those who are in harmony with the land and if we take solace in all that there is to love in the world, nature might meet us halfway. The planet and our fates hang in the balance. SA

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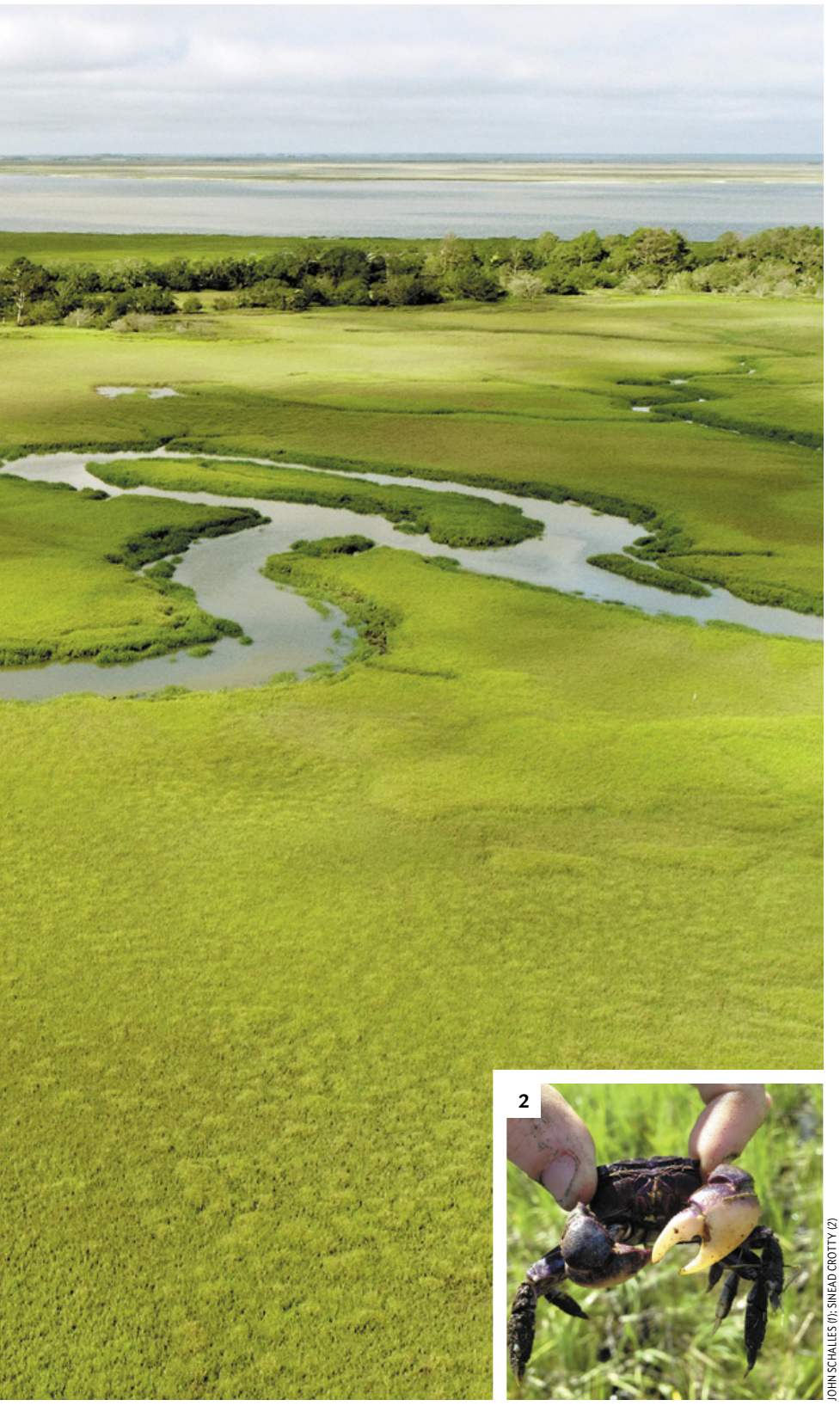
Illustration by Brooke VanDevelder

ADVANCES

Tidal creeks cross Sapelo Island's salt marshes (*1*) and are expanding because of purple marsh crabs (*2*).



2

JOHN SCHALLES (1); SINEAD CROTTY (2)

INSIDE

ECOLOGY

Losing Ground

Sea-level rise is letting a tiny crab drastically alter marsh landscapes

Halfway down Georgia's coastline, Sapelo Island is surrounded by more than 4,000 acres of salt marshes, with vast stretches of lush grasses that blaze gold in the colder months. But this beautiful barrier island is experiencing some of the harshest effects of climate change: seawater intrusion, intense storms and flooding.

And scientists have noticed something more subtle and unusual happening to the island in the past several years. A once inconspicuous burrowing crab is suddenly wiping out swaths of marsh cordgrass, a plant that holds much of the South's coastal marshland in place and protects vulnerable species. The tiny purple marsh crab, *Sesarma reticulatum,* seems to be reshaping—and fragmenting—the island's marshes.

Sinead Crotty, an ecologist and project director at Yale University's carbon-containment laboratory, used aerial images to document the crab's impact on marshland along the U.S.'s southeastern coast. To investigate the cause of the changes, Crotty and her colleagues combined analysis of the aerial imagery with historical tide data and numerical models of sea-level rise.

Their results, published in the *Proceedings of the National Academy of Sciences USA,* show the crabs are altering salt marshes' response to sea-level rise by gorging on cordgrass at the heads of tidal creeks. The researchers say rising water levels caused by climate change have softened marsh soil, creating optimal burrowing conditions for the crabs. The crabs' increased activity then results in longer and broader creeks that drain the marshes into the ocean. Over years

this process transforms marshes from contiguous grasslands into patches fractured by crab-grazed creeks.

This finding challenges the long-standing paradigm that only water flow, sediment, plants and human activity—not animals—shape how salt marshes respond to sea-level rise. The researchers say this crab may be the first identified organism to reach the status of a keystone species, an organism that has disproportionate importance and influence in its ecosystem, because of climate change. It is unlikely to be the last.

Crotty says it is mind-boggling that "this very small organism, an inch or two in diameter, can alter something as large as an entire marsh landscape visible from Google Earth images."

Scientists working on Georgia's coast already knew *Sesarma* crabs were enlarging tidal creeks by grazing cordgrass, says Meryl Alber, director of the University of Georgia Marine Institute on Sapelo Island. But this new work suggests the crabs' actions may be accelerating the long-term loss of the marsh to rising seas. "This shows that our marshes may be more vulnerable than we thought," she says. Alber was not directly involved in the study, but the institute provided logistical support to the research team.

Crotty first encountered *Sesarma* as an undergraduate in co-author Mark Bertness's Brown University lab. In 2011 Bertness's team discovered that the crabs were behind sudden marsh die-offs on Cape Cod, after overfishing had diminished predator populations such as striped bass. Marsh soils farther south had previously been too hard for the crabs to gain a significant claw-hold, and Crotty and her colleagues wondered if sea-level rise could be making them softer.

The team analyzed tidal data and found that southern marshes are now submerged up to an hour longer a day than they were in the 1990s. The researchers say this process has indeed softened the soil, helping the burrowing crabs thrive. Aerial photographs along the U.S.'s southeastern coast indicate the number of *Sesarma*-grazed marsh creeks increased by an average of two and a half times from the 1990s to late 2010s. In study areas, the team found that the rapid expansion of crab-grazed creeks increased drainage of the marsh by up to 35 percent.

By wiping out cordgrass, crabs also destroy protective cover for ecologically critical animals, including snails and other mollusks. The researchers checked predation levels on Sapelo Island by tethering snails and mussels to fishing line near grazed and ungrazed creeks. They found this loss of cover can make small invertebrates—which provide food to commercially important species such as blue crab and redfish—more vulnerable to predator feeding frenzies, Crotty says, potentially disrupting entire ecosystems.

Human activities are resetting which species hold the most sway over ecosystem behavior, says Christine Angelini, an ecologist at the University of Florida and principal investigator for the study. Because of overfishing and climate change, she observes, purple marsh crabs are "wreaking havoc everywhere" across their range.

Climate change has given several species a dangerous advantage. Ocean warming and acidification make it easier for predators such as sea urchins to gnaw away at coral. Native plants are losing ground to exotic varieties that can bloom earlier as weather warms. Higher temperatures in the Caribbean could help invasive, reef-destroying lionfish expand their range there. But scientists have not previously documented such organisms exerting the kind of influence purple marsh crabs do over the way an ecosystem functions, from its actual shape to the interplay between predators and prey.

"I have no reason to doubt that climate change will alter species' interactions such that new keystone species emerge," says Linda Blum, an ecologist at the University of Virginia, who was not involved in the study. But, she adds, the team's conclusion that sea-level rise creates new crab habitat by softening marsh soil is built on "a lot of circumstantial evidence." She suggests it should be tested with field experiments to determine whether the crabs' own activities could contribute to easier burrowing.

Now the researchers are investigating how increased activity from Sapelo Island's *Sesarma* crabs might be exposing buried carbon to the air, as well as if the crabs are raising concentrations of contaminants from a nearby superfund site by accumulating these chemicals in their bodies.

More work is also needed to understand whether the crabs are influencing how quickly seas move inland, Angelini says: "We don't know if it's the first step toward, ultimately, the marsh drowning or if marshes will stay stable and persist for decades in this fractured state." —*Stephenie Livingston*

PSYCHOLOGY

Applied Ethics

One college class discussion had weeks-long effects on meat consumption

Although ethics classes are common around the world, scientists are unsure if their lessons can actually change behavior; evidence either way is weak, relying on contrived laboratory tests or sometimes unreliable self-reports. But a new study published in *Cognition* found that, in at least one real-world situation, a single ethics lesson may have had lasting effects.

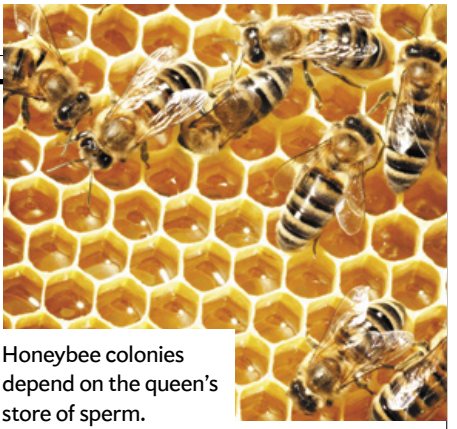
The researchers investigated one class session's impact on eating meat. They chose this particular behavior for three reasons, according to study co-author Eric Schwitzgebel, a philosopher at the University of California, Riverside: students' attitudes on the topic are variable and unstable, behavior is easily measurable, and ethics literature largely agrees that eating less meat is good because it reduces environmental harm and animal suffering. Half of the students in four large philosophy classes read an article on the ethics of factory-farmed meat, optionally watched an 11-minute video on the topic and joined a 50-minute discussion. The other half focused on charitable giving instead. Then, unbeknownst to the students, the researchers studied their anonymized meal-card purchases for that semester—nearly 14,000 receipts for almost 500 students. "It's an awesome data set," says Nina Strohminger, a psychologist who teaches business ethics at the University of Pennsylvania and was not involved in the study.

Schwitzgebel predicted the intervention would have no effect; he had previously found that ethics professors do not differ from other professors on a range of behaviors, including voting rates, blood

BIOLOGY

Clues to Collapse

Analyzing fluid from queen bees' specialized sperm sacs can expose colony stressors



Honeybee colonies depend on the queen's store of sperm.

GETTY IMAGES

A honeybee queen mates only during one brief period of her life, storing sperm in a sac inside her body for later use. But if she fails to keep that sperm viable, her colony may collapse. This "queen failure" is a main factor in the U.S. drop-off in bee numbers. Identifying reasons for queen failure has proved difficult; queens show no clear symptoms when it happens. But a new study offers a way to zero in on causes, which could lead to a valuable diagnostic tool for beekeepers.

The queen is the only female in her colony who can reproduce. Without viable sperm she cannot lay eggs, and the colony's population plummets, says Alison McAfee, first author of the study and a bee researcher at North Carolina State University. This is a significant concern for humans; as pollinators of crops such as blueberries and apples, "honeybees are responsible for around between $16 billion and $20 billion worth of economic contribution to agriculture," says McAfee, who also works for the University of British Columbia. Climate change further threatens honeybees' survival, with previous research showing that high temperatures are also associated with colony loss.

To examine queen failure, McAfee and her colleagues performed a "molecular autopsy" in which they analyzed the fluid inside sperm-storing sacs after exposing queens to extreme heat, extreme cold or pesticides. They found that each stressor was associated with elevated levels of different proteins in the fluid.

The researchers identified the two most elevated proteins as indicators for each stressor. When they looked for these in failed queens donated by British Columbia beekeepers, they found proteins indicating exposure to pesticides and extreme heat but not extreme cold. The results were published in *BMC Genomics*.

McAfee and her colleagues are using these results to develop a diagnostic test that distinguishes between different causes of queen failure. This tool is in its early stages, but Susan Cobey, a bee researcher at Washington State University, who was not involved in the study and runs a bee-insemination business, is excited by its potential: "If you can determine what's going on with [the queens] and take some preventive control … and avoid losses in the field, that would be awesome." —*Karen Kwon*

donation and returning library books. But among student subjects who discussed meat ethics, meal purchases containing meat decreased from 52 to 45 percent—and this effect held steady for the study's duration of several weeks. Purchases from the other group remained at 52 percent.

"That's actually a pretty large effect for a pretty small intervention," Schwitzgebel says. Strohminger agrees: "The thing that still blows my mind is that the only thing that's different between these two cases is just that one day in class." She says she wants the effect to be real but cannot rule out some unknown confounding variable. And if real, Strohminger notes, it might be reversible by another nudge: "Easy come, easy go."

Schwitzgebel suspects the greatest impact came from social influence—classmates or teaching assistants leading the discussions may have shared their own vegetarianism, showing it as achievable or more common. Second, the video may have had an emotional impact. Least rousing, he thinks, was rational argument, although his co-authors (University of Kansas's Bradford Cokelet and Princeton University's Peter Singer) say reason might play a bigger role. Now the researchers are probing the specific effects of teaching style, T.A.s' eating habits and students' video exposure. Meanwhile Schwitzgebel—who had predicted no effect—will be eating his words. —*Matthew Hutson*

GENETICS

Getting Dirt

Ancient DNA preserved in soil may rewrite Ice Age knowledge

Based on bone and tooth records, the Yukon's last mammoths were thought to have gone extinct about 12,000 years ago. But a new genetic sampling technique suggests the great beasts may have stuck around a lot longer, plodding through the Arctic tundra with bison and elk for thousands of years more. The story is in the soil.

Bones are rich sources of prehistoric genetic information, but not the only ones; items ranging from shed Ice Age skin cells to pine needles can contribute to the genetic record stored in dirt. Paleogeneticists have been extracting and analyzing "environmental DNA" from soil for a long time, but getting rid of non-DNA material without destroying these fragile clues is daunting.

"Environmental samples contain a huge range of other chemical substances that are challenging to separate from the DNA we're interested in," says McMaster University geneticist Tyler Murchie. "We can't afford to lose whatever we can get." In *Quaternary Reports*, Murchie and his colleagues describe gentler techniques that recover up to 59 times as much genetic material as other methods do.

In the new approach, soil samples are extracted with a sterilized chisel and then broken into smaller portions, stirred and run through a "cold spin method" to separate as much DNA as possible. The DNA is then compared against an existing genetic library to detect species matches.

"Not only do these techniques get more DNA, but they get more diverse DNA," says East Tennessee State University paleontologist Chris Widga, who was not involved in the new study. "It's becoming more nuanced, and it looks like there is actually the potential to document larger slices of the ecosystem."

This big picture comes from smaller samples, Murchie explains: "With a combination of our novel extraction and enrichment techniques, we can pull out entire genomes of multiple extinct organisms simultaneously from less than a gram of sediment."

The methodology is limited because researchers using it need to know what DNA to look for. If a saber-toothed cat species is not already in the genetic library, for example, the analysis cannot detect that animal. For known species, however, the process may yield exciting information. In their study, the researchers detected about 2,100 kinds of plants and 180 animals—including American horses and woolly mammoths, in samples from soil dated to thousands of years after their supposed extinction.

Not yet published results from other field sites are yielding similar results, Murchie says, and future fossil discoveries could strengthen the case. "We can use this approach to identify species in places and times we never knew they existed," he adds, "helping our efforts to find their fossils in places we wouldn't have thought to look." —*Riley Black*

ALAMY

TECH

Sequoia Secrets

Lasers illuminate carbon-storing capacity of world's tallest trees

California's coastal redwoods, *Sequoia sempervirens,* are the tallest trees on earth. But measuring their precise dimensions—which is key to determining how much climate-altering carbon they store as biomass—is fraught with uncertainty. Widely used scaling equations between trunk diameter and volume are based on limited sampling of much smaller trees, given that few people are willing to don a climbing harness and take measurements 30 stories off the ground. Now, for the first time, researchers have surveyed *S. sempervirens* using laser scanning, an automated technique that yields accurate measurements of a tree's structure and volume.

Mathias Disney, an environmental scientist at University College London, and his colleagues assessed 145 redwoods for a study published in *Scientific Reports*. They fired billions of near-infrared laser pulses at the trees from multiple directions, recording the time it took the pulses to bounce back. This process let them assemble a detailed map of each tree, showing burrs, twigs and other features as small as a few centimeters. "This is giving us a new perspective on the three-dimensional structure of trees," Disney says.

The researchers found that coastal redwoods tended to be about 30 percent larger by volume than published scaling relations predicted. The authors suggest this discrepancy might be because some *S. sempervirens* sprout additional trunks as they age, a process called reiteration. Based on their observations, Disney and his colleagues have established new scaling relations between tree diameter and volume for the species.

Laser scanning can increase knowledge of old-growth forests that is important in conservation efforts, says Anil Raj Kizha, a forest operations scientist at the University of Maine, who was not involved in the research. "In the coming five to 10 years," he says, "this is going to be more widespread." —*Katherine Kornei*

FROM "NEW 3D MEASUREMENTS OF LARGE REDWOOD TREES FOR BIOMASS AND STRUCTURE," BY MATHIAS DISNEY ET AL., IN *SCIENTIFIC REPORTS*, VOL. 10; OCTOBER 15, 2020

Mount Tarawera's explosive 1886 eruption may have been spurred by "nanolite" crystals.

ROD HILL Getty Images

VOLCANOLOGY

Crystal Power

Tiny crystals play a big role in unexpectedly violent eruptions

Volcanoes that are thought to be mild-mannered, releasing steady lava flows, can sometimes erupt explosively without warning—as New Zealand's Mount Tarawera did in June 1886, causing widespread damage and death. Geologists have long wondered why volcanoes make this sudden and dangerous transition, says earth scientist Danilo Di Genova of the University of Bayreuth in Germany.

Di Genova and his colleagues propose in *Science Advances* that such a catastrophic switch may begin with crystal grains called nanolites, which can form in rising magma and are only about 1/100th the size of an average bacterium. The researchers say these grains make magma more viscous, preventing volcanic gases from escaping the molten rock. This builds up pressure, setting the stage for a violent explosion.

Using electron microscopy and spectroscopic imaging tools, the scientists found nanolites in the ashes of active volcanoes, including Mount Etna in Italy and Tambora in Indonesia.

The team then examined how nanolites form in a comparatively runny type of magma that becomes basalt when it cools. Such low-viscosity magma usually allows gases to escape easily, leading to smooth lava flows. The researchers produced nanolites in the laboratory by melting basalt and then cooling it rapidly. The cooling process is critical: during eruptions, magma loses heat as it rises toward the top of a vent. The study found nanolites will form only if the heat loss rate is just right, Di Genova explains.

"Magma is a multicomponent system, mainly made by silicon and oxygen," Di Genova says. "It has other elements such as aluminum, calcium and iron, the last of which seems to be the most important element in forming nanolites." Most of the nanolites are iron oxide crystals with traces of aluminum, he adds. And because iron is found in all magmas, such crystals can form in various magma types.

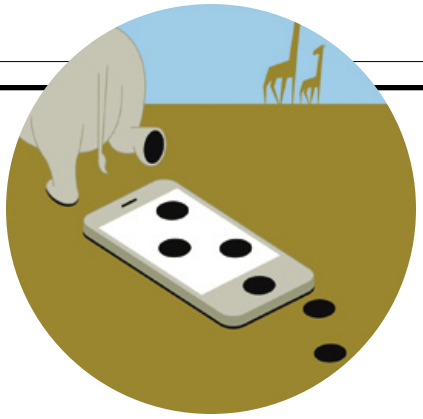
Next the researchers created an artificial magma to show that nanolites boost viscosity. They used silicon oil (which is as viscous at room temperature as basalt magma is during an eruption), adding glass spheres to mimic nanolites in shape and size. The team found that even at relatively low concentrations nanoparticles tend to clump together, disrupting the free flow of the liquid. In a real volcano, this sudden increase in the magma's viscosity would trap bubbles of escaping gas. Eventually enough pressure would build to push out blobs of magma all at once rather than in a steady stream—resulting in an explosion.

"This is an exciting study that addresses a question we have had for a long time," says Columbia University geologist Einat Lev, who was not involved in the new research. "It will be important and challenging to figure out how to incorporate this information in future volcanic models." —*Harini Barath*

CONSERVATION

Footsteps of Giants

Indigenous trackers inspire a safer way to help rhinos



International demand for black rhinoceros horn has seen the animals killed relentlessly for decades in countries such as Namibia, Zimbabwe and South Africa. In 1960 there were an estimated 100,000 left, and by 1995 fewer than 2,500 remained. Conservation efforts have brought the number up to around 5,600 today—but the species is still critically endangered, and poaching is among its biggest threats.

Scientists have worked to protect these rare creatures by tracking them with GPS devices strapped to their necks or ankles or implanted in their horns. The resulting data let researchers monitor the rhinos' numbers and when they enter poaching hotspots. But the devices can fail, and drugging the animals to attach them may cause harm.

A recent study in *PeerJ* describes a new tracking technology that uses smartphones to record rhino footprints. Called the footprint-identification technique (FIT), this system includes software that can analyze the animals' movements from a distance to help keep them safe from poachers.

The idea came from working with local trackers in Zimbabwe. These footprint-reading experts can identify individual black rhinos from the shape of their feet and, when visible, the impressions left by cracks in the animals' heel pads, which are as distinctive as a human fingerprint. "We probably wouldn't even have looked at the footprints in the first place if we didn't have Indigenous trackers," says Sky Alibhai, co-founder of conservation organization WildTrack and study co-lead.

To use the system, scientists gather rhino footprint images with a smartphone application and upload the pictures to a global database. FIT software analysis can then identify the individual animal and determine its age and sex with up to 99 percent accuracy. Researchers can also estimate the number of black rhinos in an area and watch their movements.

Alibhai and Zoe Jewell, WildTrack co-founder and study co-lead, are training wildlife conservationists, land managers, local guides and antipoaching agents to use FIT in Namibia, home to about 28 percent of the world's black rhinos. Since the paper's publication, researchers have adapted the technology for diverse animals, including African lions, Bengal tigers and Eurasian otters.

"I think this is great for monitoring animals and counting individuals," says Ciska Scheijen, a conservation scientist at Rockwood Conservation in South Africa, who was not part of the study. Scheijen wonders about FIT's performance during the rainy season, when footprints are often indistinct, and if it can track larger herds as opposed to the 35-odd individuals this study monitored.

Endangered animals often live in small groups or are sparsely distributed, Jewell says, but "FIT is scalable, and we would always welcome the opportunity to use it with endangered species that are in larger populations." —*Helen Santoro*

IN THE NEWS

Quick Hits

By Sarah Lewin Frasier

LATVIA
DNA harvested from a 700-year-old public toilet in Riga (as well as a 600-year-old cesspit in Jerusalem) will help researchers examine how human microbiomes have evolved over time. Microbial DNA from both sites matches some species common in modern hunter-gatherers and some in today's city dwellers.

MADAGASCAR
In a Madagascar garden, researchers found several Voeltzkow's chameleons—a rare species whose females can change from green to a vivid black, white and blue when excited. The short-lived species had not been documented for more than 100 years, and no females were previously recorded at all.

COSTA RICA
Researchers embedded GPS devices in decoy sea turtle eggs to track poaching patterns. In their first field test, five of the 101 decoys (which had similar size, weight and texture to real eggs) traveled significantly, potentially reaching consumers.

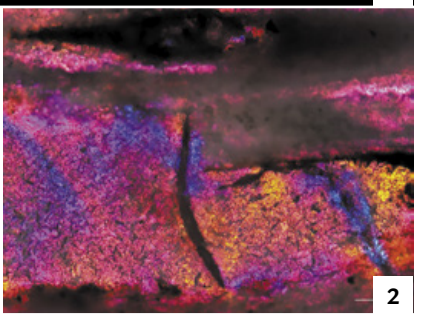
INDONESIA
New research shows that fluffy but venomous slow lorises frequently bite one another to settle territorial disputes—a rarity in venomous animals.

AUSTRALIA
An enormous, newfound coral reef off the continent's northern coast is taller than the Empire State Building, rising more than 500 meters above the seafloor. Considered part of the Great Barrier Reef, it is the first detached reef structure discovered there in 120 years.

ANTARCTICA
New analysis suggests a 50-million-year-old foot bone found on Seymour Island comes from a species of bird whose wingspan reached 6.4 meters across. The researchers also attributed part of a large jawbone with toothlike structures to the species.

For more details, visit www.ScientificAmerican.com/jan2021/advances

Illustration by Thomas Fuchs



A titanosaur's nasty bone condition (*1*) may have come from wormlike invaders (*2*).

PALEONTOLOGY

Blood Worms

A titanosaur fibula hosts what look like dozens of tiny parasites

Around 80 million years ago in what is now Brazil, a sick dinosaur limped along—but its days were numbered. Its leg bone was so diseased that it had turned spongy, and a particularly gruesome culprit may have been to blame: wormlike parasites wriggling through its blood vessels. The dinosaur in question was a long-necked titanosaur.

Aline Ghilardi of Brazil's Federal University of Rio Grande do Norte and her colleagues examined the bone's surface and ran a CT scan to rule out cancer and tuberculosis as the titanosaur's ailment. They eventually concluded it had a rare bone condition called osteomyelitis, which leads to severe inflammation. When they examined thin slices of the fossil under a powerful microscope, they found a startling potential cause: scattered through the bone's blood vessel cavities were fossilized remains of what appeared to be around 70 tiny worms, each roughly the length of a dust mite.

"We were totally shocked," Ghilardi says. "We can't compare it to anything." To her knowledge, such organisms have never before been found inside dinosaur bone fossils. In *Cretaceous Research*, she and her team suggest these "worms" could be ancient parasites whose arrival caused the infection. The researchers note, however, that osteomyelitis can also be caused by bacteria, fungi and single-celled organisms called protozoa.

The wormlike structures look similar to a known prehistoric parasite called *Paleoleishmania*, but they are between 10 and 100 times bigger. Forthcoming research compares them with a broad range of parasites.

There could be other explanations, however. "I'm not entirely convinced that those things are parasites or, if they are parasites, that they have anything to do with the bone diseases," says Tommy Leung, an ecologist who studies parasite evolution at the University of New England in Australia. Leung was not involved with the new finding, although he was a reviewer on a previous version of the paper.

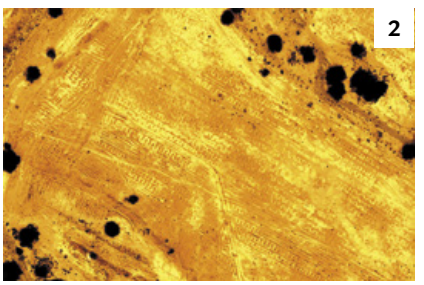
If the wormlike forms were indeed living organisms, they could have entered the bone to feed on the dinosaur after it died. But the researchers argue this is unlikely because there were no obvious fractures through which such creatures might have invaded.

Parasites afflict birds and reptiles today, so it makes sense that they troubled dinosaurs, too, says paleobiologist Paul Barrett of the Natural History Museum in London, who also was not involved with the research. To him, the fossilized objects look like nematodes.

"It's a really neat study," Barrett says, "and it shows that by applying a range of techniques in this case to a single bone, you can get a lot of information about the biology of an individual dinosaur." —*Chris Baraniuk*

HUGO CAFASSO (*1*); FROM "BLOOD PARASITES AND ACUTE OSTEOMYELITIS IN A NON-AVIAN DINOSAUR (SAUROPODA, TITANOSAURIA) FROM THE UPPER CRETACEOUS ADAMANTINA FORMATION, BAURU BASIN, SOUTHEAST BRAZIL," BY TITO ALEXANDRE ET AL., IN *CRETACEOUS RESEARCH*, VOL. 118; FEBRUARY 2021 (*2*)

A modern-day field growing in water and sediment left in the aftermath of an El Niño event near Pampa de Mocan (*1*). Views of an ancient field and canals (*2* and *3*).



ARCHAEOLOGY

Channeling El Niño

Peruvian farmers harnessed this weather pattern's floods on a large scale 2,000 years ago

Peru's northern coast has one of the world's driest deserts, except for when El Niño drowns it with massive flooding. Rainwater from the nearby Andean foothills gushes down parched ravines, often tearing up modern agricultural fields in the sediment-rich plains of the desert's few rivers. Ari Caramanica, an archaeologist at the Universidad del Pacífico in Lima, had long been intrigued by what looked like lengthy, straight, ancient "structures" crisscrossing parts of this desert, which is called the Pampa de Mocan. Caramanica first noticed these strange lines in 1940s photographs. Now she and her colleagues say farmers built them in pre-Hispanic times as a sophisticated, flexible-use canal system—which usually used river water but harnessed the wild El Niño floodwaters when they came—to survive and prosper in a challenging environment.

El Niño events occur in irregular six- to 20-year cycles, and they are difficult to predict. But Caramanica and her team's new research suggests farmers working between A.D. 1 and 1476 were prepared. The scientists examined the Pampa de Mocan archaeological record to chart how these farmers benefited from El Niño floods; their research is reported in the *Proceedings of the National Academy of Sciences USA*.

By studying clues such as discarded agricultural tools, plant remains and field locations, the team determined that farmers selectively used the canals to direct El Niño floodwaters to fields farther from the rivers, beyond the reach of their typical river-water irrigation. Some of these fields were enclosed by low mounds that allowed water to collect. The ancient farmer-engineers also built small rock piles to slow the water's movement and to collect fertile silt.

Caramanica says she had initially assumed that the photographed structures were used for more common river-water agriculture. But her team found they had multiple roles—some even acting as fields themselves when El Niño damage cut them off from rivers. "They were being constantly patched together to take advantage of whenever and wherever water appeared," she says.

In contrast, Caramanica says modern agriculture in the area is dominated by large landholders or companies that repeatedly plant one crop and use fields year-round, not adapting as flexibly as smaller operations could.

"It's about the perception of the environment," says Jason Nesbitt, an archaeologist at Tulane University, who was not involved in the study but wrote an accompanying commentary. Today many view El Niño events as extraordinary, he says, whereas the ancient residents of the Pampa de Mocan viewed them as an expected phenomenon—and that kind of mindset could help today's farmers deal with extreme weather. When officials draw up agricultural or disaster-management policies, he adds, "maybe it'd be good to have archaeologists and anthropologists at some levels of decision-making about these things." —*Lakshmi Supriya*

ARI CARAMANICA (*1* and *3*); LUIS JAIME CASTILLO BUTTERS (*2*)

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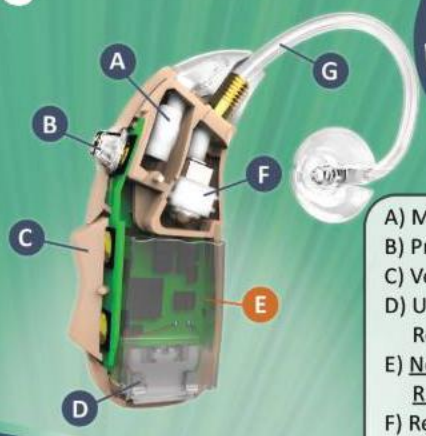

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Alison Hawthorne Deming is Regents Professor of Creative Writing at the University of Arizona, where she also holds the Agnese Nelms Haury Chair in Environment and Social Justice. She has won many awards for her nonfiction, as well as for her poetry collections, the most recent of which is *Stairway to Heaven* (Penguin, 2016).

LETTER TO 2050

The Squamscott River
grew lazy in early summer—
muskrat rose and dove
heron swept the air and landed
and hemlocks that had survived
another century's practice
of harvesting their bark
were thriving. Some suffered
beaver girdles and the predation
by woolly adelgids but still
the pileated woodpeckers
found what they required
in the snags. This is how it was
for us—pulling threads of hope
out of the air as if we had
the skill to weave them
back into webs. We surprised
ourselves when it worked—
so much needed to be undone.
And I promise you that
as paltry as our efforts
may seem to you—no.
I won't justify our failures.
The story of the alewives'
return—that's what I wanted
you to know because it helps
to think of desires that last
for centuries without being
satisfied. How far inland
did the alewives come,
I wondered, the dam removed
after three hundred years
and in the first year then
they came in a rush.
Locals could hear the gulls
gathered in the estuary
in their joy and the alewives
swam and swam to the reaches
of their ancestors—eleven miles
and three hundred years
of appetite for place
their genes remembered
and knew how to find.
The Abenaki offered
a welcome back ceremony.
And fishers gathered—human
cat and bird to feast
and the memory that had been
thwarted for centuries
became a fertile flow.

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Claudia Wallis is an award-winning science journalist whose work has appeared in the *New York Times, Time, Fortune* and the *New Republic*. She was science editor at *Time* and managing editor of *Scientific American Mind*.

Is 70 *Really* the New 60?

People are aging better in many ways but not across the board

By Claudia Wallis

We often hear that 60 is the new 50 and 70 the new 60. It is a bromide borne out by old photos. Just check out images of your grandparents or great-grandparents (depending on your age) and notice their stooped and soft bodies, their lined faces and how they seem anchored in their chairs when they were barely pushing 60. What a contrast with vigorous, gym-going sexagenarians of today!

Recent studies comparing populations born in different decades have looked beyond these surface impressions to nail down actual physical and mental differences in the ways we are aging. This research has identified particular areas of improvement. But these gains are not across the board, and they appear to depend on social, behavioral and economic factors.

A pair of new studies from Finland—one looking at physical aging and one looking at cognitive aging—strikingly demonstrates some of the details of generational change. The research, overseen by gerontologist Taina Rantanen of the University of Jyväskylä, compares adults born in 1910 and 1914 with those born roughly 30 years later. The two age groups were assessed in 1989 and 1990 and in 2017 and 2018, respectively. The beauty of this work is that both birth cohorts were examined in person at age 75 and again at 80 with the same substantial battery of six physical tests and five measures of cognition. Most cohort studies look at a narrower range of measures, and many of them rely on self-assessments.

The later-born group could walk faster, had a stronger hand grip and could exert more force with their lower leg. Such metrics are reliable predictors of disability and mortality. On cognitive tests, the later cohort had better verbal fluency (naming more words beginning with a K in three minutes), clocked faster reaction time on a complex finger-movement task, and scored higher on a test matching numbers to symbols.

But not everything changed across the generations: measures of lung function were surprisingly static, and there was no improvement in the short-term-memory task of recalling a string of digits—possibly because rote memorization has been de-emphasized in school and in daily life in recent decades, the researchers suspect.

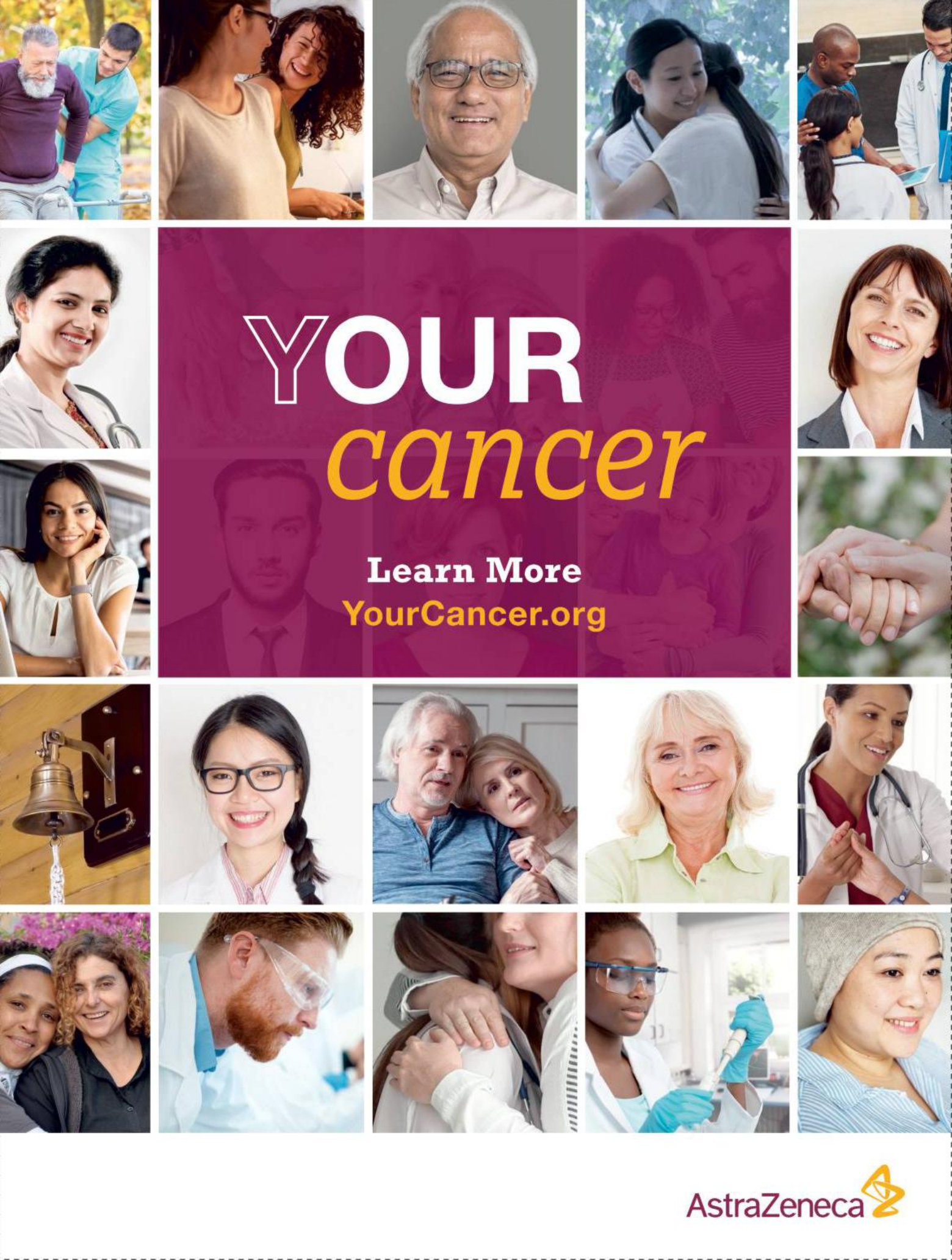
Many of these findings fit with other cohort studies. For example, a Dutch study of cognitive aging published in 2018 showed that elderly adults born between 1931 and 1941 outshone same-age adults born in the 1920s on a range of cognitive measures—though again not on short-term memory. And a 2013 Danish study revealed that birth-year-related differences can persist into very old age: 95-year-olds born in 1915 outperformed 93-year-olds born in 1905 on a cognitive exam. The later-born Danish cohort did no better on measures of gait speed and grip strength, but its members were more adept at activities of daily living—such as bathing and dressing—perhaps because of their improved cognitive status.

There are many reasons that people are aging better, including improved medical care and a drop in smoking, but the factors that loomed largest in the Finnish study of physical function, lead author Kaisa Koivunen says, were that the later-born adults were more physically active and had bigger bodies, which suggests better nutrition. (In 1943 Finland became the world's first country to require universal free lunch at school.) For brain function, the key seems to be more years of education. In both the Finnish and the Dutch studies, cognitive differences between the cohorts largely disappeared when researchers controlled for this factor.

Education is a powerful influence on aging and health, says Luigi Ferrucci, scientific director of the U.S. National Institute on Aging: "With more education, you are probably going to have a larger income, which means you are more likely to go to the doctor, have good nutrition and have a job that is not eviscerating your body." In prosperous countries, these advantages have not only lengthened life but most likely ensured that more years are spent in good health.

In the U.S., Ferrucci says, the benefits of prosperity are less equally distributed than in Finland or Denmark. He points out that the average life span is seven years shorter in a poor state such as Mississippi than in a wealthier one such as California. "Here we still have lots of people who cannot take the drugs they need because they cannot pay for them," he says, and they may lack access to healthy foods and opportunities for exercise and learning that extend our vital years. In short, 70 may be the new 60 for many of us but not for all. SA

Illustration by Fatinha Ramos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ASTRONOMY

TOO BIG FOR THE UNIVERSE

Ancient galaxy clusters seem to have grown so quickly that they would have broken the laws of the cosmos

By Arianna S. Long

Illustration by Ron Miller

Arianna S. Long is a graduate student in physics at the University of California, Irvine, who investigates the history of galaxy clusters.

JUST LIKE TREES, PEOPLE AND STARS, GALAXIES HAVE LIFE CYCLES. A galaxy is born when enough gas and stars coalesce to form a coherent structure—perhaps it starts as one cloud of gas and slowly gathers mass, or maybe it builds up from the collision of two or more clouds. Either way, once formed, a galaxy spends its lifetime making stars, using its reservoirs of gas to create tiny furnaces where nuclear fusion burns elements to release light and energy. A galaxy deemed "alive" shines strongly in ultraviolet light, a signal of young, bright and hot stars. As those stars age, their light changes from hot and blue to cool and yellow or red. When a galaxy contains mostly yellow and red stars and emits little to no ultraviolet light, we consider it retired, or "red-and-dead." Eventually, if massive enough, it becomes a spheroidal blob, known as an elliptical galaxy, that will likely never birth a new star again.

All around us in nearby space—within, say, 300 million to 600 million light-years—astronomers see dead or dying elliptical galaxies gathered in great ensembles called galaxy clusters. These clusters hold the fossilized remains of the most massive galaxies ever formed—hundreds to thousands of them slowly dancing around one another, gravitationally bound forever in their permanent graves.

But galaxy clusters present a problem for astronomers. Most clusters seem to have been established by the time the universe was only half of its current age. That means the galaxies within those clusters must have birthed most of the stars they contain early in cosmic history. It appears that these galaxies grew to the size of the Milky Way or larger but up to 10 billion years more quickly. The young galaxy clusters, called protoclusters, where these galaxies formed must have been incredibly violent and active places, full of galaxies producing stars at a furious pace. Our current understanding of physics cannot quite explain how they could have grown so big so quickly.

Only recently have astronomers had the telescopic tools necessary to find protoclusters, which are very distant (their light often travels 10 billion years or more to reach us) and frequently hide their most massive galaxy members behind dust. In the past few years scientists have discovered two protoclusters that are providing an unprecedented window into cluster growth. Follow-up observations have revealed that they are, in fact, active and huge—so huge that

CLUSTERING GALAXIES: The Hubble Space Telescope captured this image of the galaxy cluster RXC J0032.1+1808. Astronomers are hunting for progenitors of such groups in the early universe.

ESA, HUBBLE, NASA AND RELICS

they challenge our understanding of galaxy formation. If we can solve the riddle posed by galaxy clusters, we may redefine our understanding of the evolution of the universe.

HUNTING FOR STARBURSTS

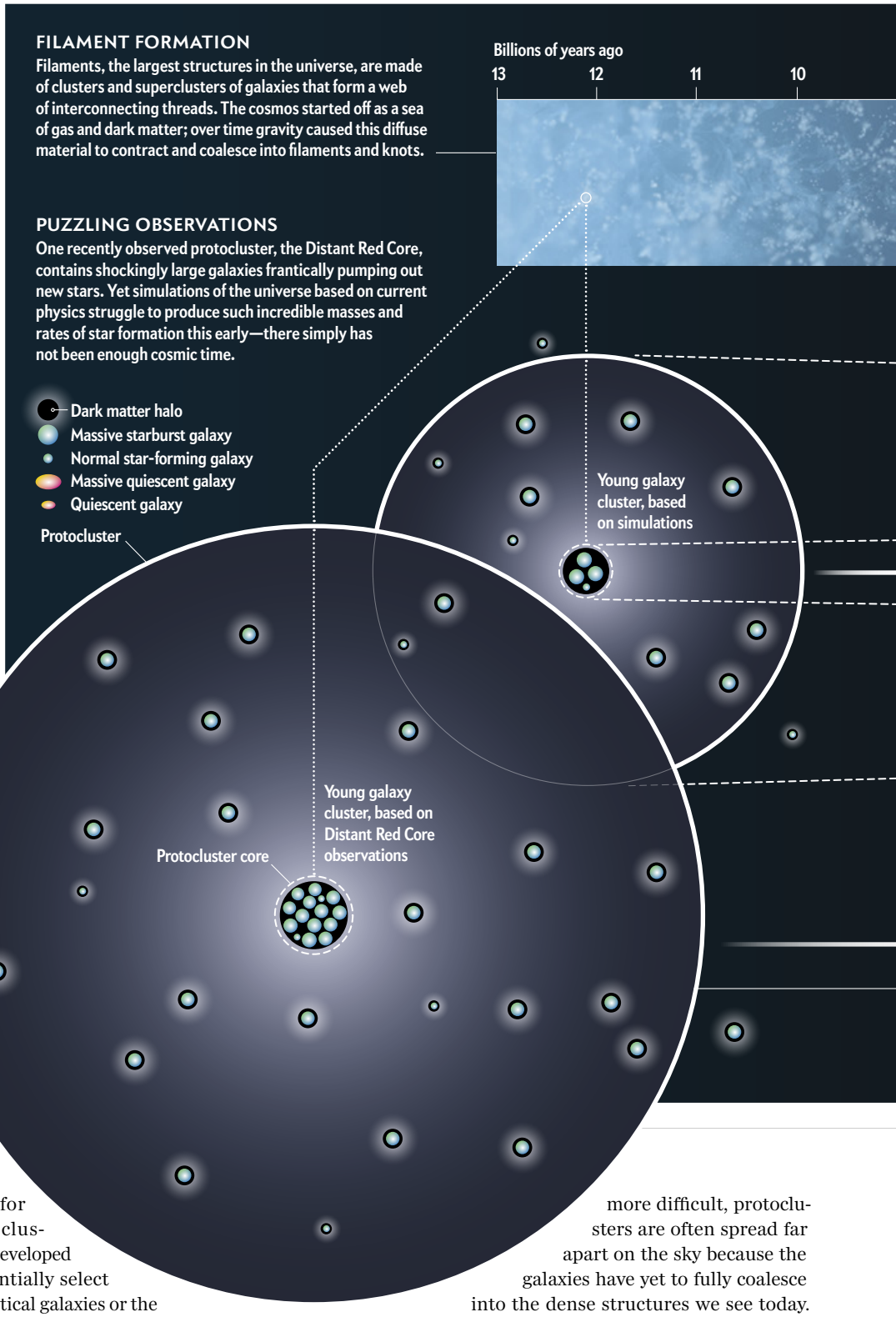
THE MOST COMMON TYPE of star-forming galaxy produces roughly one to tens of suns' worth of stars a year. These are often called normal star-forming galaxies. The Milky Way is in this class. Normal star-forming galaxies are metaphorical tortoises, forming stars slowly and steadily over the course of 10 billion years or so, remaining blue and disklike and depleting their reservoirs of fresh gas (fuel for new stars) at a leisurely pace.

Galaxies that produce hundreds to thousands of stars every year are known as starburst galaxies. These are the hares in galaxy evolution. In perhaps 300 million years at most, these galaxies burst into existence, form as many stars as possible as quickly as possible and, in a cosmic blink of an eye, run out of fuel. Starbursts live fast and die young. Astronomers think that they are the best candidate ancestors for the massive, dead elliptical galaxies we see in clusters today.

It stands to reason that if we looked deep enough into space, we would find protoclusters filled with starburst galaxies—tomorrow's clusters of dead galaxies. Yet finding starburst galaxies in protoclusters has proved challenging. Until recently, most of our

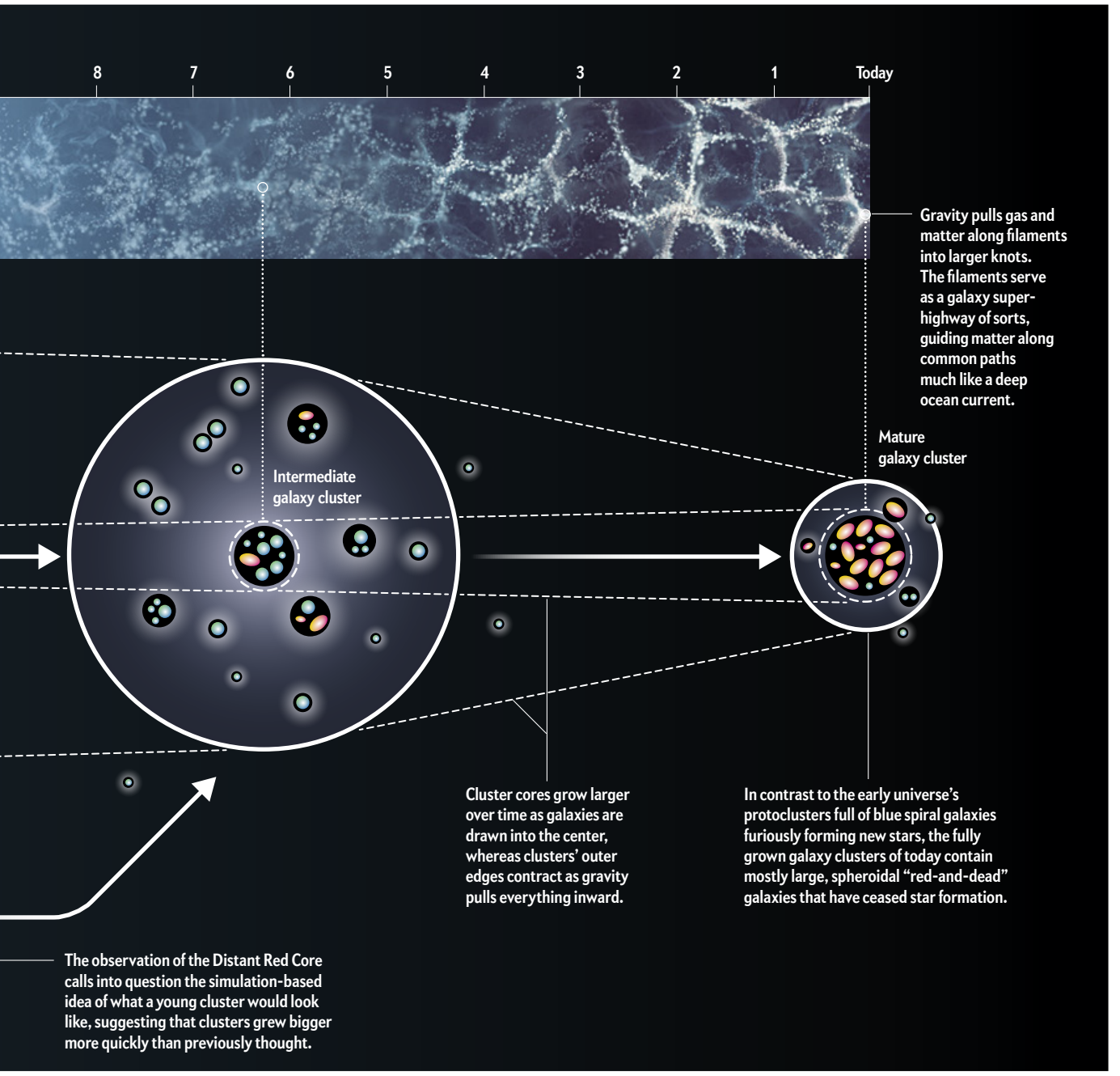
How Galaxy Clusters Grow

Today galaxy clusters are filled with huge, mostly dead galaxies—meaning they have stopped forming new stars. Astronomers have recently discovered several distant "proto-clusters," the progenitors of modern clusters, full of young galaxies rapidly birthing stars. But the proto-clusters are so big, and their galaxies so massive, that scientists question how they could have grown to such a point so early in cosmic history.



methods for spotting clusters were developed to preferentially select dying elliptical galaxies or the hot gas that pervades the space between them. Elliptical galaxies and hot intracluster gas appear at the later stages of galaxy cluster evolution, so we need new methods to find their bluer, more star-forming infant counterparts. To make things more difficult, protoclusters are often spread far apart on the sky because the galaxies have yet to fully coalesce into the dense structures we see today.

When our most famous and precise telescopes have cameras that span only the width of a pencil (the Hubble Space Telescope, for instance), it is not surprising that we cannot piece together protocluster puzzle pieces that are spread across the sky at distances more



SOURCE: "GALAXY PROTOCLUSTERS AS DRIVERS OF COSMIC STAR FORMATION HISTORY IN THE FIRST 2 GYR," BY YI-KUAN CHIANG ET AL., IN *ASTROPHYSICAL JOURNAL LETTERS*, VOL. 844; AUGUST 2017 *(simulations)*

than 100 times greater than our telescope's field of view.

Other methods of searching, such as systematically surveying large swaths of the sky, tend to miss starburst galaxies because they are often obscured by dust. The exceptional stellar growth in starbursts generates an overabundance of heavy metals that are produced in the explosive deaths of stars. Once dispersed into space, heavy elements such as iron, carbon and gold collide to form complex dust molecules that absorb and obscure ultraviolet and optical light. Think of the reddening sun during wildfire season: dust dims hotter, more energetic blue light while letting redder light sneak through. The result is that starburst galaxies are nearly invisible when viewed with optical and ultraviolet telescopes, but they shine like beacons when viewed in the cooler infrared spectrum.

All of this means that until recently, the tools to find and study protoclusters usually missed a key population of galaxies. From the late 1990s through the early 2010s the Submillimeter Common-User Bolometer

Illustration by Ron Miller and Jen Christiansen

Array, the Herschel Space Observatory, the South Pole Telescope and the Spitzer Space Telescope revolutionized our understanding of the dust-obscured universe by unveiling millions of galaxies that were previously invisible. Starting about 15 years ago, astronomers began studying the clustering properties of dusty starbursts, and they found that these powerhouses live preferentially near other large and actively star-forming galaxies. But the state of technology was still behind our ambitions; the resolution of infrared and millimeter telescopes was still so low that multiple galaxies would get blended into one large object, even if those galaxies were far apart but lay along the same line of sight. The age of the infrared universe was here, but we needed sharper and more sensitive instruments to fully comprehend what we were seeing.

No words can describe how it feels to be the first person to glimpse a part of the universe that no one else has seen. I felt compelled to take a moment to inspect each star and galaxy in the field.

Finally, in 2013, the Atacama Large Millimeter/submillimeter Array (ALMA) arrived. High in the Chilean desert, this collection of nearly 70 radio dishes works together as a single telescope, reaching resolutions up to 600 times sharper than that of the Herschel telescope. ALMA has transformed many corners of astronomy, including galaxy evolution. (I know several people with tattoos dedicated to this telescope.) The observatory is excellent at detecting dusty, gaseous stellar nurseries throughout star-forming galaxies. With it, astronomers have discovered systems that are both shocking and exciting.

SURPRISING BEHEMOTHS

IN 2018 TWO SEPARATE TEAMS OF ASTRONOMERS used ALMA to study the brightest infrared objects they could find in the distant universe. Each team discovered a different conglomeration of dusty, starbursting galaxies that were previously blended together, hiding as one in surveys taken by the first generation of infrared telescopes. SPT2349-56, a group of 14 galaxies, and the Distant Red Core (DRC), a group of 10 galaxies, were both found growing and thriving in different corners of the universe, when the cosmos was only 10 percent of its current age. We see both these budding protoclusters undergoing extreme bursts of star formation—each group birthing nearly 10,000 times as much stellar mass a year as the Milky Way does—across volumes only half the size of our Local Group (which includes our own galaxy plus Andromeda and several smaller galaxies). Estimates of the protoclusters' individual gas reservoirs tell us that if these galaxies continued to form stars at such excessive rates, they would exhaust their fuel supply in just a few hundred million years and become the massive red-and-dead elliptical galaxies that are ubiquitous in fully grown clusters. Moreover, they would complete this cycle well before the present era.

The discovery of these two dust-obscured protocluster cores presented a promising new lens for studying cluster growth, but we were still missing an important part of the picture. The best way to "weigh" a galaxy is to measure the light from its adult star population, which requires data from across the electromagnetic spectrum. But until recently, all observations of protoclusters living in the first two billion years of the universe had been conducted within a narrow energy spectrum (in either the optical or the infrared). Then, in September 2018, my colleagues and I were able to observe, for the first time, ultraviolet and optical emission from a dusty, starbursting protocluster as seen 12 billion years ago: the Distant Red Core. Using the Hubble Space Telescope, the Gemini Observatory and the Spitzer Space Telescope, we captured the multiwavelength perspective necessary to more deeply understand this structure's past and future.

Waiting for Hubble Space Telescope data can be anticlimactic. You know the day that your patch of the sky is scheduled for observation, but you have no idea when you will actually receive your data: you just have to wait for an e-mail notification to tell you to check the archive. The day our protocluster observations were scheduled, I checked my e-mail what felt like every two minutes. I was disappointed when it was time to go to bed and nothing had arrived in my in-box.

The following morning, against my partner's protests, I rolled out of bed immediately and went straight to my computer to see if the data had at last come in. Fortunately, it had been delivered a few hours past midnight. I commenced the download, dancing impatiently like a child waiting for her turn to unwrap presents. Finally, I opened the image. No words can describe how it feels to be the first person to glimpse a part of the universe that no one else has seen. I felt compelled to take a moment to inspect each star and galaxy in the field, to acknowledge their existence. Eventually I snapped back to myself and zoomed

in on the part of the sky I was interested in. I saw something remarkable.

This little region of space is violent. At least half of the galaxies there were so messily shaped they must have recently crashed into other nearby galaxies or were still in the process of doing so. When we measured the population of adult stars in these galaxies, we found something incredible—so incredible that it may pose a problem for our current understanding of the universe. Already at this early era, some of the Distant Red Core galaxies had formed three times more stars than our own Milky Way has—but in just a fraction of the time. Yet simulations of the universe based on known physics struggle to produce galaxies this massive so early on. This incongruity between simulation and observation exacerbates a problem that we have known about since the discovery of dusty, star-forming galaxies. Modeling the extreme pace and density of star formation seen in starbursts is difficult because physics predicts the simulated galaxies should either shred themselves apart or heat up so much that they blow out all of the fresh gas needed to grow large enough to match what we see today.

The protocluster as a whole presents another problem: it is shockingly massive. When I first measured it, I could not believe my numbers. I knocked on doors in my department to make sure I was doing the calculations right. Two weeks later I brought the results to a conference to show my collaborators. One said, "There must be a bug in your code." Another asked, "Are you sure you're not double counting somewhere?" (Turns out there was a small bug in my code, but it was not enough to explain the huge measurement.) Eventually, after double-checking my calculations and trying out different methods, the measurements became undeniable. The Distant Red Core seems to be too big for our universe. We do not know how it could have gotten so large in such a short time.

To better understand its bulk and how much mass was in the form of stars, we zeroed in on the size of the dark matter halo around this protocluster. Dark matter is the most abundant form of matter in any given galaxy and in the universe as a whole. All galaxies and clusters are thought to be surrounded by blobs, or halos, of this mysterious stuff. And although it is invisible and poorly understood, dark matter leaves a clear gravitational signal. There are a variety of ways to infer the amount of dark matter in a given astronomical object, and to cover those methods would require an additional article (or five).

Suffice it to say, we weighed the dark matter component of the Distant Red Core, and according to our simulations, it contains nearly the largest allowable halo mass at that period in the history of the universe. This apparent overabundance of dark matter means that the DRC may be so large that it violates the laws of our universe as we understand them. When we fast-forward our simulations to estimate what the DRC may look like after evolving 12 billion years to the present day, we find that it may grow to be larger than the largest known galaxy cluster, El Gordo. Although we have a healthy margin of error on our dark matter calculation (meaning it could be overestimated), the discrepancy looks even worse when we consider the fact that our observations are capturing only a small percentage of the likely galaxy cluster members; there are probably more galaxies in the DRC that were simply out of the narrow field of view of our telescopes and thus not included in our calculations. This mismatch most likely will grow as we continue surveying and studying this protocluster.

RETHINKING THE TIME LINE

OUR INVESTIGATION OF THE DISTANT RED CORE, along with the discoveries of other potentially similar protoclusters, forces us to reconsider our understanding of galaxy cluster formation. Because the galaxies in clusters are likely to be some of the first galaxies ever, we must determine how such massive objects can form so quickly. Doing so is not just an issue of constraining the physical mechanics and chemistry of star formation inside the first galaxies. It is also a matter of investigating the timing of the conditions that lead dark matter to gravitationally collapse into halos, seeding galaxies. Is it possible that galaxies and structure began forming earlier in the universe than we thought? What does that mean for our understanding of the formation of the first elements? Could these galaxies have forged the right ingredients to build stars with habitable planets around them—and perhaps hosted some of the first forms of life in the universe?

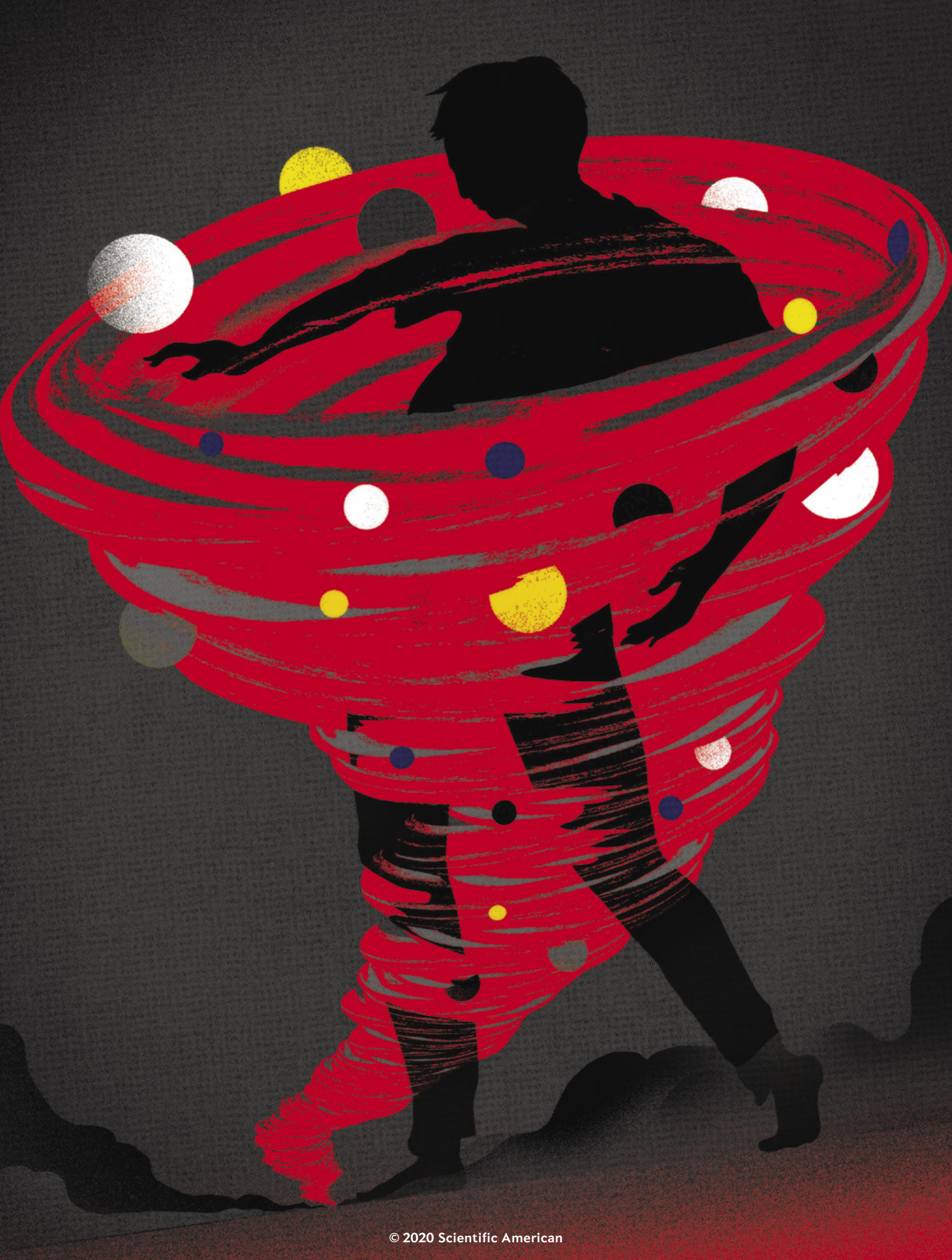
Some of these questions probably will not be answered during my lifetime, but I and other astrophysicists are working hard and fast to address the others. Already we are carrying out more observations of these known protoclusters across the electromagnetic spectrum. We are also developing new methods for identifying large samples of dusty protocluster candidates. With more examples, we may be able to determine whether protoclusters such as the Distant Red Core are examples of a common, yet previously invisible, phase of galaxy evolution that all clusters go through or just rarities. Observers and theorists are forming new collaborations to learn how early in the history of the universe conditions were right for protoclusters akin to those we have discovered—pockets of space overdense with tremendous rates of star formation and outsize masses.

The best way to test our physical models is to look at extremes. In the next few years these colossal congregations of exceptional galaxies will be putting humanity's grasp on the cosmos to the test. SA

FROM OUR ARCHIVES

Back in Time. Dan Coe; November 2018.

scientificamerican.com/magazine/sa



IMMUNOLOGY

The Immune Havoc of COVID-19

The virus flourishes by undermining the body's chemical defense system

By Akiko Iwasaki and Patrick Wong

Illustration by Brian Stauffer

Akiko Iwasaki is Waldemar Von Zedtwitz professor in the department of immunobiology and the department of molecular, cellular and developmental biology at Yale University and an investigator of the Howard Hughes Medical Institute.

Patrick Wong is a graduate student in the Iwasaki Lab at Yale University.

We may well remember the 21st century in two halves: the time before SARS-CoV-2 and the time after. Despite decades of warnings about the potential for a deadly global pandemic, public health systems worldwide were completely outmatched. The first COVID-19 patients were admitted to a hospital in Wuhan, China, on December 16, 2019, and several of them died. Many Americans assumed that even if China failed to contain the virus on its own soil, the span of an ocean would protect them. This complacent view ignored the fact that previous coronavirus outbreaks—caused by SARS-CoV (for severe acute respiratory syndrome coronavirus) and MERS-CoV (for Middle East respiratory syndrome coronavirus)—reached several continents; MERS-CoV has yet to be eradicated. And so SARS-CoV-2 arrived on American shores in early 2020. The public health response was chaotic and varied from region to region. Some cities and state governments invoked stay-at-home orders and mask-wearing mandates. Others simply hoped for the best. At the timing of writing, 1.3 million had died globally.

Despite the disorganization at the national level, medical professionals and research scientists launched an all-out effort to counter the new threat even before it arrived in the U.S. Less than a year later this global collaboration has generated unprecedented insight into the coronavirus and its impact on the human body. We are now beginning to understand why SARS-CoV-2 results in wildly different degrees of illness. Some people exhibit no symptoms; others develop a cough or a fever. Most gravely, some fraction of patients suffer a life-threatening pneumonia and a condition called acute respiratory distress syndrome (ARDS).

Researchers now know that the virus, like SARS-CoV and MERS-CoV, can provoke the immune system to misfire—and the resulting inflammation may lead to ARDS and an array of perilous symptoms. Readily available clinical tests show clear evidence of high levels of immune proteins—IL-6, TNF-α and CRP—in the blood of seriously ill patients. A few months into the course of the pandemic, the welcome but limited success of broad immune-suppressing drugs, such as the corticosteroids prednisone and dexamethasone, confirmed suspicions that in the sickest patients the immune system had gone into hyperinflammatory overdrive. These same anti-inflammatory treatments were widely used for severe infections with the preceding coronavirus outbreaks.

We now know that in a certain fraction of COVID patients, an unbridled immune response causes damage throughout the body, producing blood clots, heart damage and even organ failure. The most severe cases require hospitalization in intensive care units. The standard retinue of steroids are not enough for treating severe COVID: these patients will require more targeted treatments. We also badly need rapid tests that can examine tissue samples for biological indicators, or biomarkers, that predict the course of the disease—for example, the likelihood that a patient diagnosed with mild COVID will go on to develop a severe case.

IMMUNOLOGICAL MISFIRES

Developing biomarkers and drug treatments requires a deep understanding of how SARS-CoV-2 interacts with cells throughout the body and how the immune system then responds to the virus's arrival. This past spring our laboratory, in collaboration with many others, began to examine the dysregulated immune reactions that underlie severe COVID cases. We knew when we started that the immune system choreographs an intricate chain of events in response to invading pathogens. We also knew that if any of the steps in the immune response are mistimed, it can lead to exaggerated levels of inflammation that damage the body's own tissues.

The immune system has in its arsenal a fast, emergency response and a slower but longer-lasting defense against viruses, bacteria, fungi and other pathogens. The "innate" immune system acts as a first responder. Some receptors on and inside these

immune cells sense invaders, activating an elaborate signaling cascade using proteins called cytokines. The cytokines warn nearby cells to put up defenses, initiate the death of an infected cell or heighten the alarm to bring in other types of cytokines. Innate immune cells also summon certain white blood cells to build more durable immunity to the pathogen. Within a week or two these members of what is called the "adaptive" immune system become active by increasing levels of highly targeted antibodies and T cells that eventually disable or kill an invader.

In most COVID patients, the innate immune system performs as it evolved to, disarming and killing SARS-CoV-2. In about 5 percent of cases, however, the body's counterattack does not proceed as planned. When this carefully timed cascade of signals goes awry, innate immune cells react by making too many cytokines. The overproduction of diverse signaling molecules in COVID-19 resembles "cytokine storms" that turn up in other medical conditions and were thought to be a factor in severe COVID. The most recent research suggests that, in most cases, inflammation differs from that of a cytokine storm, even though it still poses a threat to patients. It can bring about ARDS, resulting in lasting damage to the lung or other tissues. It can also lead to the buildup of fibrin, a protein that causes clotting. If this were not enough, it can induce fluid leakage from blood vessels, triggering respiratory failure.

LABORATORIES the world over have adapted their research to target the fight against COVID-19. In Siena, Italy, a biosafety lab develops an antibody test.

PAOLO LAZZERONI Getty Images

Viruses harness the human cell's machinery to reproduce themselves. One innate immune system strategy undermines the virus's ability to multiply, but it appears to falter against SARS-CoV-2. In recent months researchers have devoted attention to a class of cytokines known as interferons, a first line of defense that can block the various steps of viral replication in a cell. Rapid production of type I interferon (IFN-I) by the immune system may enable a virus to be brought under control and check any progression beyond mild disease. But some studies suggest that in older adults or patients exposed to large amounts of a virus, the immune system may lag in its response, allowing the virus to continue reproducing. Further, when interferons finally do arrive on the scene, they may overreact, spurring the manufacture of high levels of diverse cytokines, which can lead to inflammation and severe illness. Measuring the interferon response may furnish vital knowledge about whether a COVID-19 case will progress to a life-threatening illness, and it may provide clues about how to treat the infection.

The science is still in flux, however, and there are many ways the immune response could run askew. For example, the virus may hamper a person's ability to make interferons. Alternatively, a given patient might produce less IFN-I because of genetic factors. It is even possible for a person's immune response to be so erratic that their body makes antibodies against IFN-I. We and others are investigating the presence of these "autoantibodies" as a possible cause of long-term COVID symptoms. Detection of autoantibodies could serve as a useful biomarker to predict whether a patient's condition will worsen. Some patients might also benefit from an infusion of lab-made interferon, and clinical trials of such treatments have already begun, but the results are still unclear.

AN INFLAMMATORY ERUPTION

CYTOKINE STORMS made headlines in severe cases of the previous coronaviruses (SARS-CoV and MERS-CoV), so when SARS-CoV-2 emerged it was natural for scientists to suspect that a similar mechanism was at play. Early in the pandemic, physicians did detect elevated cytokines in patients, but the amount of these proteins and the subsequent inflammatory state they evoked differed from that of a classic cytokine storm.

Whipping around inside these patients were high levels of cytokine proteins that, depending on the cell receiving them, could lead to a range of outcomes, some of them detrimental. Some cytokines, such as IL-6, TNF-α, IL-1β and IL-12, amplify inflammation and tissue damage. Diane Marie Del Valle of the Icahn School of Medicine at Mount Sinai and her colleagues reported significantly elevated levels of some of these cytokines in the blood of nearly 1,500 patients from the New York City area. Findings from this group indicated that abnormally high levels of IL-6 and TNF-α could serve as reliable predictors of disease severity and death.

We saw the same changes in the patients we were tracking. Moreover, our lab and others began to recognize some unusual outliers among patients' cytokine profiles compared with a typical cytokine storm. We observed high levels of IL-5 and IL-17, cytokines not classically associated with antiviral immune activity. Instead these cytokines initiate a seemingly misguided response—one better suited for infections by parasites and fungi. We have yet to understand whether this response causes damage to tissue or just diverts resources the body needs to fight the virus.

In some COVID patients we also found elevated levels of chemokines, a subclass of cytokines that guide immune cells to where they are needed. High concentrations of the chemokines CCL2, CCL7, CXCL9 and IL-8 generated at infection sites served as a rallying trumpet. Not only were cytokines and other immune

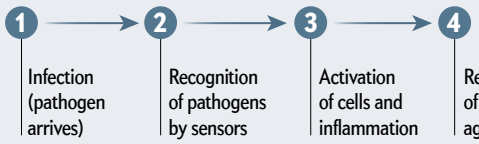
Three Ways COVID-19 Subverts the Immune System

A virus devises ingenious strategies to outwit the body's defenses. SARS-CoV-2, the virus that causes COVID-19, is adept at circumventing the body's intricately coordinated counterattack of immune cells and proteins. The virus's evasive tactics can lead to raging inflammation that damages tissue—not only in the lungs but throughout the body. SARS-CoV-2, by unknown means, also interferes with T cells that kill infected cells. Therapies under development are designed to restore the immune system's normal antiviral function.

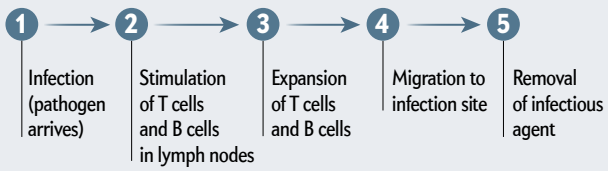
Self-Defense, Fast and Slow

The immune system mounts an immediate and a long-term response to invading pathogens. Rapid-response cells and proteins sound an alarm and organize chemical defenses to extirpate microbial invaders. Later, within a week or two, white blood cells—T cells and antibody-making B cells—multiply and move to the sources of infection, providing a long-lasting response if a pathogen reappears.

Innate Immunity (immediate)



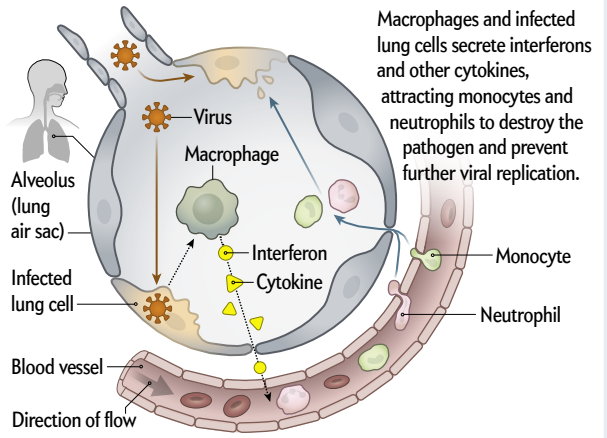
Adaptive Immunity (long-term)



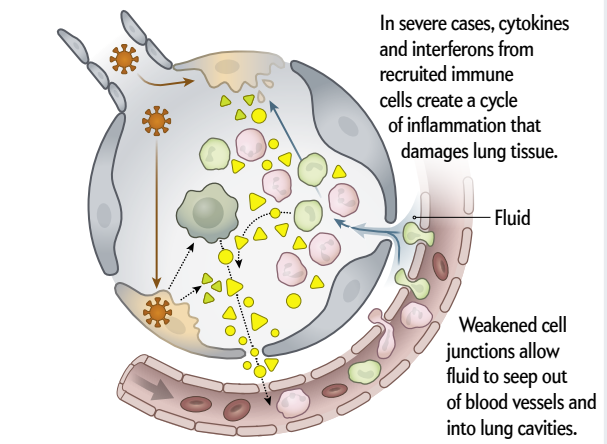
Innate Immunity and Inflammatory Storms

When the innate immune system works as it should, it disarms and destroys the SARS-CoV-2 virus. But the novel coronavirus is able to bypass this finely calibrated process, and chaos results in its wake. Too many of the signaling proteins, called cytokines, then descend on an infection site, exacerbating damage. Inflammation in infected lung cells damages tissues, and nearby blood vessels start to leak and fluid enters the lung cavity.

Beneficial Inflammatory Immune Response



Damaging Inflammatory Response



messengers causing local damage, chemokines were also calling in cells from throughout the body to join the fray.

To identify the source of tissue damage, a number of research groups decided to look at cells in the blood and lungs. In the field of immunology, we commonly use flow cytometry, a technique that allows us to tag subsets of cells in the blood with fluorescent antibodies. Using these markers, our group was able to detect a sizable shift in the populations of immune cells circulating in patients, as compared with healthy donors. Two innate immune cell types—monocytes and neutrophils—were particularly abundant.

To take one example: In healthy donors, monocytes make up between 10 and 20 percent of peripheral blood mononuclear cells, a category of commonly studied white blood cells. But in COVID patients, we often found that the proportion of monocytes exceeded the normal range by threefold or more.

As an integral component of the innate immune system, monocytes normally patrol the blood and arrive first on the scene to eliminate or sequester pathogens. When they sense a microbial threat, the cells can respond by differentiating into macrophages and dendritic cells—specific types of white blood cells. Macrophages consume pathogens and cellular debris. Dendritic cells identify and flag a pathogen for other immune cells to respond to.

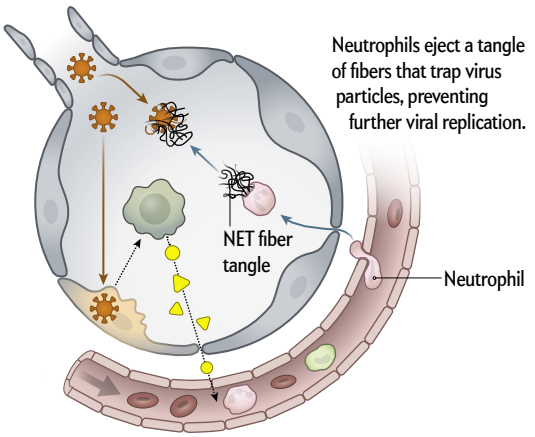
To ensure that the immune system does not overreact, levels of monocytes are usually tightly regulated, but this control is lost in severe COVID cases. In the worst disease outcomes, monocytes and macrophages infiltrated the lungs. When Mingfeng Liao and others at the National Clinical Research Center for Infectious Disease in Shenzen, China, peered into the lungs of

Illustration by Jen Christiansen

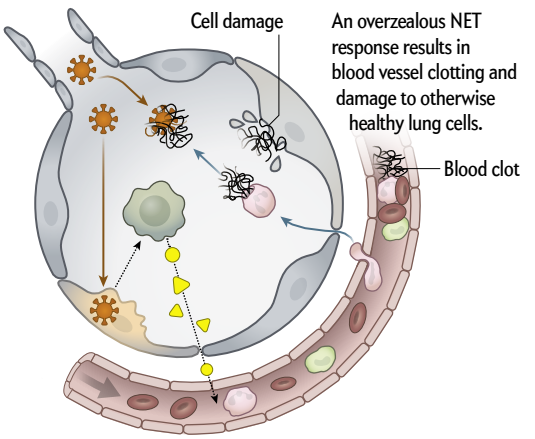
Spider Web–Like Microbe Traps

Pathogen-ingesting white blood cells called neutrophils expand to excess levels in some COVID-19 patients' blood. Their presence can help predict progression to severe disease. These cells can extrude weblike fibers—neutrophil extracellular traps (NETs)—that sequester and eliminate viruses and other pathogens. But with severe inflammation, NETs can turn toxic and produce blood clots and damage to lung cells.

Beneficial Neutrophil Extracellular Trap (NET) Response



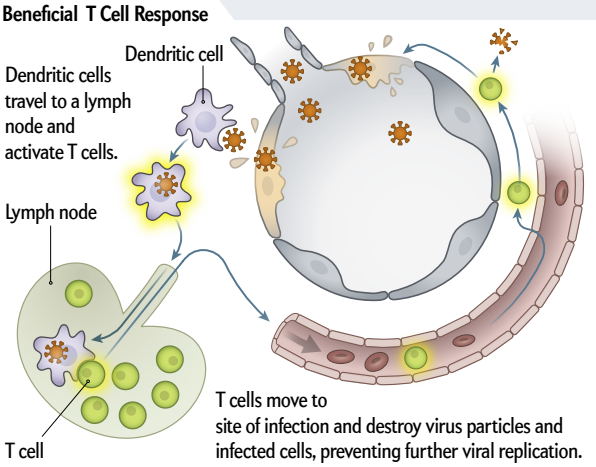
Damaging NET Response



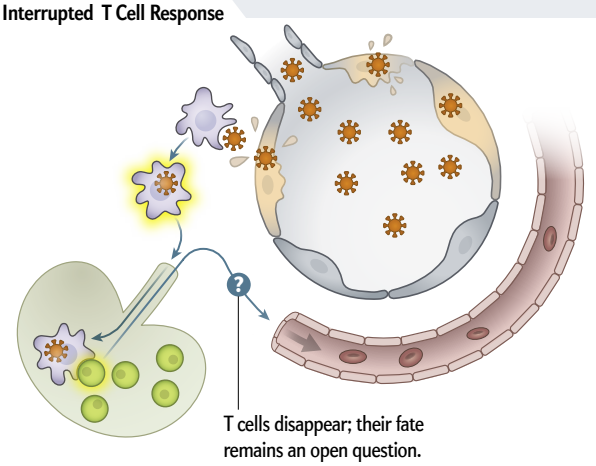
Lasting Protection and the Loss of T Cells

The adaptive immune response triggers production of T cells, which arrive to fight the virus with some lag time after an initial infection. T cells move to infected cells from lymph nodes after being called into action by dendritic cells. In COVID-19, the number of T cells able to come to the scene to take on virus-fighting roles is greatly diminished for unclear reasons that researchers continue to investigate.

Beneficial T Cell Response



Interrupted T Cell Response



severe COVID patients (by sampling cells in fluid from the lower respiratory tract using a technique called bronchial alveolar lavage, or BAL), they found an abundance of monocytes and macrophages. In agreement with some other findings, both cell types expressed cytokines at comparable levels to those found in severe inflammation. Assuming cytokines, largely produced by monocytes and macrophages, are responsible for enhancing all of this damage, interventions that block their inflammatory activity might prevent severe infection.

If cytokines are in fact major drivers of severe COVID, it would be logical to try to reduce their presence in patients. There are drugs that do this: tocilizumab, for example, is a therapeutic that blocks the receptor where a major cytokine called IL-6 docks. Unfortunately, clinical trials with tocilizumab show little evidence of improving disease outcomes. A growing number of scientists and clinicians have therefore begun to look beyond just cytokine storms for a more complete explanation for the damaging hyperinflammatory responses in COVID-19.

An additional contributor to COVID-19 immune pathology may be a peptide, or small protein, called bradykinin. By reanalyzing lung-fluid data from patient BAL samples, Michael R. Garvin of Oak Ridge National Laboratory in Tennessee and his colleagues formulated a hypothesis that bradykinin may, like cytokines, induce an inflammatory response. These "bradykinin storms" can, in fact, be exacerbated by inflammatory cytokines. In excess, bradykinin may lead to massive dilation of blood vessels and many of the surprising symptoms seen in COVID patients, such as cardiac arrhythmias and sudden cardiac arrest. Researchers also began to find massive

Our Lab's Makeover

On March 1, New York City confirmed its first case of COVID-19, in what would eventually become the most devastating series of community-acquired infections in any city in the U.S. Just 80 miles away, we waited in New Haven, Conn., for Yale University to confirm an impending shutdown, which happened on March 18. In many ways, our initial experiences mirrored those of all Americans placed under state lockdowns. Undergraduates were instantly sent home. Postdocs and graduate students were prohibited from working in shuttered laboratories. As coronavirus spread across the globe, university departments full of trained scientists were sidelined—though not for long. Our lives were about to change dramatically.

In the same breath, scientists at all levels in academia laugh about and lament the gossamer-thin line separating our research lives from our personal lives. The arrival of a global pandemic erased that boundary for those of us who chose to shift our research to address SARS-CoV-2. The sudden influx of patients and the urgency of the pandemic eliminated the luxury of planning experiments or leisurely reading papers. We rapidly shifted from investigating the immune response in cancer, herpes and influenza infection to uncovering its role in COVID-19. Of course, home lives were upended as well.

As a lab that specializes in the immune responses against viral infections, we were ready to contribute our insights—or at the very least our knowledge of the immune system and our skills in working with sophisticated lab equipment. In a flurry of activity, our facility began collaborations with doctors, nurses and administrators across multiple schools and departments at the university and the Yale New Haven Hospital. As soon as the first patients started to roll in, we had a chance to contribute to helping understand how SARS-CoV-2 makes us sick.

Our newly formed team, called IMPACT (*I*mplementing *M*edical and *P*ublic Health *A*ction against *C*oronavirus *CT*), performed numerous PCR (polymerase chain reaction) tests daily to supplement our area's capacity for testing suspected cases of COVID-19. Compared with reports of two-week turnarounds from commercial testing facilities, we were blazingly fast, with samples arriving in the early afternoon and results available less than 12 hours later. Although we did not realize it at the time, this pace was to become the new norm for research in the lab and represented only our first steps in a pivot toward SARS-CoV-2 research.

Academic research typically proceeds at a slower, more considered pace, but doing science in the time of COVID-19 required us to be just as careful in a fraction of the time. Our efforts to pitch in with PCR testing blossomed into a comprehensive study of the immune cell changes occurring in SARS-CoV-2-infected patients. We received batches of patients' blood daily, and within hours we translated these crimson tubes into hard data. From each patient, we took a sample roughly every four days, and over the course of months we assembled these daily snapshots into a comprehensive record of the immune system's fight against SARS-CoV-2. Most important, we were learning in real time what turned a bad infection into a lethal one.

At the same time, all across the world, other labs were racing to perform similar experiments. As you can expect, these parallel efforts can generate comparable data and—more often than you would like—conflicting numbers. But when dealing with patients, it is extremely important to validate a discovery from one lab with the work of another and another on top of that.

People are unique and so, too, are their medical treatments, their underlying conditions and other factors outside of our ability to accurately track, such as how much virus they were exposed to. It is a testament to the robustness of our scientific enterprise that researchers around the globe came to similar conclusions.

The pandemic has also changed the way we disseminate our findings. Instead of waiting for months to publish our work in peer-reviewed journals, scientists quickly shared their findings through preprint servers such as BioRxiv and MedRxiv. This enabled rapid exchange of information and ideas in real time and changed academic publication practices overnight. COVID-19 has brought about a fundamental transformation in the way that basic research is conducted. *—A.I. and P.W.*

increases in the production of hyaluronic acid in very ill patients. Aggregates of this molecule can hold remarkable amounts of water. Postmortem analyses of heavily saturated lungs have shown that the combination of these conditions, along with the leakage of fluid from blood vessels, has proved fatal for some patients.

The involvement of bradykinin in COVID-19 requires further confirmation. Direct measurement of the peptide remains extremely difficult. But some success from an exploratory study with icatibant, an inhibitor of a bradykinin receptor, lends weight to the hypothesis that lower levels of the peptide may alleviate severe illness.

ROGUE MICROBE TRAPS

BRADYKININ ALSO TURNS UP in another inflammatory pathway found in patients' blood. Neutrophils, which ingest pathogens, can activate bradykinin production. Various labs, including our own, have found neutrophils to be plentiful in some patients' blood. High circulating levels of a cytokine called IL-8 in COVID-19 can attract neutrophils to sites of infection, including the lungs, and assist in increasing the numbers of these cells as well. Crucially, the presence of elevated neutrophils on the first day of hospitalization reliably predicts later admission to the ICU.

Recent papers suggest why neutrophils may be a culprit in COVID-19 pathology. These cells extrude neutrophil extracellular traps (NETS), which consist of webs of DNA, antimicrobial proteins and enzymes that sequester and kill pathogens. Unfortunately, NETS can also damage tissue.

Looking at lung autopsy specimens, Moritz Leppkes of Friedrich-Alexander University in Germany and his colleagues discovered striking blockages of small blood vessels by aggregated NETs. They also observed NETs in the blood vessels of kidney and liver samples. In addition to physical obstructions, NETs can degrade proteins that inhibit blood coagulation, contributing to the high

levels of clotting in severe cases. Acknowledging a possible role for these aggregates, McGill University has announced a pilot study of a cystic fibrosis drug that clips apart the DNA in NETs.

These varied studies have made it apparent that SARS-CoV-2 turns the immune system against itself. Innate immune defenses—cytokines, monocytes, neutrophils and others—spin out of control. The adaptive immune system also goes off-kilter. One of the most apparent differences between the blood of some COVID patients and healthy individuals is the stark loss of T cells, key components of long-term adaptive immunity.

Researchers have observed that T cells from patients with moderate disease behave differently than those from severely ill patients. Normally T cell populations that target a specific invader, or antigen, grow more abundant as a protective measure, but this was not the case in the sickest patients.

There are two types of T cells—those that directly eliminate virus-infected cells and those that coordinate a response to an invader after receiving signals from cytokines. Loss of both types has been observed in hospitalized COVID patients, but it also occurs in other respiratory infections. Diminished cell levels persist, however, for an exceptionally long time—up to weeks in some COVID patients. From research with other respiratory viruses, we know T cells can travel from the blood into infected tissues. Patients with these viruses show elevated levels of chemokines, such as CXCL9 and CXCL10, which direct T cells to infected sites. While we found plenty of chemokines in the blood of COVID patients, we did not find a similar abundance of T cells.

A number of studies have investigated the lungs of patients with severe COVID, where the virus has taken up residence. With a genetic sequencing method called single-cell RNA-seq, researchers identified several subsets of immune cells, including a sizable cluster of T cells. But this finding did not provide a full explanation. Neither these lung experiments nor autopsy studies looking at multiple organs could account for the low T cell numbers overall in the blood. It was likely these missing cells simply died, and indeed many research groups have found evidence to support this conclusion.

How then are T cells disappearing? COVID patients had an expanded number of T cells bearing receptors that indicated that the cells were susceptible to an early demise. Another possibility is that the bone marrow might not make enough of the precursor cells that give rise to T cells, which could diminish the pool of mature cells. Studies of aging and other diseases have established firm evidence that cytokines modulate the bone marrow's production of T cells. A similar connection has yet to be definitively proved in COVID-19, despite the presence of the same inflammatory cytokines. Finally, it is possible that the virus itself is directly killing T cells. Testing these competing hypotheses may lead to therapies that can enhance T cell numbers.

Many of the severe immunological manifestations seen in COVID-19—drastically elevated cytokines, inflammatory cells that infiltrate the lungs, NETs and diminished numbers of white blood cells—appear in other serious viral respiratory infections. SARS-CoV-2 presents its own special challenges. What stands out is its unprecedented spread during the presymptomatic phases and among asymptomatic people who never show symptoms.

SARS-CoV, the virus responsible for the 2003 epidemic, has a relatively late viral peak of 10 days after the onset of symptoms. MERS-CoV's viral load peaks seven to 10 days after symptoms set in. But SARS-CoV-2's viral load tops out three to five days after symptoms begin. The early peak translates to extremely high viral levels even before symptoms appear (which for most people happens four or five days after exposure). These numbers mean an infected person can spread significant amounts of virus before feeling even the smallest tickle in the throat.

The wide array of organ systems involved in COVID symptoms also seems unique among respiratory viruses. SARS-CoV-2 can cause loss of smell, brain fog, gastrointestinal problems, blood clots, cardiovascular problems and even "COVID toes." The virus can also infect neurons in the brain. Among those who recover, tissue damage can linger for months.

Never has the transition from lab bench to patient bedside proceeded as rapidly—a legacy that will remain to help counter future pandemics.

These observations may not be entirely surprising. Three cell types that make up blood vessels—endothelial cells, pericytes and vascular smooth muscle cells—wind through every tissue. All of them are studded with the ACE2 receptor, the portal through which SARS-CoV-2 enters cells. They practically lay out the welcome mat for SARS-CoV-2. Making matters worse, cytokine and bradykinin storms can damage tissue made up of these cells.

Even though the earlier coronavirus, SARS-CoV, uses the same receptor and can cause cytokine storms and ARDS, there are few reports of the sort of serious extrapulmonary injuries caused by COVID-19. The viruses are an 80 percent genetic match; it is reasonable to suspect that the other 20 percent of their genomes accounts for the differences between them. But a simpler explanation might be that SARS-CoV-2 has infected more than 6,700 times as many people as its eponymous predecessor and has done so before the eyes of the world's scientific community.

The past nine months of discovery and innovation stand as testament to the dedication of scientists and medical professionals. The research and medical communities have never been more united in their efforts—and never before has the transition from lab bench to patient bedside proceeded as rapidly as in the current climate. This legacy will remain after the success or failure of any of the hundreds of COVID-19-related treatment trials. These innovations will persist to counter future pandemics. SA

FROM OUR ARCHIVES

How to Boost Your Immunity. Claudia Wallis; July 1, 2020.

scientificamerican.com/magazine/sa

PUBLIC HEALTH

THE VERY REAL DEATH TOLL *of* COVID-19

President Trump and other conspiracy fantasists touted the fake claim that COVID death counts are exaggerated. But three kinds of evidence point to more than 250,000 deaths

By Christie Aschwanden

A PERSISTENT FALSEHOOD HAS BEEN CIRCULATING ON SOCIAL MEDIA: the number of COVID deaths is much lower than official statistics, and therefore the danger of the disease has been overblown. In August, President Donald Trump retweeted a post claiming that only 6 percent of these reported deaths were actually from COVID-19. (The tweet originated from a follower of the debunked conspiracy fantasy QAnon.) Twitter removed the post for containing false information, but fabrications such as these continue to spread. In September outgoing U.S. Representative Roger Marshall of Kansas—now incoming senator—complained that Facebook had removed a post in which he claimed that 94 percent of COVID-19 deaths reported by the Centers for Disease Control and Prevention "were the result of 2-3 additional serious illnesses and were of advanced age."

Illustration by Peter Horvath

Christie Aschwanden is a science writer in Colorado and author of *Good to Go: What the Athlete in All of Us Can Learn from the Strange Science of Recovery* (W. W. Norton, 2019).

Now some facts: Researchers know beyond a doubt that the number of COVID-19 deaths in the U.S. surpassed a quarter of a million people by November 2020. This number is supported by three lines of evidence, including death certificates. The inaccurate idea that only 6 percent of the deaths were really caused by the coronavirus is "a gross misinterpretation" of how death certificates work, says Robert Anderson, chief mortality statistician at the CDC's National Center for Health Statistics.

The scope of the coronavirus's deadly toll is clear, even if the exact toll varies by a small fraction depending on the reporting system. "We're pretty confident about the scale and order of magnitude of deaths, but we're not clear on the exact number yet," says Justin Lessler, an infectious disease epidemiologist at the Johns Hopkins Bloomberg School of Public Health. To understand why the figures converge, even if they contain some uncertainty, it is important to know how they are collected and calculated.

The first source of death data is called case surveillance. Healthcare providers are required to report cases and deaths from certain diseases, including measles, mumps and now COVID-19, to state health departments, which pass this information along to the CDC, Anderson says. The surveillance data are a kind of "quick and dirty" accounting, says Shawna Webster, executive director of the National Association for Public Health Statistics and Information Systems. States gather all the information they can on these diseases, but this is the first pass—no one has time to double-check the information or look for missing laboratory tests, she says. For that, you have to look for the next source of information: vital records.

This second line of evidence comes from the National Vital Statistics System, which records birth and death certificates. When somebody dies, a death certificate is filed in the state where the death occurred. After the records are registered at a state level, they are sent to the National Center for Health Statistics, which tracks deaths at a national level. Death certificates are not filed in the system until outstanding test results are in and the information is as complete as possible. By the time a record gets to the vital records system, "it is as close to perfect as it's going to get," Webster says.

A physician, medical examiner or coroner fills out the cause of mortality on the death certificate. That specialist is instructed to include only conditions that caused or contributed to death, Anderson says. One field lists the sequence of events leading to the death. "What we're really trying to get at is the condition or disease that started the chain of events leading to the death," Anderson says. "For COVID-19, that might be something like acute respiratory distress due to pneumonia due to COVID-19." A second part of the certificate lists other conditions that may have contributed to the death yet were not part of the sequence of events that led up to it, he says. These are called comorbidities, and although they can be contributing factors, they cannot be directly involved in the chain of cause and effect that ended in death. Medical conditions such as diabetes or heart disease are common comorbidities, and they can make a person more vulnerable to the coronavirus, Anderson says, "but the fact is: they're not dying from that preexisting condition."

"When we ask if COVID killed somebody, it means 'Did they die sooner than they would have if they didn't have the virus?'" Lessler says. Even a person with a potentially life-shortening condition such as heart disease may have lived another five, 10 or more years, had they not become infected with COVID-19.

The 6 percent number touted by Trump and QAnon comes from a weekly CDC report stating that in 6 percent of the coronavirus mortality cases it counted, COVID-19 was the only condition listed on the death certificate. That observation most likely means that those death certificates were incomplete because the certifiers gave only the underlying cause of death and not the full causal sequence that led to it, Anderson says. Even someone who does not have another health condition and dies from COVID-19 will also have comorbidities in the form of symptoms, such as respiratory failure, caused by the coronavirus. The idea that a death certificate with ailments listed in addition to COVID-19 means that the person did not really die from the virus is simply false, Anderson says.

The surveillance and vital statistics data provide a pretty good picture of how many deaths are attributable to the coronavirus, but

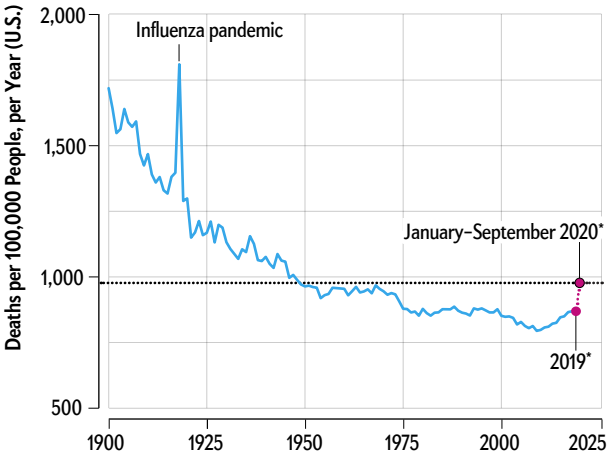
PAGE 42: TIA DUFOUR *The White House (Trump)*

COVID Has Become the Third Leading Cause of Death in the U.S.

In 2020 it killed more people than the flu and also surpassed stroke, Alzheimer's and diabetes

A Few Fateful Months Stymied Decades of Medical Gains

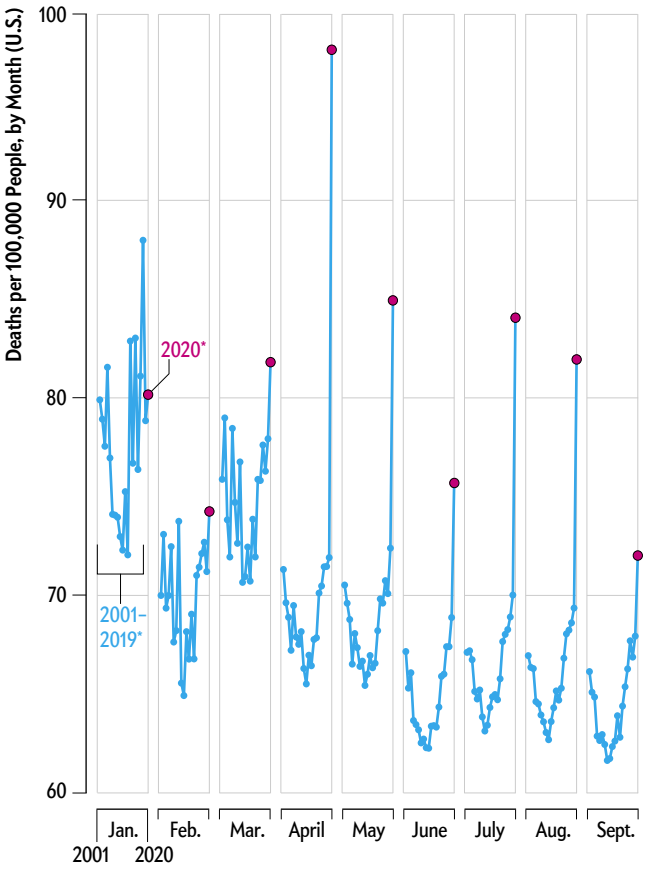
A larger share of the U.S. population died in the first nine months of 2020 than in any of the past 50 years, a period of declining mortality because of health-related improvements.



**The 2019 and 2020 figures are provisional and were likely undercounted in recent months. The rate for 2020 was calculated by annualizing the data count of January through September. Older populations die at a higher rate, and COVID-19 has been impacting such populations more than younger ones. So the age adjusted-rate for 2020 will likely be lower than the crude rate data shown here (more than 940 deaths per 100,000 people).*

April Was the Cruelest Month as COVID Became Exponential

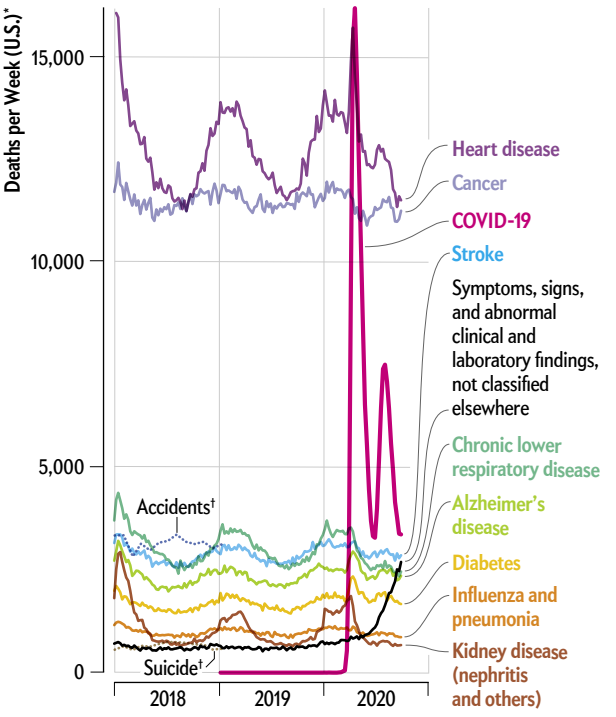
The U.S. experienced the highest monthly death rate of the past two decades in April 2020. Rates dropped in May and June, then began climbing up again in July.



*The 2019 and 2020 figures are provisional and were likely undercounted in recent months.

COVID Outpaced Seven Top Killers in 2020

This chart shows deaths per week for the top 10 causes of death—per 2017 annual rankings—plus COVID-19 and a provisional category for abnormal clinical and lab findings. (This category includes cases pending COVID-19 test confirmation and may be revised later by public health officials.) In the last two weeks of April, more Americans died from COVID-19 than from heart disease.



*The 2019 and 2020 figures are provisional and were likely undercounted in recent weeks. Deaths caused by problems with the circulatory system (including heart disease) and the respiratory system are seasonal and tend to peak in cold winter months.

† Deaths caused by accidents and suicide are rooted in monthly reports. Data are not available for 2019 and 2020.

they do not capture all of them, and that is where the final line of evidence come in: excess deaths. They are the number of deaths that occur above and beyond the historical pattern for that time period, says Steven Woolf, a physician and population health researcher at the Virginia Commonwealth University School of Medicine. In a paper published in October 2020 in *JAMA,* Woolf and his colleagues examined death records in the U.S. from March 1 through August 1 and compared them with the expected mortality numbers. They found that there was a 20 percent increase in deaths during this time period—for a total of 225,530 excess deaths—compared with previous years.

Two thirds of these cases were attributed to COVID-19 on the death certificates, and Woolf says there are two types of explanations for the rest: Some of them were COVID-19 deaths that simply were not documented as such, perhaps because the person died at home and was never tested or because the certificate was miscoded. And some of the extra deaths were probably a consequence of the pandemic yet not necessarily of the virus itself. For instance, he says, imagine a patient with chest pain who is scared to go to the hospital because he or she does not want to get the virus and then dies of a heart attack. Woolf calls this "indirect mortality." "The deaths aren't literally caused by the virus itself, but the pandemic is claiming lives," he says.

The numbers in Woolf's study come from provisional death data, the kind that the CDC has not yet checked for miscoding or other issues. What builds his confidence in these results, however, is that they have been replicated numerous times by his group and others. "All serious analyses of these data are showing that the number of deaths we're hearing on the news is an undercount," he says.

COVID-19 is now the third leading cause of death in the U.S., and the toll continues to rise as cases, hospitalizations and fatalities surge across the country. The complete number may never be known, even after the pandemic ends, but already it is a staggering number of lives cut short. SA

SOURCES: U.S. CENSUS; CENTERS FOR DISEASE CONTROL AND PREVENTION; AND NATIONAL CENTER FOR HEALTH STATISTICS

FROM OUR ARCHIVES

Eight Persistent COVID-19 Myths and Why People Believe Them. Tanya Lewis; November 2020.

scientificamerican.com/magazine/sa

Graphics by Youyou Zhou

PALEONTOLOGY

THE REAL *DILOPHOSAURUS*

The most comprehensive study of the iconic *Jurassic Park* dinosaur reveals a very different animal from the one the movie portrayed

By Matthew A. Brown and Adam D. Marsh

Illustration by Chase Stone

Matthew A. Brown is director of the Texas Vertebrate Paleontology Collections and teaches paleontology laboratory and museum methods at the University of Texas at Austin. He studies museum practices and their effect on fossils as sources of scientific data.

Adam D. Marsh is lead paleontologist at Petrified Forest National Park and a research associate at the Texas Vertebrate Paleontology Collections and the Museum of Northern Arizona. His research focuses on integrating vertebrate paleontology, stratigraphy and geochronology of early Mesozoic rocks.

THE FADING SUN BEAT DOWN ON OUR BACKS AFTER AN ALREADY LONG DAY IN THE FIELD. Exhausted, we toiled over shovels and dug with our bare hands to clear away the sand. We were in the heart of dinosaur country on the Colorado Plateau of northern Arizona, working in the middle of the Navajo Nation to determine the ages of two skeletons of *Dilophosaurus wetherilli* that had been unearthed there previously. We had spent this hot June day in 2014 hiking up and down the badlands to measure the rock beds and fill our backpacks with geologic samples. And now we had to excavate—not a new dinosaur but rather our truck, which had gotten bogged down in the sand dunes and was buried up to the axles. The life of a globe-trotting field scientist is rooted in the mundane—applying for permits, taking notes, cooking meals and washing dishes in camp, reviewing the day's data by light of the campfire—rather than the swashbuckling of the movies. We never see Indiana Jones or Alan Grant digging out a stuck pickup truck.

In the summer of 1993 dinosaurs and paleontologists exploded onto movie screens around the world. Adapted from the 1990 Michael Crichton novel, *Jurassic Park* made instant stars, and villains, of several little-known species. Names such as *Velociraptor* and *Dilophosaurus* joined *Tyrannosaurus* and *Triceratops* in the public lexicon. The dinosaurs of action movies are typically not the animals that scientists know from nature. Yet one of the elements that made the *Jurassic Park* franchise so successful (it broke box office records in 1993 and topped the charts again in the summer of 2020) was its narrative reliance on the state of the art in paleontology and genetics. Author Crichton and director Steven Spielberg brought a modern look at dinosaur science to audiences for the first time, and the image they portrayed of active, intelligent animals still resonates today.

Of course, Crichton and Spielberg took artistic liberties to tell a compelling story, dramatizing not only the scientists but also the dinosaurs. The animal that departed most from the fossil evidence was *Dilophosaurus*. In the movie, it takes the form of a golden retriever–sized creature with a rattling frill and venomous spit that kills the computer programmer–turned–dinosaur embryo smuggler, Dennis Nedry. What was *Dilophosaurus* really like?

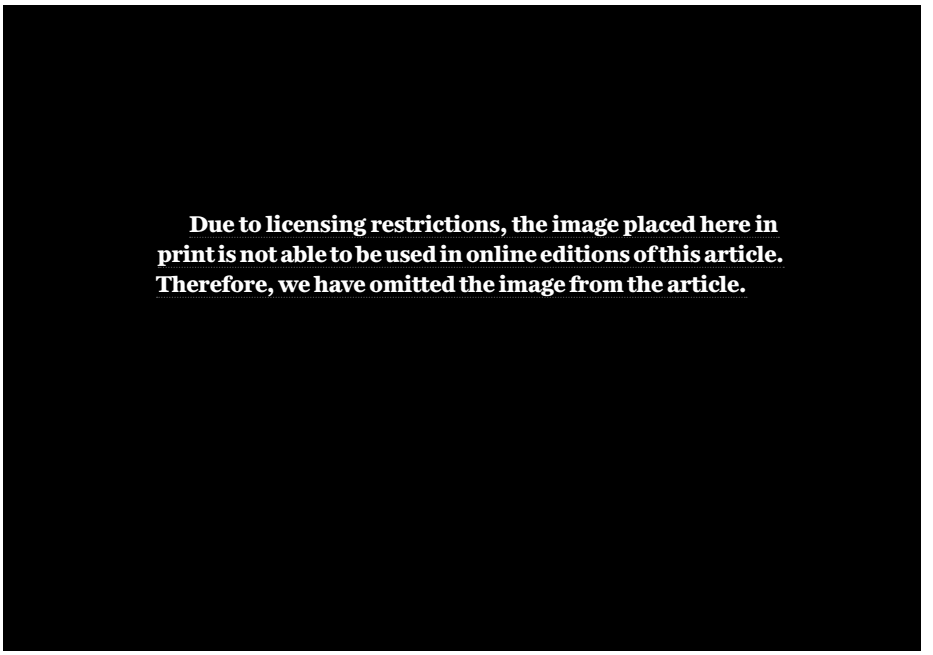
In truth, scientists did not have a complete picture of this animal back when it entered pop culture. But in the nearly three decades since *Dilophosaurus* got the Hollywood treatment, researchers have recovered significant new fossil specimens of this dinosaur and analyzed all of the remains with increasingly sophisticated methods. As a result, we can now reconstruct this dinosaur—its appearance and behavior, how it evolved, the world it inhabited—in detail. The findings show that the real *Dilophosaurus* bore little resemblance to its big-screen counterpart. They also provide the most detailed portrait yet of a dinosaur from the Early Jurassic epoch.

A STAR IS BORN

TODAY WE KNOW *Dilophosaurus* as a bipedal, meat-eating dinosaur more than 20 feet long with two distinctive parallel crests of very thin bone along the top of its head (its name derives from the Greek words for "two-crested reptile"). But in 1954, when the animal first appeared in the scientific literature, it had a different name: in a series of papers, Samuel Welles, a University of California, Berkeley, paleontologist, presented his research on two skeletons found by Jesse Williams, a Navajo man who lived near Tuba City, Ariz. The crest had not been identified among the fragmentary remains, and Welles called the creature *Megalosaurus wetherilli,* believing it to be a new species in the previously known genus *Megalosaurus*. When Welles found an additional specimen in 1964 that preserved the top of the skull, with its dual crests, he realized that the original find represented a new genus, so he renamed the animal *Dilophosaurus wetherilli*.

The basic body plan of the dinosaur in *Jurassic Park* was patterned on Welles's 1984 anatomical description and sculpted reconstructions of the bones in museum exhibits, as well as artwork by paleontologist Gregory Paul in the 1988 book *Predatory Dinosaurs of the World*. But the *Jurassic Park Dilophosaurus* departed from the scientific record of the time in several key details. Most obviously, it was depicted as half the size of the real animal. The filmmakers did this deliberately to avoid any confusion with another saurian antagonist, the *Velociraptor*.

The hallmarks of the cinematic *Dilophosaurus*—namely, its venomous saliva and collapsible frill—were also fictional traits added for dramatic effect. But these embellishments resembled the biology of other real animals, which made them believable. When Welles described the fossils of *Dilophosaurus,* he interpreted some of the joints between the tooth-bearing bones at the end of the snout as "weak" and suggested that the animals may have been scavengers or that they did most of their killing with claws on their hands and feet. When writing the story, Crichton invented a dramatic mechanism by which the animals could spit a blinding venom, based on some modern species of cobras, which can spit two meters. Inspiration for the frill, meanwhile, came from the modern-day frilled agamid lizard that lives in Australia and New Guinea. The lizard has a structure made of bone and cartilage originating from the throat that supports the frill. No evidence of such a trait has turned up in the fossil record of *Dilophosaurus.*



Other aspects of *Jurassic Park* drew from the latest science. In the early 1980s paleontologists were just starting to reach broad agreement that modern birds descended from dinosaurs and are, in fact, the last surviving dinosaur lineage. The filmmakers threw out early test animations of sinuous, snakelike velociraptors in preference of recommendations from their science adviser, dinosaur paleontologist Jack Horner, to make the animals more birdlike in their movements. The film, with its depiction of dinosaurs as quick, clever animals rather than the sluggish, more lizardlike creatures that 19th-century scholars thought them to be, was the first time many members of the general public encountered the bird-dinosaur connection.

NEW AND IMPROVED

ARTISTIC CHOICES ASIDE, scientific understanding of *Dilophosaurus* was bound to change in the years after *Jurassic Park*'s release. In the lead-up to the book and film, the field of paleontology was undergoing tremendous change. Advances in computing were revolutionizing the study of fossils, enabling researchers to process enormous data sets in ways unimaginable when *Dilophosaurus* was first discovered. Take, for instance, cladistic analysis, which identifies discrete, heritable anatomical features that can be compared between animals and that provide a statistical basis for testing hypotheses about the relationships of animals to one another. Researchers can now analyze many more characteristics much more quickly than ever before and thus develop better-supported hypotheses about how dinosaurs are related and how they evolved. Increased computing power and developments in medical and industrial CT scanning also created a nondestructive way to look inside bones and rocks at hidden anatomy.

Not only did the analytical tools available to paleontologists evolve, but in 1998 teams at the University of Texas at Austin began recovering more *Dilophosaurus* remains in the same region of northern Arizona that yielded the first finds. Every new fossil discovery can support or refute prior thinking about long-vanished organisms. In this case, the new fossils preserved parts of the *Dilophosaurus* anatomy that were missing or distorted in previously collected specimens.

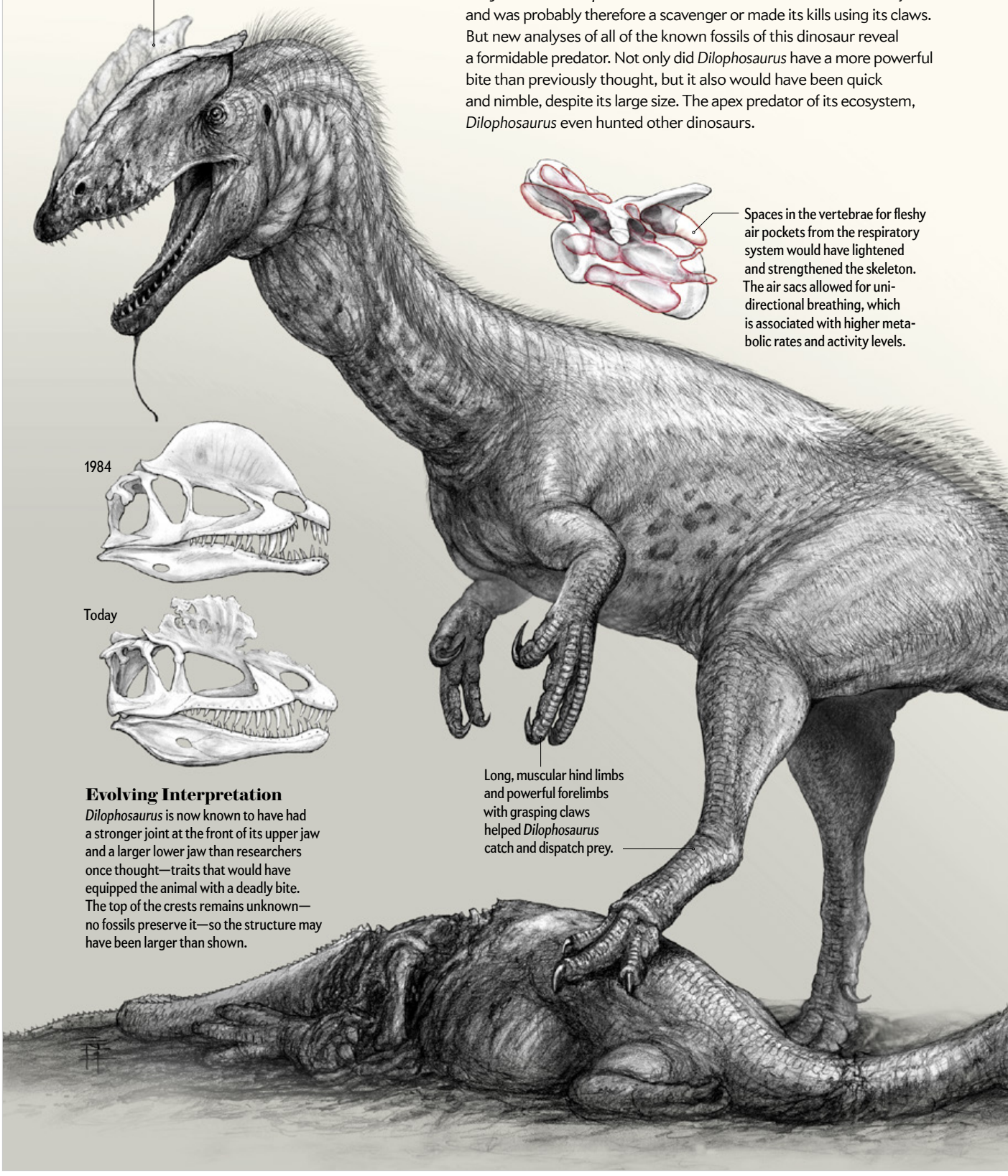
Fossils are typically collected in large blocks of rock and encased in plaster to protect them during their journey from the field to the laboratory. When they arrive in the museum, paleontologists use dental picks, chisels and miniature handheld jackhammers to carefully remove the rock and expose the fossils. After millions of years of exposure to geologic processes such as crushing and weathering, the fossils we find are most often distorted and incomplete elements. We sometimes disassemble and reconstruct broken fragments to better approximate their original condition, sculpting and adding missing material based on closely related animals.

When Wann Langston, Jr., and his colleagues prepared the first *Dilophosaurus* skeletons at U.C. Berkeley around 1950, they filled in missing skull parts with casts from the skull of a more complete carnivorous dinosaur from the Jurassic, and they sculpted missing parts of the pelvis out of plaster. No one really knew what those missing parts looked like; the reconstructions represented a hypothesis of the real form of *Dilophosaurus*—one that could be tested with new fossils.

The *Dilophosaurus* material discovered since Welles's initial description and Langston's reconstruction shows that the animal's snout and jaw were much more substantial than originally recognized. The upper jaw bones do not have the weak interface that the fragmentary first finds suggested. Instead these bones indicate a strong skull capable of biting into prey. Likewise, newly identified features of bones from the animal's lower jaw show stout ridges for muscle attachments. In modern reptiles, these ridges provide sur-

Portrait of a Predator

Early studies of *Dilophosaurus* concluded that the animal had weak jaws and was probably therefore a scavenger or made its kills using its claws. But new analyses of all of the known fossils of this dinosaur reveal a formidable predator. Not only did *Dilophosaurus* have a more powerful bite than previously thought, but it also would have been quick and nimble, despite its large size. The apex predator of its ecosystem, *Dilophosaurus* even hunted other dinosaurs.



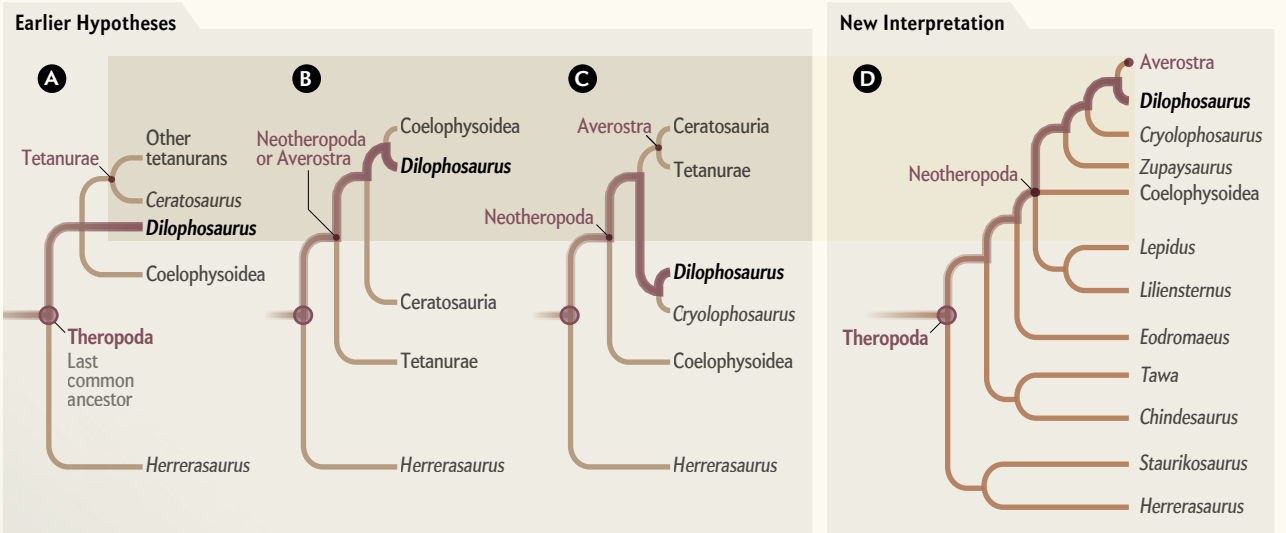
Evolving Interpretation

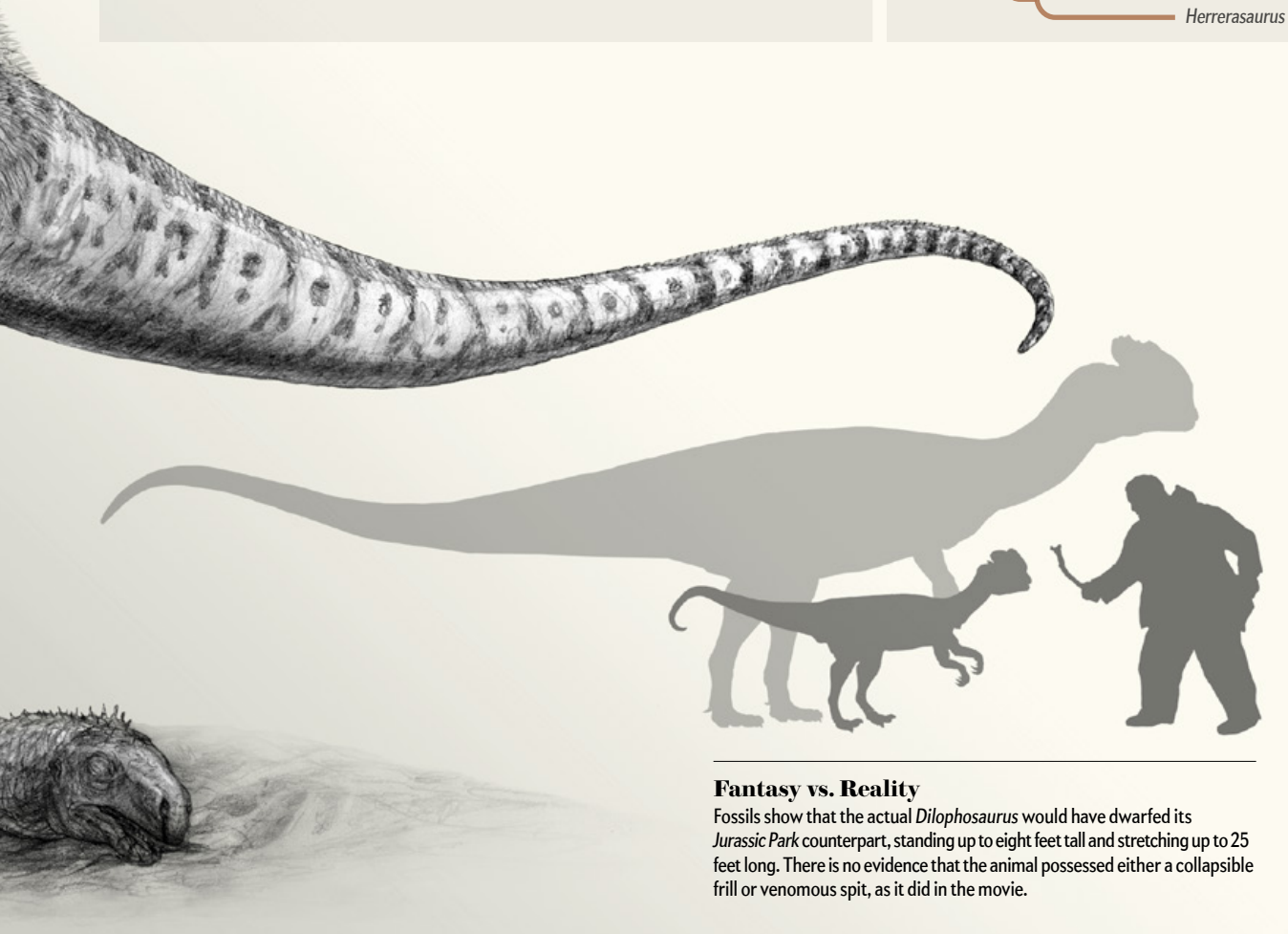
Dilophosaurus is now known to have had a stronger joint at the front of its upper jaw and a larger lower jaw than researchers once thought—traits that would have equipped the animal with a deadly bite. The top of the crests remains unknown—no fossils preserve it—so the structure may have been larger than shown.

Illustrations by Brian Engh (reconstructions) and Daisy Chung (family trees)

Redrawing the Tree

The question of how *Dilophosaurus* is related to other dinosaurs has long vexed paleontologists. Previous studies concluded that it was most closely related to coelophysoids A, ceratosaurs B or other crested theropods C. Samuel Welles, who published the first descriptions of *Dilophosaurus* fossils, suggested that the remains might even belong to more than one genus of dinosaur. The latest, most comprehensive analysis of the anatomical characteristics of *Dilophosaurus* indicates that the fossils belong to a single species. Comparing its traits with those of other dinosaurs from around the world reveals that *Dilophosaurus* is closest to large Jurassic theropods D. But the wide evolutionary gap between these two groups implies that closer relatives of *Dilophosaurus* remain to be found.





Fantasy vs. Reality

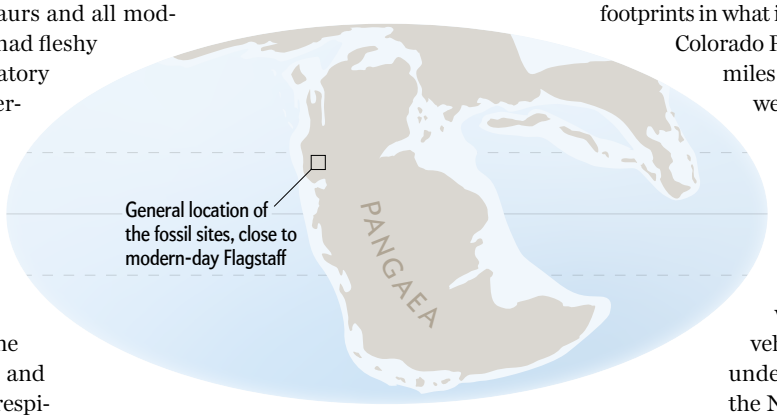
Fossils show that the actual *Dilophosaurus* would have dwarfed its *Jurassic Park* counterpart, standing up to eight feet tall and stretching up to 25 feet long. There is no evidence that the animal possessed either a collapsible frill or venomous spit, as it did in the movie.

SOURCE: "A COMPREHENSIVE ANATOMICAL AND PHYLOGENETIC EVALUATION OF *DILOPHOSAURUS WETHERILLI* (DINOSAURIA, THEROPODA) WITH DESCRIPTIONS OF NEW SPECIMENS FROM THE KAYENTA FORMATION OF NORTHERN ARIZONA," BY ADAM D. MARSH AND TIMOTHY B. ROWE, IN *JOURNAL OF PALEONTOLOGY*, VOL. 94; JULY 2020 (*family trees*)

face area for the attachment of large muscles. And the skeleton of a different dinosaur found at the U.T. Austin dig site—the plant-eating *Sarahsaurus*—features bite marks, attesting to the presence of a large meat-eating animal with jaws strong enough to puncture bone. Together this evidence supports the idea that *Dilophosaurus* was probably a predator with a deadly bite rather than a creature that had to scavenge or use its claws to kill, as Welles supposed.

Dilophosaurus was a large dinosaur, especially for its time. Most of the dinosaurs of the Late Triassic of western North America, just 20 million years earlier, were animals the size of turkeys or eagles, but *Dilophosaurus* would have towered over a human, standing up to eight feet tall and measuring up to 25 feet long when fully grown. It had much longer and stronger arms than other larger meat-eating dinosaurs such as *Allosaurus* and *Ceratosaurus,* and its legs were relatively longer as well. When the first skeletons of *Dilophosaurus* were found, scientists thought the species was related to the so-called carnosaurs *Allosaurus* and *Streptospondylus,* so they reconstructed the missing parts of the pelvis to look like they did in those animals. The better-preserved *Dilophosaurus* skeletons found later show more intermediate pelvis anatomy between *Coelophysis*-like and *Allosaurus*-like animals from the Late Triassic and Late Jurassic, respectively.

Like many early dinosaurs and all modern birds, *Dilophosaurus* had fleshy air pockets from its respiratory system growing into its vertebrae, which provided strength while simultaneously lightening the skeleton. These air sacs allowed for the unidirectional flow of air through the lungs—in other words, the entire cycle occurs in one breath, as it does in birds and crocodilians. This type of respiration provides the animal with more oxygen than does the bidirectional respiratory system that mammals have, in which air flows both in and out of the lungs. Animals that breathe unidirectionally tend to have relatively high rates of metabolism and thus high activity levels, so *Dilophosaurus* was probably a fast, agile hunter.



FOSSILS of *Dilophosaurus* all come from two sites in what is now northern Arizona that date to the Early Jurassic, shortly after the supercontinent Pangaea began to break apart.

CT imaging has revealed that these air sacs are also present in the bones surrounding the dinosaur's brain and were continuous with the sinus cavities in the front of the skull. In most meat-eating dinosaurs, a ridge of bone provides a roof over an opening in the skull in front of the eye sockets known as the antorbital fenestra. But in *Dilophosaurus,* this opening is continuous with the side of the dinosaur's unique crests, suggesting that the crests, too, had air sacs. The crests were almost certainly covered by keratin, the same material that forms horns, claws and hair, and may have played a role in helping members of this species identify one another or attract mates. But how the air sacs might have supported these or other functions of the crests is unclear.

One of the challenges of studying the evolutionary history of any species is understanding physical variation within and among taxonomic groups. Welles thought the various skeletons we now categorize as *Dilophosaurus* actually represented multiple genera. Taking advantage of the latest cladistics tools, one of us (Marsh) tested that hypothesis by identifying hundreds of anatomical features present on each individual skeleton and comparing them with one another. The results of this statistical analysis show that, contrary to what Welles surmised, all of the animals are so similar that they must represent not only one genus but one species.

Marsh also incorporated these anatomical characteristics into a much larger data set that compares *Dilophosaurus* with other specimens from around the world. This process elucidates the early evolutionary history and biogeographical distribution of dinosaur groups and has more precisely located *Dilophosaurus* on the tree of life. We now know that the evolutionary gap between *Dilophosaurus* and its closest known relatives is significant, implying that many other, closer, relatives remain to be discovered.

CONTEXT CLUES

JUST AS OUR CONCEPTION of *Dilophosaurus* the animal has grown more detailed, so, too, has our understanding of the world it lived in. The hike down the Adeii Eichii Cliffs to the *Dilophosaurus* quarry is a journey back through 183 million years to the Early Jurassic. Back then, dinosaurs roamed the landscape, leaving footprints in what is now sandstone across the Colorado Plateau. Paved surfaces end miles from the rock outcrop, so we drive on overgrown rutted two-tracks that cross the loose sandy dune fields that show up on our geologic maps as "QAL"—Quaternary alluvium. This windblown sand is what stranded our field vehicles in 2014. The bedrock under these modern dunes is the Navajo Sandstone, the lithified remains of a 180-million-year-old desert. The red rock badlands of Ward Terrace, as this area is known, spill out to the western horizon, where they meet the much younger volcanic San Francisco Peaks of Flagstaff, Ariz. To the northwest is the mouth of one of the world's most visited geologic features, the Grand Canyon.

From the sands that trapped our pickup atop Ward Terrace to the Vishnu schist—the black rock at the bottom of the canyon that is being carved away by the Colorado River—these landscapes preserve much of the past 1.8 billion years of the rock record. As paleontologists, we work to understand the life entombed in those rocks, and we use lines of geologic and biological evidence preserved within them to reconstruct the environments of deep time.

One of our objectives was to more precisely determine the age of the rock in which *Dilophosaurus* is found, known as the Kayenta Formation. This rock was laid down by rivers, lakes and streams east of a volcanic arc that was depositing ash and fine-grained particles into the area. The ash helped to both preserve the bones of *Dilophosaurus* and aid early efforts to date the Kayenta Formation. We collected new rock samples to date using radiometric methods. We processed the samples by grinding and

SOURCE: CHRISTOPHER R. SCOTESE (*paleogeographic map reference*)

Map by Daisy Chung

extracting zircon crystals, which can preserve unstable isotopes of uranium. The uranium isotope decays into lead at a constant rate, and when we vaporize the crystals with a laser and analyze them with a mass spectrometer, the relative quantities of uranium and lead we measure indicate when the rock layers were laid down. In the case of this *Dilophosaurus* site, it was around 183 million years ago, plus or minus a few million years.

Dilophosaurus thus lived during the Early Jurassic epoch, approximately five million to 15 million years after the end-Triassic mass extinction that resulted in the loss of roughly three quarters of life on Earth, including most of the large reptiles that competed for resources with the early dinosaurs. The mass extinction was probably triggered by the initial breakup of the supercontinent Pangaea as the northern Atlantic Ocean opened up like a volcanic zipper. Throughout the Late Triassic and Early Jurassic, the North American tectonic plate traveled northward from a subtropical climate belt into an arid climate belt, so the location that *Dilophosaurus* lived in moved from the approximate latitude of modern-day Costa Rica to modern-day northern Mexico. As such, the environment that deposited the Kayenta Formation was seasonally dry, with sand dunes migrating in and out of wetter environments where animals flourished.

Fossils of other organisms found in the Kayenta Formation reveal how *Dilophosaurus* fit into the ecosystem. It was the apex predator in the river oasis it inhabited, a conifer-lined waterway through a sea of sand. One specimen housed at U.T. Austin was found in the same quarry as two individuals of the long-necked herbivore *Sarahsaurus*. These dinosaurs lived alongside a smaller meat-eating dinosaur called *Megapnosaurus* and a small armored dinosaur called *Scutellosaurus*. The most common animal found in the Kayenta Formation is the early turtle *Kayentachelys,* which swam alongside heavily scaled bony fish, freshwater coelacanths and lungfish. Early mammal relatives, including the beaverlike tritylodontids and ratlike morganucodontids, were also potential prey for *Dilophosaurus*.

FOSSILS FOR ALL

IN THE FOSSIL EXCAVATION depicted in *Jurassic Park,* a complete *Velociraptor* skeleton comes to light with some gentle brushing. In the real world, dinosaur fossils are typically found as broken, barely identifiable fragments. On a lucky day, a mostly complete bone might turn up. With the publication last summer of Marsh's comprehensive anatomical study, *Dilophosaurus* has become the best-documented Early Jurassic dinosaur from anywhere in the world. But it took decades to find additional remains that filled in the unknown anatomy of the animal. And it took successive generations of paleontologists to interpret the bones.

Museums play a vital role in facilitating such efforts. The public's conception of museums is a dramatically lit exhibit gallery, but the major function of a natural history museum is to conduct research into the natural world. To that end, these institutions build large collections of specimens to serve as the evidence for scientific research. Teams of specially trained conservators, archivists and collection managers carefully document and preserve the specimens, with the goal of making the collections accessible to researchers in perpetuity. Repeatability is a cornerstone principle of scientific research; other scientists must be able to corroborate our findings. In paleontology, that means that the fossils themselves must be preserved in a museum, so that future generations of scientists can revisit the specimens and double-check observations.

The Navajo Nation has partnered with museums that care for those fossils to preserve not just the bones themselves but all the archives and data associated with them. In 2015, when we went to relocate the original *Dilophosaurus* discovery site for this research, we were lucky to meet John Willie, a relative of Jesse Williams, the Navajo man who found the first bones in 1940. Willie walked us to the site and explained that the natural resources unique to the Navajo Nation are extremely important to the Diné (Navajo People). The Navajo Nation is one of the best places in the world to see terrestrial rocks from the Early Mesozoic era, and its Minerals Department has been active in facilitating scientific research, including approving permits for fieldwork and loans of fossils and reviewing scientific manuscripts.

Scientific understanding comes from building on and reevaluating prior knowledge and sometimes overturning old notions. It is exciting when this hard-won information filters into pop culture. Paleontology has close ties with cinema going to the dawn of animation. Winsor McCay's 1914 *Gertie the Dinosaur* opens with the animator and a group of friends visiting the American Museum of Natural History in New York City, looking at the skeleton of a sauropod dinosaur. McCay bets his party that he can bring the animal to life; the result is the first dinosaur to appear on film. McCay consulted with paleontologists at the museum for guidance on his reconstruction of Gertie. Later Barnum Brown, the discoverer of *Tyrannosaurus rex,* provided expertise to Walt Disney during the production of its 1940 animated film *Fantasia*. And the studio behind the 1954 *Godzilla* found inspiration for the design of its monster in the dinosaurs that appeared in a 1947 mural by Rudolph Zallinger entitled *The Age of Reptiles,* housed at the Yale Peabody Museum. With the *Jurassic Park* film franchise set to release its sixth installment in 2022, we look forward to seeing how paleontology is represented.

Dilophosaurus has become the best-documented Early Jurassic dinosaur from anywhere.

Incidentally, the reverse is also true. Pop culture filters into science, sometimes literally. Langston once recounted that while repairing fossils at U.C. Berkeley in the 1930s and 1940s, the paleontologists would dissolve cellulose acetate film strips in acetone to make glue rather than buy the more expensive Duco Cement. So, yes, *Dilophosaurus* is in the movies. But perhaps there is a little bit of the movies in *Dilophosaurus,* too. SA

AUTHORS' NOTE: Fieldwork on the Navajo Nation was conducted under a permit from the Navajo Nation Minerals Department. Any persons wishing to conduct geologic investigations on the Navajo Nation must first apply for and receive a permit from the Navajo Nation Minerals Department, P.O. Box 1910, Window Rock, Arizona 86515, and telephone no.: (928) 871-6587.

FROM OUR ARCHIVES

What Killed the Dinosaurs. Stephen Brusatte; December 2015.

scientificamerican.com/magazine/sa

HIMALAYAS loom large in Nepal.

NATURAL RESOURCES

PEAK WATER

Data retrieved from Earth's highest mountains show that the water supply to two billion people is changing

By Walter Immerzeel

The nights are long inside a tent 5,300 meters above sea level at the snout of

Nepal's Yala Glacier. At 8:00 P.M., after a meal of Nepali *dal bhat* (lentils and rice), the 10 members of our expedition take refuge against the cold in sleeping bags inside the small tents that make up our temporary camp. Falling asleep is tough because the low oxygen concentration fools our bodies into increasing their heart rates. As a consequence, I spend many overnight hours listening to distant sounds of thundering avalanches and cracking ice, contemplating whether to leave the sleeping bag to pee outside and what not to forget the next day. As soon as the sun rises, the camp is bustling, and we are on our way up the steep glacier to install special instruments at 5,600 meters.

Walter Immerzeel is a physical geography professor at Utrecht University in the Netherlands, where he leads the mountain hydrology group. He has lived in Nepal and has been measuring snow and ice high in the Himalayas since 2002.

Our team, which includes colleagues from the International Center for Integrated Mountain Development in Nepal, has been conducting field expeditions biannually in this place, called the Langtang catchment, since 2012. We have erected automated weather stations at the base camp and at higher elevations that measure precipitation, snow depth, radiation, temperature, relative humidity and wind, making Langtang one of the best-monitored high-altitude catchments in Asia. We need to visit the stations every six months to maintain the instruments and to download their data; there is no cellular network to transmit readings automatically, and the mountains tend to block satellite signals. On the current ascent we will mount new sensors on a metal frame three meters high that we will drill into the ice. The sensors will measure sublimation—the phase transition of ice directly to water vapor—by sampling temperature and vapor 10 times a second.

These expeditions help us gather the information necessary to understand the high-altitude water cycle: snow falls on mountaintops and gradually turns into glacier ice, which slowly flows downhill and melts. The water cascades down into growing rivers that supply numerous high-elevation settlements, as well as agricultural terraces, hydropower stations, forests, valley agriculture fields, and large cities and industries below. The high-altitude water cycle was largely a mystery when we started. We did not know how much rain and snow actually falls, how much water flows into and out of the snowpack, or why glaciers blanketed in detritus eroded from surrounding slopes seem to melt as fast as their debris-free counterparts. We need to know these details to determine how much water ultimately flows out of the snowpack and glaciers and how the volume and timing of flow may change in the future.

The Langtang catchment is a small river basin that drains several mountain peaks and glaciers. It feeds the Trishuli River, an important water source for recently constructed hydropower dams halfway down the mountains and the irrigated fields farther downstream. The amassing flow leads to the Ganges Delta hundreds of kilometers away, which provides water to 400 million people as it empties into the Bay of Bengal east of India. There are hundreds of catchments like Langtang across the Himalayas.

The same dynamics play out across many other mountain ranges, such as the Alps, the Andes and the Rockies. A study we published in *Nature* in December 2019 revealed that 78 of these "water towers," from the Tarim in China to La Puna in Peru, supply the bulk of fresh water for almost two billion people globally. Computer models show that climate change could threaten these vital water supplies. Changes in temperature, precipitation patterns, the accumulation and melting of snow, and the distribution of airborne particulates all help to determine how much water descends. Yet most plans and policies related to climate change and sustainability ignore the role of mountain water. Now that we have systematically studied the world's top water towers, that may begin to change.

MONSOON DOMINANCE

MOUNTAINS FUNCTION like water towers because it rains and snows more at higher elevation than it does in lower surrounding terrain and because much of the precipitation is stored temporarily as snow and glacier ice. This snow and ice melts slowly and steadily, providing a reliable, predictable supply of water and acting as a natural buffer against dry spells.

The water stored high in Nepal's mountains is crucial for the people who live below because monsoon winds between June and September provide 70 to 80 percent of the Himalayas' annual precipitation. Data from our weather stations, in conjunction with weather models, reveal how the monsoons interact with the mountains. Even across a small catchment such as Langtang, the rains vary strikingly. The valley runs east to west, and its elevation rises in that direction. Much of the warm, moist air precipitates between the entrance of the valley, at around 1,350 meters, and the village of Lama Hotel at 2,480 meters. We have measured around 250 centimeters of annual rainfall at this site, the wettest in the valley. The village of Kyanjin, at 3,900 meters, is only another 15 kilometers to the east, but there we measure around 80 centimeters of annual rainfall—more than three times as dry yet so nearby.

The position and shape of the mountains create other local effects. If we ascend from Kyanjin to the Yala Glacier at 5,300 meters, precipitation increases again by 40 percent on average.

PRECEDING PAGES: SUSAN HALE THOMAS

SCIENTISTS INSTALL a weather station at 5,600 meters on Yala Glacier in Nepal's Himalayas to help determine how snow accumulates and thaws. Meltwater beginning as trickles flows downhill, collecting into increasingly large rivers that supply water to farms, hydropower dams and millions of people on the way to the Ganges Delta and Bay of Bengal.

USMAR HELLEMAN

Together the large, medium and valley-scale processes shape the distribution of rain and snow throughout the region. If most precipitation falls as rain at lower altitudes, for example, the rivers below will flow differently than they would if most precipitation fell as high-altitude snow.

To understand the dynamics of water towers, we have extensively investigated the amount of water stored in the snowpack close to Yala Glacier. This amount, known as the snow-water equivalent, is influenced by how much snow falls, how much melts and refreezes, how much sublimates and how much is distributed by wind. Our instruments at the 5,300-meter base camp measure many of these kinds of variables. The conditions can be severe: extreme cold has caused batteries to explode, high winds have twisted sensors, and avalanches have knocked over the scaffolding holding our instruments.

It is particularly tricky to measure sublimation, a turbulent process that occurs under cold, sunny, windy and dry conditions, which are common during the Himalayan winter. Instruments such as the ones we erected at 5,600 meters have quantified sublimation by measuring variations in air humidity and temperature. We find that at windy, exposed locations, about 21 percent of the snowfall never ends up in a river because it sublimates into the atmosphere. This phenomenon suggests that even at temperatures well below zero degrees Celsius, the snowpack can shrink.

We have also found that when the air temperature two meters above the snow is below zero, there is enough energy on the snow's surface to melt it. The energy is the net result of the shortwave radiation from the sun, the longwave radiation emitted by the surface and the atmosphere, and turbulent heat fluxes. There are interesting dynamics inside the snowpack as well. At least 30 percent of the snow that melts into water during the day refreezes at night. Much more energy is required to melt snowpack than what would be expected purely on the basis of its mass.

Another instrument, which we install above the snow, measures changes in gamma radiation as a proxy for snow-water equivalent. Rock under the snow naturally emits gamma rays, and the degree to which their signal is attenuated is related to how much water is actually stored in the snowpack.

Sometimes we hit on unexpected ways to collect information. About eight years ago a colleague working in Tasmania sent

Map by Mapping Specialists

Mountains That Water the World

Ice, snow and rainfall on 78 regional mountain chains, or "water towers," deliver water to almost two billion people and surrounding ecosystems. Ranking the towers on each continent shows which ones are highly important—providing large amounts of water to many people. The most vital towers also turn out to be the most vulnerable, stressed by climate change, rising population and economic growth, suggesting that actions are needed to protect supply and ease demand.

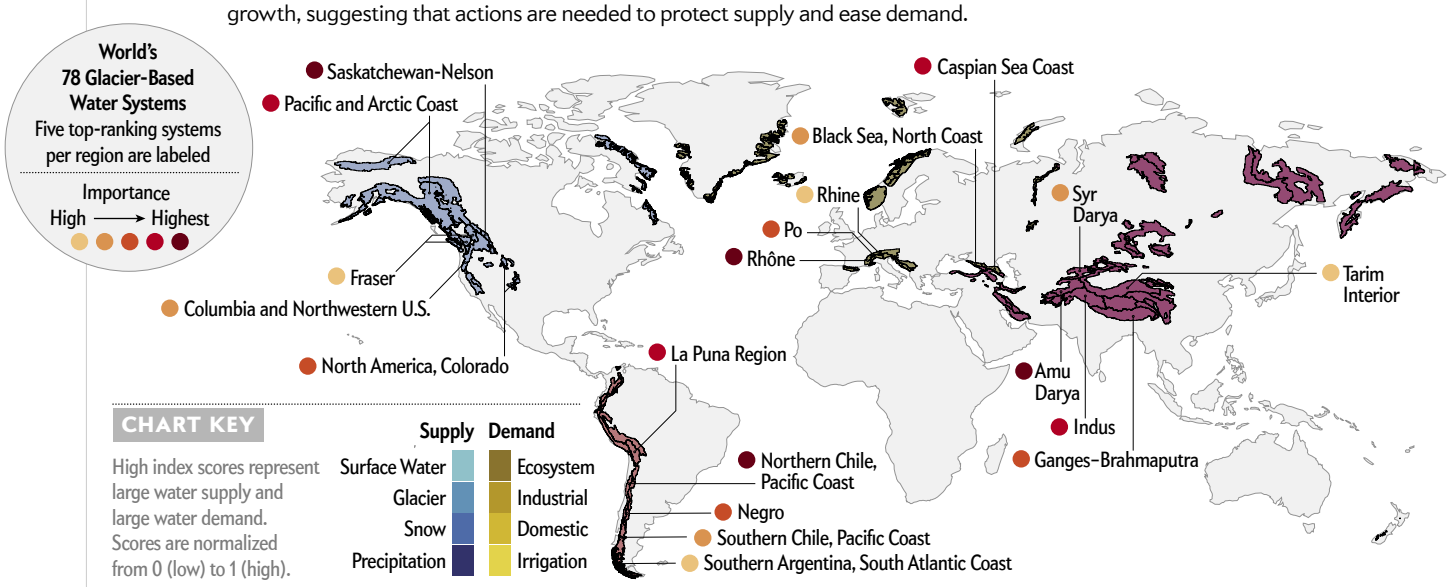
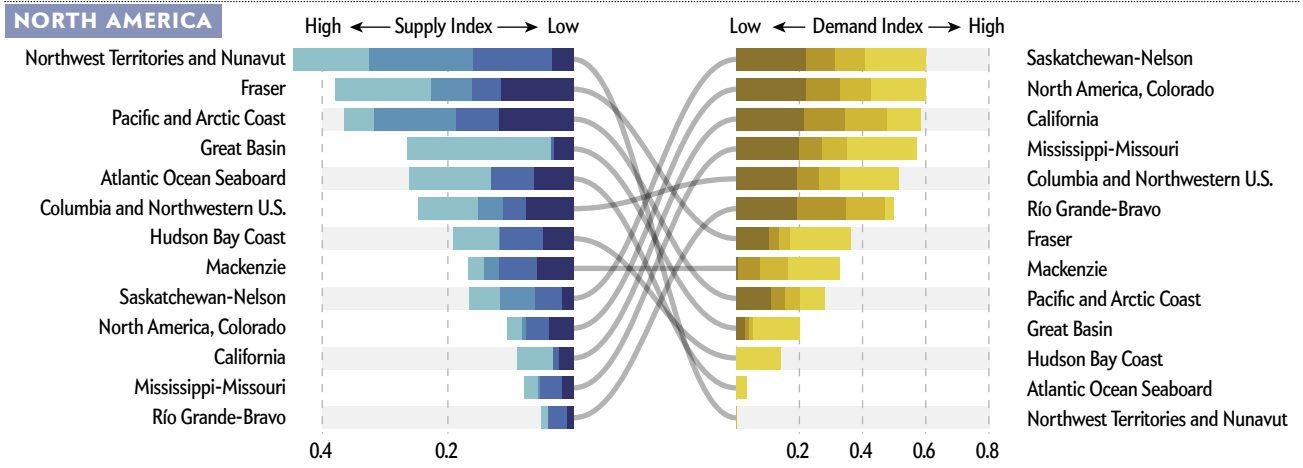


CHART KEY

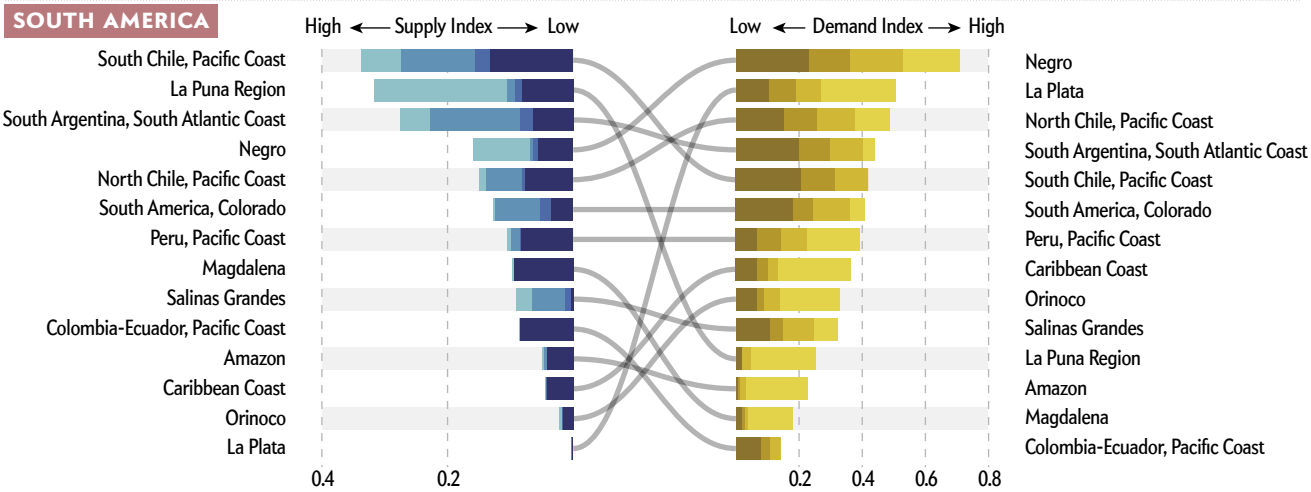
High index scores represent large water supply and large water demand. Scores are normalized from 0 (low) to 1 (high).

Supply	Demand
Surface Water	Ecosystem
Glacier	Industrial
Snow	Domestic
Precipitation	Irrigation

NORTH AMERICA

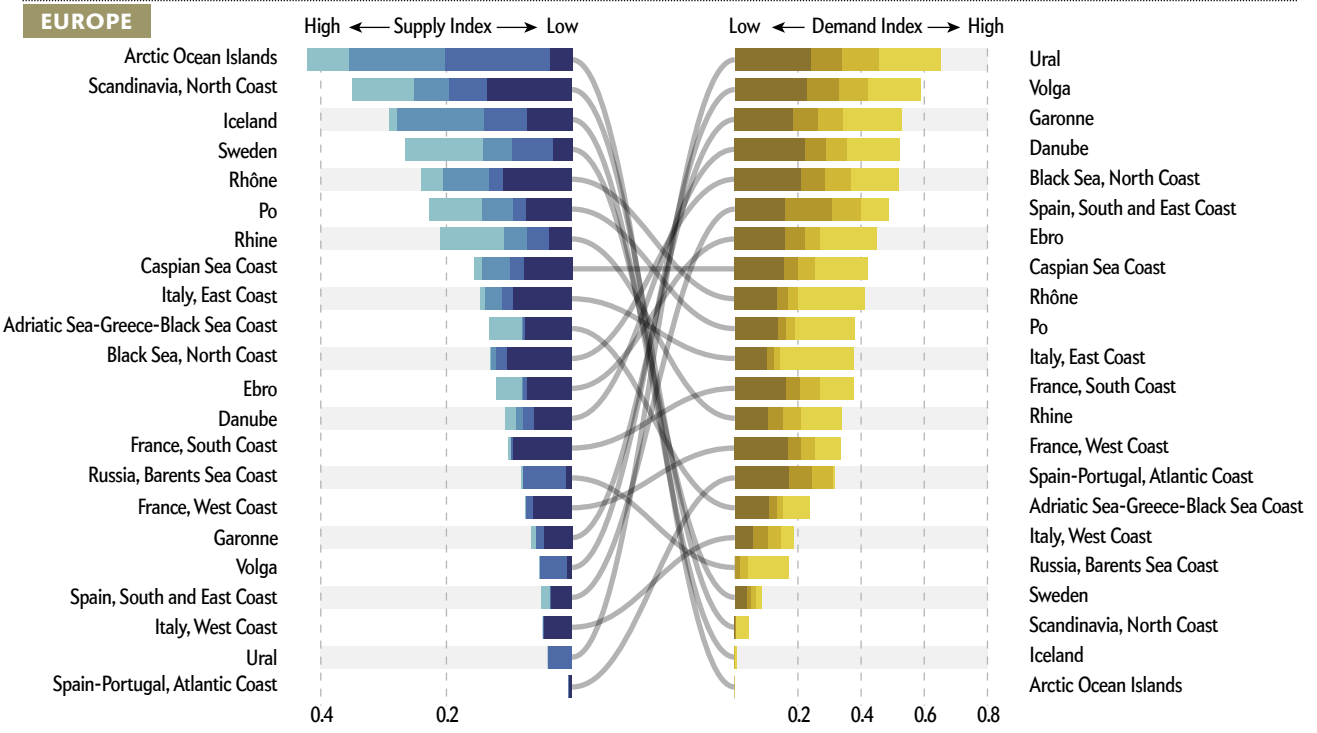


SOUTH AMERICA

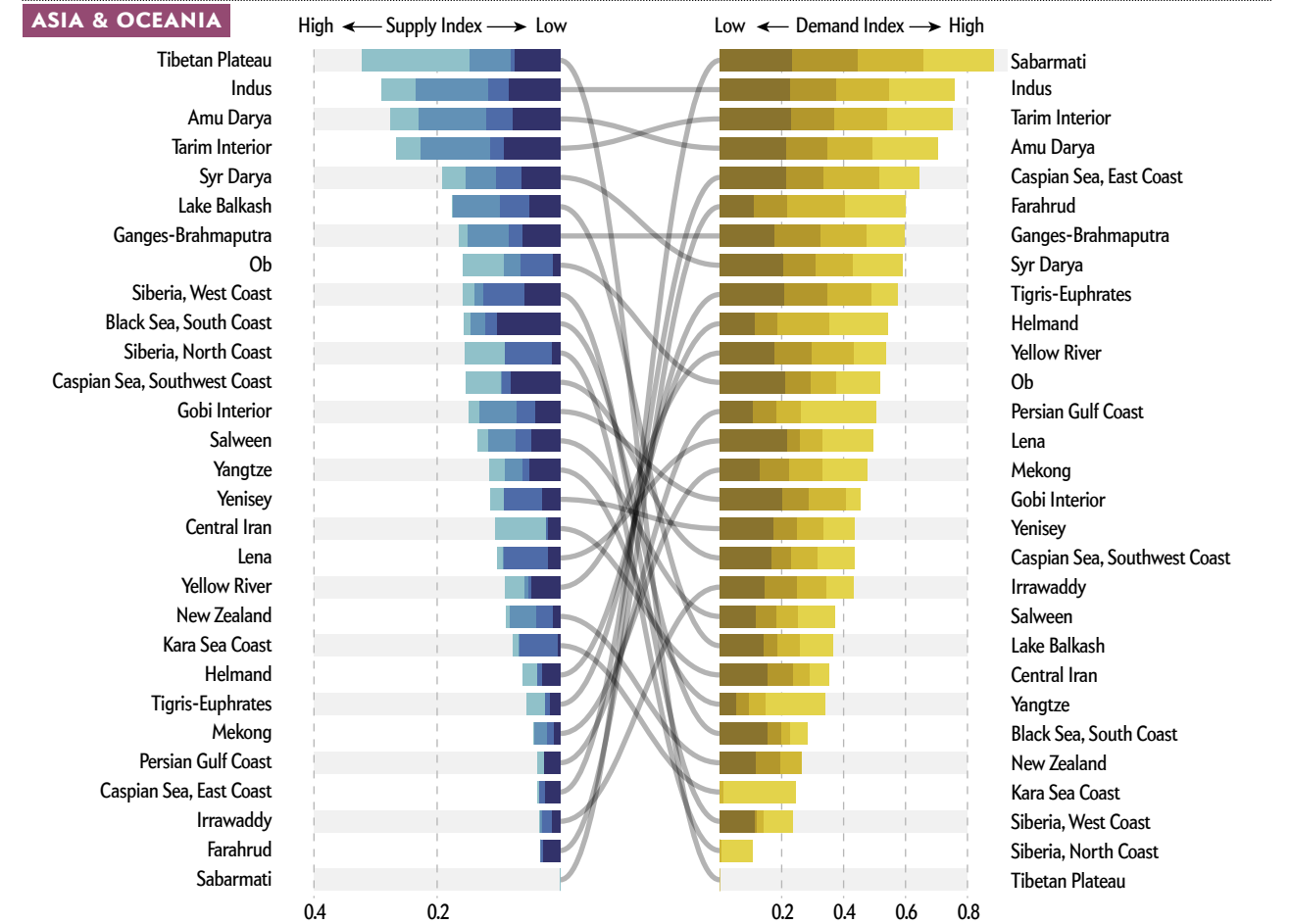


Graphic by Pitch Interactive

EUROPE



ASIA & OCEANIA



SOURCE: "IMPORTANCE AND VULNERABILITY OF THE WORLD'S WATER TOWERS," BY W. W. IMMERZEEL ET AL., IN *NATURE*, VOL. 577; JANUARY 2020

drones up over a landslide there and came back with intriguing data about the landslide's volume and velocity. We realized we could use the same approach to obtain data on glaciers covered in debris, which are hard to access. Our first outing was in 2013 on the glacier tongue just above Kyanjin.

The drone takes overlapping pictures of the glacier's surface. Software determines the surface elevation to a remarkable resolution of about 10 centimeters. We repeated the surveys every six months or so until 2019. We found that the glacier's edge is receding by about 40 meters a year, that the surface is thinning by about 80 centimeters a year and that the dwindling ice hardly flows anymore. It will not be long until the cold mass no longer qualifies as a glacier. Instead it will be a piece of dead ice slowly withering away like a heap of dirty snow plowed to the end of a parking lot. Theoretically, a debris-covered glacier should melt much more slowly than a debris-free glacier at the same altitude because the debris acts as insulation. But we identified hotspots of melt that amplified the overall process. We would never have found them with traditional satellite imagery because its resolution is too coarse.

We put all these puzzle pieces into our models to learn how much water will flow in the rivers in the future. One last big piece, though, is the amount of water flowing in the rivers today. The water height alone cannot indicate the volume of flow. We need a so-called rating curve—a relation between the water level and the river's discharge. And this curve has to represent both high monsoon flows crashing downhill and meager winter runoff. Generating reliable data is a challenge here, too. In the rivers, we either lodge a pressure transducer housed in a steel pipe at the bottom of the river or mount radar sensors on frames jutting up a few meters above the river's surface. We also throw salt into the river upstream of the gauges and measure changes in electrical conductivity at the gauge site; the extent of salt dilution can help us determine the discharge. Even though the conditions are more hospitable here than at 5,300 meters, sensors get washed away by monsoon floods. But after years of effort, we now have a fairly good idea of water flow.

AVALANCHES AND FLOODS

FOR SEVERAL YEARS we have been integrating findings from various catchments into a model that describes all the processes influencing Himalayan water flow; other scientists are doing the same in other mountain regions worldwide. Climate change presents some conspiring factors. One is elevation-dependent warming, in which mountains warm faster than lower-lying plains because of atmospheric feedbacks such as cloud formation, increased humidity and higher albedo as snowpacks retreat. Global warming of 1.5 degrees C means 2.1 degrees C of warming in the Himalayas.

Another factor is seasonality. A warmer atmosphere holds more moisture, which leads to more mountain precipitation. And more of it falls as rain than as snow, landing on rocky surfaces that were previously covered by ice, quickly running into rivers. According to research published last July, most models predict a wetter climate, although conditions can vary widely in the region. Compared with glaciers in a steady state, melting glaciers will provide more river water in the short term but less in the long term as glaciers retreat uphill and ice thins. We estimate that Langtang will hit peak water supply around 2060; after that, the supply will drop steadily.

USMAR HELLEMAN

BASE CAMP on Yala Glacier, at 5,300 meters, is the starting point for treks every six months to set up equipment and retrieve data from instruments scattered across the mountains. Researchers, including the author (*in blue jacket on frame*), also have to maintain sensors that are wracked by extreme temperatures and winds.

WALTER IMMERZEEL

We will achieve even greater understanding as we erect more sensors, making the observational network denser, particularly at high altitude, and as we integrate the data with extremely detailed models. Satellite remote sensing can also help us better estimate precipitation patterns between sensors across the basin, allowing us to fine-tune our models. Other research teams are making similar progress in mountainous terrains. Abundant data sets are available for the Alps, for example, and for the Andes. Coverage is getting better in the Himalayas as researchers at Kathmandu University and Tribhuvan University shift their attention to higher ground, taking measurements across the Annapurna and Everest mountain ranges.

My team has analyzed supply worldwide by using hydrological simulations, too. Our *Nature* study ranks mountain water towers worldwide. We consider a water tower "important" if it is rich in glaciers, snow or lakes and if water demand is great from people downstream. Significant water towers include the Colorado, the Fraser in western Canada and the Negro in Argentina, as well as the European Alps feeding the Rhine and Po Rivers.

Our modeling shows that Asia's ranges, which feed major rivers such as the Amu Darya and Indus, are the most important in the world. They are also among the most vulnerable: the models project strong rates of warming there, as well as rapidly growing populations and economies that will increase water demand tremendously. Average water availability is not likely to decrease until midcentury, in part because of greater monsoon rainfall, but the longer-term forecast is grim. We predict that 50 to 60 percent of the ice volume will be lost by the end of the century unless the world radically reduces it greenhouse gas emissions.

The big challenge for high-mountain Asia in the short term will be coping with changes in the timing of river flow and with natural hazards. In some basins, snow melt may start several weeks earlier than before, requiring farmers to change crops or sowing schedules. With snowpack providing less of a buffer, swollen rivers will increase across a region that is already facing heavy flooding every year.

Extreme rainfall in the mountains is also causing more landslides, particularly during monsoons. Greater melting is filling glacial lakes to the brim, causing disastrous floods when rocky ridges that dam the lakes burst because of the immense water pressure behind them. In the past two decades natural disasters such as avalanches, landslides and sudden floods have caused thousands of casualties and billions of dollars' worth of economic damage. Future increases in extreme rainfall and warming will exacerbate these hazards. Damages will rise, too, as growing populations build towns and hydropower dams at increasingly high altitudes.

Although these overall trends are clear, each region must be studied in detail to provide people there with useful information. One anomaly is a Central Asian region connecting the eastern Pamir and Karakoram ranges with the western Kunlun Shan. Glaciers there are stable or even gaining mass, which we see almost nowhere else on the planet. Data collected within the past year or so reveal that greater farming and irrigation in the nearby Tarim Basin may play a role. Irrigation water withdrawn from groundwater and surface sources evaporates into the atmosphere, and transpiration by crops adds even more moisture. This vapor condenses over the mountains and falls as snow—a critical reminder that human actions can alter natural systems.

MOUNTAIN POLICIES

RESEARCH ON THE high-altitude water cycle is already creating awareness of the importance of mountain water to billions of people worldwide. Officials should start acting now to safeguard it.

The first step is to include mountains in broader discussions about preserving Earth's natural resources. Locally, leaders can create national parks to protect peaks from development. They can set policies to reduce emissions of pollution and black carbon to lessen airborne debris. And they can build reservoirs to store rainwater and snowpack that melts rapidly in the spring—as long as they analyze how a structure's size and effects on water flow could interfere with ecosystems. An excellent example comes from Langtang Valley. The upper village had no electricity until two years ago, when a nongovernmental organization and the community built a hydropower plant that now provides electricity, and therefore Internet, to villagers.

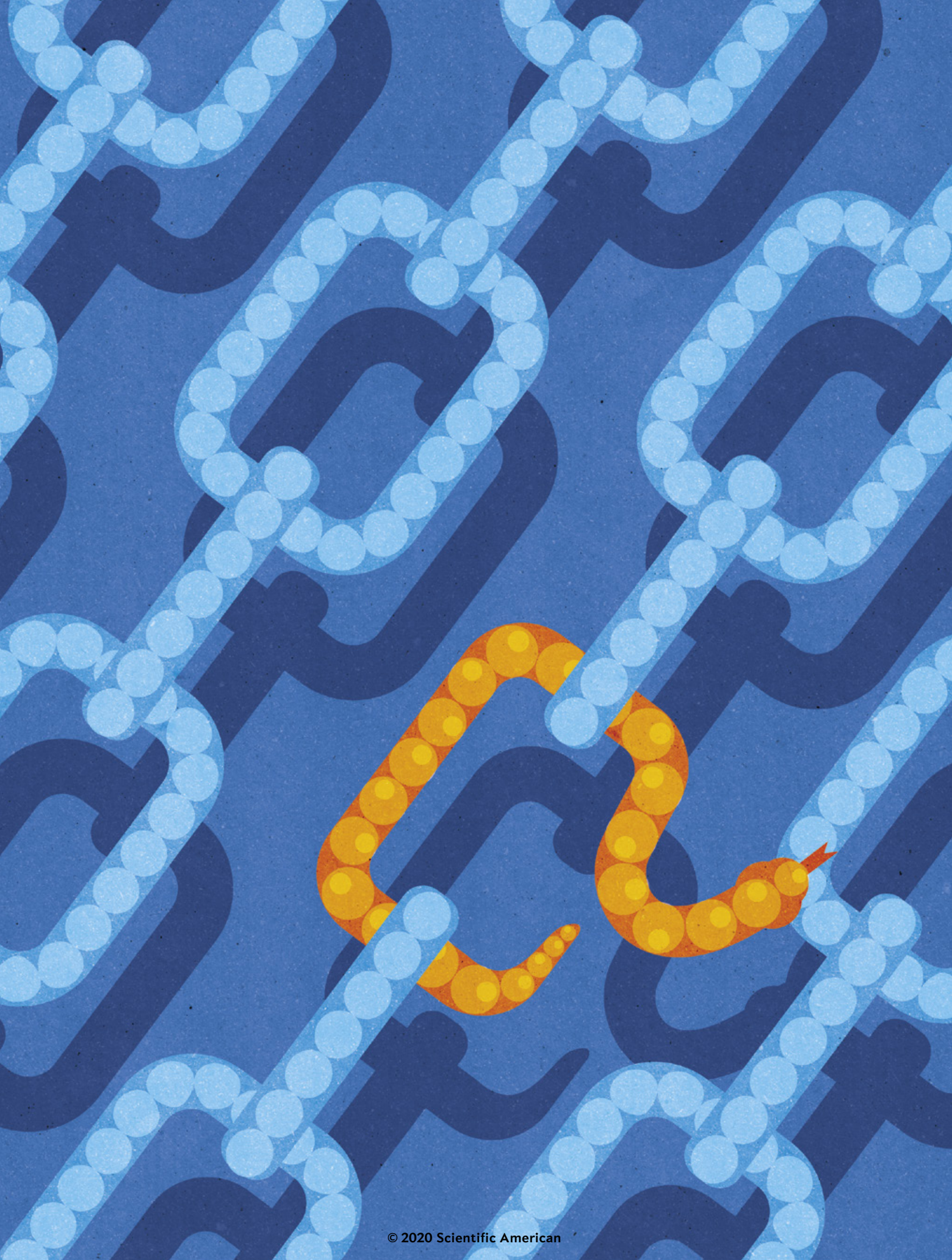
Neighboring countries can work together to reduce water demand; treaties can cover competing withdrawals from rivers flowing down from high peaks, which frequently cross national boundaries. Ministers from eight countries that touch the Hindu Kush Himalayan region set a strong example in October 2020 when they met for a mountain summit and signed a declaration pledging to use science to improve mountain policies, listen to advice from the region's highly diverse population and speak as a unified voice in global negotiations. Millions of people in Afghanistan, Bangladesh, Bhutan, China, India, Myanmar, Nepal and Pakistan depend on the Hindu Kush Himalayas for water, and rain patterns and crop yields there are already changing.

The world's high peaks are transforming rapidly. Within the next few decades many people living downstream will have to adapt to more weather extremes, greater natural risks and shifts in water supply. Scientists, engineers and policymakers should join forces and act now to ensure that sustainable mountain water resources will be available for future generations. SA

FROM OUR ARCHIVES

Is Antarctica Collapsing? Richard B. Alley; February 2019.

scientificamerican.com/magazine/sa



MEDICINE

Cells coexist by cooperating. When some break the rules, cancers result

MALIGNANT CHEATERS

By Athena Aktipis

Illustration by Kotryna Zukauskaite

Athena Aktipis is an associate professor in the psychology department at Arizona State University. She is a project leader at the university's Arizona Cancer Evolution Center and a co-director of the multi-institution Human Generosity Project. Aktipis is author of *The Cheating Cell: How Evolution Helps Us Understand and Treat Cancer* (Princeton University Press, 2020) and host of the Zombified podcast.

A HUMPBACK WHALE IS AMONG THE LARGEST ANIMALS ON THIS PLANET, now or ever. It is also a gigantic society made of quadrillions of cooperating cells. Different cell types orchestrate eating, breathing, swimming, reproduction, reacting to other animals, and all the functions that are necessary for a whale to survive and thrive. If you look inside an elephant, a person or even a saguaro cactus, you will see a similarly well-functioning cellular civilization.

Multicellular creatures evolved in the first place because cells that cooperate had advantages over loners such as a single-celled bacterium. Sharing resources allowed life-forms to become larger—a benefit that helps them resist predators—because nutrients and chemical signals that cells need could be transported around the body. Dividing labor let cells specialize and build useful parts such as a stomach or legs. And teamwork gave them the ability to maintain a healthy extracellular environment so they could live longer than they otherwise would.

But cooperation is a fragile proposition. Within multicellular life, cheaters can prosper. By hogging resources, they replicate more quickly than cooperators and take over, unless there are mechanisms to enforce cooperation. Cheating cells can take advantage of the cooperative cellular society they are living in, overproliferating, monopolizing nutrients and otherwise disrupting the harmony that makes multicellular organisms viable in the first place. This cellular cheating is what we know as cancer.

Cancer cells break the rules of normal cells. They divide when they should not, do not die when they should, rob other cells of essential supplies, shirk their cellular jobs and pollute the extracellular space. While cooperating cells curb excess growth and proliferation, cancer cells often evade growth-suppressing signals. Cooperating cells have limited lives, but cancer cells resist cell death and hide from an immune system that would typically destroy them. Normal cells distribute nutrients and chemical signals essential to survival, but cancer cells grow extra blood vessels to grab more resources for themselves. These contrasts show us that cheating is not merely a metaphor for cancer. It is a description of cancer's cellular reality.

This lens of evolution and cooperation is providing scientists with new insights into the way cancer happens—and why it does not. Giant animals such as the whale and elephant, for instance, rarely get cancer despite having multitudes of cells that can go wrong. Why? A number of researchers, including our team at Arizona State University's Arizona Cancer Evolution Center, have examined the genomes of these giants and found they have many copies of genes that destroy cells that mutate and produce aberrant proteins, a signal of cancer. The animals also have extra copies of genes that trigger DNA repair. These genes are, essentially, the cooperation police. One of them, called *TP53,* has been identified as a cancer suppressor in people—but unlike the giant animals, we have only two copies, and unsurprisingly we are more prone to malignancies. Researchers are now trying to translate the actions of such genes into therapies and looking for similar genes all across the tree of life. Oncologists have even started to use evolutionary principles to design chemotherapy that protects less aggressive, less selfish cells within a nascent tumor, reducing the cancer's danger.

THE COOPERATION GAME

I WAS FIRST DRAWN to this evolutionary interplay between cooperation and cheating when I was getting my undergraduate and graduate degrees in psychology. I wrote computer programs that tested the effects of different strategies on hypo-

thetical populations, rather like nodes in a network. Generally in such models, without countervailing forces such as genetic relatedness or social norms of reciprocity to keep them in line, cheaters outcompete cooperators. At first I was trying to understand what helps keep cooperation stable in human societies. But then I began learning about the biology behind cancer and the ways cancerous cells behave, and it became clear that lots of cancer cell activities looked like the breakdown of cooperation in a multicellular system. Cancer—which had seemed to me like a senseless disease, an organism destroying itself—started to make sense.

As I looked deeper, I discovered that cellular cheating manifests as cancer and cancerlike phenomena in many complex organisms, from humans to clams to cacti. Plants, for example, exhibit cancerlike protrusions called fasciations. One of the most striking examples is the crested cactus. Saguaro cacti can develop mutations in meristem cells (equivalent to stem cells in animals) on the growing tips of the plant. These lead to cell overproliferation and abnormal growths that fan out into crests. The fasciations can be quite beautiful, but like cancer in people, they can take a toll. Crested cacti often have disrupted flowering, which impairs reproduction, and are more vulnerable to disease and injury.

TIPS on a saguaro cactus can erupt in cancerlike protrusions called fasciations, the result of abnormal cell growth. These crests can leave plants more vulnerable to disease.

JOHN CANCALOSI *Alamy*

I realized that many of these breakdowns in cellular cooperation bore an uncanny resemblance to the "hallmarks of cancer," a framework developed by cancer biologists to describe general tendencies of malignancies. In addition to things such as excess proliferation, invasion of other tissues is one of the hallmarks of cancer, and an evolutionary approach suggests that invasion might be a consequence of cellular cheating. When cancer cells overuse resources in their local environments—producing enzymes that digest nearby tissue, for instance—the process often destroys their normal cellular surroundings. We know from ecology that organisms that deplete resources in their environments are under greater pressure to evolve the ability to move via "dispersal evolution."

Cancer cells respond to this same pressure to go mobile. I created a model of cancer that showed that higher rates of cellular resource consumption led to the evolution of cells that had a greater propensity to move, a development my colleagues and I reported in 2012 in *Cancer Prevention Research*. Our conclusion suggested that overuse of resources by cancer cells may be one of the pressures that drives cancer to metastasize, or spread. Even before invasion of other tissues happens, degradation of resources may push cancer cells to evolve the ability to move inside tumors.

This ecological and evolutionary perspective highlights new ways of identifying cancerous cells, beyond the typical hallmarks such as excessive replication. In 2017 biologist Carlo Maley and I, along with other colleagues at Arizona State, noted in *Nature Reviews Cancer* that scientists can look for cells that are not properly regulating other aspects of their behavior. These features include cells that are consuming resources too quickly or producing proteins and enzymes that damage the environment around them.

CATCHING CHEATERS

THE COOPERATION inside a multicellular body is not just about cells holding back from excessive activity. It is also about other cells working to detect and suppress cheating when it arises. Bodies have evolved ways of doing this. For instance, cells normally can replicate only with "permission" from their neighbors, which trigger the release of growth signals. And if any cells depart from the proper multicellular script, they are targeted for destruction by their cellular neighbors or the immune system.

Cancer cells also cheat detectors in their own genetic code. One such sentinel is the cancer-suppressor gene *TP53*. It codes for a protein called p53 that plays a central role in many aspects of cellular control, from halting the cell cycle and initiating DNA repair to triggering apoptosis (controlled cell death) if a cell is too damaged. Other genes in our police force include *BRCA,* a crucial DNA-repair gene; when mutated and unable to perform its normal function, *BRCA* increases the risk of breast, ovarian and prostate cancer.

Genes in the *TP53* family (others include *TP63* and *TP73,* both of which help to maintain the integrity of the genome) evolved very early in multicellular organisms, first appearing in primitive creatures such as sea anemones, and offered enough of a survival advantage to subsequently spread widely across the tree of multicellular life. In 2019 Anna Trigos of the Peter MacCallum Cancer Center in Australia and her colleagues reported that common mutations in cancer overwhelmingly affect signaling pathways that involve genes such as *TP53*. Furthermore, they found that there was a loss of communication between these genetic regulatory systems that evolved even before the evolution of multicellularity and those that evolved to keep these more selfish cellular behaviors under control during the transition to multicellularity. The scientists figured out the age of the genes using a technique called phylostratigraphy,

which compares features of genes of existing organisms to determine a likely common ancestor, ultimately showing where and when in the evolutionary tree of life these genes emerged. They then looked at the mutated genes in tumors from more than 9,000 patients, finding that the genes that help to regulate multicellular cooperation were often compromised.

How do genes such as *TP53* spot cheating? They appear to function as information collectors about cellular activity. For example, signals about an increased number of mutations in a cell or heightened production of aberrant proteins flow to these genes from elsewhere in the cell and from other parts of the genome. Such signals most likely indicate that the cell is no longer cooperating properly with the multicellular body. And they trigger action by *TP53* and similar genes, which can halt the cell-replication cycle and initiate DNA repair. If these measures are insufficient, the genes induce cellular death to protect the organism from the potential threat that the cell might pose.

The two copies of *TP53* in humans come from our parents: one from our mother, one from our father. If one of these copies of *TP53* becomes mutated itself, this leads to a much higher overall cancer risk over a person's lifetime. People who have a rare condition called Li-Fraumeni syndrome inherit just one copy of *TP53,* which leaves them extremely susceptible to cancer.

Elephants, in contrast, have 40 copies of their version of *TP53*–it is called *EP53*—and several scientists, including myself, think this explains why the giant animals rarely get cancer. That absence of malignancy has been a long-standing oncological puzzle known as Peto's paradox**.** In 1977 Richard Peto, an epidemiologist at the University of Oxford, and his colleagues pointed out that larger (and longer-lived) organisms should get more cancer than smaller creatures because bigger ones have more cells and logically that should raise the chances of malignant mutations. Yet cancer risk and body size do not track, he noted. In a study published in 2017 and in ongoing work, our research group has found that this paradox exists throughout the animal kingdom. We created a large database of zoo and veterinary pathology records and learned that species that are larger and longer-lived have essentially the same rates of cancer as species that are smaller and shorter-lived; our continuing analysis has pointed to more instances of this pattern.

To us, this suggests that large and long-lived organisms have particularly good mechanisms for suppressing cellular cheating, such as those extra copies of *EP53*. Evolutionary geneticists have compared elephant genomes with reconstructed genomes of several related species—the woolly mammoth, for one—and found that as animals in this lineage got bigger, they kept adding more copies of *TP53*-like genes**.** This repeated occurrence suggests that the genes played an important role in the evolution of large body size. Big bodies helped the elephants and their kin survive predators, and the cancer-suppressor genes helped the elephants survive cheating cells in those big bodies.

This seems to be a pattern. Jumbo bodies have evolved many times in the history of life, both on the land and in the sea, and genes to check cheaters evolved with them. For instance, among cetaceans, the taxonomic group that includes dolphins and whales, a huge variation in body size exists. The humpback whale is approximately four times larger than the closely related common minke whale, and the orca whale can be 20 times larger than the closely related bottlenose dolphin. And the numbers of genes in the cellular-cooperation police force go up with body size in this group. Members of our research team, looking closely at the genomes of humpback whales, found duplications of genes involved in the process of apoptosis, when cells recognize they can no longer function properly and essentially kill themselves. Smaller cetaceans do not have as many copies of these genes. In the big whales, our group also found evidence of evolutionary selection for a number of genes involved in cancer suppression, such as cell-cycle checkpoint genes, cell-signaling genes and genes involved in proliferation. One of these genes, called *PRDM2*^{T}, regulates the expression of the cetacean version of *TP53,* which again shows the central role of that particular sequence of DNA.

Cooperating cancer cells seem like the stuff of nightmares, but understanding their roles lets us think about new ways to stop these cheaters.

Stopping cheating cells is not an easy task, because, in an ironic twist, malignant cells that stop cooperating with normal cells can start to cooperate with one another. That makes things even worse for healthy cells. For example, cancer cells can produce growth factors for one another. They can also help shield their fellow cheaters by producing molecules that help malignant cells cloak themselves, making it harder for immune cells to detect them. My group's computational models of cell populations in the body show that this kind of cancer cooperation can evolve and is more likely to happen when cancer cells interact with their genetic clones, events that often occur in tumors. This cooperation among rule breakers may drive their ability to successfully metastasize and invade other tissues. Cancer cells can move as a group, using electrical and chemical signaling, sometimes forming a long line of cells that works its way into other parts of the body. One study found that groups of tumor cells in the bloodstream are 23 to 50 times more likely to successfully generate metastases compared with individual cancer cells in the blood.

IMPROVING NATURAL DETECTION

COOPERATING CANCER CELLS do seem like the stuff of nightmares, but understanding the role of cooperation among cells does let us think about new ways of stopping the cheaters, even when they band together. It may be possible, for example, to strengthen our natural cheating-detection systems. Some members of our research center are currently working on

MORE CELLS IN A BODY should raise the odds of cancer, but giant humpback whales have multiple genes that suppress the disease.

developing cancer treatments using elephants' abundant *EP53* genes. In test-tube experiments, they have already shown that splicing *EP53* into the genome can restore damaged p53 functions in cells taken from human and dog osteosarcomas. The addition of *EP53* enhanced the usual apoptosis response that helps to protect the body from cancer cells. Treatments called immune system checkpoint blockades are another exciting area. These drugs block the ability of cancer cells to send misleading signals to immune cells—signals that hide their escalating cheating behavior—and they have shown some success in treating cancers such as melanoma.

Still another approach, called adaptive therapy, tries to weaken groups of cancerous cells by maintaining cells among them that are not so far gone. Instead of bombarding a tumor with heavy doses of chemotherapy—which ultimately favors the evolution of cells that resist the drugs, just as constantly spraying pesticides on crops leads to pesticide-resistant insects—oncologists have been trying a more restrained approach. They employ only enough chemotherapy to keep the tumor small. Allowing drug-sensitive cells to survive and compete for resources with drug-resistant cells can keep the latter population low. In early clinical trials with patients who had aggressive prostate cancer, this method kept tumors under control for at least 34 months, compared with the average for standard therapy of about 13 months. These tests are ongoing.

And because cooperation among cheaters—a warped honor among thieves—seems to be an important strategy among cancer cells, my colleagues and I have proposed blocking the molecules these cheaters pass back and forth as they signal one another to grow or develop new blood vessels to supply tumors. It would make it harder for cancer cells to work together. Given the ability of groups of cells to invade and metastasize more effectively, interfering with molecules that cancer cells use to stick to one another could be another future direction for therapies. Patients who have higher levels of some of these sticky proteins called plakoglobins have more metastases and lower survival rates, suggesting that the proteins are targets worth investigating.

The social lives of cancer cells within the body are much more complex than we could have anticipated. But our normal healthy cells are arguably even more sophisticated, and within them lie many weapons against cells gone rogue. We are not only bastions of cellular cooperation: each and every one of our cells contains within it a complex genetic network that can detect and respond to cheating. We are, after all, descended from a line of multicellular ancestors that suppressed cancer long enough to reproduce, and their offspring carried these traits onward.

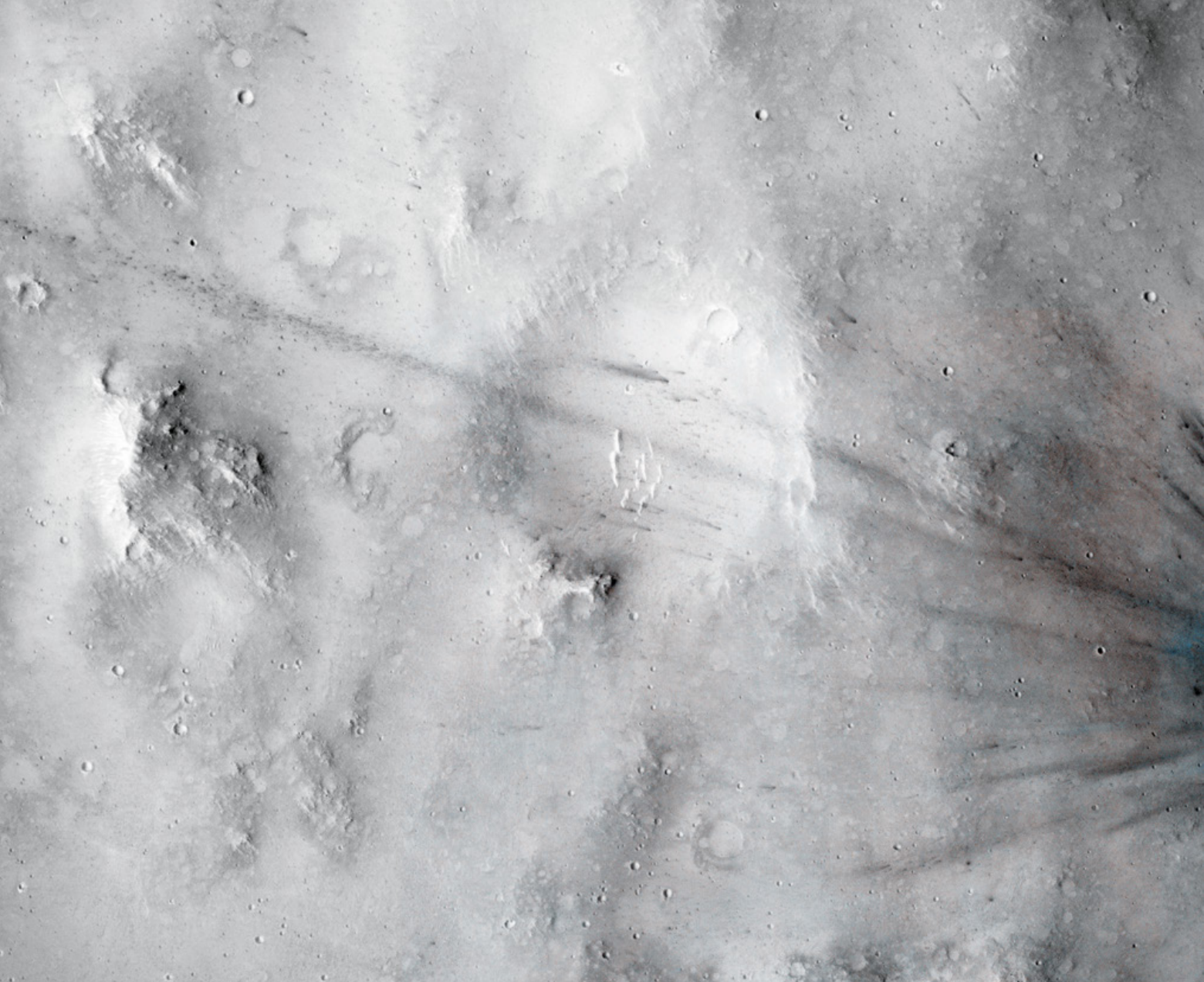
In the big picture of the evolution of life, cellular cooperation has been wildly successful despite the persistence of cellular cheating. Cancer may break the body's rules, but we—along with whales and all other forms of multicellular life on this planet, honed by billions of years of natural selection—hold the tools for restoring peaceful coexistence. SA

FROM OUR ARCHIVES

Darwin's Cancer Fix. James DeGregori and Robert Gatenby; August 2019.

scientificamerican.com/magazine/sa

GEORGE KARBUS *Getty Images*

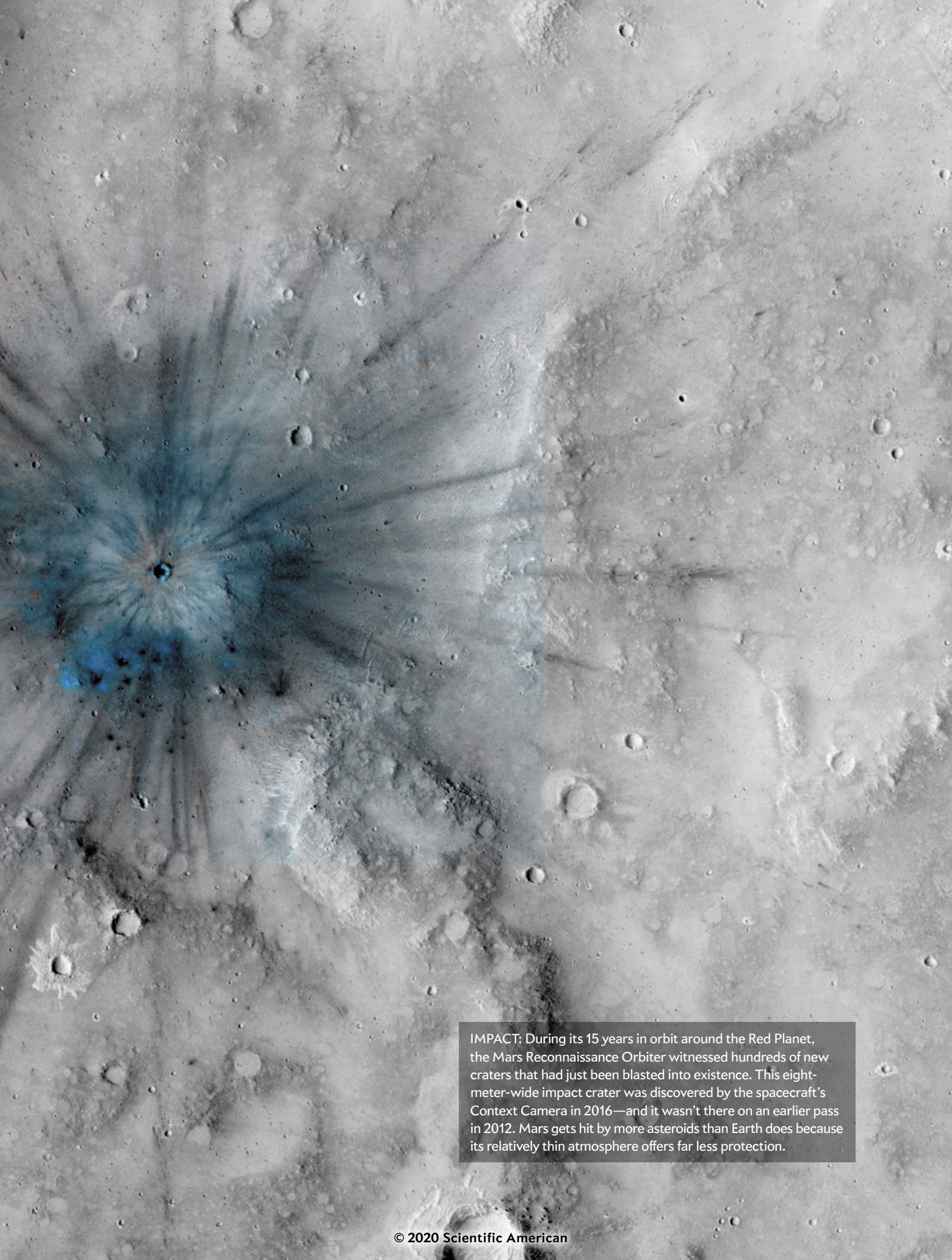


SPACE SCIENCE

DYNAMIC PLANET

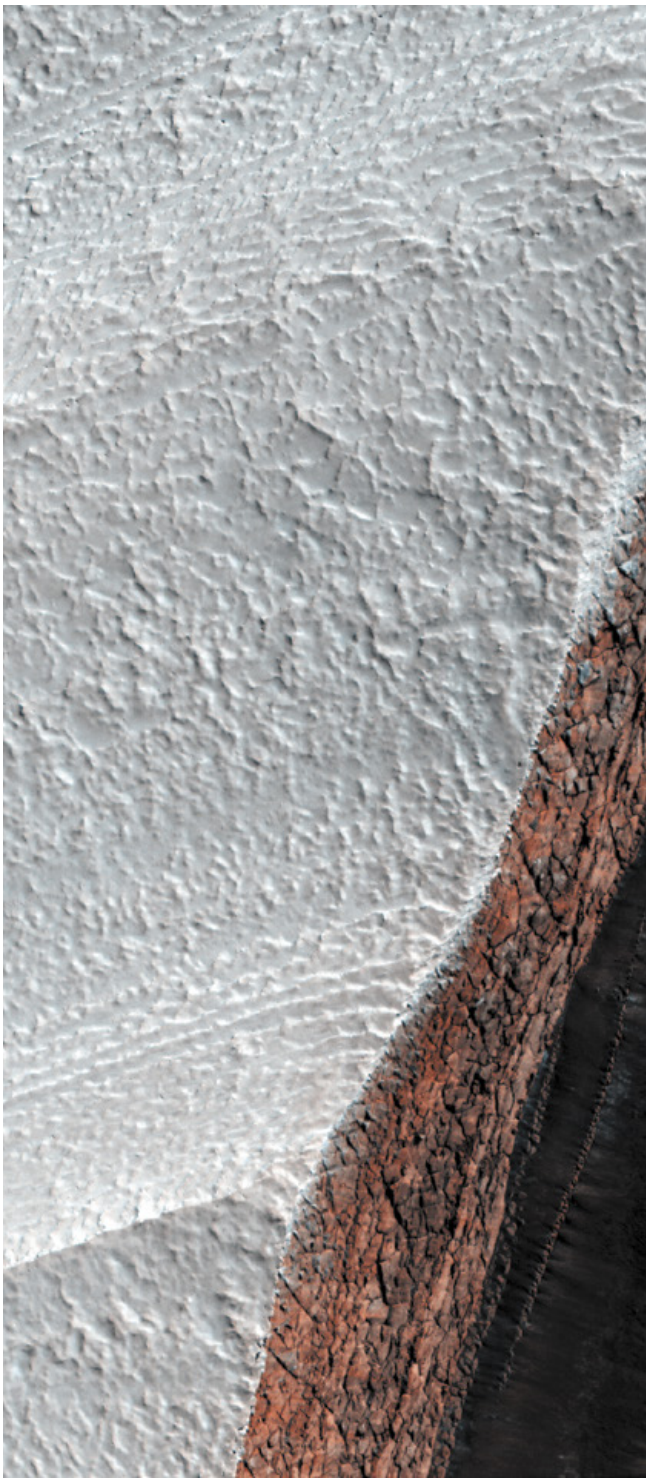
For 15 years the Mars Reconnaissance Orbiter has transformed our view of the Red Planet

By Clara Moskowitz



IMPACT: During its 15 years in orbit around the Red Planet, the Mars Reconnaissance Orbiter witnessed hundreds of new craters that had just been blasted into existence. This eight-meter-wide impact crater was discovered by the spacecraft's Context Camera in 2016—and it wasn't there on an earlier pass in 2012. Mars gets hit by more asteroids than Earth does because its relatively thin atmosphere offers far less protection.

Clara Moskowitz is a senior editor at *Scientific American*, where she covers space and physics.



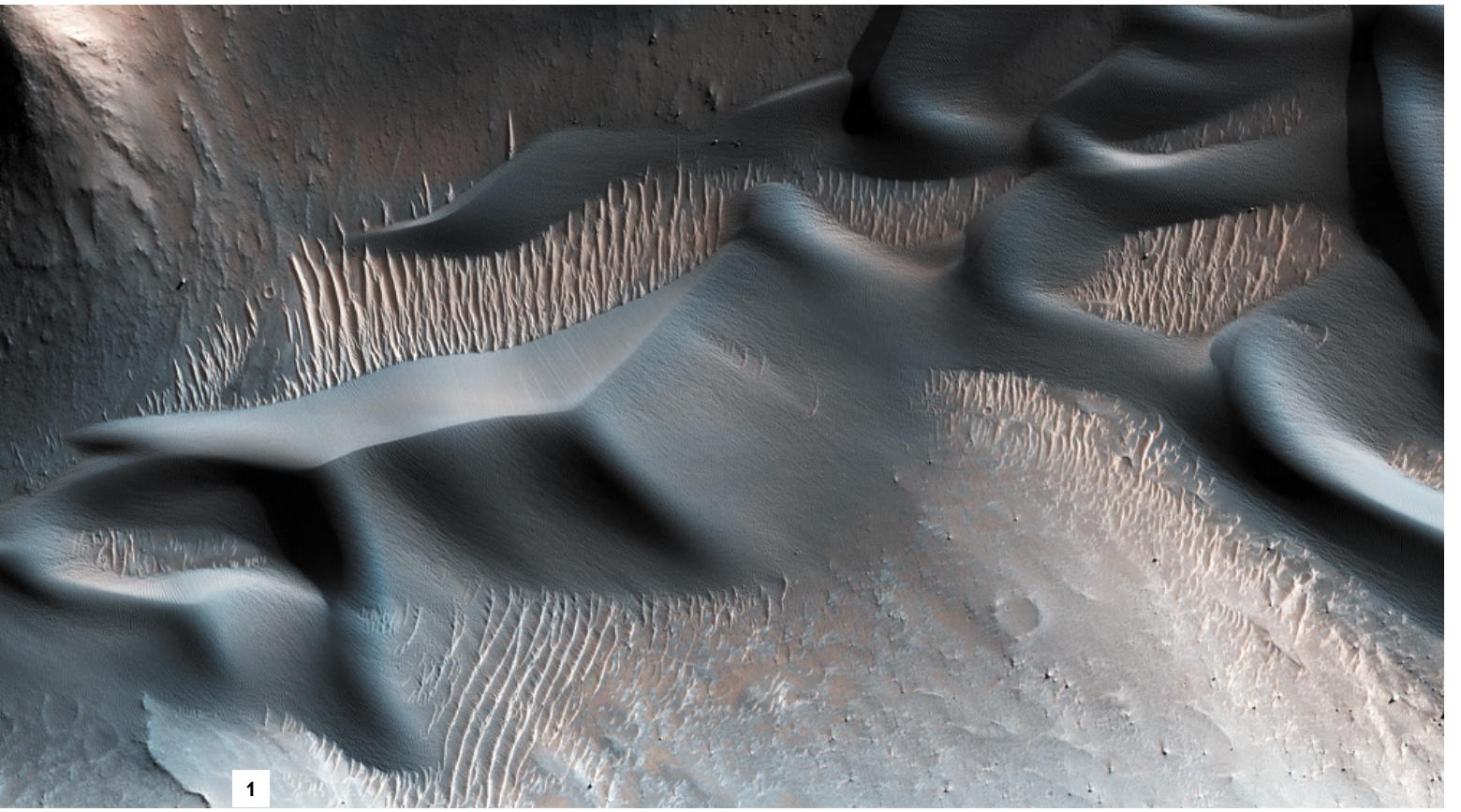
MARS, ONCE THOUGHT TO BE A STATIC, DUSTY LANDscape, is ever changing. It wasn't until NASA's Mars Reconnaissance Orbiter (MRO) showed up that we observed shifting dunes, seasons coming and going, and dust devils swirling across the planet. The spacecraft recently marked its 15th anniversary in orbit around our neighboring world, where it has used a suite of four science instruments and three cameras to catalogue a diverse array of geologic features. "Before we had never had enough resolution over a long-enough period to see changes on the surface," says Richard Zurek, MRO's project scientist at the Jet Propulsion Laboratory (JPL) in Pasadena, Calif. "Today we can see that Mars is dynamic."

The orbiter, launched in 2005, has led to many discoveries about Mars's atmosphere, its surface and, thanks to radar, even what is buried underneath, via a cumulative 400 terabits of data beamed back to Earth over the years. It has also served as a communications satellite, relaying messages from the various landers and rovers that have visited the Martian ground during its stay. After 15 years the spacecraft is still in good health and has enough fuel to keep going for another 15 years at least—as long as its instruments hold out. "Of course, things could break unexpectedly, just like an old car," says MRO deputy project scientist Leslie Tamppari of JPL. "But having that longevity can really deepen our understanding of Mars. There are things we can learn only by having a long baseline history of observations."

The spacecraft's instruments have also exposed the beauty of Mars, revealing an alien world that bears surprising similarities to our own. Like many of the great photographs by the Hubble Space Telescope and other observatories, MRO's images aren't just science—they're art. Zurek recalls when Alfred McEwen, principal investigator of the High Resolution Imaging Science Experiment (HiRISE), came to visit JPL: "The secretary told him, 'Go down the hallway, and when you come to the Impressionist painting, turn left.' He went down the hall and saw that the Impressionist painting was actually a photo of Mars, taken by his camera."

AVALANCHE: A cloud of debris hovers over a spot on Mars's North Pole in this 2010 photo from the orbiter's High Resolution Imaging Science Experiment (HiRISE). Most likely the aftermath of an avalanche, the scene shows a steep cliff made of layers of water ice topped with bright white carbon dioxide frost. Such vistas are common in this area every Martian spring, suggesting that the northern pole sees a recurring avalanche season when sunlight and heat break up the winter's frost.

ALL IMAGES BY NASA, JPL AND UNIVERSITY OF ARIZONA



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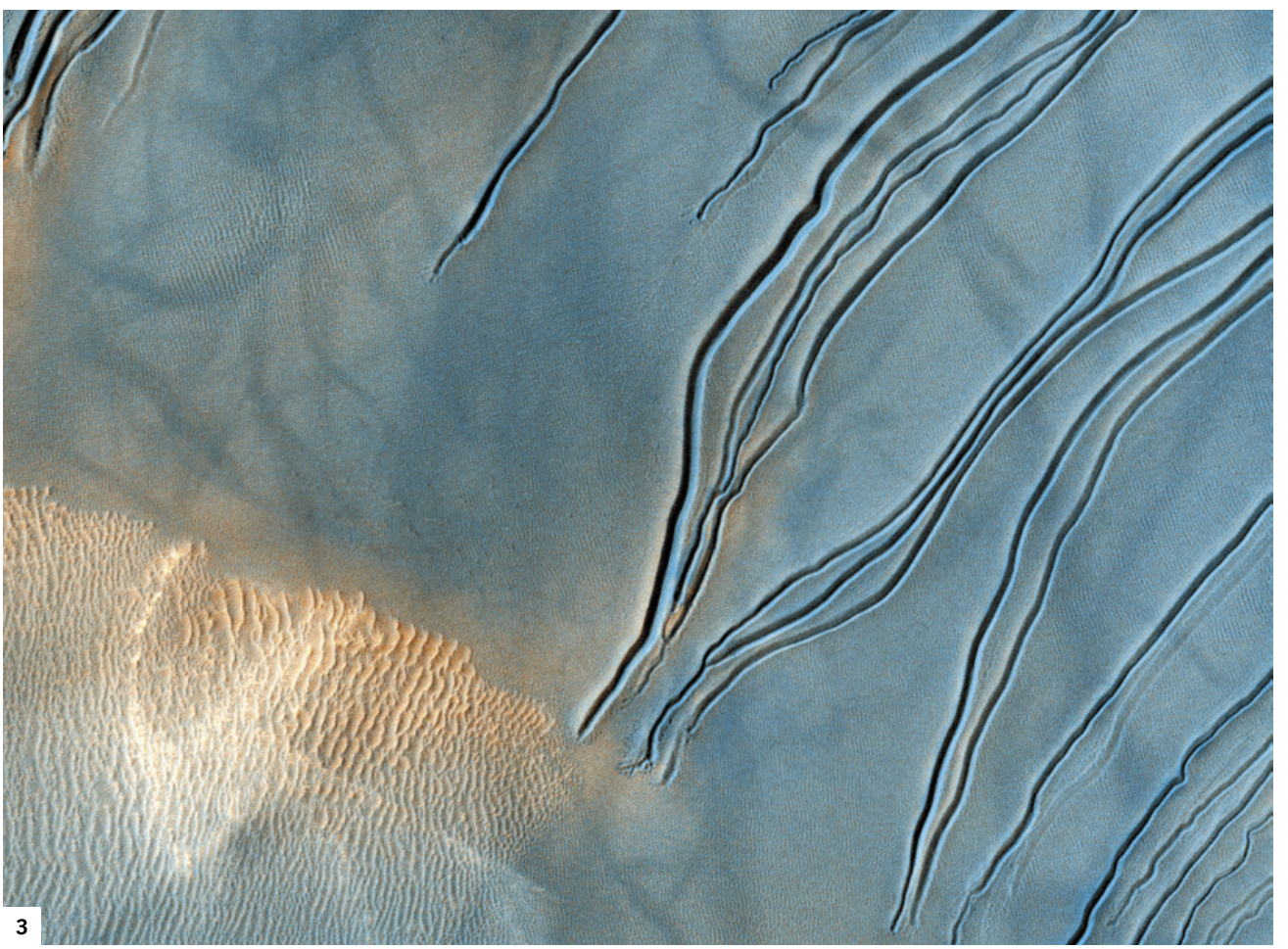

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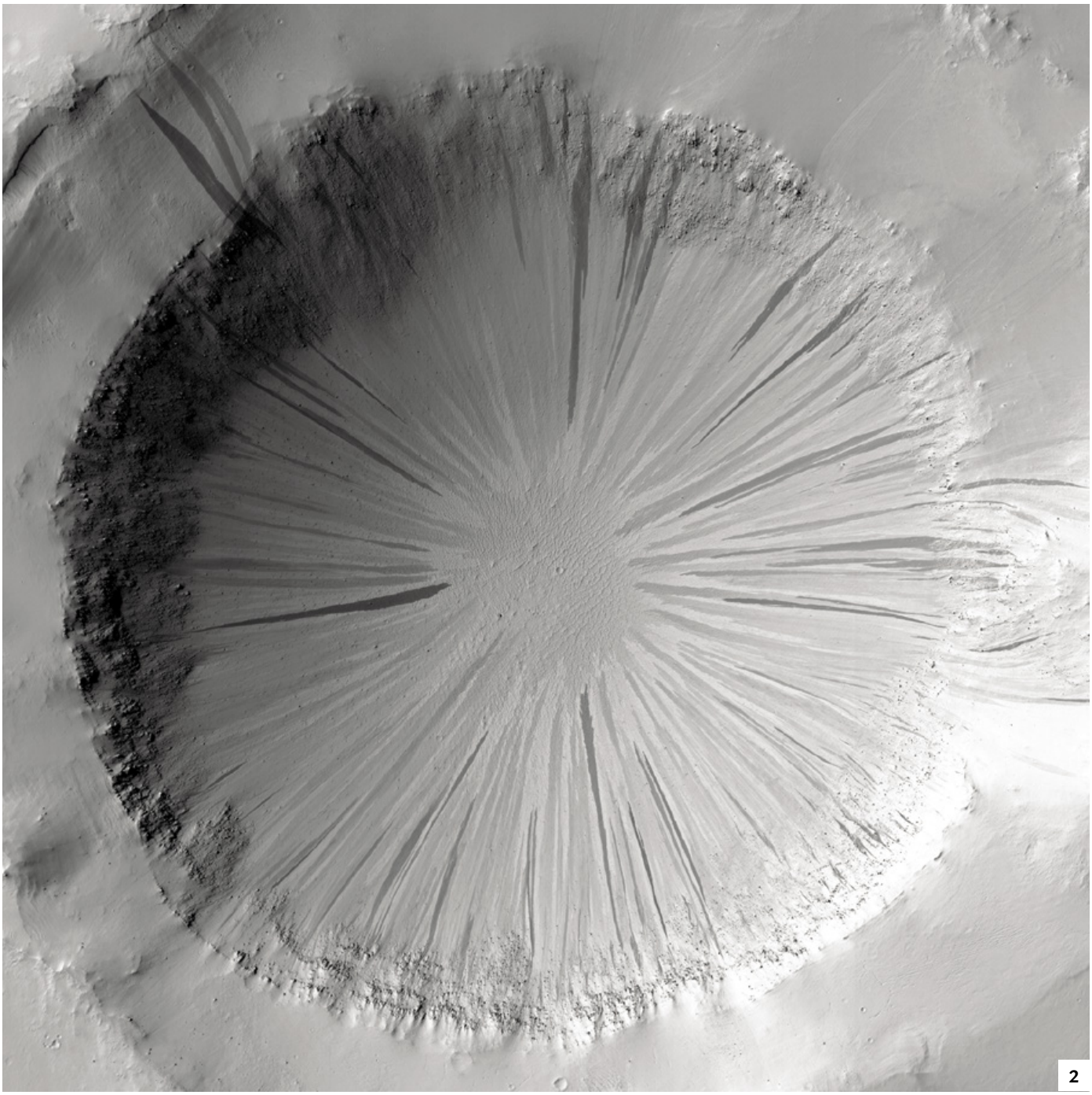
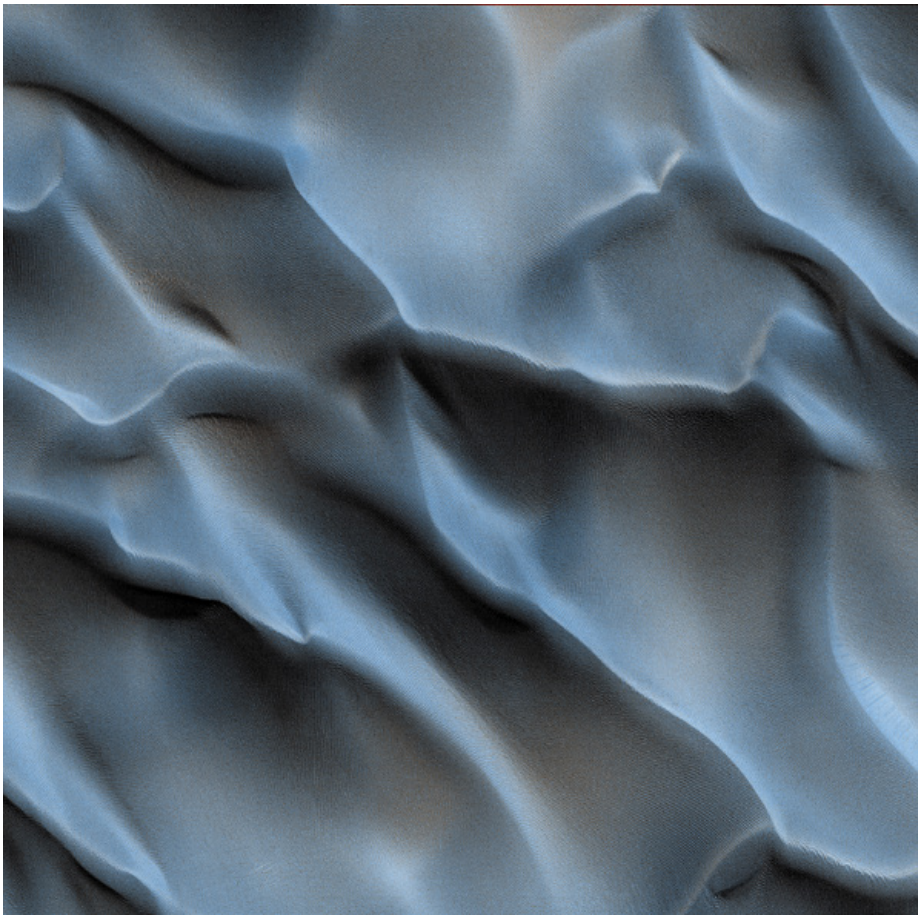
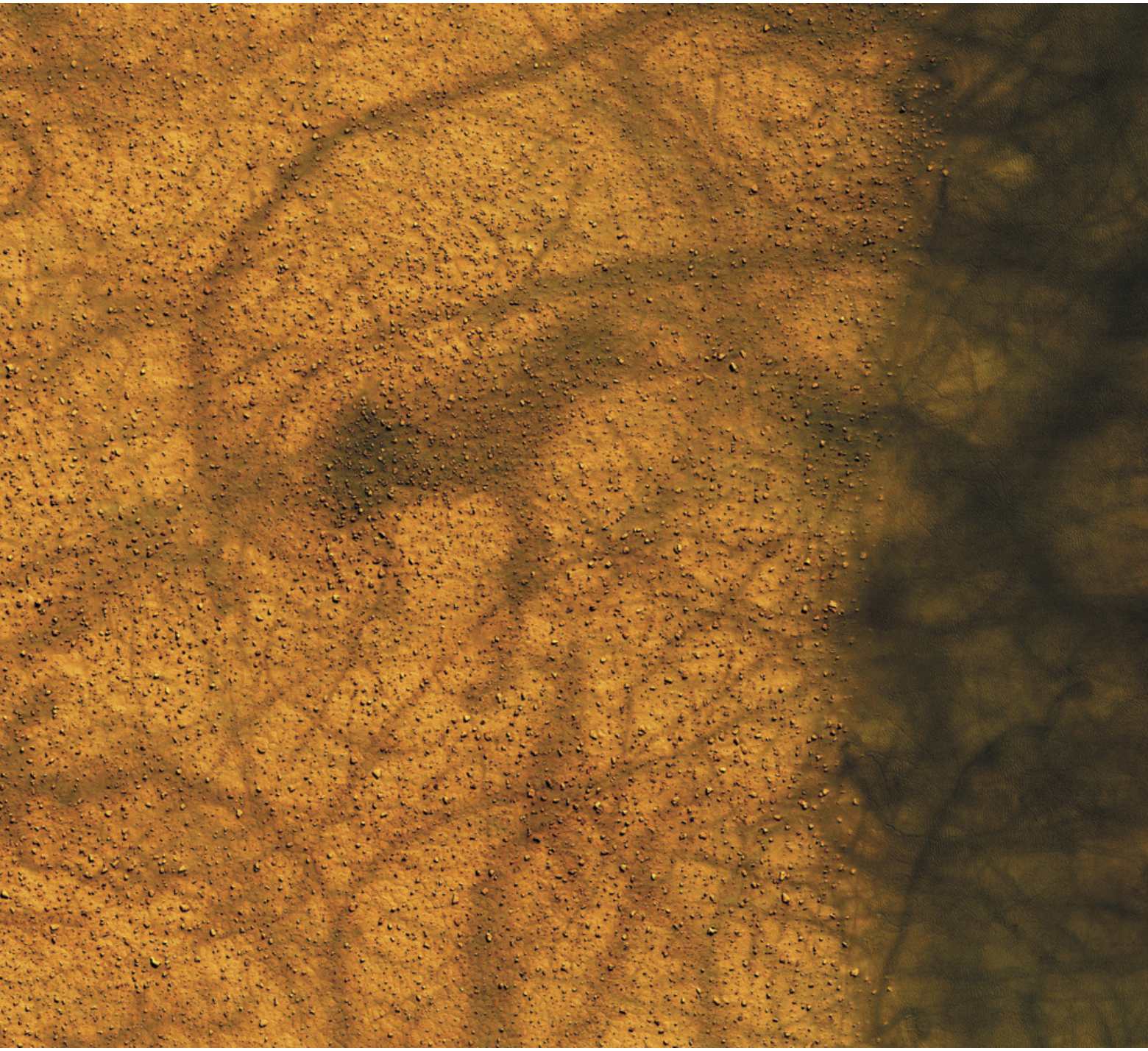


STRIPES AND WAVES: Varied and gorgeous textures blanket Mars's dusty surface. Large dunes (*1*) undulate over the Nectaris Montes slopes within the Coprates Chasma canyon. Narrow troughs along the dunes in Russell Crater (*2*) form when chunks of carbon dioxide ice slide down the steep faces of the dune. The dark channels seem to appear and rearrange with the seasons, creating different patterns each year the orbiter has photographed them. Lines of a different kind run down the slopes of a crater in the Arabia Terra region (*3*). These streaks form when dust cascades along the sides of the circular crater.



DUNES: Resembling wrinkled satin, dune crests rise from the Martian floor. Scientists study the shapes of dunes to understand Red Planet geology. Some curved peaks here, for instance, look like so-called barchan dunes, which form when the wind blows steadily in a single direction—a feature common in Earth deserts as well as those on Mars.



DUST DEVILS: A weather feature shared by Earth and Mars is the dust devil—a vertical column of dust spiraling upward around a pocket of lower air pressure. This one (*1*), captured by HiRISE in 2019, soars some 650 meters into the sky, as evidenced by the length of its shadow stretching off to the right. Dust devils carve meandering dark tracks (*2*) into the Martian surface when they pass over it by lifting away bright dust from the ground.

FROM OUR ARCHIVES

Mars in Motion. Alfred S. McEwen; May 2013.

scientificamerican.com/magazine/sa

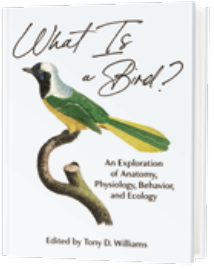
RECOMMENDED

By Andrea Gawrylewski

What Is a Bird?

An Exploration of Anatomy, Physiology, Behavior, and Ecology

edited by Tony D. Williams. Princeton University Press, 2020 ($35)

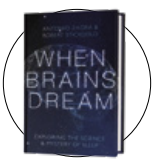


BALTIMORE ORIOLE prepares to take off.

Many housebound quarantiners have recently discovered a new—or renewed—interest in birds. Through windows overlooking gardens or fire escapes and in small parks or dense woods, birds occupy nearly every habitat on earth and are our constant, if sometimes unnoticed, companions. This welcoming compendium is part coffee-table book and part deep dive into the science of ornithology—the team of biologist-authors, edited by biology professor Williams, elucidates all things bird: from their evolution and anatomy to their social and migration patterns. Even casual bird-watchers will be drawn into the fascinating mechanisms of feather coloring of house finches (*Haemorhous mexicanus*) or the science that gives the bill of a helmet vanga (*Euryceros prevostii*) its unmistakable blue.

When Brains Dream: Exploring the Science and Mystery of Sleep

by Antonio Zadra and Robert Stickgold. W. W. Norton, 2021 ($27.95)



Dreams feel significant while they are happening, but do they have any scientific relevance? Sleep researchers Zadra and Stickgold propose their own framework for dreams' purpose in the brain, detailing the history of dream research and lessons learned. Dreaming acts as a memory-processing mechanism, they write, exploring images and thoughts weakly associated with moments from the previous day. Scientists have used compelling methods to probe dreams' inner workings: for example, dreamers can signal via eye movements to observers while their dreams are in progress to help researchers measure the length of dream actions. The book wields dreamy anecdotes and complex neuroscience to try to grasp the importance of these phantasms. —*Sarah Lewin Frasier*

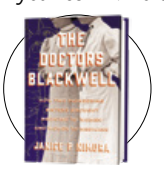
Fundamentals: Ten Keys to Reality

by Frank Wilczek. Penguin Press, 2021 ($26)

To see the world through the lens of physics we must be "born again," physicist Wilczek writes—we have to lose our preconceptions and approach the universe with the same open-minded curiosity and acceptance as a baby first learning about her surroundings. In this overview of modern physics, Wilczek describes the known and the unknown—what scientists have managed to learn about reality and what they still struggle to explain. In 10 chapters covering space, time, matter, energy and other basic concepts, he tells the stories behind the major players and turning points in the development of physical knowledge. The result is a lucid and riveting narrative of the fundamentals—what Wilczek calls "the central messages of modern physics," which are not just facts about how the world works but also "the style of thought that allowed us to discover them." —*Clara Moskowitz*

The Doctors Blackwell: How Two Pioneering Sisters Brought Medicine to Women—and Women to Medicine

by Janice P. Nimura. W. W. Norton, 2021 ($27.95)



In the mid-19th century the view of most male physicians was that women did not have the intellect or emotional fortitude to be their colleagues. Despite this skepticism, Elizabeth Blackwell was accepted by the Geneva Medical College in New York State and in 1849 became the first woman in America to obtain a medical degree. Writer Nimura uses an extensive collection of journal entries and letters to trace Blackwell's trailblazing journey through medical school and her further training in the U.S. and abroad. Her younger sister, Emily, followed in her footsteps and became a doctor in 1854. In 1857 the sisters established the New York Infirmary for Indigent Women and Children—the first hospital staffed by women and intended to offer both care for women and training for future women doctors.

GETTY IMAGES

Naomi Oreskes is a professor of the history of science at Harvard University. She is author of *Why Trust Science?* (Princeton University Press, 2019) and co-author of *Discerning Experts* (University of Chicago, 2019).

Can Exploring Space Unite Us?

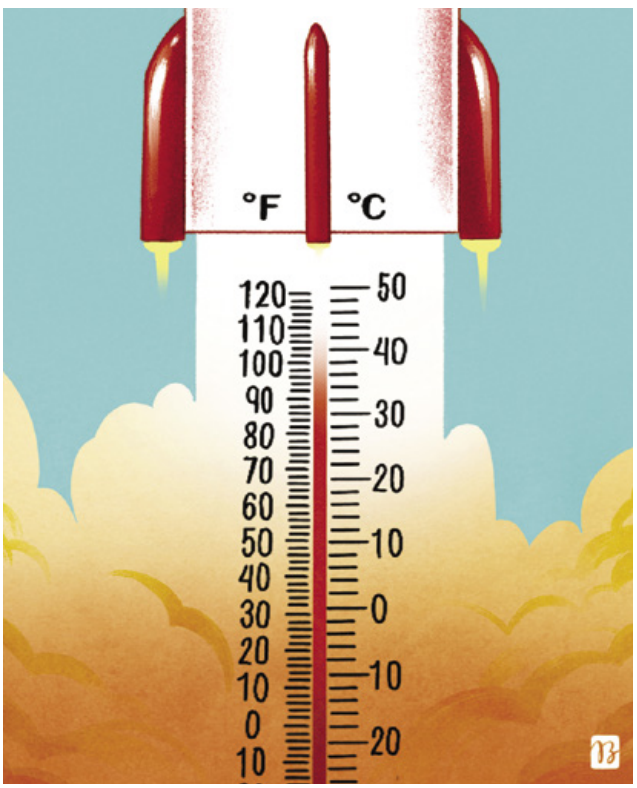
It might—but probably not in the way some people think

By Naomi Oreskes

On January 31, 1958, the U.S. put its first satellite into space. As the Jupiter C rocket carrying the satellite burned its way through the upper atmosphere, engineers at Cape Canaveral in Florida were "shouting, singing, cheering." At the National Academy of Sciences, there was "hardly elbow room among the crowd on hand to hear that the first U.S. satellite, Explorer 1, was up."

But how did Americans feel about what followed? Many people think that NASA's programs of the late 1950s and 1960s were extremely popular, and it is often said that the Apollo program unified an otherwise disunified nation. In our current moment of national disunity, it is tempting to imagine a reinvigorated program of space exploration bringing us back together.

Sadly, however, space exploration has never been the national unifier that many people think. While scientists cheered Explorer 1, a good many would oppose the human spaceflight programs that followed, viewing them as expensive, dangerous and largely symbolic. Many military leaders also opposed human spaceflight, preferring to see resources focused on the crucial challenges of rocket propulsion and guidance, essential for the emerging intercontinental ballistic missile (ICBM) program.



Dwight Eisenhower, president at the time of Explorer 1, was a reluctant space warrior, rejecting the idea of a space race since "race" implied haste, which would likely lead to waste. Moreover, while it was often asserted that space superiority would translate into military superiority, Ike believed that the U.S. could be fully defended with existing nuclear weapons and conventional forces. In later years he would dismiss the Apollo program as a "stunt."

And then there was the cost. Under Presidents John F. Kennedy and Lyndon B. Johnson, NASA's budget skyrocketed—Apollo would be the largest peacetime expenditure in American history—and Congress got increasingly uneasy. NASA's 1964 budget was $5.1 billion; the 1965 budget was $5.3 billion. For Democrats wanting to expand spending on social problems and Republicans wanting to shrink the federal budget, these were disturbing numbers.

They disturbed the American people, too. Throughout the 1960s polls showed that a majority of Americans did not believe the Apollo program was worth the cost. But after Americans made it to the moon, most of the earlier skepticism was forgotten, and as Apollo receded into history people had an increasingly favorable view of it. According to one poll, in 1989, 77 percent of Americans thought the moon landing was worth the cost, in contrast to 1979, when only 47 percent felt that way. Perhaps it is the *idea* of space that unifies us, particularly when burnished by the glow of nostalgia. Or perhaps it is simply that nothing succeeds like success: after the lunar landing, Americans decided that it had been worth the price after all.

NASA now plans to go again to the moon, with a goal of landing astronauts—among them at least one woman—near the lunar south pole in 2024, and once again the American people, including many scientists, are skeptical of the value. Most Americans are pro-NASA and pro-space. A 2018 Pew Research poll found that 80 percent think the space station has been a good investment, 72 percent think it is essential for the U.S. to remain a global leader in space, and 65 percent say that should happen through NASA, not primarily through private companies. But only 18 percent think it should be a top priority to send humans to Mars, and only 13 percent support sending humans to the moon.

So what do Americans want NASA to do? The answer may come as a surprise: 63 percent say NASA should make monitoring global climate a top priority. If we include those who think it should be an important (but not top) priority, the percentage increases to a whopping 88 percent. The second-highest priority is looking for asteroids or other objects that might hit our planet. Many of us have been loath even to talk about climate change because it is seen as divisive—but one thing that seems to unify us is the belief that the most important thing we can do in space is to collect information to protect ourselves and our one and only Earth. SA

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Illustration by Jay Bendt

JANUARY

1971 Antievolution Evolves

"In recent months the teaching of evolution has come under attack in a number of states. The revival of fundamentalism in biology takes a somewhat new form: the emphasis is on opposition to current theories of the origin of life and the diversity of species not by theologians but by scientists. The movement is led by the Creation Research Society, whose members have appeared before state boards of education and textbook committees in California, Texas, Arkansas and Tennessee. The society's credo says that it is 'committed to full belief in the Biblical record of creation and early history' and that its goal is 'the realignment of science based on theistic creation concepts.'"

Joy of Pulsars

"The origin of the energy input to the Crab Nebula had been a puzzle that had long defied attempts at solution. Among the various possibilities considered, John Archibald Wheeler at Princeton in 1966 and Franco Pacini at Cornell in 1967 had independently put forward the apparently far-fetched idea that a rotating neutron star might be the energy source. Now, after the fact, it is possible to use the observations of the Crab Nebula and its pulsar (NP0531) to invert the problem and show that if the pulsar is a rotating star, it must have the mass and radius of a neutron star. In other words, even without the theory developed over the past 40 years, it is possible to assert that stars of approximately one solar mass and radii on the order of 10 kilometers must exist since the pulsar in the Crab Nebula is such a star. —Jeremiah P. Ostriker"

1921 Truck Transport

"This is the era of the motor truck. Yesterday it was the railroad; before that it was the stage coach; and still further back it was the canal. The motor truck, and by that we mean modern highway transportation, has come into its own. It has defined its field of service and established itself therein. In the field of short-haul transportation, the motor truck is the last word in efficiency. The use of the motor truck, both in the handling of passengers and freight, is entirely an economic proposition for the benefit, according to the leading motor truck authorities, of the country at large. If that is so, there is no justification for limiting by legislation the use of the highway by that vehicle."

1971

1921

1871

Connecting Coast to Coast

"The Longest Land Line in the United States was recently opened to the public by the Western Union Telegraph Company, giving direct communication between New York and Seattle. Four messages simultaneously each way can be sent over the single copper strand."

1921: Loading and unloading machinery keeps the truck a productive link in the transportation system.

1871 Thomas Henry Huxley

"Although Dr. Huxley is profoundly learned in natural history, he has also found time for general literary culture, and is fond of poetry, fiction, and fine writing. It is this wide culture that gives him such power in his controversial writings. He seems to like nothing better than a regular set-to with some members of the old-school scientists, and he has sometimes been accused of exhibiting a pugnacious and acrimonious spirit. On one occasion Samuel Wilberforce, Bishop of Oxford, blandly asked him in the presence of a large audience: 'Is the learned gentleman really willing to have it go forth to the world that he believes himself to be descended from a monkey?' Professor Huxley rose and replied in his quiet manner, 'Whether I should be descended from a respectable monkey, or from a bishop of the English church, who can put his brains to no better use than to ridicule science and misrepresent its cultivators, I would certainly choose the monkey!'"

Camels in Nevada

"On a ranch on the Carson river is to be seen a herd of twenty-six camels, all but two of which were born and raised in this State. The camel may now be said to be acclimated to Nevada. The ranch upon which they are kept is sandy and sterile in the extreme, yet the animals feast and grow fat on such prickly shrubs and bitter weeds as no other animal would touch. When left to themselves, their great delight, after filling themselves with the coarse herbage of the desert, is to lie and roll in the hot sand. They are used in packing salt to the mills on the river, from the marshes lying in the deserts, some sixty miles to the eastward. Some of the animals easily pack 1,100 pounds."

SCIENTIFIC AMERICAN, VOL. CXXIV, NO. 2, JANUARY 8, 1921

Text by Mark Fischetti | Graphic by Jen Christiansen

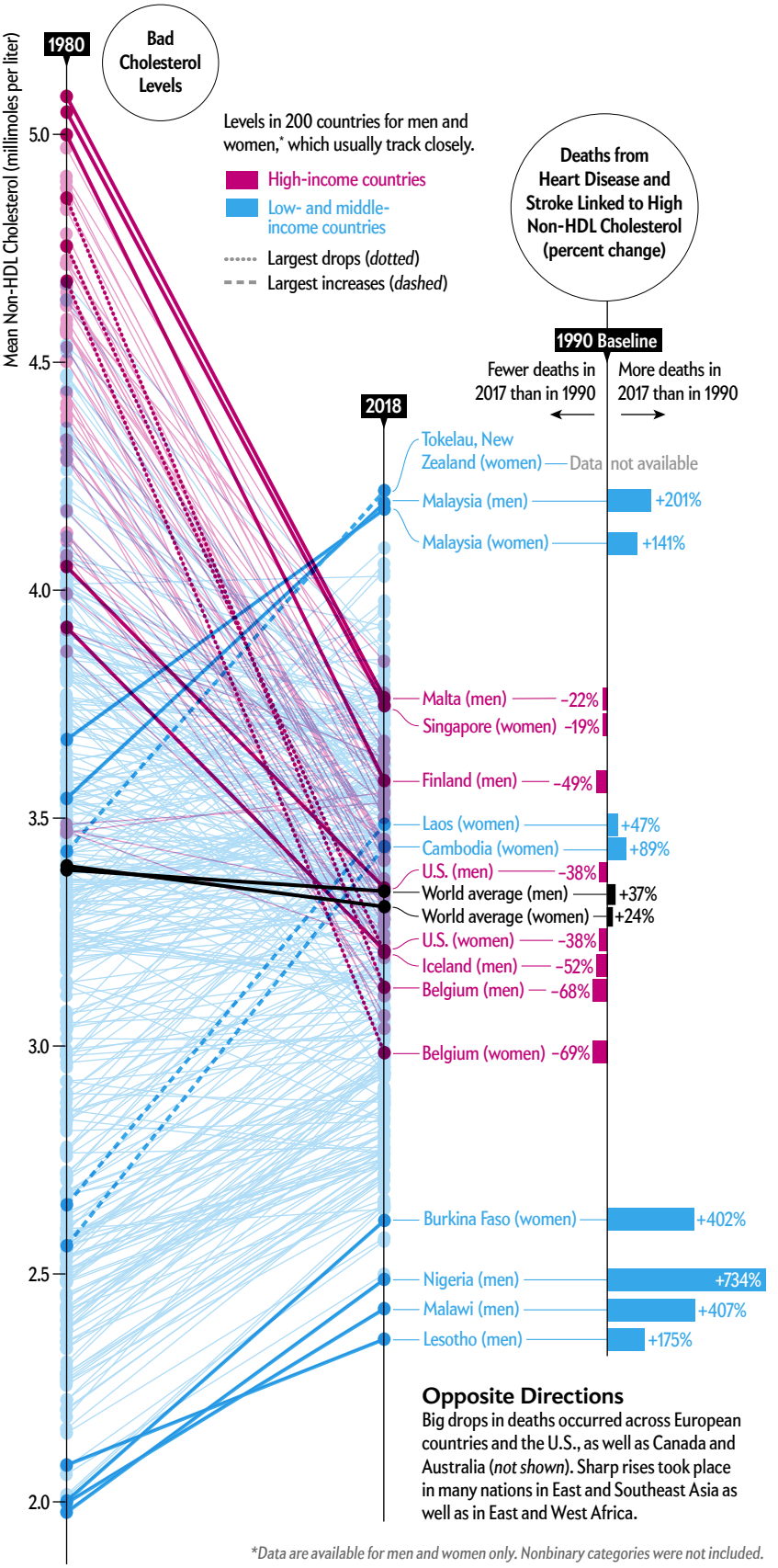
Cholesterol Trades Places

Blood levels drop in the West but climb in Asia and Africa

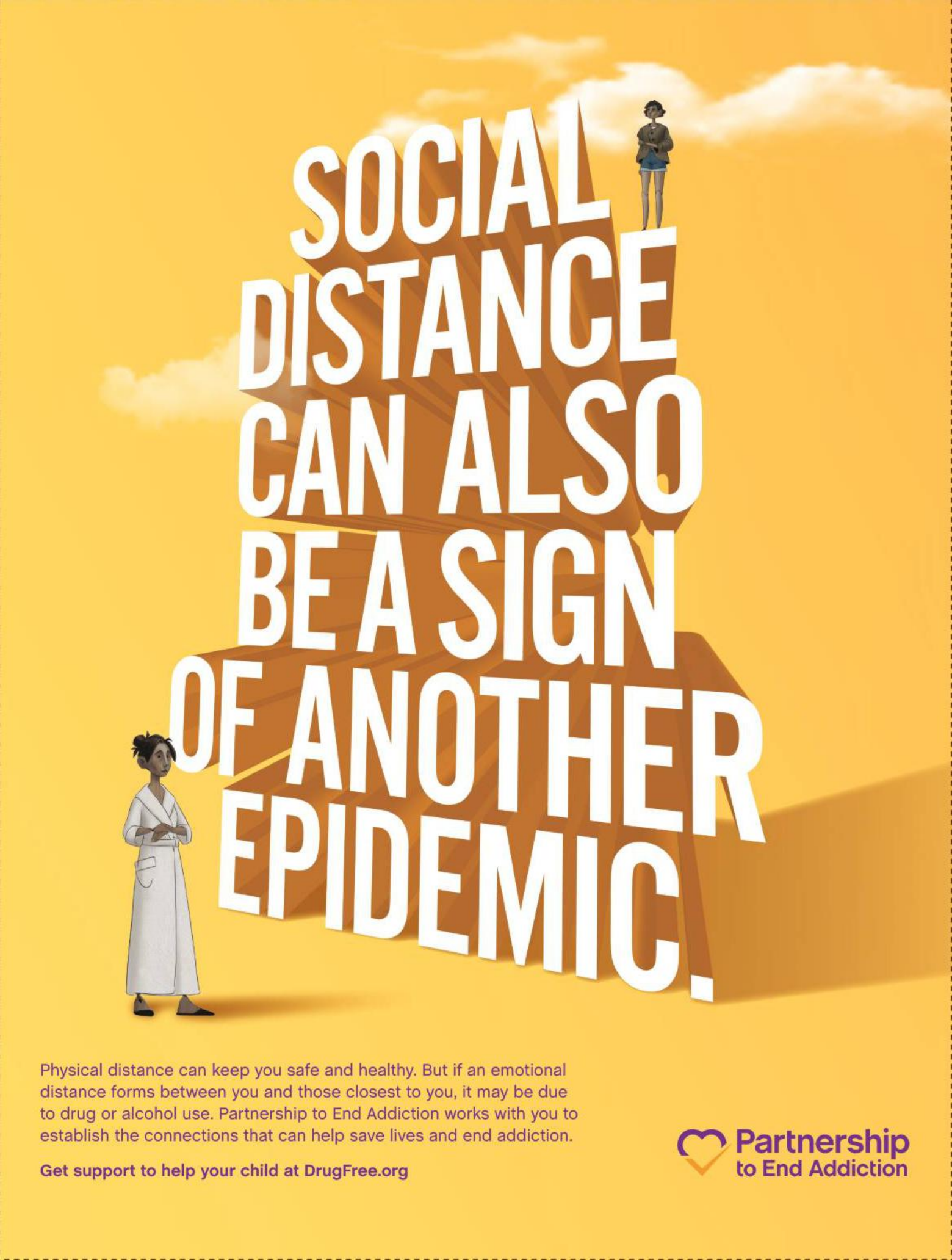
Cardiovascular risk from the accumulation of "bad" cholesterol in blood vessels is shifting from high-income Western countries, especially those in Europe, to low- and middle-income countries, particularly in East and Southeast Asia. A meta-analysis of 1,127 studies comprising 102.6 million people worldwide shows a significant drop in bad cholesterol from 1980 to 2018 in countries such as Finland, Belgium and the U.S. and a strong rise in Thailand, Malaysia, Nigeria and Malawi. Two major factors in high-income populations are less consumption of saturated fats and widespread use of lipid-lowering medications, notably statins, says study co-leader Majid Ezzati of Imperial College London. In lower-income populations, eating saturated fats is rising, and statins are not common. Researchers had limited evidence about an emerging shift a decade ago, but Ezzati is surprised to see "just how far it has gone."

Good and Bad

"Bad" cholesterol—low-density lipoprotein, or LDL—can build up inside blood vessel walls, narrowing arteries and raising the chance of stroke or heart attack. "Good" cholesterol—high-density lipoprotein, or HDL—can remove LDL as well as other minor forms of bad cholesterol. Isolating LDL in studies is difficult, so researchers typically assess HDL and non-HDL as the telltale factors.



SOURCE: "REPOSITIONING OF THE GLOBAL EPICENTER OF NON-OPTIMAL CHOLESTEROL," BY NCD RISK FACTOR COLLABORATION (NCD-RISC), IN *NATURE*, VOL. 582; JUNE 4, 2020



Physical distance can keep you safe and healthy. But if an emotional distance forms between you and those closest to you, it may be due to drug or alcohol use. Partnership to End Addiction works with you to establish the connections that can help save lives and end addiction.

Get support to help your child at DrugFree.org

Partnership to End Addiction

Too many people are getting injured playing hockey, and it's putting the game's future at risk.

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