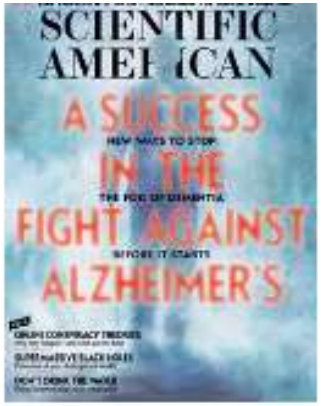
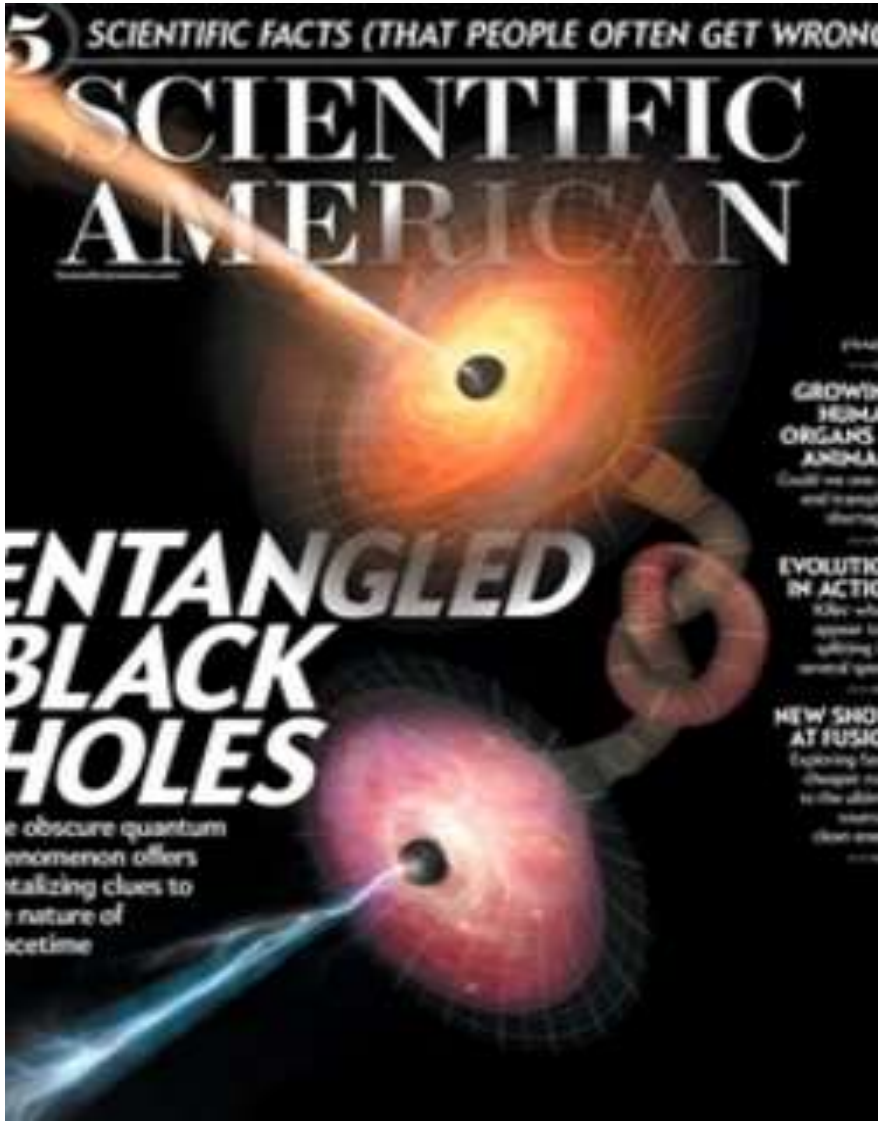


PULLING BACK THE CURTAIN:

Writing for SCIENTIFIC AMERICAN

Josh Fischman
Senior Editor

We didn’t always look like this…



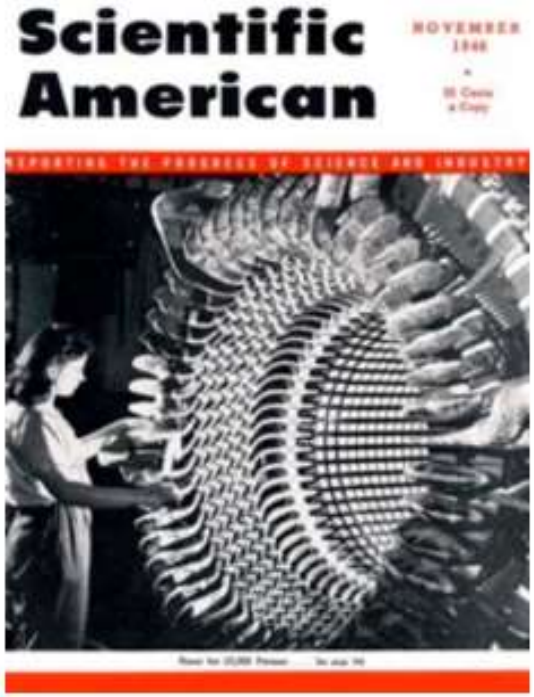
We used to look like this….

SCIENTIFIC
AMERICAN

September 11, 1845

And this….

SCIENTIFIC AMERICAN
December 29, 1900



SCIENTIFIC AMERICAN
November 1946

SCIENTIFIC AMERICAN
February 1950

Now its mostly this…

OCEANOGRAPHY

waves of destruction

Scientists chase towering seas that smash Arctic ice, with far-flung effects on climate and ecology

By Mark Harris

IN BRIEF

Arctic sea ice has been melting faster than predicted by global warming models, puzzling scientists.

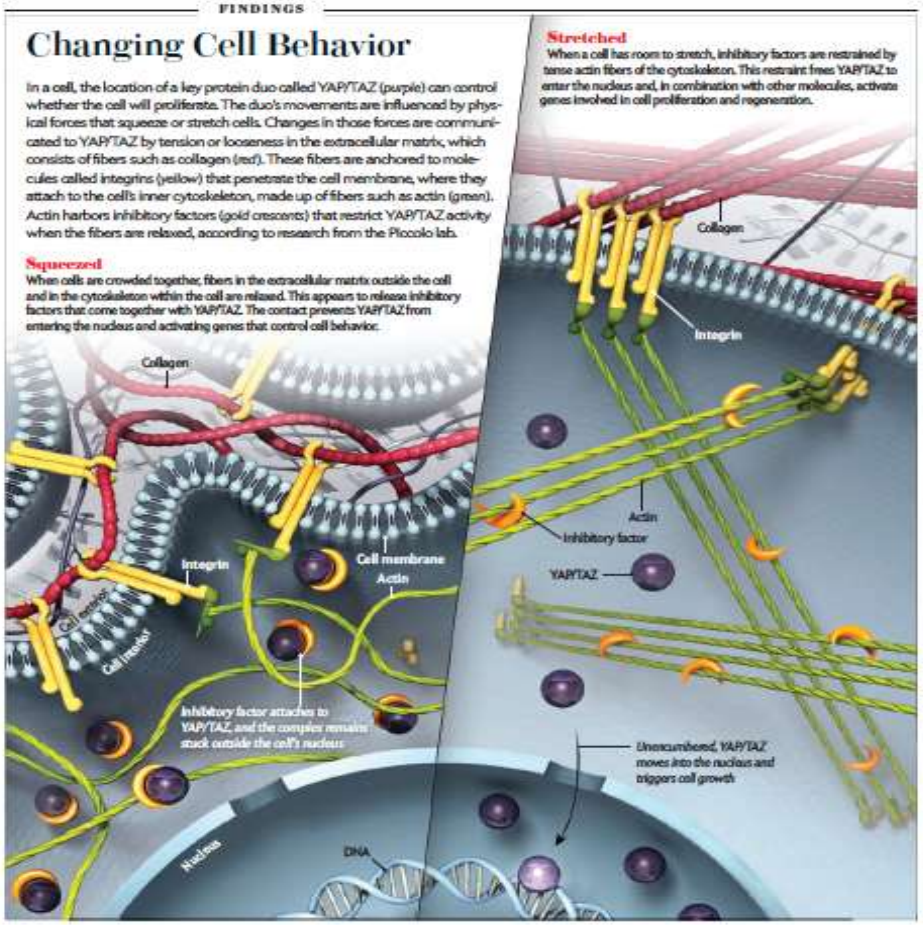
Giant waves, never seen before, may be the reason. A little ice melt gives waves room to grow large and powerful.

The large waves can smash still more ice as they crash around, creating more open water and more waves.

Newly opened Arctic seas may affect faraway weather patterns, erode coasts and challenge national security.

ROUGH SEAS: North of Russia, near the Arctic islands of Franz Josef Land, open waters—which had more ice cover in the past—toss up choppy waves.

And mostly this too…



mechanical forces and affect a cell's fate. A second is the different types of ground that a cell can encounter. The extracellular matrix to which cells are secured is indeed not monotonous but has different textures. Some tissues, such as bone, create a stiff, dense matrix, like solid rock. Other tissues, such as brain tissue or fat, develop a much softer version. In other words, each organ's matrix has its own signature.

These signatures appear crucial in organ development and regeneration. Notably, their differing mechanical properties guide the efforts of a very important cell type: mesenchymal stem cells. These cells are found in many adult organs and contribute to repair after an injury. They differentiate into a strikingly diverse array of cell types, including bone, fat, nerve and muscle cells. For years biologists assumed that the cocktail of

Which is one reason for this…

We reach over

9	0	0	0	0	4	8

people per month

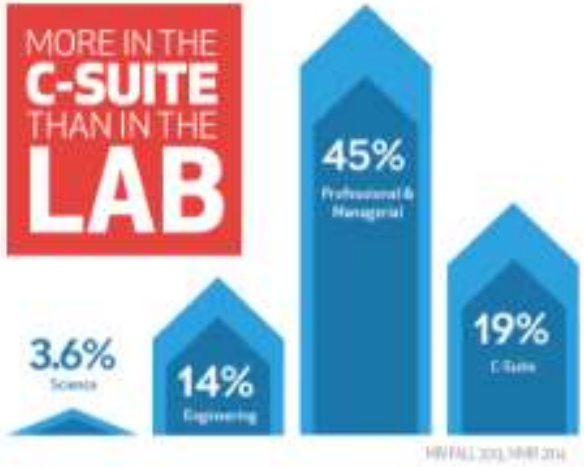
That's roughly 6 times the population of Manhattan.

SCIENTIFIC AMERICAN has 3.5 million print a
readers worldwide, 5.5 million global on
visitors monthly, a social reach of
translated into 14 languages.

They're also educated.

We're in the top 10 among MRI measured publications
for readers with college degrees and #4 in postgraduate education.

(MRI FALL 2013)



Which explains why 19% of them are in the C-Suite.

This is another reason…

158 Nobel Prize-winning scientists have written 251 articles for SCIENTIFIC AMERICAN.

It's not unusual to find Nobel-quality work in SCIENTIFIC AMERICAN months or years before it's recognized by the Nobel committees. This record of editorial excellence and prescience – covering such a diverse range of subject matter – is unequalled in publishing. All these prizes underscore the credibility and authority of SCIENTIFIC AMERICAN. And the excellence continues. In every issue of SCIENTIFIC AMERICAN, readers are likely to find the work of authors who are the Nobel laureates of tomorrow.

Most authors haven't gone to Stockholm…

Stefano Piccolo is a professor of molecular biology at the University of Padua in Italy. His laboratory studies how cells sense their environment and use this information to build tissues.

Frédo Durand works on computational photography as a professor of electrical engineering and computer science at the Massachusetts Institute of Technology.

William T. Freeman, a professor of electrical engineering and computer science at M.I.T., studies how machine learning can be applied to computer vision.

Michael Rubinstein is a research scientist at Google, working on computer vision. His motion microscope work was done while he was at Microsoft Research and M.I.T.

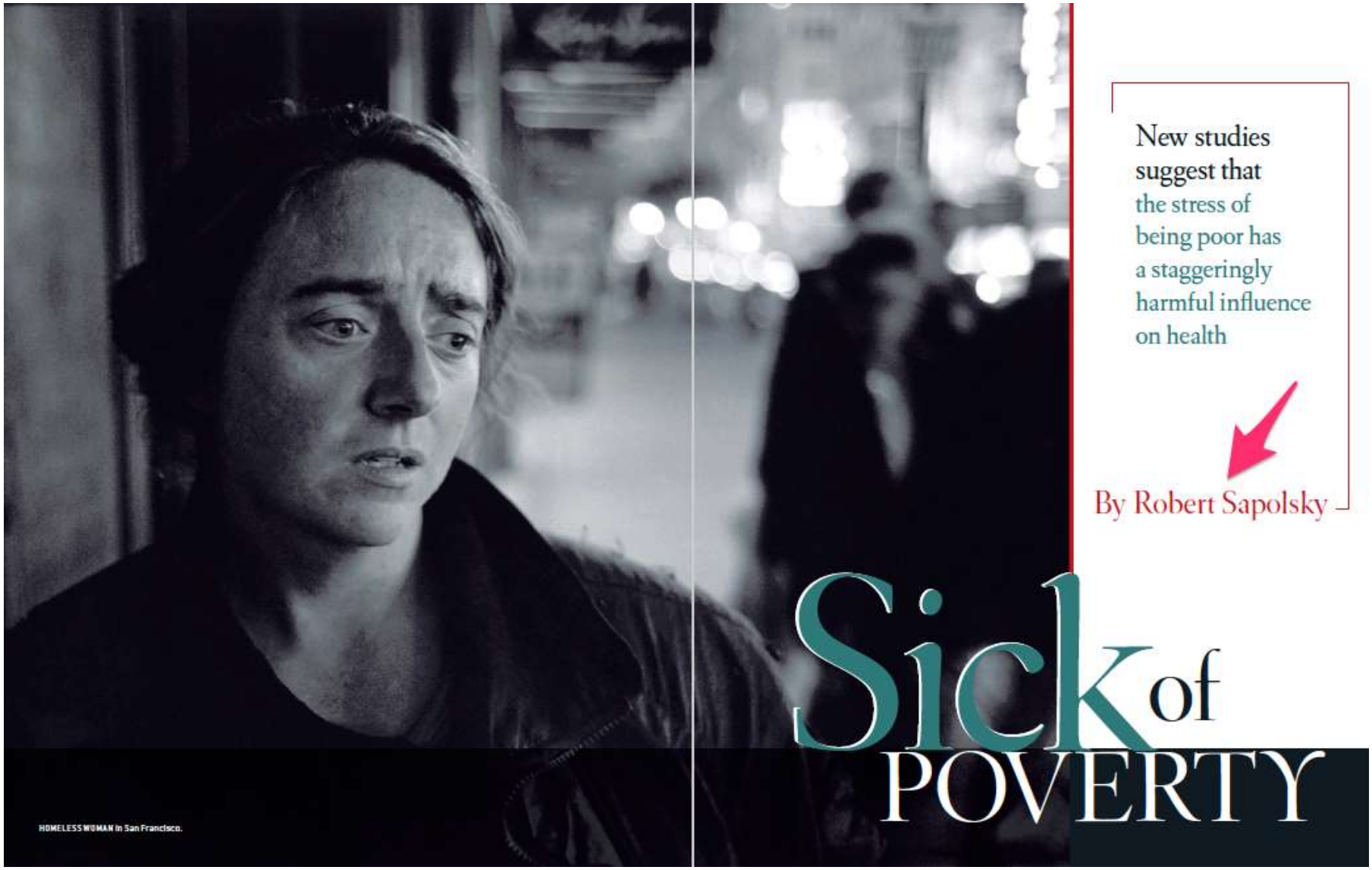
Robert J. Lee is an assistant professor in the department of otorhinolaryngology–head and neck surgery and in the department of physiology at the Perelman School of Medicine at the University of Pennsylvania. A molecular biologist, he has spent more than a decade studying cells that line inner surfaces in the nose and lung.

Noam A. Cohen is an associate professor in the department of otorhinolaryngology at the University of Pennsylvania. He is a surgeon with 15 years of clinical experience and director of rhinology research at the school.

Linda T. Elkins-Tanton, a planetary geologist specializing in the evolution of terrestrial planets, is director of the School of Earth and Space Exploration at Arizona State University.

About 60 % of our feature stories are written by scientists!

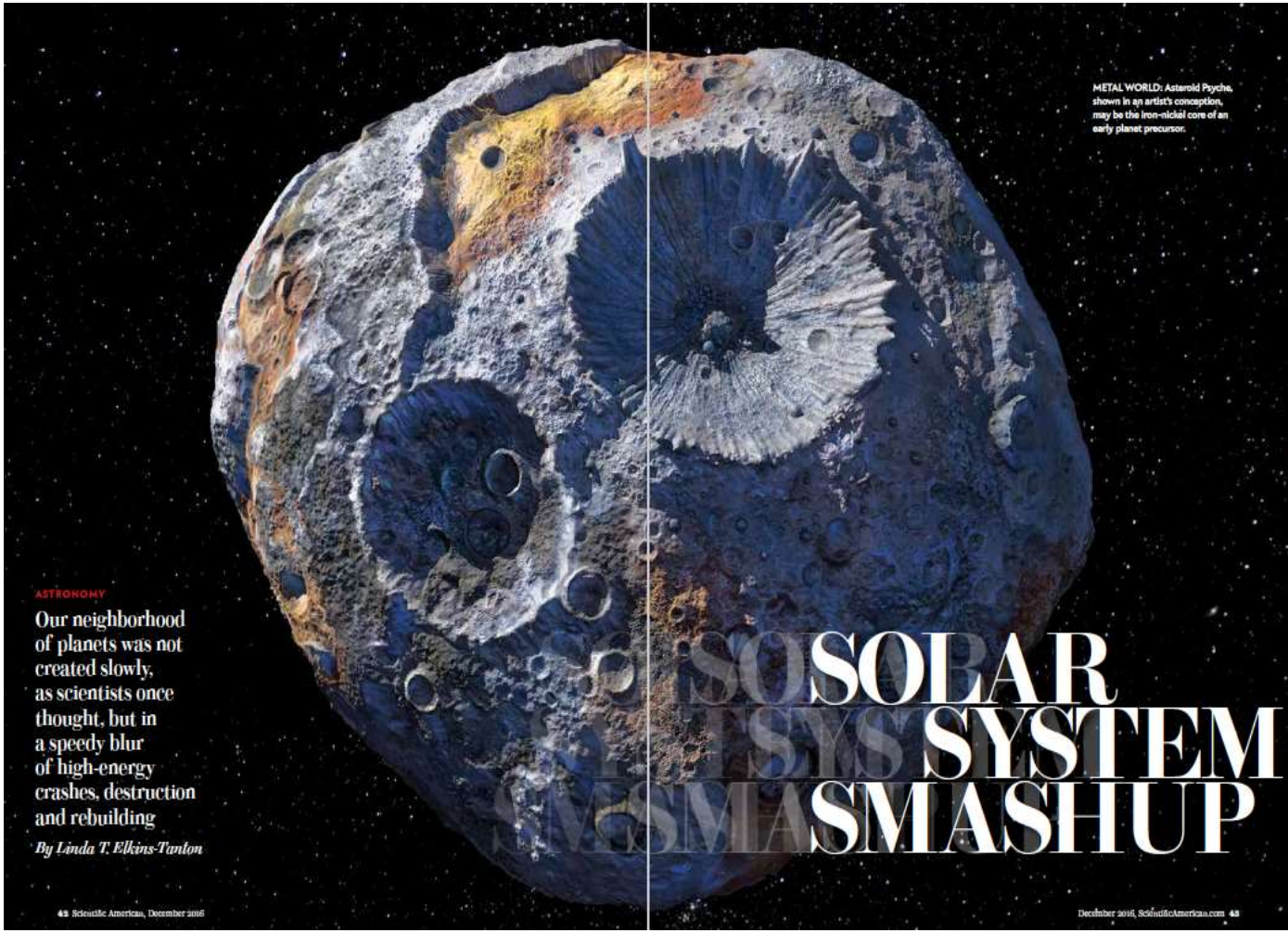
They are people committed to enthralling non-scientists with science



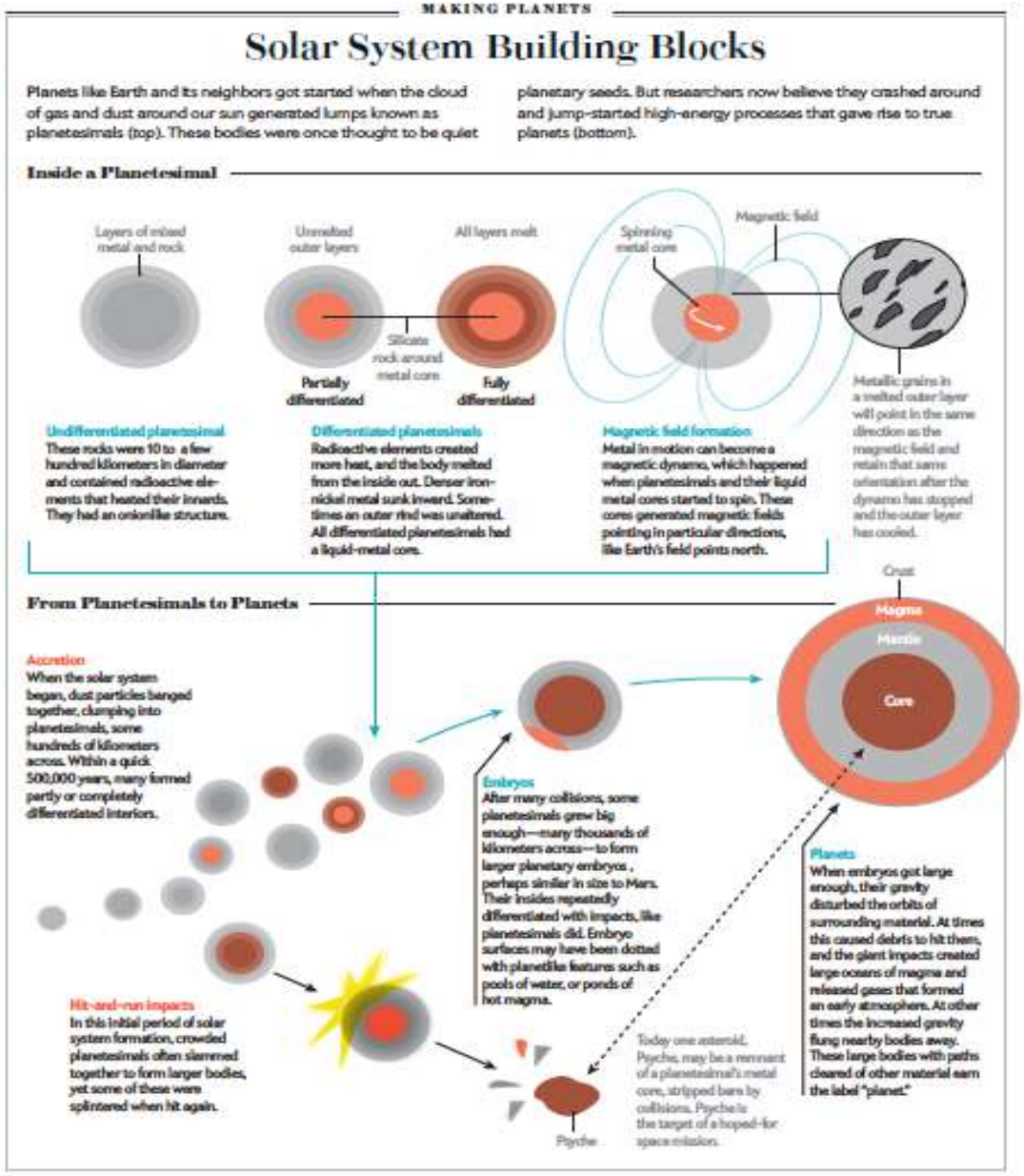
How do you get from this….

(That's "The accretion and impact history of the ordinary chondrite parent bodies" in *Geochimica et Cosmichimica Acta)*

…to this?



And tell readers about this?



Start here: scientist suggests story

SCIENTIFIC AMERICAN™

Article Proposal

from Lindy Elkins-Tanton, director, School of Earth and Space Exploration, Arizona State U.

Proposed article title/headline: The High-speed, Hit-And-Run Solar System

Primary Topic (Select ONLY one, with an X): Space/Physics X

Anticipated type: (Select ONLY one, with an X): Feature (1,800words or more) → X

Anticipated word count: 2500-3000

Proposed subtitle/dek: The story of building planets in our Solar System is not one of slow, graceful growth, as once believed, but a fast-moving tale of violent heat, giant impacts, and migrating spheres just two million years from the start, We can get an "inside view" of these processes by looking at the metal asteroid Psyche, which can reveal details of a core-mantle boundary like the one in the middle of Earth.

1. Editors ponder story, debate story, try and figure out why general readers would be interested in the story.

2. Author writes first draft of story.

3. Then the fun stuff happens

Authors get edits…

¶

STORY STARTS ¶

4/30/15 ¶

The high-speed, high-energy, hit-and-run Solar System ¶

¶

¶

[[Lindy, as I mentioned, you need a more engrossing, accessible start to the story to draw readers in. I think you can do this by moving the story of the Allende asteroid up from lower down. Go from the end of the Allende anecdote that I marked to this: ¶

~~The Universe began 13.8 billion years ago ago with the Big Bang, an unimaginably violent, unimaginably rapid expansion of matter and energy.~~ ¶

¶

Sometimes its more, um, involved.

[[I appreciate the basic background but what's smarter is to start with something that gets you faster to what the story is really about: the search for enhancers, and how distant parts of DNA affect one another. because of structure. So I suggest cutting this background and starting off with that nice bit about scientists being bummed they didn't have more genes than a worm, and they realized the other DNA must be important, and needed some structure that allowed distant parts to regulate other parts.]]~~DNA is a twisted ladder. It is made up of two complementary strands, each comprising a sequence of four chemical letters, or bases – Adenine (A), Cytosine (C), Guanine (G), and Thymine (T). Every rung of the ladder is a pairing of these bases: always A with T, always C with G. So it's easy to replicate: By breaking the bonds that hold the base pairs together, the strands can be pulled apart, and the unpaired bases along each strand can be used as a template in order to fill in the missing strand. Together, the bases of the double helix comprise the genetic code, or genome, that orchestrates the existence of every living thing.~~ ¶

~~The discovery of the double helix in 1953 raised a series of questions which have influenced biological research ever since. Like: what did the letters mean? It took about a decade or so for scientists to determine how the letters in certain stretches of DNA, called genes, encoded the instructions for making proteins, the biochemical machines that are responsible for doing nearly all the work of the cell. They found that genes are made up of codons, groups of three consecutive base pairs, like A-A-A. Each codon specifies a particular amino acid, one of the chemical building blocks from which proteins are made. For instance, A-A-A indicates the amino acid Lysine. Together, the codons in a gene are a recipe for making a protein.~~ ¶

~~But, in decoding genes, scientists had only just begun to unravel DNA's secrets.~~ ¶

~~It soon became clear that, structurally, DNA was more than just a double helix. Efforts led by Roger Kornberg in the early 70s showed that short stretches of the double helix – hundreds of base pairs in length – wound about a combination of proteins called "histones" to create a structure called the nucleosome.~~ ¶

~~It also became clear that DNA was more than just genes. Scientists began to find regulatory elements, bits of DNA sequence that can control a gene, even if they aren't part of the gene itself. One important class, called "promoters," work like the buttons on your television: they sit at the front of every gene, and turn it on or off. A second class, called "enhancers," were more mysterious. They worked like a remote control, activating genes that lay millions of base pairs away. The discovery of enhancers was totally unexpected, and no one knew for sure how they worked. The most plausible explanation was the existence of yet another, hitherto unseen DNA structure. Perhaps, at scales far larger than the nucleosome, DNA might loop back on itself, bringing the enhancer close to its target gene?~~ ¶

~~But these questions of structure would have to wait. By 1977, a team led by Frederick Sanger had chromosomes, that comprise the 6 billion letters of the human genome. The effort would take decades, during which thoughts of DNA looping drifted increasingly far from the limelight.~~ ¶

¶

~~The draft sequence of the human genome – announced in XXX 2000 – was, of course, a titanic achievement. But it was also a huge surprise: it didn't resemble any of the smaller genomes that had been sequenced before. Ninety-eight percent of our genome had no obvious function. The remaining 2% comprised only 20,000 genes. This may sound like a lot, but it was, by all accounts, a downer: It put *homo sapiens* roughly on a par with the 1mm-long roundworm *c. elegans*.~~ ¶

~~This startling observation had profound implications. For nearly half a century, S~~scientists used to believe they were ~~had thought that the secret of why scientists are~~ so much more clever than ~~round~~worms because, among other things, ~~is that we~~ humans have a bigger genome with room for more genes. Then, early this century, geneticists finally got around to counting. The results were kind of a downer. Ninety-eight percent of our genome had no obvious function. The remaining 2 percent~~s~~ comprised only 20,000 genes. This ~~may sound like a lot, but it was, by all accounts, a downer: It~~ put *homo sapiens* roughly on a par with the 1mm-long roundworm *c. elegans*. [[in absolute number of genes or percentage of genetic DNA?]]¶

~~But that obviously wasn't so. Instead, scientists realized,~~ Then we[[you are part of this effort, right?]] thought of something that made us feel better. Ggenes were like instruments in a grand orchestra. ~~What made us human was the music they made: how genes turned on and off in a complex, coordinated fashion in order to direct heart cells to beat, neurons to think, or immune cells to fight infection.~~ Buried in the vast spaces between genes – the 98 percent~~s~~ of the genome we could not account for – was a grand musical score, a ~~gene regulatory~~ program for controlling our genes ~~far more complex than anything we had ever contemplated~~. What made us human was thise music ~~they made~~: ~~how~~ genes turninged on and off in a complex, coordinated fashion in order to direct heart cells to beat, neurons to think, or immune cells to fight infection. ¶

Before everyone caught sequencing fever, a few researchers in the 1960s [[or 1970s]] had taken some note of these orchestral conductors. One important class of non-gene DNA, called "promoters," appeared to work like the buttons on your television: they sit at the front of every gene, and turn it on or off. A second class, called "enhancers," was more mysterious. Enhancers activated genes that lay millions of base pairs away. Perhaps, at scales far larger than the windings of a double helix, DNA might loop back on itself, bringing the enhancer close to its target gene. ¶

~~Figuring it out was the obvious next step.~~ ¶

~~Now, if all genetic regulation took place at promoters, this might have been pretty easy. You could just~~

Sometimes there are just a few tweaks

we can turn our tsunami of data into accurate 3-D images of a molecule. ¶

¶

¶

Step by step, we improved our technique. And by 2014 **[[ok? Need some date advancing beyond 2009-jf]]** our work gave us the first real-time look at the transfer of electrons between two key players in photosynthesis: the large sunlight-catcher photosystem I and a protein called ferredoxin. ¶

¶

When light hits photosystem I, it converts the light into electrons, which ferredoxin then carries away for further reactions that build molecules. At this point, the crystallized proteins quickly dissolve, making the reaction difficult to follow. Only the superfast process of SFX[9] could see the rapid change. ¶

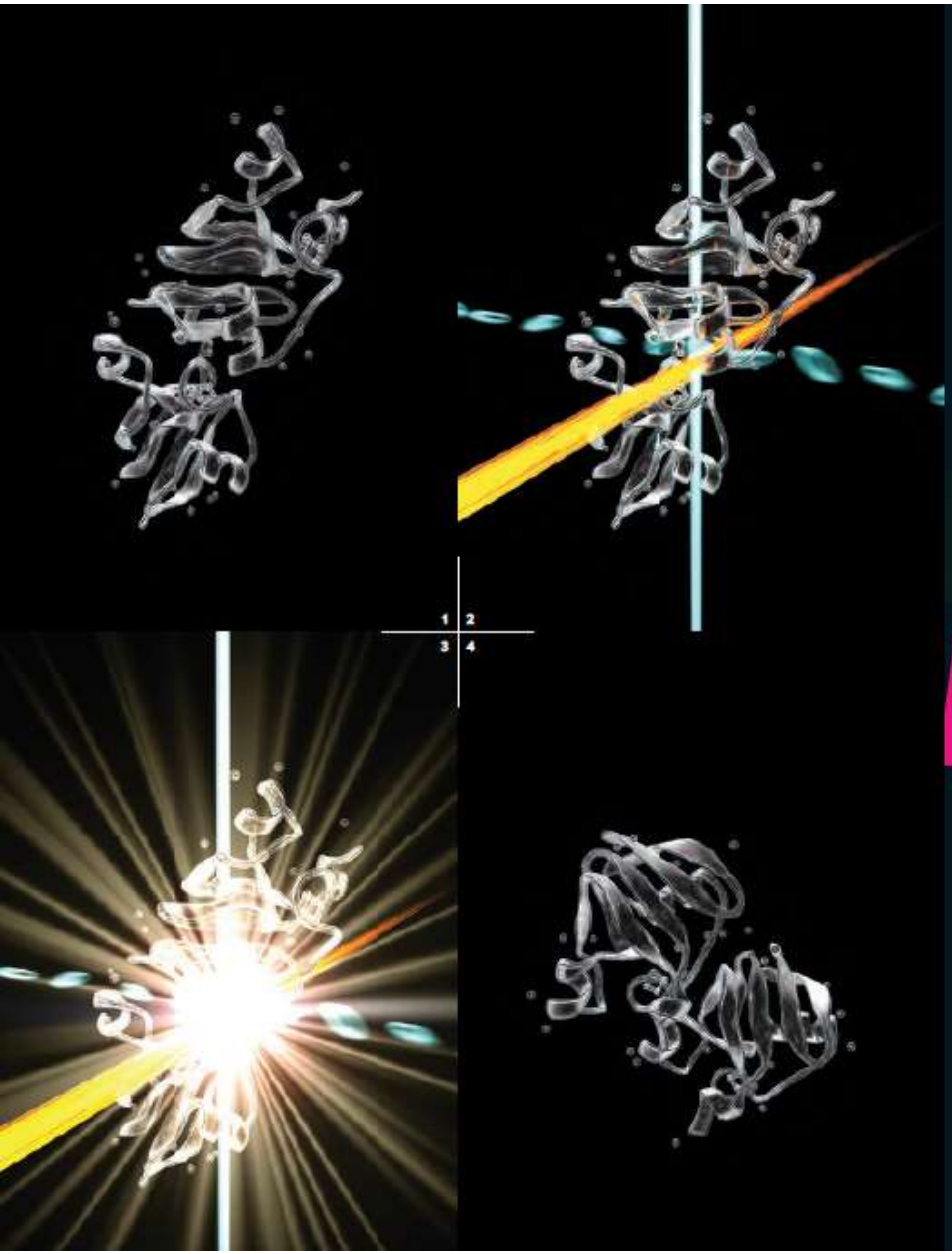
¶

Not reaching out to your readers, and using language you and your fellow specialists understand but non-scientists do not, is a prime reason for lots of red ink

When you spend the time to think about how your readers perceive your words, and meet them on their level and not force them to struggle to yours, you combine the smarts of a scholar with the craft of a storyteller.

You don't **dumb down**. You **wise up**. You combine accessibility with knowledge.

You are able to tell people why what you do matters.



CHEMISTRY

SPLIT-SECOND REACTIONS

New images of drug proteins or photosynthesis in action, in millionths of a billionth of a second, show how the molecules work—or fail

By Petra Fromme and John C. H. Spence

IN BRIEF

Proteins are in constant motion, carrying out the reactions that make life possible. These movements happen on a scale too small, and too fast, to be seen with microscopes.

Using x-ray laser pulses lasting just millionths of a billionth of a second, researchers have created "molecular movies" that show how proteins change structure when they interact.

These movies can reveal biological reactions in unprecedented detail and show why drugs sometimes do not hit target proteins and how plant photosynthesis creates clean energy.

Illustration by Bryan Christie

Petra Fromme is Paul V. Galvin Professor in the School of Molecular Sciences and director of the Center for Applied Structural Discovery at Arizona State University.

John C. H. Spence is Richard Snell Professor of Physics at Arizona State and director of science at the BioXFEL Science and Technology Center.

BURROWED DEEP UNDER THE FOOTHILLS NEAR PALO ALTO, CALIF., SCIENTISTS SCURRIED THROUGH AN UNDERGROUND LABORATORY, making final preparations for a series of explosions. ***THEIR PLAN:*** blow up tiny crystals of proteins that could reveal one of nature's best-kept secrets—how plant photosynthesis turns light into chemical energy. The potential payoff: a step toward unlimited clean power.

It was December 2009, and a sleep-deprived team of researchers and students at SLAC National Accelerator Laboratory had been working nonstop for days to set up this experiment at the world's most powerful x-ray laser, the Linac Coherent Light Source (*LCLS*), which accelerates electrons to nearly the speed of light. One group feverishly adjusted injectors that would shoot tiny crystals of protein into the x-ray beam. Another locked and loaded the injector with fresh crystals of a protein complex called photosystem I, which is key to photosynthesis.

At the end of the two-mile accelerator tunnel, the crystals began their march into the intense laser light. But before each of them exploded, its snapshot was taken with a newly developed scientific technique. Today that method promises to reshape our understanding of biology on the tiniest scale because we can now assemble a rapid sequence of such images—shot in femtoseconds, or millionths of a billionth of a second—into movies.

Physicist Richard Feynman once said, "Everything that living things do can be understood in terms of the jigglings and wigglings of atoms." But never before have we been able to directly see the wiggling of atoms and molecules within living things at this speed. Our method, called serial femtosecond crystallography (SFX), lets us watch high-speed molecular dances that determine how medicines affect diseased cells and how chemical reactions convert energy to different forms.

Already research teams around the world have used SFX to reveal fine details of how an experimental drug regulates blood pressure—paving the way to better hypertension medications. SFX has also shown the structure of the enzyme that destroys red blood cells in sleeping sickness, a fatal disease caused by parasites. And it has yielded the first look at the initial steps during photosynthesis that split water into hydrogen and oxygen.

Back in that underground lab in 2009, the stakes were high as x-ray pulses began to annihilate our carefully formed crystals. Many scientists had said SFX would never work and rejected our requests for funding. But then beautiful images of scattered x-rays began to emerge on computer screens. We still remember our cheers erupting around the room as we watched what would become proof that a new field of x-ray science had been born.

X-RAY VISION

BEFORE SFX, scientists made amazing advances in detecting the changes of certain chemical structures, but they could not actually observe the most delicate and complex biological structures in action. In the 1980s, for instance, the late chemist Ahmed H. Zewail invented a way to watch atoms move during chemical reactions using ultrafast pulses of visible laser light. Yet the light's wavelength was too long to distinguish the tiniest details of protein structure. More recently, dramatic advances in microscope technology have produced near-atomic-resolution images of proteins and viruses. But they are not quick enough to capture rapid reactions such as photosynthesis.

We decided to use x-rays, which have the necessary speed and resolution to record biological reactions in action. Key to our work was developing a technology that would allow x-rays to form pictures of molecules in the instant before destroying them. Traditionally scientists who do this work painstakingly grow large crystals of proteins and other molecules into large crystals to map the positions of atoms within them. Then they bounce x-rays off the crystals and record the pattern of x-ray scattering, or diffraction. In a crystal, molecules are held in place in an orderly arrangement, so the x-rays scatter in predictable ways, allowing scientists to interpret the position and identity of atoms. This method is called x-ray crystallography, and our serial femtosecond crystallography uses the same principle to see atomic structure but far faster.

X-rays ultimately destroy the molecules we are trying to see, however. It was commonly believed that the x-ray laser, which concentrates high-energy x-rays into a powerful beam, would only make matters worse. The laser's bright light alone can punch a hole through steel. A fragile biomolecule, one would think, would not stand a chance. We needed to outrun the x-rays' damage and capture an image in femtoseconds. For perspective, the difference between one femtosecond and one full second is equivalent to the difference between a second and 32 million years.

The key to the SFX technique lies in that imperceptible sliver of time between the molecule being struck by the x-ray laser pulse and electrons being ripped off its atoms by x-ray energy. Stripped of electrons, the positively charged remnants repel one another, causing the molecules to expand and ultimately explode.

Here is how it works: First, we prompt molecules to interact to form a tiny crystal. Then we shoot a powerful x-ray beam at the crystal in an extremely short pulse, just long enough for some of the x-rays to scatter off the crystal before the beam's energy rips the molecules apart. Finally, a detector captures the bounced x-rays, whose pattern reveals the type and position of

PLANT CHEMISTRY

Moviemaking on a Molecular Scale

Photosynthesis makes life on earth possible by converting sunlight into chemical energy. A new kind of molecular movie has given scientists their first glimpse of the process in action. Researchers use visible light, simulating sunlight on a leaf, to spur proteins to begin photosynthesis, then use a powerful x-ray laser to take snapshots of changes in these proteins in the fractions of a second before they are destroyed. Snapshots are made in five steps (shown below) and combined into a movie.

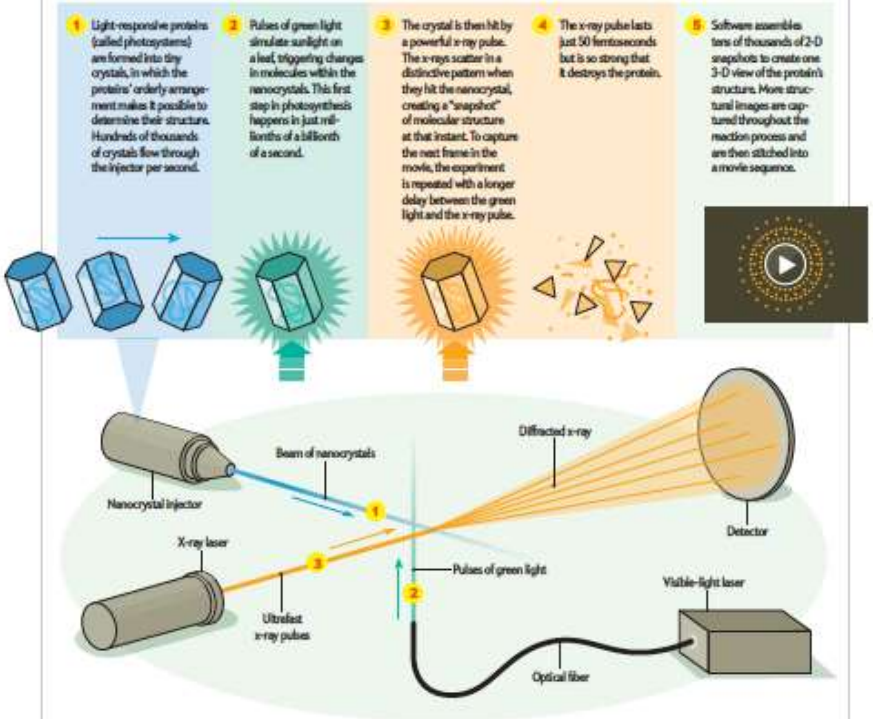


Illustration by Lucy Reading-Ikkanda

the nitric oxide produced by supertaster nasal cells during T2R38 activation caused faster ciliary beating and directly killed more bacteria than nontaster nasal cells. We next discovered that the same class of bacterial compounds that were previously shown to activate mouse nasal chemosensory cells, AHLs, directly activates human T2R38 receptors. Nasal cells from supertasters detect bacterial AHLs through T2R38 and produce nitric oxide, whereas cells from nontasters do not. These properties make cells from supertasters much better at killing AHL-producing bacteria than cells from nontasters. From these observations, we concluded that the T2R38 bitter receptor is used by airway epithelial cells to detect bacterial activity and activate defenses.

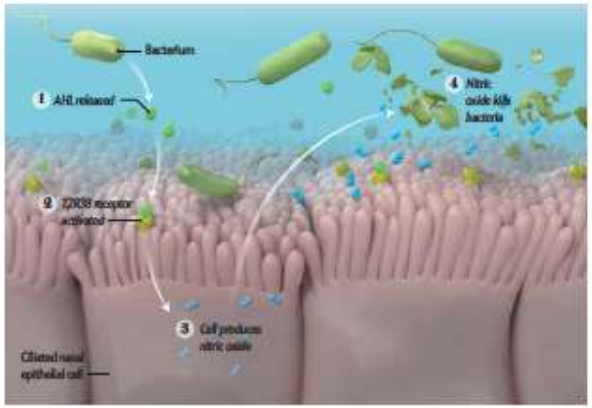
IMMUNE RESPONSE

Two Defense Systems That Use Bitter Receptors

Cells in the human airway play roles in defending the body against invading bacteria. Two of these cell types (diagrams) have been shown to use bitter taste receptors in different ways to detect and repel the invaders.

Hair-Trigger Reaction

Bacteria of the gram-negative class, when they infect the nose, release chemicals called acyl-homoserine lactones, or AHLs (1). This chemical is detected by bitter taste receptors from a group called T2R38s, which sit in hairlike projections called cilia that extend from cells on the inside surfaces of the nose (2). The cells, known as nasal epithelial cells, release a gas, nitric oxide, in response (3). The gas diffuses into the bacteria, killing them (4). Cilia on the cells also beat back and forth, flicking bacteria away.



Start and Stop

Other cells, called solitary chemosensory cells, have both bitter taste receptors (T2Rs) and sweet taste receptors (T1Rs). Infectious bacteria release a compound that contacts bitter receptors (1), and then the cells release calcium (2). The calcium signals nearby cells to release compounds called defensins (3). Defensins hurt and kill bacteria (4). Then sweet substances such as glucose (5) increase because they are no longer eaten by bacteria. Glucose is detected by sweet receptors (6), which reduce bitter receptor activity, probably preventing an overreaction.

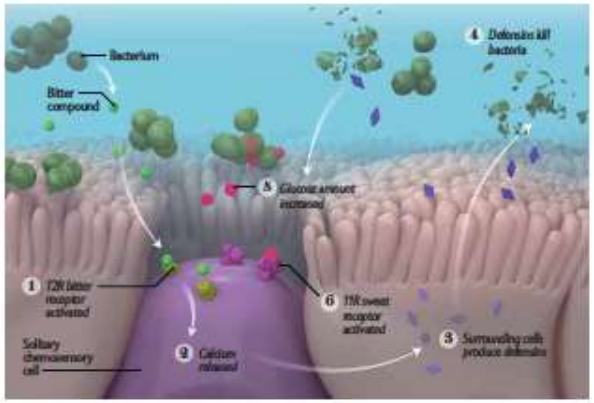


Illustration by ATA Studio

Since our discovery of T2R38 in cilia of human nasal epithelial cells, our knowledge of the role of taste receptors in the nose has expanded even further. These receptors also appear in solitary chemosensory cells in the human nose, similar to those found in mice. Solitary chemosensory cells really are solitary, dispersed widely throughout the nasal cavity and making up about only 1 percent of the cells there. The cells have both T2R bitter receptors and T1R sweet receptors. When T2Rs in these cells get stimulated, we have found, the solitary cells release a signal to surrounding cells that prompts them to release antimicrobial proteins called defensins into the airway mucus. Defensins are capable of killing many illness-causing bacteria, including *P. aeruginosa* and MRSA.

The sweet taste receptors, when stimulated, shut down the activity of the bitter ones, probably to prevent cells from releasing too many proteins at an inappropriate time. Sweet receptors had already been found in other body parts, such as the pancreas, where they sense sugars in the blood and stimulate cells to produce insulin that regulates glucose levels. Our work on the nasal cells, however, showed that sweet and bitter receptors in the same cell have opposing roles.

These experiments suggest to us that taste receptors constitute an early-warning arm of the airway immune response. They seem different from the most well-studied class of early-warning proteins, which are known as toll-like receptors (TLRs). The TLRs also activate immune responses when stimulated by certain bacterial molecules, as the T2Rs appear to do. But there is at least one important difference: some TLR responses—such as signaling genes to start creating antibodies that mark invaders for destruction—are much slower, taking several hours or even days. T2R38 and its bitter cousins, in contrast, produce responses within seconds to minutes. These taste receptors might be most important during the onset of infection by activating a kind of "locked and loaded" instant reaction. Other immune receptors may be more crucial for fighting a prolonged infection, calling up the troops when the first response is not sufficient.

VULNERABLE PEOPLE

THE LARGE NUMBER of genetic varieties in T2R bitter taste receptors makes their role in immunity even more intriguing. Most of the 25 bitter receptors have genetic variations that increase or decrease their abilities, thus making people who have them more or less sensitive to bitter-tasting substances. If a reaction to bitterness is indeed part of the immune response to invading bacteria, these same genetic variations may also create differences in the way people combat infections. Increased bitter receptor function may confer greater protection against infection, whereas lower function may increase susceptibility.

We have begun to test this idea in people and have hints that it is correct. The millions of patients with chronic rhinosinusitis constitute a natural test population and a group in need of help. When given quality-of-life questionnaires, rhinosinusitis patients report worse scores than patients with several heart and lung diseases. Plus, rhinosinusitis patients can develop dangerous lung infections and exacerbate lower airway diseases such as asthma. We have looked at microbiology cultures from patients with the condition. Supertasters did get rhinosinusitis—they are not immune—but they had a much lower frequency of nasal infections with gram-negative bacteria than did nontasters. That makes sense because gram-negative bacteria produce AHLs, the compounds that, by triggering receptors, lead cells in these people to release microbe-killing nitric oxide. Other bacteria do not produce AHL, so they would not run afoul of these immune defenses.

Further clinical research has supported the role of T2R38 in sinusitis. Two studies from our group at Pennsylvania demonstrated that people with two copies of the T2R38 supertaster gene are less likely to get severe rhinosinusitis than are patients with two nontaster copies or even patients with one copy of each. A study by otolaryngologist Martin Desrosiers of CHUM in Montreal and his colleagues verified that the T2R38 nontaster gene occurs more often in patients than in healthy people. That study found that rhinosinusitis severity is also associated with variants in two other T2R receptor types, T2R14 and T2R49.

In organs beyond the nose, connections between taste receptors and immunity are starting to show up. In 2014 scientists showed that when confronted with pathogenic *Escherichia coli*, chemosensory cells in the urinary tract use T2Rs to stimulate the bladder to release urine. This could be the body trying to flush bacteria out and prevent bladder infections. Another recent study has shown that white blood cells—which include neutrophils and lymphocytes and are crucial components of the immune system—also use T2R38 to detect *Pseudomonas* AHLs.

Right now we want to learn whether chemicals that activate T2R receptors can work as medicine for rhinosinusitis patients by stimulating stronger bacteria-killing responses. The vast array of bitter compounds in foods we eat and drink every day could be potential therapeutics, including humulones and lupulons from hoppy beers, isothiocyanates from green vegetables such as Brussels sprouts and bitter chemicals from citrus such as limonin. Absinthin, a bitter chemical isolated from the wormwood plant and found in the liquor absinthe, has been shown to stimulate solitary chemosensory cell T2Rs. In our lab, we are investigating several formulations that could work as drugs. Novel medications based on bitter compounds might someday be used to combat infection without using conventional antibiotics.

We believe it is also possible that taste or genetic testing of T2Rs might eventually be used to predict susceptibility to infections. The natural variations in these taste receptors may help us answer an age-old question: Why do some people frequently get respiratory infections, whereas others never seem to get sick? Using bitter receptors to solve this puzzle would be sweet indeed.

MORE TO EXPLORE

Taste Receptor Signaling—From Tongues to Lungs. S. C. Kinnamon in *Acta Physiologica*, Vol. 204, No. 2, pages 158–168; February 2012.

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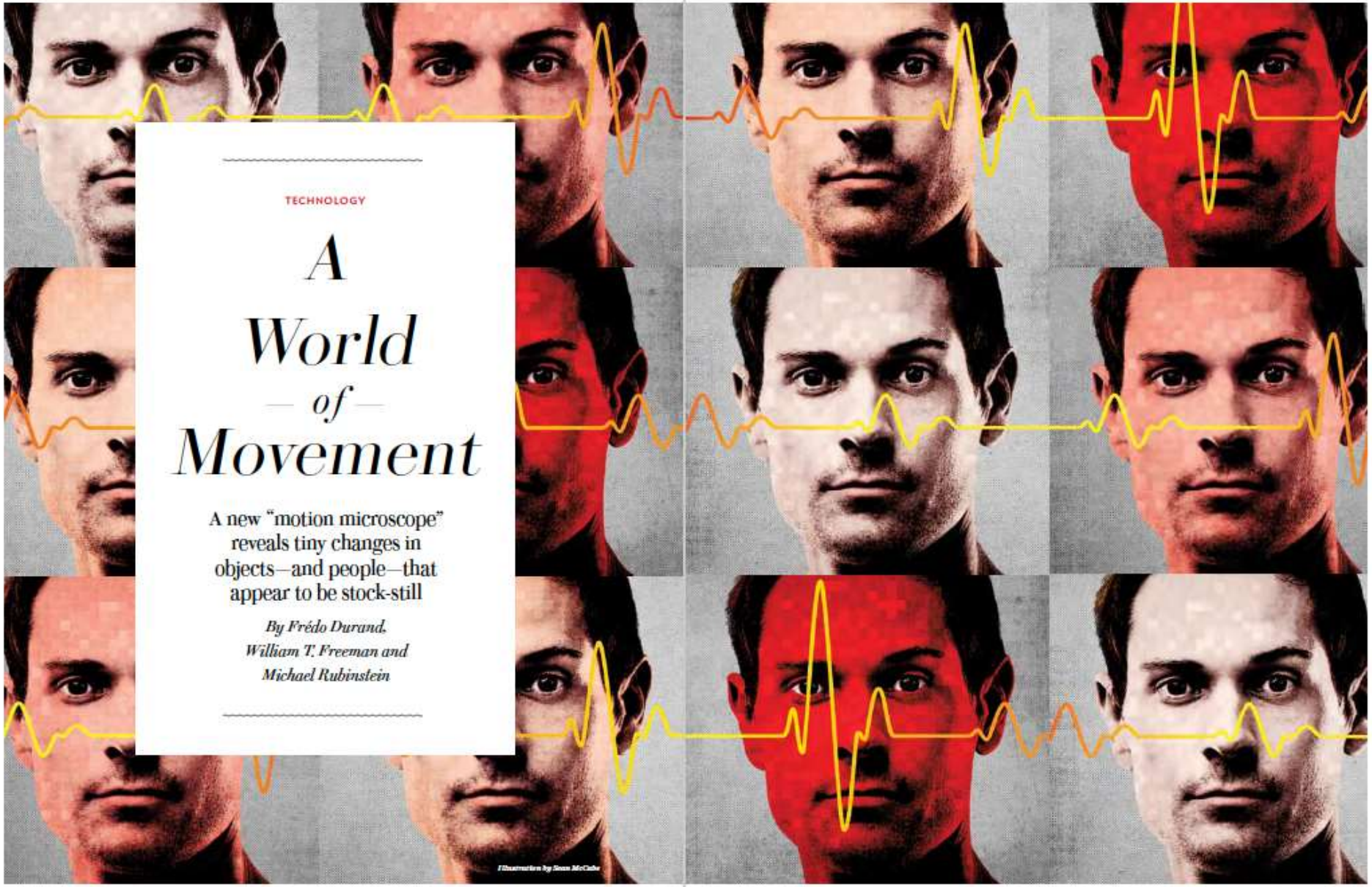


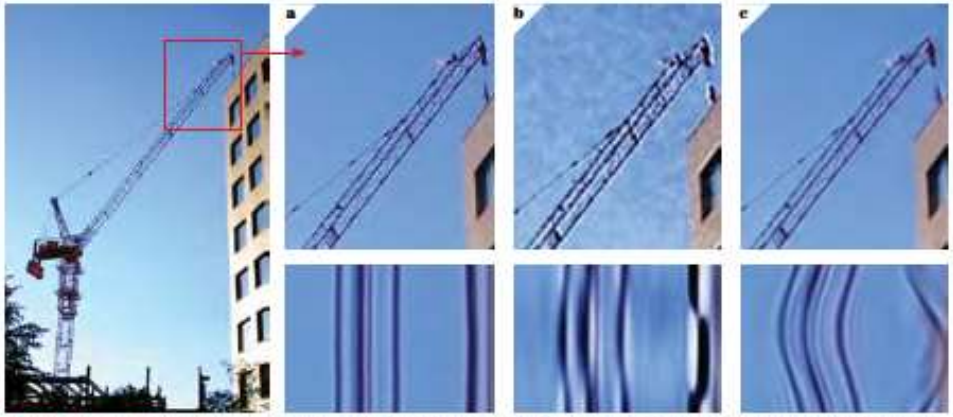
TECHNOLOGY

A World of Movement

A new "motion microscope" reveals tiny changes in objects—and people—that appear to be stock-still

By Frédo Durand, William T. Freeman and Michael Rubinstein

Illustration by Sean McCabe



MOVING TARGET: This construction crane seems motionless (*a*). Amplifying video color changes reveals swaying, but pixels look jagged (*b*). A computer smooths pixel transitions, showing motion (*c*). The bottom images show one crane feature moving over time.

called color gradient. Mathematically, we can say that the change of a pixel's color over time is the product of the speed of the object multiplied by this color gradient.

Our algorithm, of course, does not know about speed or color gradients. Nevertheless, because it amplifies the color change at any particular point as the ball moves a fraction of an inch to the right, it also amplifies that fractional motion of the ball for your eyes to see. In a similar way, the colors of pixels representing specific points on a baby's chest will change as the baby breathes, and making the color change more dramatic also makes the tiny movement of the chest more obvious.

A FLUID LOOK

THE DIFFERENCES BETWEEN our earlier work that used vectors and our new approach based on color changes over time are a matter of perspective. It is the difference between going with the flow and standing still amid the current, and that change in viewpoint is what makes our newer calculations simpler to do. The idea comes from scientists who watch fluids and model how they move. There are two contrasting ways to do this: Lagrangian and Eulerian methods. Lagrangian approaches track a given portion of fluid as it travels through space, like an observer on a boat following the flow of a river. In contrast, Eulerian approaches use a fixed location in space and study the fluid passing by it, as if the observer was standing on a bridge.

Our earlier work followed a Lagrangian philosophy, acting like the observer on a boat, where pixels are tracked in the input video and then moved—as the boat moves—according to magnified vectors from point to point to point. In contrast, our new approach considers color changes only at a fixed location, similar to the observer who stays on the bridge. This local perspective applies to only small motions but makes it much simpler and robust. A computer can quickly process a video using this technique, whereas our earlier work required a lot of computer-processing time and often contained mistakes.

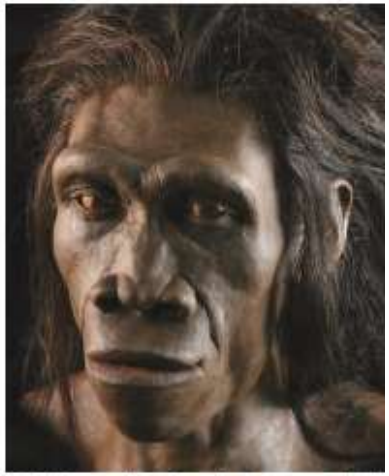
In 2012 we published a paper on this new method, called Eulerian video magnification. It showed how the blood flow changed a face. It also contained a variety of other examples, such as the breathing motions of an infant, which can be amplified so that parents of newborns could check an enhanced video signal to see if the baby was moving. We also took a high-speed video of a guitar where all the strings were vibrating and selected narrow bands of frequencies around given notes, such as 72 to 92 hertz for a low E string vibrating at 82 Hz. This amplified the motion of a single string, whereas the others looked like they were absolutely still.

We created a Web site where people could upload their videos and run them through this motion-magnification process. (See the videoscope at https://videoscope.qrilab.com.) People used it in ways we had not thought about, which was exciting. One person posted a video showing fetal movements in a late-term pregnancy. Another person amplified the breathing motion of her pet guinea pig. An art student made a video showing the imperceptible movements and expressions of her friends trying to stay still.

We also learned, however, that our Eulerian approach does have limits. If a given input pixel gets much darker from one frame to the next, the computer will enhance this change to an excessive degree, producing a fully black pixel, kind of a runaway amplification effect. This type of issue can cause dark or bright halos around motion areas. Input color variations from sensor noise are also a challenge because—even though we smooth them out by averaging many local pixels—the noise still gets magnified.

This result prompted us and our graduate student, Neal Wadhwa, to develop a new algorithm that preserves the benefits of simple Eulerian approaches but provides a better view when changes get more extreme.

We realized that the root of our original method's limitations is a false assumption. It acted as if the color difference between each pixel and all its neighbors—pixels to the left, the right, above, below—was the same, which unfortunately is not always true. Edges, for example, correspond with much bigger pixel differences (higher gradients) than their surrounding smooth areas. So if

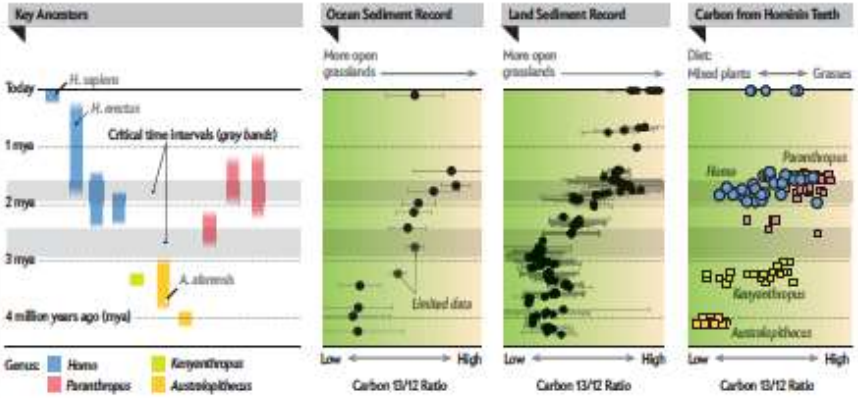


STONE AGE EATING: A distant ancestor, *Paranthropus boisei* (*left*), lived in open plains and mostly ate grasses or related f as indicated by chemical analysis of fossil teeth. But *Homo erectus*, sometimes called *Homo ergaster* (*right*), a member of our genus that lived in the same landscape, had a more varied diet, and adaptability may have helped its evolutionary success.

FINDINGS

A Climate for Change

Two moments in our evolutionary history show a tantalizing connection between climate swings and the life and death of key members of our family tree. Starting just after three million years ago, the species *Australopithecus afarensis* vanished, and the groups *Paranthropus* and *Homo* (our own genus) appeared. During this period, changes in carbon isotope ratios from land and ocean sediments show dry grasslands rapidly expanded and wetter woodlands shrank. Starting after two million years ago, *Homo erectus*, one of our direct ancestors, appeared and migrated out of Africa. Again, the carbon evidence shows grasslands got another boost. Yet carbon in the teeth from *H. erectus* indicates the consumption of a mixed diet and an ability to find food from a variety of sources even as grasslands enlarged. *Paranthropus* teeth, however, showed the group (like an earlier extinct forebear, *Kenyanthropus*) was restricted to eating from grassy surroundings.



WET AND DRY CYCLES

EVIDENCE FOR THESE BURSTS of landscape change and evolution comes not just from land but from the sea. African ground sediments are often hard to analyze because of erosion and other geologic disturbances. In the deep oceans, however, they remain undisturbed. By drilling into the seafloor near the African coasts, geologists like myself have been able to penetrate a multimillion-year time capsule, recovering long cores of sediments that preserve complete records of past African environments. To get these cores, we need a special ship. That is why a team of 27 scientists and I spent two months in the fall of 1987 on the 470-foot drilling vessel *JOIDES Resolution*.

"Core on deck!" squawked the driller over the PA system in his Texan twang. We scientists groaned, donned our hard hats, and marched out of the ship's cool, comfortable laboratories into the blinding Arabian sun to carry yet another 30-foot segment of deep-sea sediment core inside for analysis. The *Resolution* is an internationally funded research ship designed to explore and drill the ocean bottom and recover the earth's history recorded there. We were drilling through layers of deep-sea sediment in the Arabian Sea in a mile and a half of water, taking cores nearly half a mile into the sea bottom. Since the divergence of great ape and human lineages several million years ago, the ocean bottom here had accumulated nearly 1,000 feet of deep-sea mud in the dark, peaceful abyss, at a rate of about one and a half inches every 1,000 years.

The sediments here consist of mixtures of fine-white calcium carbonate fossil shells from ancient ocean plankton and darker, silty grains of dirt blown from areas of Africa and Arabia by windy monsoons. When the mix looks darker and gritty, it indicates drier, dustier times. When it looks lighter, that reflects wetter, more humid conditions.

Laying the split sediment core on a table inside the spacious research labs, we could see that the alternatin and dark layers repeated every three feet, more or less, meant they changed about every 23,000 years. It was cle African climate history had been one of continuous swi tween wetter and drier times. That was nothing like a sharp shift to a savanna.

These swings reflected the known sensitivity of Afric Asian monsoonal climates to the earth's orbital wobble, occurs as a regular 23,000-year cycle. The wobble chan amount of sunlight hitting our planet in a given season. Fo Africa and South Asia, more or less beat during the sum creased or decreased monsoon rainfall, making these reg ther much wetter or drier as our planet wobbled back an

Just how wet things got is recorded in magnificent r drawn between 10,000 and 5,000 years ago by humans the most recent wet period in North Africa. Art found acr Sahara depicts lush landscapes filled with elephants, hip amases, giraffes, crocodiles and bands of hunters chas zelles. The Sahara was covered with grass and trees; lake now overrun by sand dunes, were filled to the brim with A swollen Nile River rushed into the eastern Mediterr and black, organic-rich sediments called sapropels accum on the Mediterranean seafloor. They alternated with whi ers laid down during dry periods, a bar-code message te African climate cycles reaching deep into the past, just l changing dust layers recovered from the Arabian Sea.

THE LAST OF LUCY

SUPERIMPOSED ON THESE orbital wet-dry cycles were large toward dry and open grasslands. Small patches of gra first expanded in East Africa nearly eight million years a vast grassy plains such as the Serengeti were only established permanently after three million years ago. Just about this time, our evolutionary history was given a jolt as well.

We lost Lucy. Her extremely successful species, *A. afarensis*, had survived in East Africa for 900,000 years, starting at about 3.9 million years ago. But just under three million years ago, Lucy's kind disappeared from the fossil record.

Next the *Paranthropus* group appeared, followed 2.6 million years ago by the first signs of stone choppers and scrapers and then in a few hundred thousand years by early *Homo* fossils.

We know these changes in our family tree and in technological invention happened during a shift in overall climate because of some clever scientific detective work, tracing the fingerprints left by some plants that flourished in wetter environments and others that thrived in drier times.

Savannas are open tropical ecosystems composed of grasses and sedges, sometimes spotted with clusters of woody trees. Savanna grasses do very well in hot, dry regions because, to take up carbon from the atmosphere, they use a specific photosynthetic pathway called C4. This set of reactions is miserly with carbon and water, an adaptation to life in dry and low-CO_2 environments. Woody vegetation such as trees finds homes in wetter ecosystems because it uses another photosynthetic pathway called C3, which requires much more water.

Thure E. Cerling and his colleagues at the University of Utah developed a way to reconstruct the vegetation history of ancient landscapes. Some years ago researchers discovered that C4 grasses have a greater abundance of the heavier but rarer carbon 13 isotope relative to the lighter, more abundant carbon 12 isotope. But C3 shrubs and woody plants have a lower carbon 13/12 ratio. The scientists discovered that they could take samples of soil or nodules of rock from a given landscape, analyze the carbon ratios, and use them to accurately estimate the percentage of C4 grasses versus C3 woody plants that were once in that area.

When they looked at the East African sediment from sites that had yielded fossil hominins, the researchers learned that East African landscapes were predominantly C3 forest and shrublands before eight million years ago. After that, the proportion of C4 grasslands increased gradually. Then a relatively large and fast shift occurred between three million and two million years ago.

During this shift, grasslands expanded rapidly across present-day Kenya, Ethiopia and Tanzania. The spread was accompanied by a rise in the proportion of grazing mammals, shown by their abundant fossils. As time ticked forward, closer to two million years ago, African antelopes—their horns, whose different shapes indicate different species, are well preserved—seem to have undergone extensive speciation, extinction and adaptation, rather like our hominin forebears.

BIOMECHANICS

SWIMMERS UNDER PRESSURE

Jellyfish manipulate physics to be the most efficient ani… …ng in the sea

By Josh Fischman

Jellyfish never stop. Twenty-four hours a day, seven days a week, they move through the water in search of food such as shrimp and fish larvae, on journeys that can cover several kilometers a day. They are more efficient than any other swimmer, using less energy for their size than do graceful dolphins or cruising sharks. "Their cost of transport—the oxygen they use to move—is 48 percent lower than any other swimming animal," says Bradford J. Gemmell, a marine biologist at the University of South Florida. By studying moon jellies, the species *Aurelia aurita*, Gemmell and other researchers have recently found that jellyfish pull off this feat by creating zones of high and low pressure around their body that alternately suck and push them forward.

Scientists once believed that jellies traveled so easily because they were light, mostly water. But water has mass, and mass still has to be moved. So Gemmell, with engineer John Dabiri of Stanford University and their colleagues, took a close-up look. They put a jellyfish in a tank and dropped tiny glass beads into the water. By illuminating the beads with lasers, they could track their movements with a high-speed camera, essentially making the water velocity and pressure visible around the animal.

When the animal contracted its bell—the dome that forms much of the jellyfish body—it created lower pressure outside the bell and higher pressure within. Because objects move from high to low pressure, the moon jelly got pulled forward, the scientists noted in November 2015 in *Nature Communications*.

Then the researchers got a surprise. When the jelly relaxed the bell margin, letting it flare out, the high-pressure water below the animal rose up into the bell. "It gave the moon jelly a secondary bump forward, even while it relaxed," Gemmell says. To make these moves, the jelly needs to flex the bell margin up and down. Jellyfish have muscles, but most go around the bell like a stack of rubber bands. That arrangement is good mainly for squeezing. Recently, though, Richard Satterlie, a biologist at the University of North Carolina Wilmington, discovered other muscles at the margin that stick out at angles. Those fibers let the jelly bend its edge, moving water around it, and make for a very effective swimmer. ■

Josh Fischman is a senior editor at *Scientific American*.

SCIENTIFIC AMERICAN ONLINE ▸ Watch engineer John Dabiri explain how jellyfish inspire submarine design at ScientificAmerican.com/may2016/jellyfish-physics

Muscle fibers generally circle the bell, but at the margin, they form a mesh, helping it bend.

The Jellyfish Swim Stroke

Jellyfish create zones of high and low pressure around themselves, then move from one to the other. By dropping tiny glass beads in a water tank with a jellyfish, a team of scientists was able to see that a vortex—a relatively low-pressure spinning doughnut of water—rolls down the jelly. Seen here as a cross-sectional slice, the vortex (blue) appears as two wheels of water (1). As the jelly contracts, it creates higher pressure within its bell, and the animal is pulled ahead to lower pressure (2). By flexing the edge of its bell, the jelly moves the vortex down and underneath (3). There the vortex pushes water up as it spins, giving the jelly an extra shove (4).

Illustrations by Eleanor Lutz