SPECIAL FOOD ISSUE

SCIENTIFIC AMERICAN

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SEPTEMBER 2013

It started as fuel, became a passion, ignited a global crisis—and made us human

food

INSIDE

You Don't Know CALORIES

The Truth About GMOs

Why We Needed BBQ

ON THE COVER

To celebrate this special issue on food, the cover of this month's *Scientific American* comes in three flavorful variations on a culinary theme.

Photographs by Dan Saelinger. Prop styling by Dominique Baynes.

SCIENTIFIC AMERICAN

September 2013 Volume 309, Number 3

the food issue

feast

CELEBRATING OUR LOVE OF FOOD

fuel

HOW FOOD CHANGES US

farm

THE FUTURE OF THE FOOD SUPPLY

Photograph by Grant Cornett

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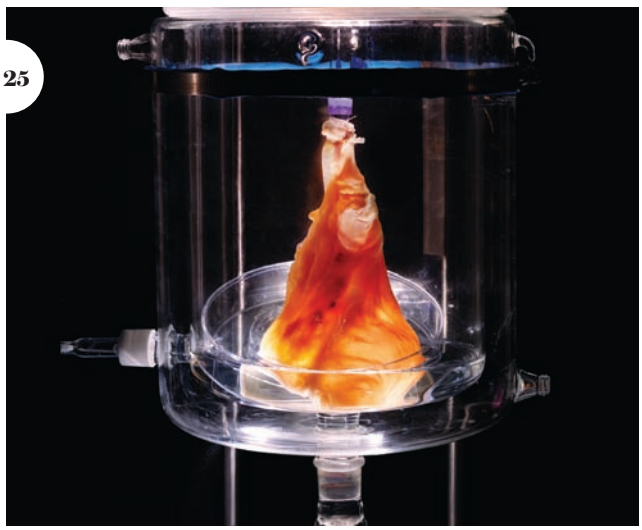

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DEPARTMENTS

ON THE WEB

What to Eat?
If your appetite for food science is stimulated by this special issue, explore more resources—on everything from fad diets to GMOs to misleadingly labeled foodstuffs—to help navigate the sea of confusing information.
Go to www.ScientificAmerican.com/sep2013/food-resources

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Mariette DiChristina is editor in chief of *Scientific American*. Follow her on Twitter @mdichristina

A Scientific Feast

PERHAPS THE MOST intimate relationship each of us will ever have is not with any fellow member of our own human species. Instead—as you have no doubt already guessed from the subject and title of this special issue—it is between our bodies and our food.

In fact, when our editorial team first discussed pursuing this edition's theme several months ago, we all became intrigued by the intricate reciprocal interactions between us and our chow. On the one hand, we certainly are what we eat. Food constitutes our very being. It can serve up sensory delights, as senior editor Michael Moyer, who organized this single-topic issue, explains in his essay on the nature of deliciousness, starting on page 34. It affects our long-term health, as nutrition researcher Gary Taubes discusses, beginning on page 60, in "Which One Will Make You Fat?" It even helped make us human; turn to page 66 for senior editor Kate Wong's interview with Richard Wrangham of Harvard University, "The First Cookout."

On the other hand, we intensively manage our sources of sustenance, shaping them to our needs and desires and affecting the environment on a global scale. "Are Engineered Foods Evil?" David H. Freedman asks regarding our modern breeding techniques, beginning on page 80. On page 50, culinary expert Evelyn Kim traces "The Amazing Multimillion-Year History of Processed Food" that has led to the present-day nutritional outputs of the food-industrial complex. What we ingest can even work as an ecosystem corrective, as chef Bun Lai points out in his story, "How (and Why) to Eat Invasive Species," on page 40.

As one editor put it, the issue ultimately is about how we play with our food and how food, in turn, plays with us. We invite you to dig in. SA

SCIENCE IN ACTION

Congrats, Elif Bilgin

We have announced the 2013 winner of the $50,000 Science in Action prize, sponsored by *Scientific American* as part of the Google Science Fair, the annual global competition for students ages 13 to 18: Elif Bilgin, age 16, hails from Istanbul, Turkey. See "Turning Peels into Plastic" [Advances], on page 19, to learn about her remarkable work. —M.D.

Photograph by Dan Saelinger (food collage), Illustration by Nick Higgins (DiChristina)

editors@sciam.com

May 2013

"It is a common misunderstanding that if something happens once a century, it won't happen again for 100 years."

MARK NICOLICH *WEST AMWELL, N.J.*

FIREARMS FACTS

In discussing gun control in "Gun Science" [Skeptic], Michael Shermer first cites a 1998 paper in the *Journal of Trauma and Acute Care Surgery* concluding that guns in the home are much more likely to be used in criminal assaults or homicides than for self-defense. Oddly, that study accounted only for cases where criminals were killed or wounded and not the more typical scenario in which an attacker is scared away. Cases where the attacker is killed or wounded account for well less than 1 percent.

Shermer ignores the 2004 National Academy of Sciences report that rejected the 1998 study and similar ones. As the NAS report noted, this type of public health research fails to account for the endogeneity problem—that it is especially people who feel threatened who tend to acquire guns. Fixing this reverses the claims.

JOHN R. LOTT, JR.
Author of More Guns, Less Crime, *third edition (University of Chicago Press, 2010)*

SHERMER REPLIES: The 1998 paper is one of several that determined how often guns in the home are used to injure or kill in self-defense, compared with how often they are involved in accidental shootings, criminal assaults and suicide attempts. Tragic shootings outnumber defensive ones by more than 40 to one. In addition to cases in which a homeowner chased a bad guy away, the 1998 study did not count how often guns in the home were used to threaten or to intimidate a family member, a spouse, a girlfriend or a neighbor.

The self-defense figures Lott cites were derived by extrapolating low-frequency responses to public opinion polls to the entire U.S. population and are thus wildly inflated. Police reports suggest the use of guns in self-defense is much less common. An audit of Atlanta Police Department reports of 198 home-invasion crimes identified three cases in which victims successfully defended themselves. Intruders got to the homeowner's gun twice as often.

As for the NAS report: when the committee opined about case-control research, it was criticizing an analytical method most, if not all, of its members had never employed. The endogeneity issue is a case in point. The committee speculated that any statistical association between guns in the home and violent death may exist because people acquire firearms in response to specific or perceived threats or because gun owners may be more or less violence-prone.

But Arthur Kellermann, the lead author of the 1998 study that had been referred to in the committee's report, has mitigated against that possibility. For instance, in a 1993 case-control study, he questioned households about a wide range of risk factors for violence and took any differences into account through logistic regression. In addition, every household was matched with a control in the same neighborhood, which ensured similar socioeconomic status and exposure to crime.

Interestingly, the study found that a household's risk of homicide from an intruder was neither higher nor lower if a gun was kept there but that the risk of homicide from a family member or an intimate acquaintance was much higher.

STELLAR STATISTICS

In discussing potential meteor strikes in "Preventing the Next Chelyabinsk" [Advances], John Matson writes, "Fortunately, impacts on the scale of Chelyabinsk occur only once a century, so perhaps humankind will have figured out even better techniques by then," thus falsely supporting the common misunderstanding that if something happens once a century, it won't happen again for 100 years. That would require collective memory among meteors.

As a statistician, I can offer a calculation: if we assume that meteor strikes are random and independent and follow a Poisson distribution with a mean of one per 100 years, then the probability of a strike in any given year is about 1 percent irrespective of recent strikes.

MARK NICOLICH
West Amwell, N.J.

THE "GIF" OF LAUGHTER

I enjoyed "The Strange Magic of Micro Movies," by David Pogue [TechnoFiles], but Pogue discounts the human contribution to why "micro movies" such as GIFs and six-second Vine movies are so popular. The primary use of micro movies I've seen is to communicate emotion or a reaction to a situation, comment, picture or otherwise previously uploaded statement. One could communicate emotions through words or emoticons, but micro movies allow for a more profound message.

Furthermore, while GIFs are used to communicate many emotions, they are typically made with the additional intention of making individuals laugh. I have read multiple theories that state laughter has allowed our species to form larger and more connected social networks. What if these micro movies are the next revolution in such communication?

EDWIN E. RICE IV
Columbus, Ohio

ANCESTRAL ASSOCIATION

According to "Human Hybrids," by Michael F. Hammer, modern humans and extinct archaic human species such as Neandertals were able to create fertile offspring. Doesn't this make modern humans and Neandertals part of one species?

CHANTELLE TAIT
Clifton, Va.

Hammer notes the open question as to how *Homo sapiens* replaced Neandertals and shows that it is likely that some contemporary non-Africans received an antiviral stretch of chromosome (*STAT2*) from *H. sapiens* interbreeding with Neandertals. Is it possible that modern humans wiped out Neandertals by bringing viral disease with them (such as when Europeans "conquered" the New World) and that the reason why modern humans carry *STAT2* is that only those Neandertals that had it survived long enough to mingle significantly with the virus-laden newcomers?

DOUG MCAFEE
Bothell, Wash.

HAMMER REPLIES: Regarding Tait's question, although some paleoanthropologists believe Neandertals should be classified as a separate species of Homo, *many still regard them as a subspecies of* Homo sapiens. *Opinions on this correlate with views on whether anatomically modern humans (AMH) originated via Replacement or via Assimilation and Hybridization. Many other mammals that diverged as recently as Neandertals and AMH are considered distinct species but can interbreed and produce fertile offspring.*

While McAfee's idea is interesting, in all likelihood, AMH from Africa were more vulnerable to the novel environmental pathogens they encountered as they moved into Europe than Neandertals were to pathogens from the African migrants. In contrast to the Europeans who came to invade the New World, early AMH from Africa probably carried comparatively few pathogens because they would have lived in small hunter-gatherer groups with far less density than agricultural populations that formed tens of thousands of years later and with none of their exposure to pathogens from domesticated animals. People today probably retain STAT2 *and other immune-related variants acquired from Neandertals because that DNA helped those early AMH from Africa survive new habitats.*

ERRATUM

"My Boss the Robot," by David Bourne, should have referred to Bourne as principal systems scientist at the Robotics Institute of Carnegie Mellon University.

LETTERS TO THE EDITOR

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Letters may be edited for length and clarity. We regret that we cannot answer each one. Post a comment on any article at ScientificAmerican.com/sep2013

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Fight the GM Food Scare

Mandatory labels for genetically modified foods are a bad idea

This past June, Connecticut and Maine became the first states to pass bills requiring labels on all foods made from genetically modified organisms (GMOs). In November 2012 California voters rejected the similar Proposition 37 by a narrow majority of 51.4 percent. "All we want is a simple label/For the food that's on our table," chanted marchers before the elections. The issue, however, is in no way simple.

We have been tinkering with our food's DNA since the dawn of agriculture. By selectively breeding plants and animals with the most desirable traits, our predecessors transformed organisms' genomes, turning a scraggly grass into plump-kerneled corn, for example. For the past 20 years Americans have been eating plants in which scientists have used modern tools to insert a gene here or tweak a gene there, helping the crops tolerate drought and resist herbicides. Around 70 percent of processed foods in the U.S. contain genetically modified ingredients.

Instead of providing people with useful information, mandatory GMO labels would only intensify the misconception that so-called Frankenfoods endanger people's health [see "Are Engineered Foods Evil?" on page 80]. The American Association for the Advancement of Science, the World Health Organization and the exceptionally vigilant European Union agree that GMOs are just as safe as other foods. Compared with conventional breeding techniques—which swap giant chunks of DNA between one plant and another—genetic engineering is far more precise and, in most cases, is less likely to produce an unexpected result. The U.S. Food and Drug Administration has tested all the GMOs on the market to determine whether they are toxic or allergenic. They are not. (The GMO-fearing can seek out "100 Percent Organic" products, indicating that a food contains no genetically modified ingredients, among other requirements.)

Many people argue for GMO labels in the name of increased consumer choice. On the contrary, such labels have limited people's options. In 1997, a time of growing opposition to GMOs in Europe, the E.U. began to require them. By 1999, to avoid labels that might drive customers away, most major European retailers had removed genetically modified ingredients from products bearing their brand. Major food producers such as Nestlé followed suit. Today it is virtually impossible to find GMOs in European supermarkets.

Americans who oppose genetically modified foods would celebrate a similar exclusion. Everyone else would pay a price. Because conventional crops often require more water and pesticides than GMOs do, the former are usually more expensive. Consequently, we would all have to pay a premium on non-GMO foods—and for a questionable return. Private research firm Northbridge Environmental Management Consultants estimated that Prop 37 would have raised an average California family's yearly food bill by as much as $400. The measure would also have required farmers, manufacturers and retailers to keep a whole new set of detailed records and to prepare for lawsuits challenging the "naturalness" of their products.

Antagonism toward GMO foods also strengthens the stigma against a technology that has delivered enormous benefits to people in developing countries and promises far more. Recently published data from a seven-year study of Indian farmers show that those growing a genetically modified crop increased their yield per acre by 24 percent and boosted profits by 50 percent. These farmers were able to buy more food—and food of greater nutritional value—for their families.

To curb vitamin A deficiency—which blinds as many as 500,000 children worldwide every year and kills half of them—researchers have engineered Golden Rice, which produces beta-carotene, a precursor of vitamin A. Approximately three quarters of a cup of Golden Rice provides the recommended daily amount of vitamin A; several tests have concluded that the product is safe. Yet Greenpeace and other anti-GMO organizations have used misinformation and hysteria to delay the introduction of Golden Rice to the Philippines, India and China.

More such products are in the works, but only with public support and funding will they make their way to people's plates. An international team of researchers has engineered a variety of cassava—a staple food for 600 million people—with 30 times the usual amount of beta-carotene and four times as much iron, as well as higher levels of protein and zinc. Another group of scientists has created corn with 169-fold the typical amount of beta-carotene, six times as much vitamin C and double the folate.

At press time, GMO-label legislation is pending in at least 20 states. Such debates are about so much more than slapping ostensibly simple labels on our food to satisfy a segment of American consumers. Ultimately, we are deciding whether we will continue to develop an immensely beneficial technology or shun it based on unfounded fears. SA

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Comment on this article at ScientificAmerican.com/sep2013

Illustration by Doug Chayka

George Church is a professor of genetics at Harvard Medical School and director of the National Institutes of Health Center of Excellence in Genomic Science at Harvard.

Please Reanimate

Reviving mammoths and other extinct creatures is a good idea

In its June issue Scientific American published an essay stating emphatically that reanimating species such as woolly mammoths from surviving DNA is a bad idea. This dismissal is too hasty. The idea has merit and is worth discussing with an open mind—and with multidisciplinary viewpoints.

The goal of reanimation research is not to make perfect living copies of extinct organisms, nor is it meant to be a one-off stunt in a laboratory or zoo. Reanimation is about leveraging the best of ancient and synthetic DNA. The goal is to adapt existing ecosystems to radical modern environmental changes, such as global warming, and possibly reverse those changes.

Ecosystems that depend on "keystone species" have lost the species diversity they once had because some species no longer fit. As environmental change occurs, ancient diversity may be needed again. For instance, 4,000 years ago the tundras of Russia and Canada consisted of a richer grass- and ice-based ecosystem. Today they are melting, and if that process continues, they could release more greenhouse gas than all the world's forests would if they burned to the ground. A few dozen changes to the genome of a modern elephant—to give it subcutaneous fat, woolly hair and sebaceous glands—might suffice to create a variation that is functionally similar to the mammoth. Returning this keystone species to the tundras could stave off some effects of warming.

Mammoths could keep the region colder by: (a) eating dead grass, thus enabling the sun to reach spring grass, whose deep roots prevent erosion; (b) increasing reflected light by felling trees, which absorb sunlight; and (c) punching through insulating snow so that freezing air penetrates the soil. Poachers seem far less likely to target Arctic mammoths than African elephants.

"De-extinction" is not a novel idea. Medical researchers have resurrected the full genomes of the human endogenous retrovirus HERV-K and the 1918 influenza virus. Insight into these reanimated species could save millions of lives. Several other extinct genes, including for mammoth hemoglobin, have been reconstructed and tested for novel properties. Moving from these few genes to most of the 20,000 or so in a bird or mammalian genome may not be necessary, and even if it is, it may not be hard to do. The costs for a variety of relevant technologies are low—and dropping.

Breeding animals and raising them until there are sufficient numbers to release into the wild is an ambitious undertaking, but the expense should be comparable to breeding livestock or preserving other endangered wildlife. These costs could be reduced if we used genetic means to improve the species we revive—boosting their immunity and fertility and their ability to draw nutrition from available food and to cope with environmental stress.

Aside from bringing back extinct species, reanimation could help living ones by restoring lost genetic diversity. The Tasmanian devil (aka *Sarcophilus harrisii*) is so inbred at this point that most species members can exchange tumor cells without rejection. A rare transmissible cancer spread via facial wounds is driving the species toward extinction. Reanimating ancestral, diverse *Sarcophilus* histocompatibility genes, which govern tissue rejection, could save it. Similar arguments could be made for amphibians, cheetahs, corals and other groups. Ancient genes could make them more tolerant of chemicals, heat, infection and drought.

Reanimation is not a panacea for ecosystems at risk. Preventing ongoing extinction of elephants, rhinoceroses and other threatened species is critically important. By all means, we must set priorities for allocating finite conservation resources. But it is a mistake to look at this issue as a zero-sum game. Just as a new vaccine can free up medical resources that would otherwise be spent on sick patients, reanimation may be able to help conservationists by giving them powerful new tools. That this is even a possibility is reason enough to explore it seriously. SA

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Illustration by Carlo Giambarresi

ECOLOGY

"Disgusting Bird" Is Dying Off

The much reviled but extremely valuable vulture needs a public relations makeover

Vultures have an image problem. Charles Darwin did them no favors when he saw a turkey vulture from the deck of the *Beagle* in 1835 and called it a "disgusting bird" whose bald head was "formed to wallow in putridity." Despite their vital cleanup role, vultures are not nearly as cute as polar bears, nor do they inspire the same interest when extinction looms—as it does for more than half of the world's 23 vulture species.

In Asia, livestock carcasses laced with the painkiller diclofenac wiped out 95 percent of three vulture species in just 15 years before nations began banning the drug in 2006. African vultures are vanishing just as rapidly. A study in 2012 reported up to 33 percent annual mortality rates for some species in East Africa. The crisis spurred the first Pan-African Vulture Summit last year, but political action has failed to materialize.

"A Kenya Wildlife Service scientist recently told us, 'We are so busy trying to save elephants and rhinos, when it comes to vultures we are just tired,'" says Darcy Ogada of the nonprofit Peregrine Fund. Ogada and her colleagues have documented staggering die-offs: in rural West Africa, for instance, populations of almost all vulture species have fallen by 95 percent in 30 years.

Saving African vultures will require more than a simple drug ban. In East Africa, vultures are both targets (slaughtered by ivory hunters to conceal poaching sites) and collateral damage (poisoned by pastoralists out to kill livestock predators, such as hyenas and lions). In West Africa, vulture parts are sold as meat or as clairvoyance drugs in the indigenous medicine trade. Wind farms and electrical lines pose growing additional threats.

As the vulture die-off continues, raptor specialists assess the consequences. In India it seems to have sparked population booms for rats and feral dogs, which carry leptospirosis and rabies. Ecological economists estimate the health-associated costs from India's dearth of vultures at $34 billion over 14 years. African vultures consume carcasses of livestock and migrating wildebeests, breaking down pathogens such as anthrax in the process. "If they were gone, we'd be left with a huge disease-transmission time bomb," says Munir Virani of the Peregrine Fund.

For her part, Ogada is now developing ways to track the spread of carrion-borne disease. By quantifying the public health cost of the vulture die-off, she hopes to spur governments to do something about it. *—Shruti Ravindran*

SHEM COMPION Getty Images

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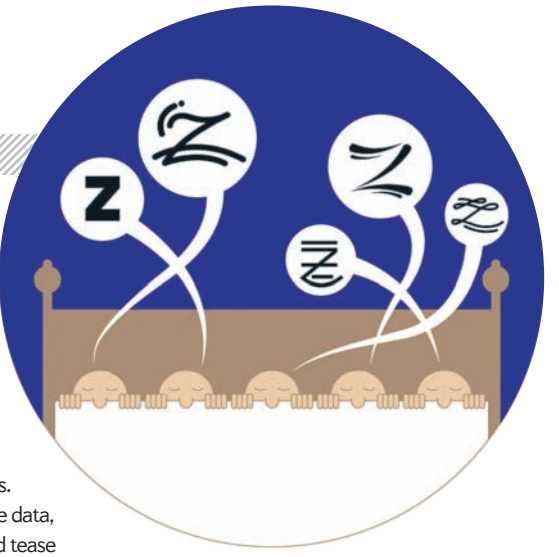


HEALTH

Wisdom of the Sleepyheads

What we could learn by monitoring sleep patterns of the entire world

Everyone knows the crankiness, puffy eyes and excessive yawning that follow a bad night's sleep. Those chronically sleep-deprived also have increased risks of heart disease, obesity and early death. Because sleep patterns are difficult to monitor in large populations, researchers do not know what causes many sleep problems or how exactly these problems affect us. Till Roenneberg, a chronobiologist at Ludwig Maximilian University of Munich, thinks a global "human sleep project" could finally solve some of these mysteries.

A common way to collect sleep data is through retrospective surveys of sleep habits, but they are unreliable because people tend to overestimate how much sleep they get. Laboratory studies are accurate but do not reproduce real-life behaviors. A global sleep project, proposed in a June issue of *Nature* (*Scientific American* is part of Nature Publishing Group), would outfit people with a variety of sensors to track their sleep patterns in real-time and, as a bonus, provide detailed feedback to the subjects.

"If people can actually see their own data on their own Internet-based platform, I think we will not get 100 or 1,000 but a million people who will participate," Roenneberg says.

With all those data, researchers could tease out the lifestyle factors that ensure healthy sleep. "Much like satellite studies of weather, it is the larger view that will reveal global patterns, limits and interactions between factors we typically hold constant in the lab," says Max Hirshkowitz, a spokesperson for the National Sleep Foundation. He believes a global sleep project would also illuminate how culture, occupation and geography all influence sleep patterns.

A project on this scale would cost about $30 million, Roenneberg says, which is a lot for a field that is, like sleep itself, chronically undervalued. "Sleep is unconscious and not apparently productive—it's not like making money or making children—so people think that they can neglect it," he says. His hoped-for global data could be a wake-up call on the importance of shut-eye.

—Melinda Wenner Moyer

Illustrations by Thomas Fuchs

Tarantula preys on an unlucky katydid.

DAVID MAITLAND Getty Images

ENVIRONMENT

The Spider in the Grass

Plants store more carbon where predators roam

The carbon cycle is essential to life on earth, but scientists still struggle to grasp its complexities. Most research to date has focused on major sources of the greenhouse gas carbon dioxide, such as deforestation and the use of fossil fuels. Now some scientists have begun to explore subtler factors, such as the interplay between plants and animals. A new study has come to the counterintuitive conclusion that plants might accumulate more carbon in the presence of predators and herbivores than they do in isolated locales, where they are less likely to be eaten or trampled.

To tease out interactions between the plant and animal worlds, Yale University ecologist Oswald Schmitz and his colleagues built three enclosed grassland environments—one with meadow vegetation only; one with vegetation and herbivorous grasshoppers; and one with carnivorous spiders, along with the grasshoppers and plants. The researchers found that plants in the third environment, the home to both herbivores and carnivores, stored 40 percent more carbon than plants in the grasshopper enclosure.

It makes intuitive sense that arachnid predators, which eat the herbivorous grasshoppers, would limit the consumption of vegetation and thus free plants to store more carbon than they would in the enclosure where herbivores grazed unchecked. Surprisingly, however, the plants sharing an enclosure with grasshoppers and spiders also packed away significantly more carbon—20 percent more—than did the isolated plants. "You would think that an environment with no herbivores or predators would lead to peak carbon storage," says Schmitz, a co-author of the study, published in June in the *Proceedings of the National Academy of Sciences USA*. "But that's hardly the case."

Why? Perhaps a degree of herbivory stimulation—a light nibble here and there—causes some unidentified physiological change in plants that boosts carbon uptake, Schmitz says, "but the truth is that we really don't know."

The research highlights how ecological changes can have significant, and unforeseen, climate impacts. "Right now there is a crisis in terms of predator diversity loss," Schmitz says. "And that may mean that we are losing the potential to help regulate the carbon cycle in ways that go far beyond just growing more trees." —*Arielle Duhaime-Ross*

ASTRONOMY

Galaxies Bright and Beautiful

The search for the Milky Way's missing satellites

The giant spiral galaxy Andromeda and the slightly smaller Milky Way are king and queen of our place in space, a gathering of some 75 galaxies called the Local Group. Andromeda and the Milky Way each rule an empire of dozens of lesser galaxies that orbit them the way moons do a giant planet. Recently astronomers have discovered many more of these galactic runts. In that time, a surprising disparity has emerged. Andromeda is only 30 percent more luminous than our galaxy and so should boast a retinue only slightly grander than our own, yet most of Andromeda's newfound satellites are much brighter than ours. Clearly, we are missing something in our own backyard.

"There should be more bright satellites around the Milky Way," says Basilio Yniguez, an astronomer at the University of California, Irvine. His team's computer simulations of how galaxies form suggest the Milky Way should host eight to 20 additional bright satellites that are as yet undiscovered.

"Bright" is defined relative to the faintest galaxies now known: Yniguez does not think the unseen satellites rival our galaxy's most flamboyant companions, the Large and Small Magellanic Clouds. Instead they are probably dim, diffuse "classical" dwarfs akin to Sculptor and Fornax, two Milky Way satellites astronomers spotted in 1938. Faint though they are, the classical dwarfs outshine a new breed of even dimmer "ultrafaint" dwarf galaxies that observers have turned up in the past decade.

Since 2004 astronomers have found about two dozen galaxies orbiting Andromeda and about a dozen orbiting the Milky Way. But whereas 16 of Andromeda's new satellite galaxies are classical dwarfs, emitting more than 100,000 times as much light as the sun, only one of the Milky Way's newfound satellites shines this brightly—the rest are ultrafaint. Nevertheless, Andromeda and the Milky Way possess nearly identical distributions of satellites out to 330,000 light-years, suggesting that our galaxy's missing satellites probably lie farther out.

Finding them will be a challenge. "Our position within the Milky Way hurts us," Yniguez says. "We're looking at Andromeda from the outside, whereas with the Milky Way we're looking from the inside." Future searches, Yniguez hopes, will expand the Milky Way's known galactic empire so that it more closely matches that of its partner in Andromeda. —*Ken Croswell*

INNOVATION

Turning Peels into Plastic

A teen scientist's banana-based bioplastic won the Science in Action Award in the Google Science Fair

"Genius," Thomas Edison famously said, "is 1 percent inspiration and 99 percent perspiration." He would have found a kindred spirit in Elif Bilgin, 16, of Istanbul, winner of the 2013 Science in Action Award, part of the third annual Google Science Fair. The $50,000 award, sponsored by *Scientific American,* honors a project by teens from 13 to 18 that can make a practical difference by addressing an environmental, health or resources challenge.

Bilgin spent two years developing a robust bioplastic from discarded banana peels, enduring 10 failed trials along the way. As she noted in her project description: "Even Thomas Edison said, 'I have not failed. I have just found 10,000 ways that won't work.'" Bilgin hopes that her material will someday supplant some petroleum-based plastics.

She is also a finalist in the Google Science Fair and will join 14 other contenders at the company's California headquarters in September for the awards event. Bilgin talked with *Scientific American* about her inspirations and her aims for the bioplastic project. Excerpts follow.

PROFILE

NAME
Elif Bilgin

TITLE
Student

LOCATION
Istanbul, Turkey

How does your project affect your community?
Istanbul is a very big and crowded city. With so many people using electricity on a daily basis—whether it is to charge their cell phones or to use their TV—a lot of cables must be used throughout the city. My project makes it possible to use banana peels, a waste material that is thrown away almost every day, in the electrical insulation of cables. This has the potential to decrease the amount of pollution created from the use of plastics.

Who are your scientific heroes?
Marie Curie has been a major inspiration and a role model, being a female scientist who devoted her life to her study of radioactivity and challenged gender norms along the way.

If you could travel through time, what innovation would you introduce 100 years early?
As a huge science-fiction fan, I wouldn't accept the opportunity to go back in time and introduce an invention or discovery. I wouldn't want to disrupt the space-time continuum! But if I had to give an answer, it would be introducing a treatment for cholera. This act would save many lives. —*Mariette DiChristina; interview by Rachel Scheer*

COURTESY OF ABDULLAH BILGIN (*Bilgin*); THINKSTOCK (*banana*)

FUNDING

Courtesy of the U.S. Taxpayer

Remember that $800 billion Uncle Sam spent during the Great Recession? Here are some of the consequences

Four years ago, as the world reeled from its most severe economic crisis in almost a century, the U.S. federal government poured roughly $800 billion into the economy, including $15 billion for scientific research and tens of billions more for green energy and environmental protection. The money must be spent by this month or returned to the government. Here are some highlights of what it bought us.

BETTER MILESTONES FOR FETAL GROWTH

To determine if a baby is properly developing in the womb, obstetricians use measurement standards devised decades ago. A five-year national study followed more than 3,350 healthy women of various ethnicities over the course of their pregnancies, carefully measuring fetal growth and health through ultrasound scans, blood tests and nutritional data. The study's findings will help establish new standards for prenatal care for every clinician in the country.

Stimulus funding: $20 million

A ROAD MAP TO THE GROWING BRAIN

To create an atlas for the developing human brain, six institutions collaborated to map genetic expression in over 40 brains of weeks-old fetuses up to middle-aged adults. The open-access BrainSpan provides an accessible database of the shifting activation of genes in the brain over a lifetime, allowing researchers to scrutinize how this variability contributes to diseases such as schizophrenia and depression.

Stimulus funding: $35 million

A CUSTOM-BUILT ARCTIC RESEARCH VESSEL

American scientists bound for the Arctic Ocean have had to borrow a U.S. Coast Guard icebreaker. Now the National Science Foundation is building its own ship that can cut through thick sea ice like so much white frosting. It will carry researchers north to investigate marine life and climate change.

Stimulus funding: $148 million

AN OCEANIC ALL-SEEING EYE

The Ocean Observatories Initiative is deploying a vast network of sensors across the world's oceans. The data gathered from sonar instruments, water-column and seafloor sensors, and open-ocean gliders will be freely available online. Researchers hope it will transform our understanding of how global warming, nutrient cycling, ocean acidification and other complex processes are shaping the planet's largest ecosystem.

Stimulus funding: $106 million

A TELESCOPE TO GLIMPSE THE UNIVERSE'S FIRST MOMENTS

The Cosmology Large Angular Scale Surveyor telescope is being designed and built in Maryland but will soon perch on a high plateau in the Chilean Atacama Desert. From there it will search the skies for indirect evidence of gravitational waves, delicate ripples in spacetime that could help substantiate inflation—the idea that the universe drastically ballooned its expansion rate in the first trillionth of a trillionth of a second after the big bang.

Stimulus funding: $5 million

FASTER, BETTER, CHEAPER DATING OF ANCIENT ARTIFACTS

Researchers at California State University, Long Beach, used stimulus funds to test a dating technique for old ceramics, called rehydroxylation. Because clay loses hydroxyl molecules when fired and regains them at a set rate over time, simply reheating and then weighing ceramic can yield its age. The method could provide pinpoint dates for archaeological sites worldwide.

Stimulus funding: $310,000

—Katie Worth

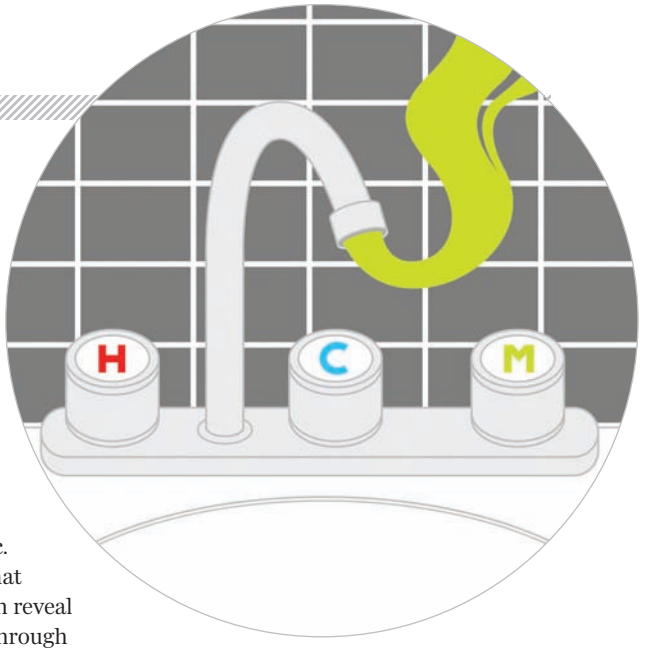
UNIVERSITY OF ALASKA FAIRBANKS PHOTO COURTESY OF TODD PARIS (*boat*); COURTESY OF CLASS SCIENCE TEAM/JOHNS HOPKINS UNIVERSITY (*telescope*)

COMMENT AT ScientificAmerican.com/sep2013

TOXICOLOGY

Fracking and Tainted Drinking Water

Well water in Pennsylvania homes within a mile of fracking sites is found to be high in methane



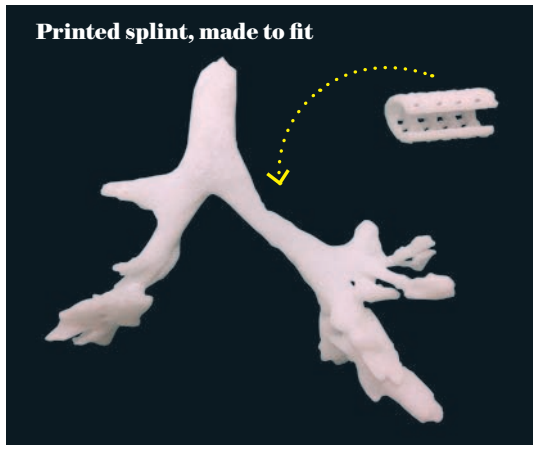
In Pennsylvania, the closer you live to a well used to hydraulically fracture underground shale for natural gas, the more likely it is that your drinking water is contaminated with methane. This conclusion, in a study published in the *Proceedings of the National Academy of Sciences USA* in July, is a first step in determining whether fracking in the Marcellus Shale underlying much of Pennsylvania is responsible for tainted drinking water in that region.

Robert Jackson, a chemical engineer at Duke University, found methane in 115 of 141 shallow, residential drinking-water wells. The methane concentration in homes less than one mile from a fracking well was six times higher than the concentration in homes farther away. Isotopes and traces of ethane in the methane indicated that the gas was not created by microorganisms living in groundwater but by heat and pressure thousands of feet down in the Marcellus Shale, which is where companies fracture rock to release gas that rises up a well shaft.

Most groundwater supplies are only a few hundred feet deep, but if the protective metal casing and concrete around a fracking well are leaky, methane can escape into them. The study does not prove that fracking has contaminated specific drinking-water wells, however. "I have no agenda to stop fracking," Jackson says. He notes that drilling companies often construct wells properly. But by denying even the possibility that some wells may leak, the drilling companies have undermined their own credibility.

The next step in proving whether or not fracking has contaminated specific drinking-water wells would be to figure out whether methane in those wells came from the Marcellus Shale or other deposits. Energy companies claim that the gas can rise naturally from deep formations through rock fissures and that determining a source is therefore problematic. Yet some scientists maintain that chemical analysis of the gas can reveal whether it slowly bubbled up through thousands of feet of rock or zipped up a leaky well. Jackson is now analyzing methane samples in that way.

Another way to link a leaky fracking well to a tainted water well is to show that the earth between them provides pathways for the gas to flow. Leaky wells have to be identified first, however. Anthony Ingraffea, a fracking expert at Cornell University, is combing through the inspection reports for most of the 41,311 gas wells drilled in Pennsylvania since January 2000. Thus far, he says, it appears that "a higher percentage" of Marcellus Shale fracking wells are leaking than conventional oil and gas wells drilled into other formations. Stay tuned. —*Mark Fischetti*



MEDICINE

A Baby Breathes Easier

A 3-D-printed windpipe signals a future of body parts manufactured on demand

Kaiba was six weeks old in 2011 when he stopped breathing and turned blue. His parents rushed him to the hospital, where they learned that his left bronchial tube had collapsed because of a birth defect. The attacks recurred for weeks until January 2012, when surgeons implanted a 3-D-printed tube to hold the baby's airway open. The tube will dissolve after a few years inside the boy's body, giving his bronchus time to grow strong enough for normal breathing. This is the first use of such an implant to aid tissue reconstruction and was reported in a May edition of the *New England Journal of Medicine.*

The trachea's 20 interlinked rings of cartilage keep a newborn's airway open as it branches into the lungs, rather like the steel rings that support a vacuum cleaner hose. But rarely, a portion is floppy and collapses. Implanted stents can prop open the airway from within but often still result in compromised breathing from the irritation they cause. Kaiba's doctors contacted Glenn Green, a doctor who, along with his colleagues at the University of Michigan, was developing custom-fit tubes to wrap around a collapsed trachea as an irritant-free way to hold the airway open.

Green and his colleagues thought 3-D printers could make an artificial trachea because of the ease of manufacturing the rings that make up the organ's tubular structure. The researchers printed tracheae from biocompatible plastic and tested them in piglets.

For Kaiba, the team first took a CT scan of the infant's airways and used the data to print a cast. Using that cast, the scientists next created a fitted, flexible sleeve to stabilize the airway. The final step was to sew the tissue of Kaiba's bronchus to the inside of the sleeve, which required an emergency-use approval from the U.S. Food and Drug Administration. "When we put the splint on, we saw his lungs move for the first time," Green says. Like Kaiba, the use of 3-D-printed medical devices and body parts is still in its infancy, but Green believes the technology has "gigantic potential." —*Marissa Fessenden*

FROM "BIORESORBABLE AIRWAY SPLINT CREATED WITH A THREE-DIMENSIONAL PRINTER" BY DAVID A. ZOPF, MARC E. NELSON AND RICHARD G. OHYE, IN *NEW ENGLAND JOURNAL OF MEDICINE*; VOL. 368; MAY 23, 2013. REPRINTED WITH PERMISSION FROM MASSACHUSETTS MEDICAL SOCIETY

BY THE NUMBERS

5% *Average annual increase in U.S. research and development funding from 1999–2009.*

China's increase in research and development funding each year for the past decade. **20%**

DATA SOURCES: NATIONAL SCIENCE FOUNDATION (*U.S.*); NATURE PUBLISHING INDEX 2012 GLOBAL (*China*)

COMMENT AT ScientificAmerican.com/sep2013

BIOLOGY

Sizing Up Sustainable Seafood

A simple shift in fishing practices could keep shrimp on the menu

Pity the pandalid shrimp. Fisheries not only harvest this cold-water crustacean in ever growing numbers but also ignore critical details of its life cycle. Pandalid shrimp are protandric hermaphrodites: all juveniles develop testicular tissues and spawn by releasing sperm into the water for external fertilization. Each shrimp can live for up to five years, and during breeding seasons hormonal changes can transform the animal into an egg-bearing female.

An individual usually becomes a female once it has reached a threshold body size. If there are too many males or females in a given year, however, an individual will often spawn as a member of the rarer sex, keeping the sex ratio balanced. Males "choose" to stay male or become female during the "maturing season" of early summer.

With autumn's arrival, the window of opportunity to change sex closes. The shrimp breed and are harvested using traps that retain only large specimens—that is, only females. A study in the *Journal of Animal Ecology* confirmed that those left behind are mostly male, thus erasing the benefits of sex swapping.

These skewed sex ratios cripple the species' reproductive capacity for each breeding season. Worse yet, smaller females are less fertile than large ones, meaning the few remaining females have suboptimal reproductive rates.

The study's authors suggest a simple solution. If harvest season were moved from fall to spring, before the shrimp's summer sex changes, postharvest populations would have time to adjust for the lack of females. This approach could help ensure sustainable fisheries—and a more continuous supply of shrimp cocktail.

—Anne-Marie C. Hodge

SPACE

Stop Pampering the Red Planet

It's time to relax constraints on Mars exploration, researchers argue

Does Mars need protection from our microbes? Conventional wisdom says yes, as does space law—the United Nations Outer Space Treaty prohibits the contamination of potentially fertile worlds with earthly bacteria. Yet some researchers disagree: Mars will be just fine on its own, they say, and the stringent safeguards now in place discourage scientists from exploring the Red Planet. On missions dedicated to searching for life, costs "could easily double because of planetary protection procedures," says Cornell University astrobiologist Alberto G. Fairén.

Protecting Mars is not worth the effort and expense, Fairén and Dirk Schulze-Makuch of Washington State University argue in a recent issue of *Nature Geoscience*. After all, some Earth bacteria are probably already there, having hitched a ride on debris from ancient meteor impacts or more recently on NASA's Viking landers. Besides, any life-form already on Mars would easily fight off the poorly adapted invasive microbes.

The odds of NASA changing course are low. "If you want to study life elsewhere, you have to make sure not to bring Earth materials along" or else risk mistaking stowaways for alien life, says Catharine Conley, NASA's planetary protection officer.

John Rummel, Conley's predecessor at NASA, says simulations and experiments suggest Earth bacteria actually could survive on Mars. Adds Rummel: "We don't know everything that Earth organisms can do." —*Nathan Collins*

GENETICS

Pathogens Decoded

New DNA recovery and sequencing technology is at last allowing scientists to assemble entire genomes of ancient scourges—and elusive modern ones

Over the past millennium the bacterium *Mycobacterium leprae,* which causes leprosy (Hansen's disease), has changed very slowly. Yet in less than a century it has given rise to strains resistant to a heavily used antibacterial treatment.

This genetic history has come to light with a little fishing—a technique known as DNA fishing, developed in part by geneticist Johannes Krause of the University of Tübingen in Germany. Beginning with old bones and teeth, the researchers trawl for ancient bacterial DNA using strands of contemporary DNA as "bait." The old DNA that sticks to the bait is then studied through genetic sequencing. Krause and his co-authors detailed the work in June in *Science.*

Based on a pathogen's evolutionary history, researchers hope to spot the modern emergence of antibiotic-resistant strains. The data can also reveal when changes in human conditions—such as improved sanitation—influenced infection rates more than a pathogen's innate traits. These insights will be, according to University of South Carolina epidemiologist Sharon DeWitte, "important for understanding how diseases can evolve and what forms they might take."

The next big target for DNA fishing, Krause notes, is *M. tuberculosis,* the bacterium behind the world's most widespread deadly infectious disease after HIV.

DNA fishing may miss ancient genetic fragments absent from modern strains, says Helen Donoghue, a microbiologist at University College London. But it could allow scientists to study even poorly preserved pathogen genomes from remote time periods. "As long as there are sufficient nucleic acids preserved in the specimen, there is really no limit," says Alison Devault, a researcher at McMaster University studying ancient cholera. Soon the scourges of bygone centuries—and millennia—might be laid bare to help future generations avoid the worst torments of those past. —*Katherine Harmon*

Hallmark hand deformities caused by leprosy

ANDREW HENDERSON *Redux Pictures*

COURTESY OF OTT LAB/MASSACHUSETTS GENERAL HOSPITAL/HARVARD MEDICAL SCHOOL

WHAT IS IT?

Its original owner is no longer alive, but this heart may soon beat again. Harald Ott and researchers at Massachusetts General Hospital are engineering the organ to be used for a transplant. They stripped the heart down to a scaffold of structural proteins so that they can repopulate it with new stem cells that are compatible with a transplant patient's immune system. If it works, the technique would boost the supply of transplantable hearts, in part by allowing human cells to be grown on cardiac scaffolds sourced from pigs and other animals.

A similar engineering process has worked with simpler, hollow organs such as bladders, but the heart is a work in progress. The key challenge is choreographing the growth of intricate vascular networks and specialized cells that must act as one to produce not just a single heartbeat but another lifetime's worth. *—Sophie Guterl*

Best of the Blogs

HEALTH

The Case for Milk Is Going Sour

Armed with new evidence, nutritionists are rallying against dairy-rich diets

The USDA, the American Academy of Pediatrics and other august institutions recommend that calorie-containing beverages should be limited in people's diets. Pretty much all, that is, except for low-fat milk. The U.S. dairy industry made the "Got milk?" slogan one of the most famous of all time—and standard dietary guidelines embrace that entreaty: three cups a day, less the saturated fat, do well by both child and adult.

Experts are starting to have second thoughts about that recommendation. Less milk than what current daily requirements call for may in fact be more healthful, and forgoing milk altogether may be fine. What's more, even low-fat milk may not be as healthy as commonly believed.

The latest broadside against the most wholesome of foods appeared in July's *JAMA Pediatrics*, in a commentary from nutrition scientists David Ludwig and Walter Willett of Harvard Medical School. Their rationale is simple: foods with less fat often make you feel less full. The child who drinks low-fat milk but then grabs an extra cookie because of lingering hunger pangs winds up consuming more refined carbohydrates and risks gaining extra pounds. As for the cholesterol-raising saturated fat in whole milk, Ludwig and Willett note that milk fat increases both artery-clogging cholesterol as well as the more beneficial kind, making the whole thing somewhat of a wash.

The authors' antimilk manifesto also has an evolutionary component. Grazing animals evolved to supply milk to their young, keeping them close to protect against predation. But this necessary closeness stops when calves and kids turn into cows and goats. Human adults who chug the preferred drink of suckling grazers thrice daily for decades may not fare so well. A hormone called insulinlike growth factor 1 that is found in milk products has been tied to prostate and other cancers. If bone-strengthening calcium is what you seek, the researchers suggest, you can meet your daily requirements by eating leafy greens, nuts and seeds.

More work remains to be done, but until then, Ludwig and Willett say that milk drinking should not be mandated. And there's no need to seek out the skim carton on the market shelf. *—Gary Stix*

Adapted from Talking Back at blogs.ScientificAmerican.com/talking-back

CONSERVATION

In Search of a Wild Song

An elusive, endangered dog may be saved by dint of a recent photograph

COURTESY OF TOM HEWITT

The New Guinea singing dog is arguably the rarest *Canis* species in the world, more endangered than any other wild dog, jackal, coyote or wolf. The dogs' distinctive vocalizations—imagine a wolf howl crossed with whale song—can occasionally be heard echoing down from their homes in the rugged mountain ranges of the island of New Guinea, but the shy, agile animals have eluded many efforts to find them. They have been photographed in the wild only twice—once by Australian mammalogist Tim Flannery in 1989 and again by wildlife tour guide Tom Hewitt (*above*) in August 2012.

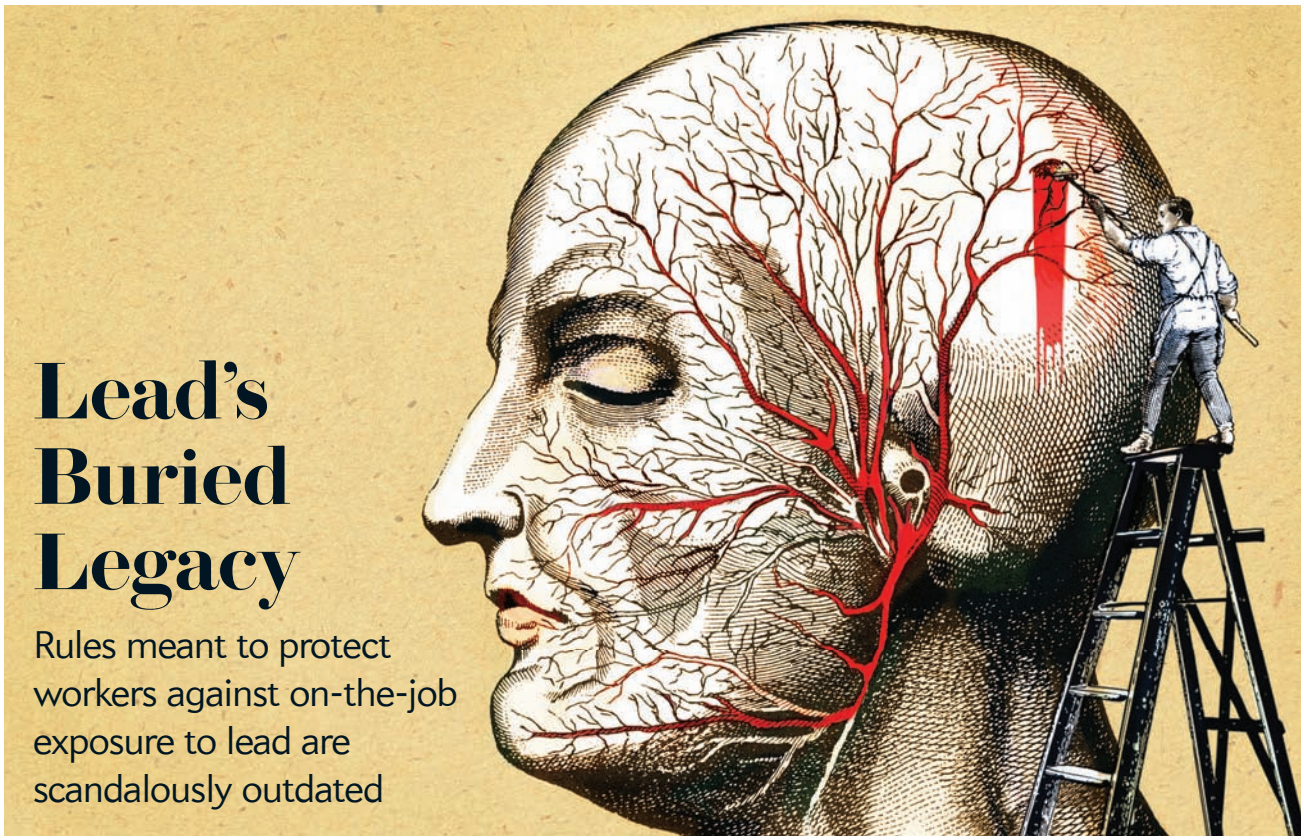
Hewitt snapped his photograph in the remote Star Mountains of West Papua, where some of the small wild population may have made the region's cloud forests their home. Inspired by Hewitt's picture, a team of researchers will be heading next year to the base of Mount Mandala in the region to seek the wild population. Members plan to collect DNA samples from sources such as scat piles and shed hairs to confirm Hewitt's sighting. Ultimately, they hope to capture a wild New Guinea singing dog.

The expedition isn't about trophy hunting, says leader James "Mac" McIntyre, director of the Southwest Pacific Research Foundation in Fernandina Beach, Fla. "Even though the dog photographed in August 2012 had the phenotype, or the physical 'look' of a New Guinea singing dog, science always requires definitive proof," McIntyre notes. That proof can come only through matching a dog's genotype, or genetic makeup, to that of purebred captive dogs, which number only about 200 individuals worldwide. Capturing a wild specimen may be crucial to the survival of the species because its bloodline could be infused into captive populations that are compromised by generations of inbreeding.

—Becky Crew

Adapted from Running Ponies at blogs.ScientificAmerican.com/running-ponies

Ingfei Chen is a freelance writer based in Santa Cruz, Calif. Her articles have appeared in the *New York Times*, *Science* and *Smithsonian*, among other publications.



Lead's Buried Legacy

Rules meant to protect workers against on-the-job exposure to lead are scandalously outdated

One of America's great public health achievements in the 20th century was removing lead—an extremely useful but incredibly toxic metal—from gasoline, paint, water pipes and food cans. Children are particularly vulnerable to the damage the element inflicts on nerve cells and the brain. Swallowing very large amounts can trigger convulsions and ultimately kill someone in a matter of days, but eating or inhaling a little lead here and there over longer periods can result in lower IQ, hearing loss, and behavioral problems, such as hyperactivity. Indeed, many researchers think there is no safe level of lead for children.

The more scientists learn about the dangers of lead, however, the more they realize that they may have underestimated how even small amounts of the element poison adults. Studies conducted over the past 20 years have documented a wide range of subtle, long-term medical issues—from an increased risk of high blood pressure and heart disease to various kidney and immune system problems—in men and women who were exposed either to the metal on the job or to lingering traces in soil, in air and in buildings constructed when lead paint was still in use.

The new insights raise concerns for older generations that accumulated lead in their bones during the leaded-gas-and-paint era. Although most of that lead is locked away in the skeleton for years to decades, the metal can leak back into the blood in small increments as people age and lose bone density. And many people currently working in such industries as metal smelting, lead-battery manufacturing and building renovation continue to routinely absorb the toxic element.

Under regulations that have not been updated since they were first established in 1978, the Occupational Safety and Health Administration (OSHA) permits blood lead concentrations in workers of 40 to 60 micrograms per deciliter (mcg/dL), depending on circumstances. Yet a 2012 scientific review from the U.S. National Toxicology Program linked concentrations between 5 and 10 mcg/dL—the rough equivalent of half a grain of salt per cup of blood—to elevated blood pressure, among other problems. "We haven't done a darn thing about what's going on with exposures for adults," says Ellen Silbergeld, an environmental health scientist at the Johns Hopkins Bloomberg School of Public Health. "We have an occupational lead standard in the U.S. that has not been changed for 35 years. It's outrageous."

CHRONIC DAMAGE

EVEN TODAY lead remains a prevalent and formidable environmental contaminant. The wind can whip up old lead dust from paint or gas emissions that settled into soil, explains environmental toxicologist Russell Flegal of the University of California, Santa Cruz. However the metal gets from one's surroundings into one's body, it takes its toll on living tissues in two key ways. It

Illustration by Leigh Guldig

interferes with the production of the oxygen-toting molecule hemoglobin in red blood cells, and it mimics the behavior of calcium, without any of calcium's benefits. Brain and nerve cells depend on calcium to transmit their electrical signals; when lead barges in, it garbles the usual communication between neurons.

Studying lead's long-term effects at low concentrations became possible only after the U.S. phased the metal out of gasoline between 1973 and 1996, which resulted in a sharp drop in the amount of lead in people's blood. Average concentrations around the country fell from 13 mcg/dL in the late 1970s to 1.12 mcg/dL as of 2010. With the help of increasingly sensitive instruments and better statistical methods for studying large populations, researchers have learned that tiny doses of lead can harm an individual's health even if they do not cause any overt symptoms.

"We haven't done a darn thing about what's going on with exposure for adults. We have an occupational lead standard in the U.S. that has not been changed for 35 years. It's outrageous."
—Ellen Silbergeld, Johns Hopkins

Research to date has associated small amounts of lead stored in bones—around 10 or 20 micrograms per gram of tissue, some of which may leak into the blood over time—with a dulling of mental acuity in the elderly equivalent to cognitive decline in three to five years of aging, notes Marc Weisskopf, a Harvard University epidemiologist. Yet untangling lead's influence from that of normal aging is tricky.

In contrast, the evidence for lead's subtle effects on the heart is much stronger. Studies over the past 10 years have linked various concentrations of lead in the blood below the 40 mcg/dL threshold permitted by OSHA to high blood pressure, which is a well-known risk factor for cardiovascular disease.

Lead likely raises blood pressure through several biochemical processes. Not only does the metal directly damage the delicate layer of cells lining blood vessels, it also hinders our cells from counteracting "free radicals"—highly reactive molecules that can harm various tissues, including those found in the circulatory system. Lead also inactivates a molecule known as nitric oxide and impairs the kidney, both of which are essential to regulating blood pressure. Small amounts of lead raise blood pressure by 1 percent or less, but even such tiny changes turn out to have greater repercussions than initially recognized for people on the border of hypertension and heart trouble.

In one investigation published in 2006, Johns Hopkins's Silbergeld and her colleagues at Tulane University looked at data collected from 13,946 men and women in the National Health and Nutrition Examination Survey between 1988 and 1994. After 12 years, individuals who initially had 3.63 mcg/dL were one and a half times more likely to have died from heart attack, stroke or other cardiovascular problems than those with levels below 1.93 mcg/dL. An elevated risk of cardiovascular death was still detectable at concentrations as low as 2 mcg/dL. What remains unclear, however, is whether such health risks arise primarily from these very low exposure levels, or if past, higher lead exposures in the older adults surveyed also play a role.

Given that cardiovascular disorders are such a common cause of premature death, anything that increases their risk even a little will translate to many more deaths in the population, experts maintain. One ballpark estimate from the Institute for Health Metrics and Evaluation in Seattle attributes 20,000 of the 670,000 annual cardiovascular deaths in the U.S. and Canada to lead exposure.

Although fewer workers encounter very high amounts of lead on the job these days, many of them still have too much lead in their blood, given the latest health research. Tens of thousands of workers still have levels exceeding 10 mcg/dL, at which adverse effects can occur, according to the National Institute for Occupational Safety and Health, a division of the Centers for Disease Control and Prevention. Consequently, public health experts have urgently called on OSHA to revise its regulations, but so far the agency has taken no action. "It's a profound disappointment and disservice to American workers," says Howard Hu, a physician and epidemiologist at the University of Toronto.

OSHA head David Michaels declined an interview request from SCIENTIFIC AMERICAN but e-mailed a statement via a spokesperson: "We recognize that our standard is outdated; this is true of many of OSHA's other chemical standards," Michaels wrote. "Unfortunately, OSHA's standard-setting process is extremely slow, and there is little that can be done under our current law to speed up that process."

So it has fallen to others to push awareness and change. In 2010 the CDC made a blood lead level of 10 mcg/dL or higher in adults a "notifiable" condition, which means that physicians and state health departments must report any results that exceed that limit to federal health authorities. This decision followed recommendations published in 2007 by several experts on lead poisoning, which advised doctors to remove workers who have blood lead levels of 20 to 30 mcg/dL from further exposure until their levels drop below 15 mcg/dL.

A handful of health organizations have adopted those guidelines or a modified version, including the American College of Occupational and Environmental Medicine and the California Department of Public Health, which is currently working with the California Division of Occupational Safety and Health to toughen the state's lead standards. And many companies have taken their own initiative to reduce on-the-job lead exposures to far below OSHA standards, including RSR Corporation, a major lead-battery recycler. Progress is afoot, but the pace is much too slow for far too many people whose health is on the line or has already been permanently damaged. SA

SCIENTIFIC AMERICAN ONLINE
Comment on this article at ScientificAmerican.com/sep2013

David Pogue is the personal-technology columnist for the *New York Times* and host of NOVA's *Making Stuff* on PBS this fall.

Death to the Upgrade

Gadgets used to become obsolete a week after you brought them home, but do they have to be?

How often do you buy a new car? A new house? A new couch? A new raincoat, fridge, or washer and dryer?

And now: How often do you get a new cell phone?

Clearly, the upgrade cycle plays a much bigger role in the tech industry than in any other realm of consumer goods. Most people wouldn't be embarrassed to drive a 2009 Toyota Camry or to put their food in a 2002 refrigerator (or even a 1992 fridge). But walk around with a four-year-old iPhone, and people think you're some kind of caveman.

The tech companies are fully aware of this, of course. They exploit it. Software companies crank out new Microsoft Offices or Intuit Quickens more or less every year, counting on our fear of obsolescence to drive our dutiful upgrades. New Every Two is no longer *officially* Verizon Wireless's marketing platform, but Americans still buy new phones, on average, about every 22 months. AT&T and T-Mobile just introduced plans that encourage their customers to upgrade their phones at least every year.

It would be easy to sweep all tech companies into the same pile, to mock their cynicism and manipulation, to accuse them of planned obsolescence on a criminal scale.

Take Apple, for example. The iPad has been the best-selling tablet since its debut. We count on a revised, better, feature-enhanced iPad model every year—and that puts Apple under certain pressure. How do you improve your product every single year, especially when a large part of its appeal is simplicity?

Apple added the extraordinarily sharp, high-resolution Retina screen to the iPad 3, released in early 2012, just as it had to the 2010 iPhone 4. So what screen did the new iPad mini get in late 2012? The old screen, not the Retina. To many, it appeared that Apple withheld a valuable feature so that it would have an enticing upgrade ready for the next version.

On an industry scale, it's hard to spot obvious patterns of planned obsolescence. In the cell phone and tablet worlds, in particular, the competition is so intense that manufacturers can't *afford* to play Withhold the Feature. When a new technology is ready for prime time (and sometimes even sooner), they bake it in and start promoting it. It would be hard to imagine Samsung or Microsoft, each desperate to compete with Apple, saying, "That's an awfully attractive feature; let's save it for next year."

And there's more reassuring news when you begin to consider different *kinds* of electronics. The PC cycle was once New Every Two, too. But these days Macs and PCs chug along for five, six or seven years before we replace them. That is largely because of the rise of the tablet and partly because there's not much innovation in PCs anymore.

Finally, remember this: we're *not* a bunch of trained sheep, conditioned to buy when the tech companies command us. You are perfectly capable of resisting the lure of a new model if the previous one is still fast enough for the software you want to use; utility, not the insecurity of being left behind, should drive your decisions.

Consider whether the new features offered in this year's model are genuinely worth the upgrade. Some will make a big, time-saving difference to your life: upshifting to a 4G LTE phone with far faster, more reliable Internet connections, for example. Others, like some of the gimmicky features on the Samsung Galaxy S phones, are little more than half-baked demo-ware. (Voice translator app, anyone?)

Yes, it's true that the engine of technology upgrades—especially in phones and tablets—runs faster and hotter than in other areas of consumer-dom. But the dynamic isn't as simple as: "We're the pawns, they're our calculating overlords." The cycles are driven by even stronger factors: technological progress, the rise and fall of gadget categories, and our own lust for the new. In short, just because you're *eligible* for an upgrade doesn't mean you have to take it. SA

SCIENTIFIC AMERICAN ONLINE
The annual obsolescence calendar: ScientificAmerican.com/sep2013/pogue

Illustration by Scott Brundage

the food issue

Food is a primal, everyday part of our lives—yet rich with mystery. In this special issue of *Scientific American,* we explore seemingly basic questions that have no simple answers. What makes us fat? Why do we eat some animals and not others? Are genetically engineered crops good or bad? And who figured out how to brew the first cup of coffee? To begin, we tackle the most enduring mystery of all:

What makes food taste so darn good?

an issue in three parts

feast
CELEBRATING OUR LOVE OF FOOD

fuel
HOW FOOD CHANGES US

farm
THE FUTURE OF THE FOOD SUPPLY

By Michael Moyer

Photograph by Dan Saelinger

TASTE IS NOT WHAT YOU THINK. Every schoolchild learns that it is one of the five senses, a partner of smell and sight and touch, a consequence of food flitting over taste buds that send important signals—sweet or bitter, nutrient or poison?—to the brain. Were it so simple.

In the past decade our understanding of taste and flavor has exploded with revelations of the myriad and complex ways that food messes with our consciousness—and of all the ways that our biases filter the taste experience. Deliciousness is both ingrained and learned, both personal and universal. It is a product of all five senses (hearing included) interacting in unexpected ways, those sensory signals subject to gross revision by that clump of nerve tissue we call the brain.

Let's start at the beginning: Food enters your mouth, meets your teeth and begins to be broken down by enzymes in your saliva. The morsel soon moves over your papillae, the few thousand bumps that line your tongue. ❶ Each papilla houses onionlike structures of 50 to 100 taste cells folded together like the petals of a young flower about to bloom—taste *buds,* we call them. These cells have chemical receptors attuned to the five basic tastes—bitter, sweet, sour, salt and umami, the last a word borrowed from Japanese that describes the savory flavors of roast meat or soy sauce. ❷

These five tastes are enough to help determine if the thing we just put into our mouth should go any farther—if it's sweet or savory and thus a probable source of nutrients or if it's bitter and potentially poisonous. Yet they can't get close to communicating the complexity of the flavors that we sense.

For that, we turn to the nose. As you take in a piece of food, a little air is forced

❶ *That tongue "taste map" your schoolteachers taught you? Forget it. The map was based on a misunderstood diagram in a 1901 paper.*

❷ *Some researchers argue that we should expand the list to include fatty, metallic and* kokumi, *which translates to "mouthfulness" or "heartiness."*

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up passageways at the back of the mouth, where scent receptors in the nasal cavity detect thousands of volatile chemicals that add up to complex flavors [*see box on pages 38 and 39*]. This retronasal olfaction, as it's called, has almost nothing to do, physiologically, with the act of sniffing your food. Your brain knows where your smell signals are coming from—through your nostrils or from your mouth. And in the case of the latter, it ropes them together with the signals from the taste buds. Retronasal olfaction produces a completely unique sense—neither smell nor taste alone but a hybrid that we call flavor. It's a process as transformative and irreversible as turning fuel and oxygen into flame.

Our sense of taste doesn't end at the mouth. In recent years scientists have found taste receptors all over the body, discoveries that have solved some long-standing mysteries. For 50 years scientists had been trying to figure out why eating glucose produces a much sharper insulin release than injecting the same amount of glucose directly into the bloodstream. In 2007 they discovered that cells lining the small intestine also contain taste receptors. When these intestinal sweet sensors detect sugar, they trigger a cascade of hormones that ultimately ends with a squirt of extra insulin into the bloodstream. ❸

Our sense of taste isn't just limited to the gut. For example, your nose is lined with cells that sense bitter chemicals. If there's poison in the air, they reflexively stop you from pulling it into your lungs.

❸ *Amazingly, these taste receptors are just as fooled by artificial sweeteners as your tongue is—NutraSweet also leads to a surge of insulin.*

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If the poison does get to the throat, bitter detectors in the trachea trigger cilia to help clear the airway. ❹

This physiology may explain what we mean by flavor—but anatomy doesn't much help us understand what we like. Our flavor preferences take shape over a lifetime, beginning while we are still in the womb. Babies whose mothers consume garlic while pregnant are more likely to enjoy the flavor of garlic in breast milk. Pregnant women who drink carrot juice are more likely to have kids who like carrots. ❺ The evolutionary justification is simple enough: If Mom ate it, it's safe.

Indeed, we use our friends and loved ones in much the same way that medieval monarchs used food tasters—let them try it first, then let's see how they are doing in 20 minutes. The principle holds all the way down the food chain. Rats hate the taste of cocoa ❻, yet some enterprising scientists recently separated a rat from its brood and coaxed it to eat some anyway. The rat then returned to its group. When the other rats smelled the cocoa on its breath, they changed their minds and suddenly couldn't get enough cocoa.

Children are harder to convince—they have to try an unfamiliar food about nine times, on average, before they begin to like the taste. ❼ As any parent will attest, so much of the eventual enjoyment rests on how well Mom and Dad sell it. Moreover, the same holds true for adults, as decades of increasingly sophisticated food-marketing campaigns have demonstrated.

The environment sends many cues about how food should taste. In one experiment, researchers connected volunteers' tongues to a low-voltage electrical device, showed them pictures of food items and then sent a mild shock across their taste buds—a sensation not unlike licking a battery. The shock was supposed

❹ *The more they look, the more researchers are finding taste receptors in the most unlikeliest of places. Sweet receptors line the bladder. Your spine has sour receptors. And most bizarrely, the testes have the capacity to sense bitter taste, whereas sperm can detect umami.*

❺ *The same has been tested for anise, mint, vanilla and every kid's favorite: blue cheese.*

❻ *The unsweetened variety—too bitter, presumably.*

❼ *Your mileage may vary.*

PROP STYLING BY DOMINIQUE BAYNES

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to impart a neutral taste. Asked afterward to rate how pleasurable the shock was, those volunteers who saw photographs of sweet or fatty foods rated the stimulus far more pleasurable than those who saw a low-calorie food.

The visual and auditory triggers can be so obvious as to appear comical. Potato chips taste crisper if you hear a crunch over headphones. White wine with a drop of red food coloring tastes like red wine—even to experienced wine tasters. People will eat less food off of a red plate. A block of cheese with sharp edges tastes sharper than one with round corners.

It's not all from our mouth, or our mouth and the back of our nose, or our mouth, and nose, and taste cells in the intestine. Deliciousness comes from our mother, our childhood, the room we are eating in, the plates we are eating on and the friends we are eating with. It's mental as much as chemical.

This hunger, this quest for deliciousness, has effects that reach far beyond our taste buds (and our waistlines). In this special issue of *Scientific American,* we have set out to explore some of the amazing ways that food continues to transform the world. We have organized the articles into three sections. The first, Feast, celebrates our love for eating and our long-standing ingenuity in making food taste delicious. The second, Fuel, examines the ongoing revolution in our understanding of how food changes us, from the surprising causes of the obesity crisis to the ancient rise of cooking that perhaps fueled our evolution into big-brained *Homo sapiens*. The third, Farm, profiles novel ideas for intelligently expanding the food supply.

As you'll see in these pages, we have learned much about food. Yet there is always so much more to know. SA

Michael Moyer *is the special projects editor at* Scientific American.

MORE TO EXPLORE

Taste: Making Sense of Flavour. Special issue of *Nature,* Vol. 486, Supplement, pages S1–S43; June 21, 2012.
Can We Feed the World? The Future of Food. *Scientific American* e-book available August 19, 2013. http://books.scientificamerican.com/sa-ebooks

SCIENTIFIC AMERICAN ONLINE
For an interactive flavor map, see ScientificAmerican.com/sep2013/flavor

TASTE MAP

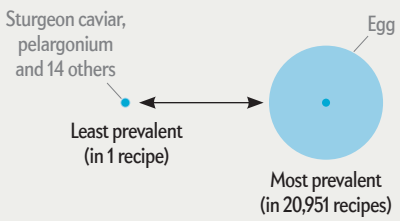
The Flavor Connection

Julia Child famously said that fat carries flavor, but perhaps instead we should give thanks to 4-methylpentanoic acid. Unique combinations of such chemical compounds give foods their characteristic flavors. Science-minded chefs have gone so far as to suggest that seemingly incongruous ingredients—chocolate and blue cheese, for example—will taste great together as long as they have enough flavor compounds in common. Scientists recently put this hypothesis to the test by creating a flavor map, a variant of which we have reproduced here. Lines connect foods that have components in common; thick lines mean many components are shared. By comparing the flavor network with various recipe databases, the researchers conclude that chefs do tend to pair ingredients with shared flavor compounds—but only in Western cuisine. Dishes from a database of recipes from East Asia tend to combine ingredients with few overlapping flavors.

How to Read This Graphic

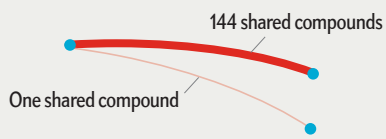
Each blue dot is a food. Similar foods are grouped into 14 category columns (listed in alphabetical order).

The size of a dot shows how popular the food is—the frequency with which it appears in a global 56,498-recipe database.



Of all the foods that share flavors outside of their own categories (and excluding roasted peanuts/peanut butter), beer and roasted beef have the most in common: 106. Close behind are apples/white wine and coffee/roasted beef, both with 105.

A line connecting two dots means the two foods share at least one flavor-related chemical compound. The more flavor compounds they share, the thicker the line. Red lines connect foods in different categories.



Wine and cheese contain many of the same flavor-producing chemicals.

Gray lines connect foods in the same category.

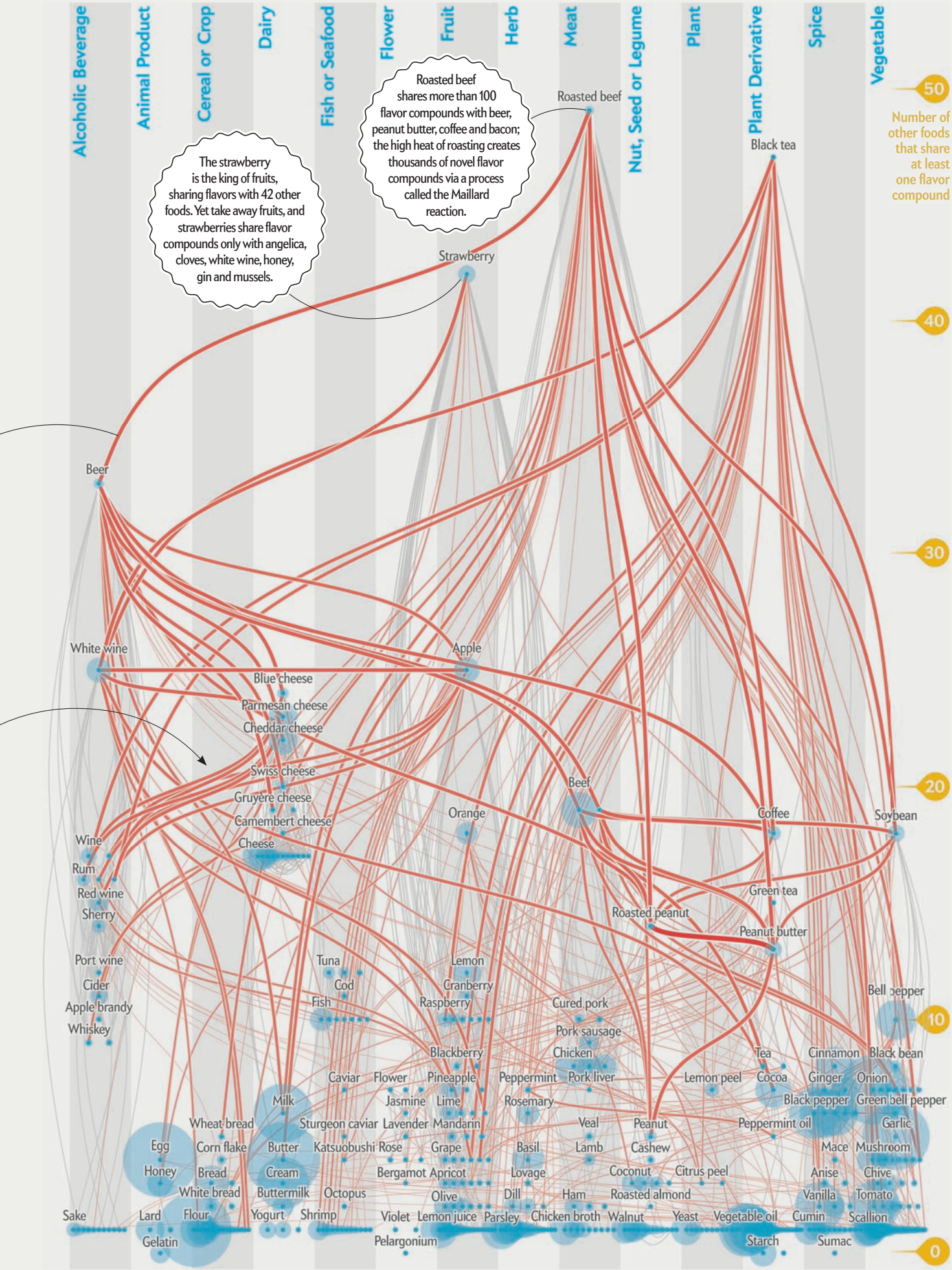
A food's vertical position on the page reveals the total number of foods that connect to it. Foods at the top of the page share flavor compounds with many other foods. Foods at the bottom of the page are completely unique—they don't share flavors with any other foods.

Because of space constraints, only the most popular ingredient in a cluster of dots is labeled.

Eggs, flour and butter were the three most popular ingredients, each appearing in more than 20,000 recipes. Rounding out the top 10: onion, garlic, milk, vegetable oil, cream, tomato and olive oil.

SOURCE: "FLAVOR NETWORK AND THE PRINCIPLES OF FOOD PAIRING," BY YONG-YEOL AHN, SEBASTIAN E. AHNERT, JAMES P. BAGROW AND ALBERT-LÁSZLÓ BARABÁSI. IN *SCIENTIFIC REPORTS*, VOL. 1, ARTICLE NO. 196; DECEMBER 15, 2011. WITH THANKS TO SEBASTIAN AHNERT

Graphic by Jan Willem Tulp


Alcoholic Beverage
Animal Product
Cereal or Crop
Dairy
Fish or Seafood
Flower
Fruit
Herb
Meat
Nut, Seed or Legume
Plant
Plant Derivative
Spice
Vegetable
Roasted beef shares more than 100 flavor compounds with beer, peanut butter, coffee and bacon; the high heat of roasting creates thousands of novel flavor compounds via a process called the Maillard reaction.
The strawberry is the king of fruits, sharing flavors with 42 other foods. Yet take away fruits, and strawberries share flavor compounds only with angelica, cloves, white wine, honey, gin and mussels.
Roasted beef
Black tea
Strawberry
Beer
White wine
Apple
Blue cheese
Parmesan cheese
Cheddar cheese
Swiss cheese
Gruyère cheese
Camembert cheese
Cheese
Beef
Orange
Coffee
Soybean
Wine
Rum
Red wine
Sherry
Port wine
Cider
Apple brandy
Whiskey
Green tea
Roasted peanut
Peanut butter
Tuna
Cod
Fish
Lemon
Cranberry
Raspberry
Cured pork
Pork sausage
Chicken
Pork liver
Bell pepper
Tea
Cinnamon
Black bean
Cocoa
Ginger
Onion
Black pepper
Green bell pepper
Lemon peel
Peppermint oil
Garlic
Mace
Mushroom
Anise
Chive
Vanilla
Tomato
Scallion
Cumin
Sumac
Starch
Yeast
Vegetable oil
Walnut
Roasted almond
Ham
Coconut
Citrus peel
Veal
Peanut
Lamb
Cashew
Caviar
Flower
Pineapple
Peppermint
Jasmine
Lime
Rosemary
Blackberry
Milk
Wheat bread
Sturgeon caviar
Lavender
Mandarin
Egg
Corn flake
Butter
Katsuobushi
Rose
Grape
Basil
Honey
Bread
Cream
Bergamot
Apricot
Lovage
White bread
Buttermilk
Octopus
Olive
Dill
Sake
Lard
Flour
Yogurt
Shrimp
Violet
Lemon juice
Parsley
Chicken broth
Gelatin
Pelargonium
50
Number of other foods that share at least one flavor compound
40
30
20
10
0

DINNER IS SERVED: Asian shore crabs have spread rapidly since their introduction on the U.S. East Coast nearly three decades ago. Here they are served on a "plate" of invasive wakame seaweed.

ECOLOGY

how (and why) to eat invasive species

What's the best way to control ecological pests? Feed them to the world's greatest predator—us

By Bun Lai

Photograph by Grant Cornett

Bun Lai, a 2013 James Beard Award nominee, is the chef at Miya's Sushi, a restaurant founded by his mother in New Haven, Conn. He dives and fishes in Long Island Sound to supply his restaurant with underutilized seafood.

MY RESTAURANT, MIYA'S SUSHI, IS JUST a few miles from Long Island Sound. An important goal of ours is to have our cuisine return to the roots of sushi, meaning simply to use what we have available where we live. Often what we find now are invasive species—unwanted plants and animals that humans have introduced to ecosystems. Nationwide, invasive species such as the wild boar and Asian carp are destroying farms and fisheries, causing economic damage that has been estimated at $120 billion a year.

Our solution? Eat them. By collecting invasive seafood on shellfish beds, for instance, we basically provide a free weeding service. I also hope to convince the world that these invasives can be delicious—if you get into the right mind-set.

Consider the stalked tunicate—also known by the delicious-sounding name "Asian sea squirt"—which has taken over what used to be blue mussel habitat from Maine to New Jersey. The alien sea squirt, which is indigenous to the Philippines, is considered a fouling organism and a pest by the shellfish industry. In South Korea, however, it is considered a delicacy and even an aphrodisiac.

I first ate sea squirt at a Korean sushi bar in New York City. The saclike squirts were arranged like a sunflower in the middle of a bright orange plate. As I bit into one of the yellow appendages, it burst with salty, viscous, warm liquid. Although I could not see the liquid, I could taste its phlegmy consistency, and it took all my willpower to keep it in my mouth and even more effort to swallow it.

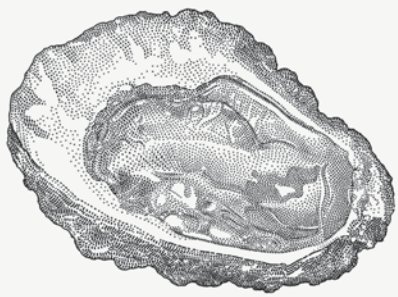
Buckminster Fuller used to say that one should "dare to be naive." I think it takes a bit of his approach to truly accept new ways of doing things—including, of course, eating. The next time I tried sea squirt, I scraped one off a pier. I sliced open its tough outer membrane, which revealed a soft, orange flesh, like mango. With barely a pause, I slurped it into my mouth from the palm of my hand. This time it was good.

Over the years I have foraged, fished and hunted lots of different plants and animals; the following are just a few of the dishes I have served in my restaurant from the invasive ones. SA

Today's Specials

La Soupe des Mean Greenies

European green crabs made their way to the U.S. in the 19th century. They voraciously consume the larvae of commercial shellfish species and are considered one of the top 100 most destructive invasive species in the world. I smoke the crabs with applewood, dehydrate with lemongrass and hot peppers, then pulverize them into a powder that I use for the base of a savory crab-miso soup. I then steam whole crabs in a hoppy beer and hot Ethiopian spices and serve them atop the soup as if they are struggling to climb out—a symbol of the durability of invasive species.



Stone Soup

The European flat oyster was deliberately introduced to Maine in the 1940s and competes with native shellfish. I simmer a rock covered in European flat oysters, rockweed and invasive wakame in a sake broth flavored with Queen Anne's lace root, wild onions and native morels. Served in a large iron pot and designed to be shared by a small village.

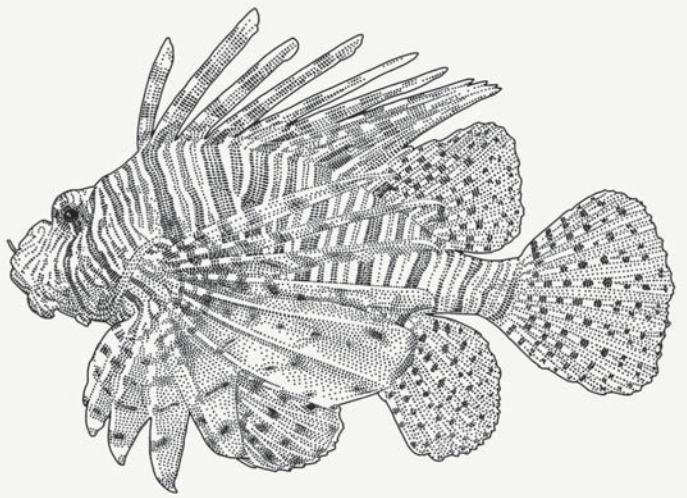
Illustrations by Keith Witmer

Knot Your Mother's Lemonade

Japanese knotweed grows quickly in clusters and crowds out other herbaceous species. It has been named one of the world's 100 worst invasive species by the International Union for Conservation of Nature and is currently thriving in 39 states. The taste is crunchy, juicy and tart—not unlike a Granny Smith apple. In a combination of mineral water and ice, I blend Japanese knotweed shoots with fresh stevia leaves, fresh kefir lime leaves and a splash of lemon juice.

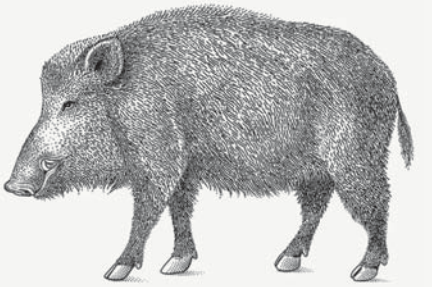
Kudzu Tchaikovsky Sushi

Native to Europe and Asia, the mute swan was introduced to the U.S. as an ornamental species. The swan's majestic looks have earned it protected status in some parts of the U.S., but the swans damage marshes and shallow-water habitats by tearing up vegetation. Kudzu, known as the mile-a-minute plant for how quickly it grows, is in the pea family and was introduced to the U.S. from Asia by gardeners in the 1930s. It creates a canopy and suffocates native forests. I rub bow-shot swan in a puree of olive oil, freshly grated ginger and Jamaican jerk seasoning, then slow-roast it. The tender dark meat is finely chopped and mixed with roasted shallots and rosemary. Served in a steamed kudzu-leaf roll stuffed with a sherry-scented sticky rice and wild morels.



Kiribati Sashimi

Lionfish is a voracious, highly poisonous, invasive predator that has been compared with the locust in its destructiveness. This fish is believed to have been introduced to the U.S. by people in the aquarium trade. Protected by highly toxic spines and resembling seaweed, lionfish have few natural enemies. Yet with their dangerous spines removed, their flesh is sweet and delicious. Served raw and sliced thin, with a squeeze of lime juice, a sprinkling of seven different types of crushed peppers, roasted seaweed flakes, toasted sesame seeds and sea salt from Kiribati, a Pacific island nation that will soon be engulfed by the ocean because of climate change.



Oinkimo

Feral hogs were introduced by European explorers in the 1500s; their numbers have exploded in recent years. The pigs consume some native and endangered species and fight for resources with others. But feral hog meat is pharmaceutical-free, unlike most commercial pork, which is raised with antibiotics. I wrap roasted invasive daylily buds in seared, thin-sliced feral hog meat, then drizzle the pig rolls with a ginger, garlic, roasted sesame and sauvignon blanc soy sauce.

Peanut Butter and Jelly

Feral rabbits are some of the most ecologically destructive animals. They procreate uncontrollably, destroy croplands and contribute to soil erosion. Jellyfish populations are expected to explode because of the acidification of the oceans, yet very few cultures appreciate them as a food source. The warty comb jelly, one of the most invasive species on earth, is linked to the collapse of a handful of fisheries. This recipe is my twist on the classic steak-house surf and turf. Invasive cannonball jellyfish, trawled off the state of Georgia, is thin-sliced and mixed with steamed invasive Australian rabbit and cucumber. The combination is seasoned with creamy, roasted peanut butter.

MORE TO EXPLORE

Eat the Invaders: www.eattheinvaders.org
National Invasive Species Information Center: www.invasivespeciesinfo.gov

SCIENTIFIC AMERICAN ONLINE
Share your recipes for invasive species at ScientificAmerican.com/sep2013/invasives

NEUROSCIENCE

the food addiction

New brain research is revealing why fats and sugars may be driving more and more people toward obesity

By Paul J. Kenny

Photoillustration by James Worrell

Paul J. Kenny is an associate professor at the Scripps Research Institute in Jupiter, Fla. His laboratory investigates the mechanisms of drug addiction, obesity and schizophrenia, as well as medications for these disorders.

WOULD A RAT RISK DYING JUST TO SATISFY its desire for chocolate?

I recently found out. In my laboratory, we gave rats unlimited access to their standard fare as well as to a mini cafeteria full of appetizing, high-calorie foods: sausage, cheesecake, chocolate. The rats decreased their intake of the healthy but bland items and switched to eating the cafeteria food almost exclusively. They gained weight. They became obese.

We then warned the rats as they were eating—by flashing a light—that they would receive a nasty foot shock. Rats eating the bland chow would quickly stop and scramble away, but time and again the obese rats continued to devour the rich food, ignoring the warning they had been trained to fear. Their hedonic desire overruled their basic sense of self-preservation.

Our finding mirrored a previous trial by Barry Everitt of the University of Cambridge—only his rats were hooked on cocaine.

So are the fat rats addicted to food? An inability to suppress a behavior, despite the negative consequences, is common in addiction. Scientists are finding similar compulsiveness in certain people. Almost all obese individuals say they want to consume less, yet they continue to overeat even though they know that doing so can have shockingly negative health or social consequences. Studies show that overeating juices up the reward systems in our brain—so much so in some people that it overpowers the brain's ability to tell them to stop eating when they have had enough. As with alcoholics and drug addicts, the more they eat, the more they want. Whether or not overeating is technically an addiction, if it stimulates the same brain circuits as drug use, in the same way, then medications that dial down the reward system could help obese people to eat less.

SUSPICIOUS HORMONES

UNTIL THE EARLY 1990S, society viewed obesity solely as a behavioral disorder: overweight individuals lacked willpower and self-control. Since then, the view has changed dramatically, in the scientific community at least.

The first change in opinion arose from pioneering work by Douglas Coleman of the Jackson Laboratory in Bar Harbor, Me., and by Jeffrey Friedman of the Rockefeller University. Experiments with two strains of mice, both genetically prone to obesity and diabetes, determined what drove the mice to overeat. The researchers discovered that one strain had a genetic defect in fat cells that secrete a hormone called leptin. Mice, like humans, normally secrete leptin after a meal to suppress appetite and prevent overeating. The obese mice had a leptin deficiency—and an insatiable appetite. Researchers later found that obesity in the second strain of mice was caused by a genetic defect in their ability to respond to leptin and regulate its actions. The findings seemed to make it clear that hormones regulate appetite and therefore body weight. A hormonal imbalance could lead to overeating; indeed, obesity runs rampant in certain human families that have a genetic deficiency in leptin.

Two observations suggest that viewing obesity as a hormone disorder is too simplistic, however. First, only a small number of obese people in the U.S. and elsewhere have a genetic deficiency in appetite-related hormones. Second, we would expect blood tests of obese people to show either a lower level of hormones that suppress appetite or a higher level of hormones that increase appetite. Yet the reverse is true. Obese individuals generally have a paradoxically high level of appetite-suppressing hormones, including leptin and insulin.

This is where the concept of food addiction comes into play. Appetite-controlling hormones affect certain pathways of neurons—feeding circuits—in the hypothalamus. They also affect systems in the brain that control feelings of reward, which makes perfect sense. If you have not eaten for many hours, you

IN BRIEF

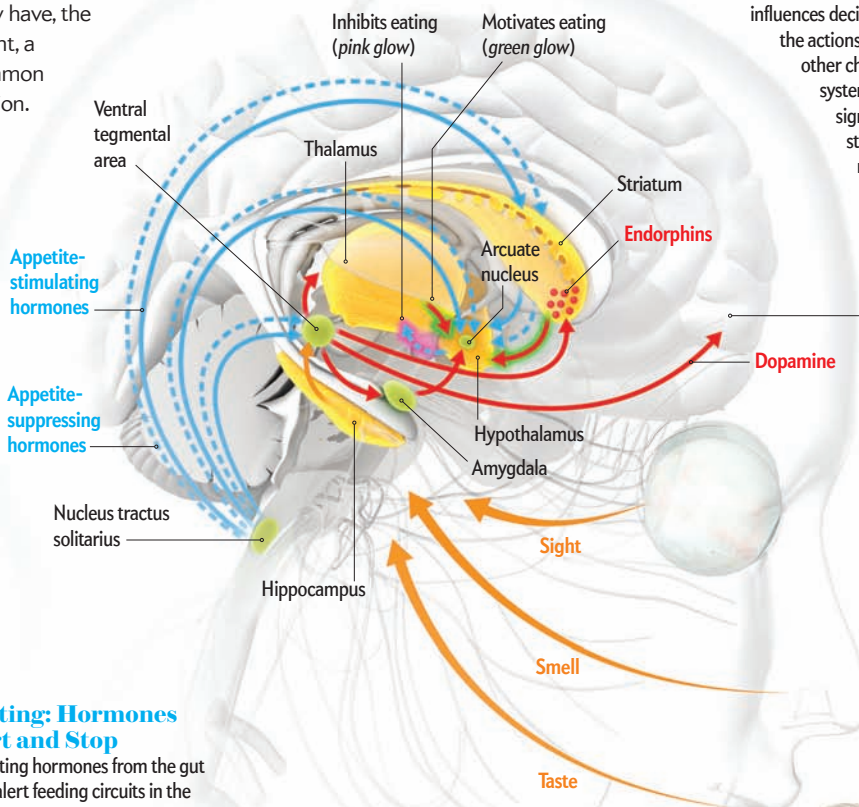
New science shows that overeating is not a behavioral disorder, such as a lack of self-control, and is not caused by a hormonal imbalance.

Instead foods rich in fat and sugar can supercharge the brain's reward system, which can overpower the brain's ability to tell an individual to stop eating. In these cases, the more someone eats, the more he or she wants.

Whether that kind of mechanism is an addiction matters only if it leads to effective treatments. The drug rimonabant, which reduces nicotine cravings in tobacco users, can reduce the desire for food, but it has dangerous side effects. More work is needed to determine whether the brain's overeating networks are the same as its drug addiction pathways and, if so, whether addiction treatments can reduce the obesity epidemic.

Hooked on Food

Our brains maintain healthy body weight by signaling when to eat and when to stop. Hormones regulate feeding circuits that control appetite and satiety (*blue*). But fatty, sugary foods can motivate some people to overeat (*red*). The more they have, the more they want, a sensation common in drug addiction.



Overeating: Brain Chemicals Hijack the Controls

Foods that are dense in fat and sugar prompt the striatum to make endorphins, "feel good" chemicals that can trigger binge eating. The foods also spark dopamine release (*red lines*) by the striatum, which motivates feeding behavior, and into the prefrontal cortex, which influences decision making. In some people, the actions of endorphins, dopamine and other chemicals that regulate reward systems can overpower hormonal signals and conscious attempts to stop eating when full. A strong motivation to eat high-calorie foods prevails despite an individual's knowledge about health consequences.

Treatment Prospects

Addictive drugs lead to dopamine release and feedback loops in the brain that can spur people to seek more and more—just as overeating high-calorie foods can do. Medications that could break this cycle could possibly ease not only drug addiction but obesity.

Normal Eating: Hormones Signal Start and Stop

Appetite-stimulating hormones from the gut (*solid blue lines*) alert feeding circuits in the hypothalamus. They also stimulate reward centers, such as the ventral tegmental area and the striatum, which increases the pleasure associated with eating. As the gut fills and blood nutrient levels rise, appetite-suppressing hormones such as leptin and insulin are released (*dashed blue lines*) in the hypothalamus and reward centers to suppress appetite and inhibit pleasure, making more food less appealing.

will spend a great deal of time, effort and money to obtain food—and it will taste very good! As the old adage says, "Hunger is the best sauce."

During periods of hunger, hormones heighten the reactivity of food-related reward circuits in the brain, particularly in the striatum. The striatum contains high concentrations of endorphins—chemicals that enhance feelings of pleasure and reward.

As you eat, your stomach and gut release appetite-suppressing hormones that decrease pleasure signals that are triggered by the striatum and other components of the reward system. This process makes food seem less attractive, and you may switch your activity away from eating and toward other pursuits. Appetite-regulating hormones control feeding, in part by modulating the pleasurable experience of consuming a meal.

Yet some modern, appetizing foods—dense in fat and sugar and often visually appealing—affect reward systems strongly enough to override the appetite-suppressing hormones, thus prompting us to eat. These foods activate our reward circuits more powerfully than leptin's ability to shut them down. All of us have experienced this effect: you have just finished a big dinner and could not possibly eat another bite. Yet when the chocolate cake appears, you can miraculously "find room" for one last morsel—one that happens to be the most calorie-laden of the day.

Therein lies the rub. We have evolved an efficient brain system to help maintain a healthy and consistent body weight by signaling when it is time to eat and when it is time to stop. But

Illustration by Bryan Christie

highly appetizing foods can often override these signals and drive weight gain.

Our body responds to the override by elevating the blood levels of appetite-suppressing hormones such as leptin and insulin higher and higher as body weight increases, yet the hormones become progressively less effective as the body develops tolerance to their actions. Moreover, brain-imaging studies by researchers at Brookhaven National Laboratory and the Oregon Research Institute show that the brain's reward systems in overweight individuals respond weakly to food, even to junk food. These muffled reward circuits depress mood. How does an individual overcome this funk? By eating more delectable food to gain a temporary boost, thereby perpetuating the cycle. Obese individuals may overeat just to experience the same degree of pleasure that lean individuals enjoy from less food.

Obesity, it seems, is not caused by a lack of willpower. And it is not always caused by an imbalance in hormones. In some cases at least, obesity may be caused by hedonic overeating that hijacks the brain's reward networks. Like addictive drugs, overeating creates a feedback loop in the brain's reward centers—the more you consume, the more you crave, and the harder it is for you to satisfy that craving.

But does that make hedonic eating an addiction?

Obesity is driven by an overpowering motivation to satisfy the brain's reward centers; hormone issues may be a consequence, not a cause.

TOLERANCE AND RELAPSE

DRUGS OF ABUSE, such as morphine, stimulate the brain's reward systems the way food does. Yet the similarities do not end there. When morphine is injected into the striatum of rats, it triggers bingelike overeating, even in rats that have been fed to satiety. This response shows that morphine and other opiates mimic the effects of neurotransmitters (brain chemicals) such as endorphins that are naturally produced in the brain to stimulate feeding behaviors.

We might expect, then, that drugs that block the action of endorphins could reduce hedonic overeating. Recent studies have shown that endorphin blockers do lessen the activation of reward circuits in humans and rodents that are presented with appetizing food—the subjects eat less. The blockers can also reduce heroin, alcohol and cocaine use in human drug addicts, supporting the idea that common mechanisms regulate hedonic overeating and addictive drug use. Strikingly, rats that binge on food every day display behaviors that closely resemble withdrawal, a symptom of drug addiction, after they are treated with endorphin blockers. This behavior raises the remarkable notion that hedonic overeating can induce a drug-dependence-like state.

These discoveries add credence to the idea that overeating in some circumstances may share core features of drug addiction. We see the same similarities with another basic neurotransmitter: dopamine. All known addictive drugs lead to the release of dopamine into the striatum. Dopamine is central to motivation, spurring people to seek the drug. Most experts maintain that this action drives the development of addiction, although the precise mechanisms are hotly debated. It turns out that appetizing food also stimulates the release of dopamine into the striatum, motivating people to focus on obtaining and consuming food. Imaging studies reveal that the striatum of obese individuals shows low levels of a receptor that responds to dopamine, termed the dopamine D2 receptor (D2R). The same holds true for those suffering from alcoholism or from opiate, cocaine or methamphetamine addiction.

We now also know that people who are born with reduced levels of D2R are at greater genetic risk of developing obesity and drug addiction. The condition results in lower levels of activity in the brain's reward systems, suggesting that these individuals may overeat just to obtain the same level of pleasure from food as those who do not have D2R deficits. These people also tend to have trouble learning to avoid actions that have negative consequences; brain systems involved in suppressing risky yet rewarding behaviors, such as consuming high-calorie food or using drugs, may not work as effectively.

Our lab study of rats backs up this idea. The obese rats that ate the cafeteria food regardless of warnings about being shocked had reduced levels of D2R in their striatum. Our study and others demonstrate that drug use in addicted rats and hedonic eating in overweight rats persist even when the animals face negative consequences. Many obese individuals struggle so badly with their poor food choices that they will voluntarily undergo potentially dangerous procedures, such as gastric bypass surgery, to help them control their eating. Yet very often they will relapse to overeating and gain weight.

This cycle of engaging in a bad habit that gives short-term pleasure, then attempting to abstain from it and eventually relapsing, sounds disturbingly like drug addiction. Given the latest research, it seems that obesity is caused by an overpowering motivation to satisfy the reward centers—the pleasure centers—of the brain. The hormonal and metabolic disturbances in obese individuals may be a consequence of weight gain rather than a cause.

NEW TREATMENTS POSSIBLE

THE SIMILARITIES between obesity and addiction have led certain experts to say that the two conditions should be treated in the same manner. Some of them recommended that obesity be in-

GETTY IMAGES

cluded in the most recent update to the *Diagnostic and Statistical Manual of Mental Disorders*—the bible of psychiatry that provides guidelines for diagnosing mental illnesses, known as the *DSM-5*. This proposal sparked lively debate among neuroscientists and psychiatrists, but arbiters for the *DSM-5* ultimately dropped the idea, largely to avoid labeling obese people, in essence, as mentally ill.

Caution may have been warranted because despite the parallels, obesity and addiction differ in important ways. For example, if food is addictive, then surely it must contain some unique component that drives the addiction—the nicotine of junk food, if you will. Work by Nicole Avena of the University of Florida, the late Bartley Hoebel of Princeton University and others lends some credence to the idea that particular fats or sugars may be responsible. A small study by David Ludwig of Boston Children's Hospital suggests that highly processed, quickly digested carbohydrates could trigger cravings. But research overall indicates that no one ingredient stokes addictionlike behaviors. Rather the combination of fats and sugars, together with calorie content, seems to maximize food's "hedonic impact."

Other experts, including Hisham Ziauddeen, I. Sadaf Farooqi and Paul C. Fletcher of the University of Cambridge, do not think that tolerance and withdrawal occur in obese people the way they do in drug addicts. They argue that obesity and drug addiction are fundamentally different. This view is debatable, however. If obese individuals must eat more and more to overcome reduced activation of reward networks in the brain, that sounds a lot like tolerance. And weight loss can trigger negative mood and depression, much like that experienced by former addicts who try to practice abstinence, suggesting that withdrawal may be in effect.

Other experts have argued that the entire notion of food addiction is preposterous because we are all, in a sense, addicted to food. If we were not, we would not survive.

The difference in obesity, I would suggest, is that modern high-calorie foods can overwhelm our biological feedback networks in a way that other foods cannot. During millions of years of evolution, the major concern of humans was not suppressing appetite but hunting, collecting or growing enough food to persist during lean times. Perhaps our feeding circuits are better at motivating food intake when we are hungry than they are at suppressing food intake when we are full. It is easy to imagine that the brain would regard overeating of high-calorie food as tremendously beneficial if it is unclear when food will again be available. Perhaps this behavior is no longer adaptive and could even be counterproductive in a world where food is bountiful.

The scientists who argue against an addiction model of obesity make reasonable points, and I also fear that the term "addiction" comes loaded with unhelpful preconceptions. Still, compulsive eating and compulsive drug use seem to share obvious features, most notably an inability to control consumption. It is up to scientists to determine if these similarities are superficial or stem from common, underlying alterations in the brain. More important will be determining whether the addiction model is useful. Unless it helps us design new treatment approaches, the debate is simply an academic exercise.

For an addiction model to have value, it should make accurate predictions about treatment options, including new medications. One example comes from Arena Pharmaceuticals, which recently obtained approval from the U.S. Food and Drug Administration to market a drug called Belviq for weight loss in obese or overweight adults. The drug stimulates a brain protein called the serotonin 2C receptor, which reduces the desire to consume nicotine in lab rats.

Another drug is rimonabant, which had been approved in Europe to help curb appetite in obese individuals. The drug exploits the well-known property of cannabis to increase desire for food—the so-called munchies. Cannabis activates a brain protein called the cannabinoid receptor 1, so researchers reasoned that inhibiting that receptor would decrease desire for food. Rimonabant does exactly that. A notable side effect is its ability to decrease tobacco users' desire to smoke. In rats, the drug also decreases the desire to use alcohol, opiates and stimulants such as cocaine.

As with all potentially therapeutic drugs, however, caution is required. Rimonabant has triggered depression and thoughts of suicide in some individuals. This finding led European authorities to suspend its use and prompted U.S. officials to not approve it. Why depression emerged is still unclear. Thus, although an addiction model of obesity could yield unexpected treatments, those modalities must be thoroughly scrutinized.

Before scientists can declare that overeating is or is not an addiction, they will have to identify precisely which networks and cellular adaptations in the brain drive compulsive drug use and then determine if the same mechanisms also motivate compulsive food intake. It is possible, even likely, that addiction networks for cocaine and for food operate in different parts of the brain yet use similar mechanisms. Scientists will also have to determine if common genetic variations, such as those that affect D2R, contribute to drug addiction and obesity. Identifying such genes may reveal new targets for medications to treat both disorders.

Even if scientists prove that obesity can stem from an addiction to food, and we find that antiaddiction medications can help people lose weight, obese individuals will have to struggle with one factor that seems now to be endemic in America: they will probably be surrounded by overweight family members, friends and co-workers who are still overeating, putting them in the same difficult environment they were in before. As we know from recovering drug addicts and alcoholics, environmental cues are a major cause of craving and relapse. Western society, saturated in fat and temptation, will make it hard for any obese person to quit. SA

MORE TO EXPLORE

Leptin Receptor Signaling in Midbrain Dopamine Neurons Regulates Feeding. Jonathan D. Hommel et al. in *Neuron,* Vol. 51, No. 6, pages 801–810; September 21, 2006.

Relation between Obesity and Blunted Striatal Response to Food Is Moderated by TaqIA A1 Allele. E. Stice et al. in *Science,* Vol. 322, pages 449–452; October 17, 2008.

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SCIENTIFIC AMERICAN ONLINE

For new insights into how eating sugar may affect insulin and therefore the brain's reward centers, see **ScientificAmerican.com/sep2013/kenny**

the amazing multimillion-year history of processed food

It is the dark force, we're told, behind the obesity epidemic, the death of the family farm and Tang. But humans have been "processing" food ever since we learned how to cook, preserve, ferment, freeze, dry or extract. Processed food has powered the evolution of the species, the expansion of empires, the exploration of space. Here are highlights

By Evelyn Kim

As early as 1.8 million years ago

ROASTED MEAT

Fire-kissed food is easier to digest and more nutritious than raw food is. Some anthropologists argue that cooking was the essential step that allowed early humans to develop the big brains characteristic of *Homo sapiens* [see "The First Cookout," on page 66].

30,000 years ago

BREAD

Agriculture began around 12,000 years ago, but early Europeans were baking bread many thousands of years before that time. In 2010 scientists found surprising evidence of starch grains on crude mortars and pestles at sites in modern-day Italy, Russia and the Czech Republic. The starches came from the roots of cattails and ferns, which early humans pounded into flour, mixed with water and baked into bread.

Bread was portable and nutrient-dense and resisted spoilage. It was also a nutritional step backward. Comparative studies show that Neolithic hunter-gatherers ate a more varied and nutritious diet than Neolithic farmers. And from the perspective of energy consumption, hunter-gatherers were far more efficient: a farmer would have to spend 10 hours to grow food with the same number of calories that six hours of foraging could provide.

Then why bother with bread at all? Anthropologists debate why farming became dominant, but one thing is certain: bread and agriculture were codependent. As societies began to rely on bread as a major foodstuff, they were also forced to expend more effort on agriculture (and vice versa).

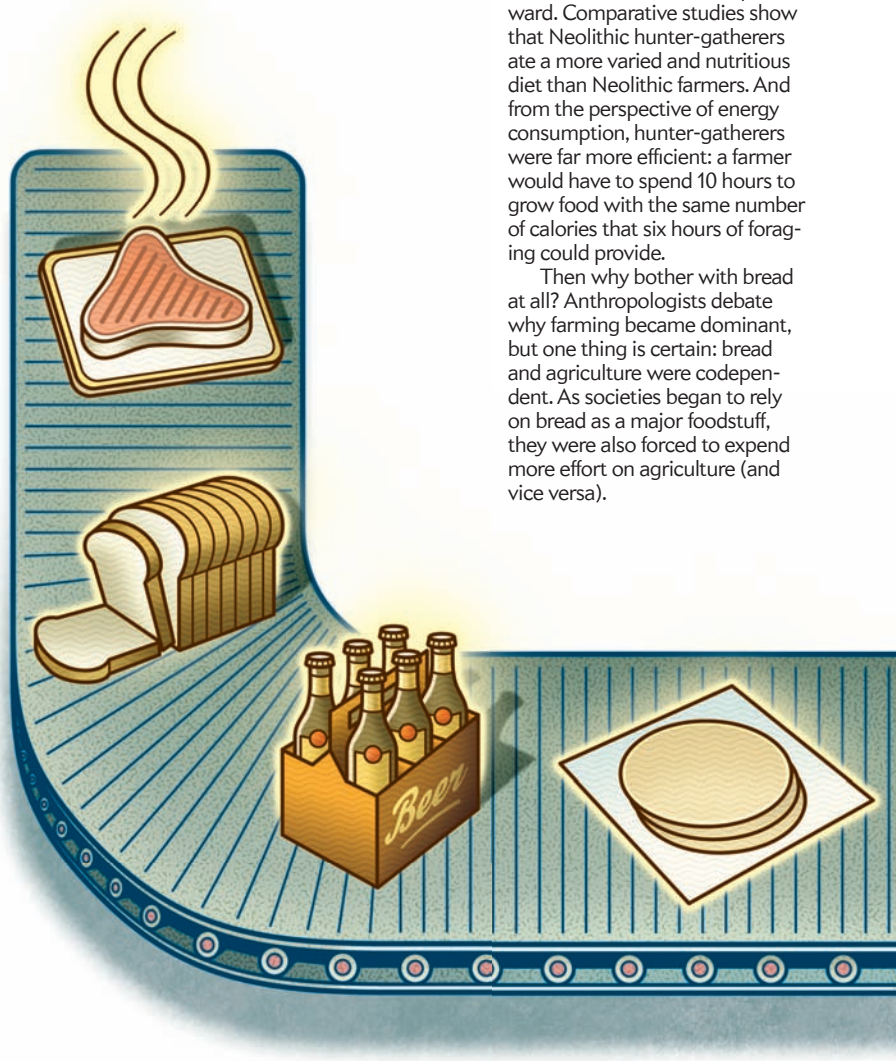

Illustration by Peter and Maria Hoey

MORE TO EXPLORE

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The Oxford Encyclopedia of Food and Drink in America. Second edition. Edited by Andrew F. Smith. Oxford University Press, 2012.

7000 B.C.

BEER

The birth of beer is hard to place. The oldest physical evidence comes from pottery shards in Iran that date back to 3500 B.C., but archaeologists such as Patrick McGovern of the University of Pennsylvania suggest that the first ale may have been produced as early as 7000 B.C. as a by-product of bread making. Early societies quickly embraced the accident: ancient Sumerians may have diverted as much as 40 percent of all grain to beer production.

Modern-day brewers, with help from archaeologists, have attempted to re-create ancient brews. McGovern has partnered with Dogfish Head Craft Brewery to ferment ancient Egyptian and Chinese beverages, whereas Great Lakes Brewing Company, with help from researchers at the University of Chicago, is brewing beer based on a 3,800-year-old ode to the Sumerian beer goddess Ninkasi.

6700 B.C.

TORTILLAS

No written records predate the arrival of Spanish explorers in the Americas, but the earliest archaeological evidence for maize domestication dates back around 8,700 years. Early Americans would soak kernels in a lime solution to create masa, releasing nutrients in the process.

5400 B.C.

WINE

The earliest evidence of wine making has been found in the Zagros Mountains in Iran. Seafaring Phoenicians then spread the practice westward from Lebanon to Egypt and the Mediterranean.

5000 B.C.

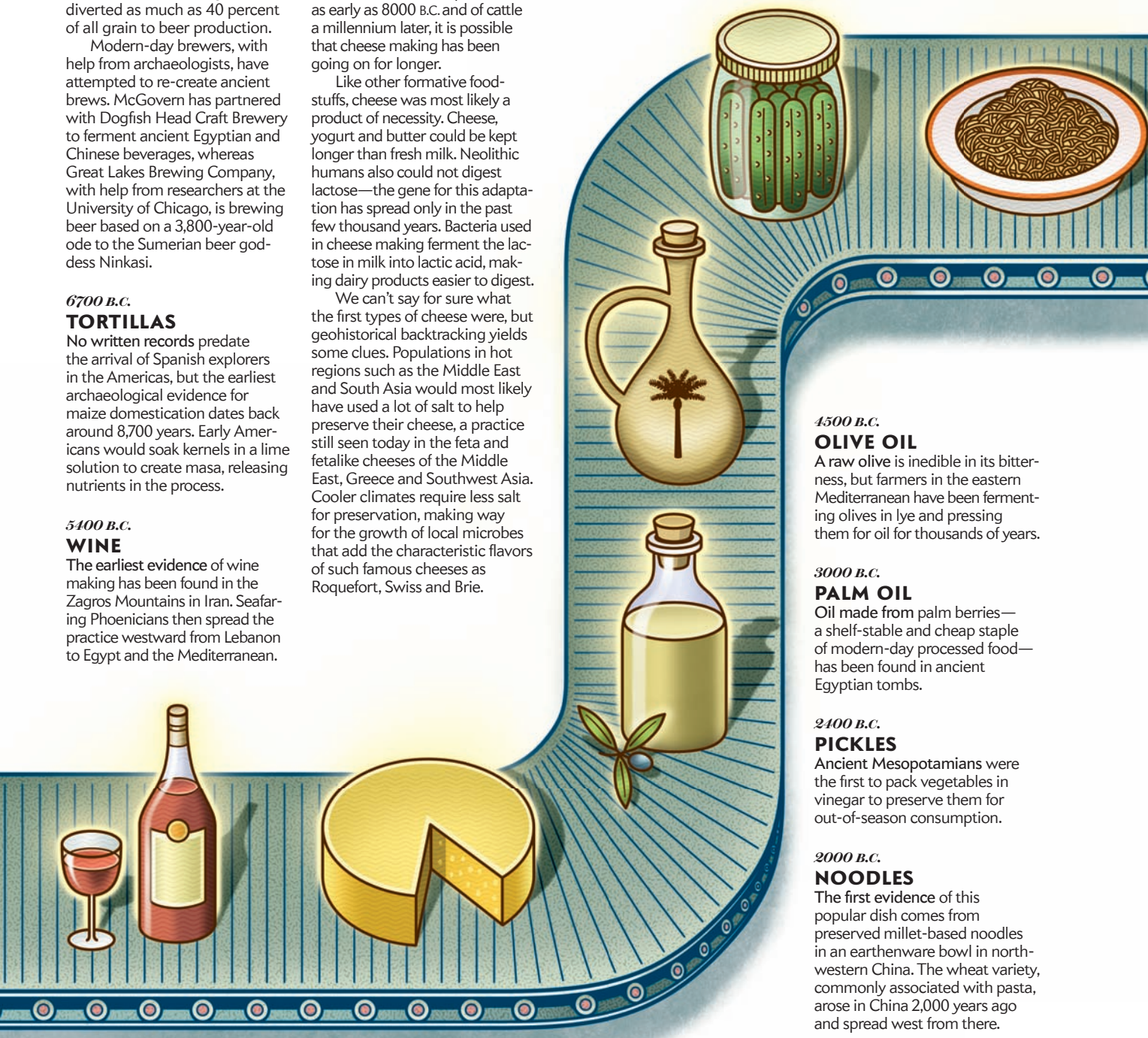
CHEESE

Take milk, place it in a ruminant's stomach, then churn. Scholars suggest this method is probably not too far off from how cheese was invented. The earliest evidence for cheese making comes from 7,000-year-old archaeological sites in Poland, where milk fat remains were found in holed ceramic containers that could have served as rudimentary strainers. Yet with the domestication of sheep and goats as early as 8000 B.C. and of cattle a millennium later, it is possible that cheese making has been going on for longer.

Like other formative foodstuffs, cheese was most likely a product of necessity. Cheese, yogurt and butter could be kept longer than fresh milk. Neolithic humans also could not digest lactose—the gene for this adaptation has spread only in the past few thousand years. Bacteria used in cheese making ferment the lactose in milk into lactic acid, making dairy products easier to digest.

We can't say for sure what the first types of cheese were, but geohistorical backtracking yields some clues. Populations in hot regions such as the Middle East and South Asia would most likely have used a lot of salt to help preserve their cheese, a practice still seen today in the feta and fetalike cheeses of the Middle East, Greece and Southwest Asia. Cooler climates require less salt for preservation, making way for the growth of local microbes that add the characteristic flavors of such famous cheeses as Roquefort, Swiss and Brie.

Evelyn Kim is a writer and educator living in Copenhagen. Her work examines the intersection of the history of science, food and the environment.



4500 B.C.

OLIVE OIL

A raw olive is inedible in its bitterness, but farmers in the eastern Mediterranean have been fermenting olives in lye and pressing them for oil for thousands of years.

3000 B.C.

PALM OIL

Oil made from palm berries—a shelf-stable and cheap staple of modern-day processed food—has been found in ancient Egyptian tombs.

2400 B.C.

PICKLES

Ancient Mesopotamians were the first to pack vegetables in vinegar to preserve them for out-of-season consumption.

2000 B.C.

NOODLES

The first evidence of this popular dish comes from preserved millet-based noodles in an earthenware bowl in northwestern China. The wheat variety, commonly associated with pasta, arose in China 2,000 years ago and spread west from there.

1900 B.C.

CHOCOLATE

Pre-Olmec civilizations in Central America ground the beans of cacao pods, mixed the powder with water and shook the mixture, producing a foamy drink. More than 3,400 years later Hernando Cortés brought the beans to Spain, where sugar was added for the first time.

1500 B.C.

BACON

Chinese cooks were the first to salt pork bellies not only as an early form of preservation but also as a way to bring out the flavor of the meat.

1000 B.C.

JIANG

Jiang was the precursor of flavorings such as miso and soy sauce that are used across East Asia today. According to the ancient Chinese text *Zhouli* (*Rites of Zhou*), *jiang* was made by mixing meat or fish with salt and *liang qu* (a fermentation starter) and leaving the mixture to mature for 100 days. Like many other fermented foods, its discovery was probably accidental, but *jiang*'s dissemination across East Asia was anything but. The rise of Buddhism across Asia in the first to seventh centuries A.D. most likely brought *jiang* to both Korea and Japan.

500 B.C.

SUGAR

According to Sanskrit texts, cooks in India processed sugarcane into giant crystals through boiling and cooling extracted sugarcane juice. Nearly a millennium later Indians invented easy-to-transport granulated sugar, which launched the global sugar trade.

A.D. 400

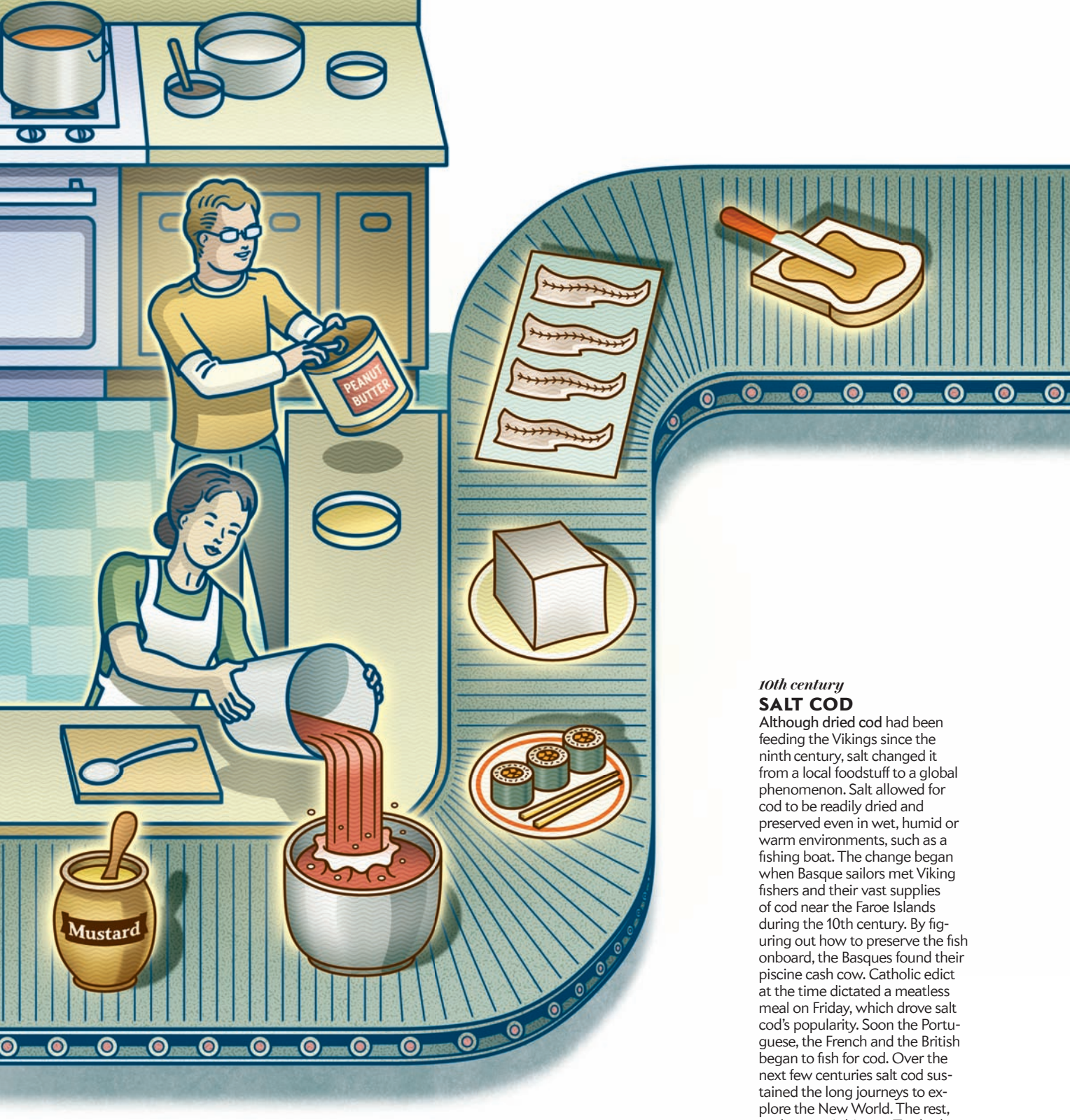
MUSTARD

One of the first mustard recipes, collected in the Roman cookbook *De Re Coquinaria,* called for a mixture of ground mustard seed, pepper, caraway, lovage, roasted coriander seeds, dill, celery, thyme, oregano, onion, honey, vinegar, fish sauce and oil.

A.D. 700

KIMCHI

The first kimchi was pretty tame: cabbage fermented with salt. Once the Japanese invaded Korea in the 16th century, taking with them red chilies that Portuguese missionaries had brought to Japan from the New World, Koreans started incorporating fiery elements into the dish.



A.D. 700

SUSHI

Sushi started as a means of fish preservation in Southeast Asia, where salted fish was covered in boiled rice and left to ferment for months. The rotting rice was then scraped off and discarded (because of the waste, sushi has always been a dish for the wealthy) and the soured fish consumed. The process is much like dry-aging beef today—you lose some of the product to rot, but the remainder is more tender and flavorful. By the time of 19th-century Japan, the process of long fermentation was eliminated and the tangy taste replaced by the introduction of vinegar into the rice mixture.

A.D. 965

TOFU

Tofu's origins are mysterious, but the first written record appears in the stories of Chinese writer Tao Ku. He writes of a vice mayor who was so poor, he was forced to buy tofu—a coagulated gel made from cooked soybeans—instead of mutton.

10th century

SALT COD

Although dried cod had been feeding the Vikings since the ninth century, salt changed it from a local foodstuff to a global phenomenon. Salt allowed for cod to be readily dried and preserved even in wet, humid or warm environments, such as a fishing boat. The change began when Basque sailors met Viking fishers and their vast supplies of cod near the Faroe Islands during the 10th century. By figuring out how to preserve the fish onboard, the Basques found their piscine cash cow. Catholic edict at the time dictated a meatless meal on Friday, which drove salt cod's popularity. Soon the Portuguese, the French and the British began to fish for cod. Over the next few centuries salt cod sustained the long journeys to explore the New World. The rest, as they say, is history. Too bad the fish that brought them there is almost history, too.

15th century

PEANUT BUTTER

Contrary to what your second grade teacher may have told you, George Washington Carver did not invent peanut butter. The Aztecs were making a paste of ground raw peanuts in the 15th century.

Mid-15th century

COFFEE

Coffee is a Western obsession, but its roots lie in the Arab world. The most credible claim to the origin of coffee comes from Yemeni Sufi monasteries in the mid-15th century. The monks wrote of a coffee trade between Yemen and Ethiopia, where the beans originated. (It is unclear exactly what was going on in Ethiopia at the time because no records survive.) Yemen eventually cultivated its own native crop of coffee from Ethiopian stock, and from there it spread to Egypt, Damascus and Mecca. By the 16th century coffeehouses, or *kaveh kanes,* had spread across the Arabian Peninsula.

Coffee was first administered for stomachaches, torpor, narcolepsy and other ailments. Yet coffee was not merely curative; several Arabic writers noted its powers of sociability. Perhaps too much so: the culture of coffee and coffeehouses, with their gossip and game playing, prompted the governor of Mecca to declare a ban on the drink in 1511. After a 13-year caffeine headache, the Turkish Sultan Selim I overturned the ban.

For European travelers and explorers of the 16th century, coffee was another curiosity of the Orient. In one of the earliest allusions to coffee by a European, in 1582 German physician and botanist Leonhard Rauwolf described a "good drink which [Turks and Arabs] greatly esteem.... It is nearly as black as ink and helpful against stomach complaints." In a move of early modern marketing, Venetian merchants started importing coffee from the Middle East in the late 16th century as a luxury drink. By the mid-17th century the French, the British and the Dutch all had the buzz.

1767

CARBONATED WATER

Joseph Priestly, the British natural philosopher who discovered oxygen, invented carbonated water after placing a bowl of water above a brewery in Leeds, England.

1894

CORN FLAKES

To satisfy the vegetarian diets advocated by Seventh-Day Adventists, John Harvey Kellogg and his younger brother, Will Keith Kellogg, developed corn flakes in 1894 as part of a diet regimen at his Battle Creek, Mich., sanitarium.

1908

MSG

German agricultural chemist Karl Ritthausen originally discovered glutamic acid, of which monosodium glutamate (MSG) is just one variation, in 1866. Much like his contemporaries in Germany, Ritthausen was part of a growing field, started by a founder of organic chemistry (and inventor of nitrogen-based fertilizers), Justus von Liebig, to look at the chemical basis for naturally occurring substances.

About 40 years later a Japanese chemist, Kikunae Ikeda, who trained as an organic chemist in Germany, tried to replicate the success of his German colleagues, especially that of von Liebig, who became wealthy from creating dehydrated beef stock. Ikeda, like Liebig, wanted to find a way to do the same for Japanese cuisine—that is, create a means of chemically reproducing the flavor of kombu dashi, a staple seaweed-based stock. In 1908, after evaporating a large quantity of dashi broth, he found a residue, tasted it and realized it was the essence of Japanese flavor. Publishing his results in the *Journal of the Chemical Society of Tokyo* in 1909, Ikeda declared that his study had found that seaweed contains glutamates and that glutamates create the familiar yet theretofore undesignated taste umami.

1926

SPAM

The first iteration of what became Spam was called "Hormel spiced ham," and it was just cured pork shoulder in a can. Competitors quickly launched their own versions. To differentiate his product, Jay Hormel changed the recipe in 1937, grinding up the pork, adding salt and spices, and encasing the meat in an aspic gelatin. Most important, Hormel rebranded the product with the catchy name "Spam"—short for "shoulder of pork and ham"—before World War II broke out. The U.S. Army, deciding that Spam was the perfect tent food, bought 150 million pounds of it over the course of the war to feed Allied troops all over the world. In the postwar years, wherever U.S. troops went, cans of Spam followed.

During the Korean War, it became unofficial currency; surplus cans flooded the black market and were used to pay for doctors' visits and military intelligence. To this day, Spam remains a popular product across Korea and the rest of Asia, with Spam added to traditional foods such as *kimbap* and *chanpuru*.

1950s

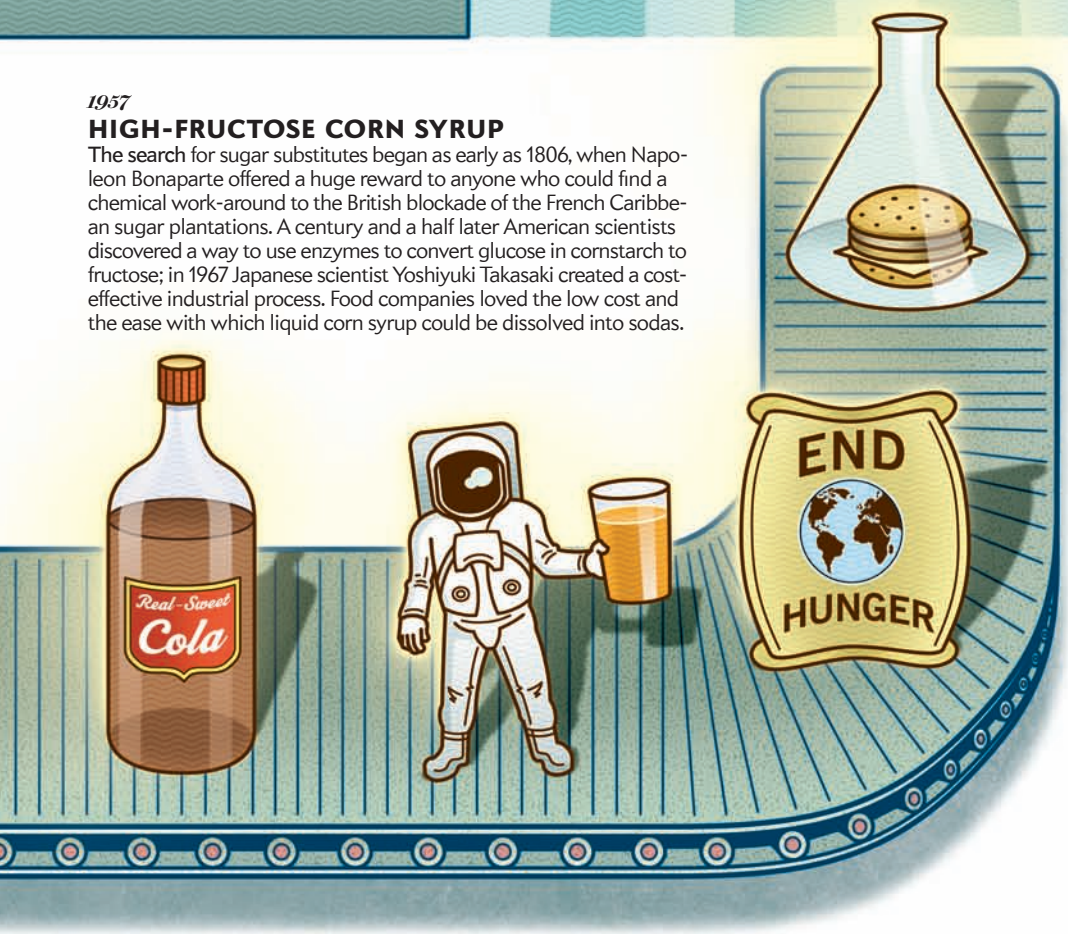
CHICKEN NUGGETS

Robert C. Baker, a food scientist at Cornell University, ground up chicken parts and coated them with breading as a way to increase demand for chickens in upstate New York.

1957

HIGH-FRUCTOSE CORN SYRUP

The search for sugar substitutes began as early as 1806, when Napoleon Bonaparte offered a huge reward to anyone who could find a chemical work-around to the British blockade of the French Caribbean sugar plantations. A century and a half later American scientists discovered a way to use enzymes to convert glucose in cornstarch to fructose; in 1967 Japanese scientist Yoshiyuki Takasaki created a cost-effective industrial process. Food companies loved the low cost and the ease with which liquid corn syrup could be dissolved into sodas.



1959

TANG

Scientists at General Foods worked for years to create a powdered orange juice substitute, but their concoctions had unpleasantly bitter tastes. They succeeded by abandoning their ambitions to include all of O.J.'s vitamins and minerals.

1996

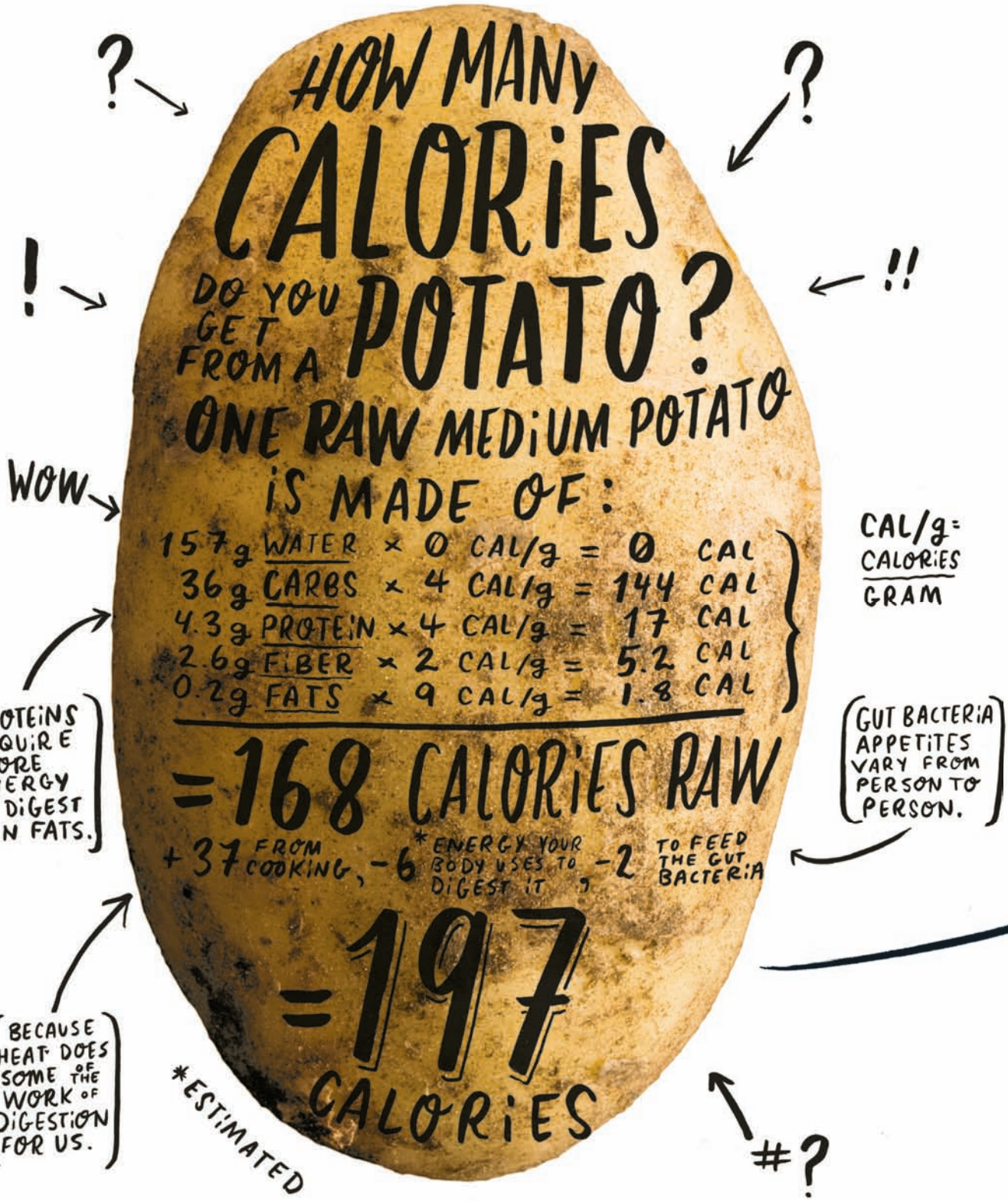
PLUMPY'NUT

This nutrient-dense, vitamin-fortified food product made from peanuts, vegetable oil, powdered milk and sugar was designed to help severely malnourished children gain weight.

2013

LAB-GROWN MEAT

The first public taste test of in vitro meat is scheduled to feature a burger grown from bovine stem cells.


HOW MANY CALORIES DO YOU GET FROM A POTATO?
ONE RAW MEDIUM POTATO IS MADE OF:
157g WATER × 0 CAL/g = 0 CAL
36g CARBS × 4 CAL/g = 144 CAL
4.3g PROTEIN × 4 CAL/g = 17 CAL
2.6g FIBER × 2 CAL/g = 5.2 CAL
0.2g FATS × 9 CAL/g = 1.8 CAL
=168 CALORIES RAW
+37 FROM COOKING, -6 *ENERGY YOUR BODY USES TO DIGEST IT, -2 TO FEED THE GUT BACTERIA
=197 CALORIES
*ESTIMATED
?
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WOW
#?
CAL/g = CALORIES / GRAM
PROTEINS REQUIRE MORE ENERGY TO DIGEST THAN FATS.
GUT BACTERIA APPETITES VARY FROM PERSON TO PERSON.
BECAUSE HEAT DOES SOME OF THE WORK OF DIGESTION FOR US.

NUTRITION

everything you know about calories is wrong

Digestion is far too messy a process to accurately convey in neat numbers. The counts on food labels can differ wildly from the calories you actually extract, for many reasons

By Rob Dunn

AT ONE PARTICULARLY STRANGE MOMENT IN MY career, I found myself picking through giant conical piles of dung produced by emus—those goofy Australian kin to the ostrich. I was trying to figure out how often seeds pass all the way through the emu digestive system intact enough to germinate. My colleagues and I planted thousands of collected seeds and waited. Eventually, little jungles grew.

Clearly, the plants that emus eat have evolved seeds that can survive digestion relatively unscathed. Whereas the birds want to get as many calories from fruits as possible—including from the seeds—the plants are invested in protecting their progeny. Although it did not occur to me at the time, I later realized that humans, too, engage in a kind of tug-of-war with the food we eat, a battle in which we are measuring the spoils—calories—all wrong.

Food is energy for the body. Digestive enzymes in the mouth, stomach and

JUSTIN LIGHTLEY *Getty Images (potato)*

Illustration by Joel Holland

Rob Dunn is a biologist at North Carolina State University and a writer whose articles have appeared in *Natural History*, *Smithsonian* and *National Geographic*, among other publications. His most recent book is *The Wild Life of Our Bodies* (Harper, 2011).

intestines break up complex food molecules into simpler structures, such as sugars and amino acids that travel through the bloodstream to all our tissues. Our cells use the energy stored in the chemical bonds of these simpler molecules to carry on business as usual. We calculate the available energy in all foods with a unit known as the food calorie, or kilocalorie—the amount of energy required to heat one kilogram of water by one degree Celsius. Fats provide approximately nine calories per gram, whereas carbohydrates and proteins deliver just four. Fiber offers a piddling two calories because enzymes in the human digestive tract have great difficulty chopping it up into smaller molecules.

Every calorie count on every food label you have ever seen is based on these estimates or on modest derivations thereof. Yet these approximations assume that the 19th-century laboratory experiments on which they are based accurately reflect how much energy different people with different bodies derive from many different kinds of food. New research has revealed that this assumption is, at best, far too simplistic. To accurately calculate the total calories that someone gets out of a given food, you would have to take into account a dizzying array of factors, including whether that food has evolved to survive digestion; how boiling, baking, microwaving or flambéing a food changes its structure and chemistry; how much energy the body expends to break down different kinds of food; and the extent to which the billions of bacteria in the gut aid human digestion and, conversely, steal some calories for themselves.

Nutrition scientists are beginning to learn enough to hypothetically improve calorie labels, but digestion turns out to be such a fantastically complex and messy affair that we will probably never derive a formula for an infallible calorie count.

A HARD NUT TO CRACK

THE FLAWS in modern calorie counts originated in the 19th century, when American chemist Wilbur Olin Atwater developed a system, still used today, for calculating the average number of calories in one gram of fat, protein and carbohydrate. Atwater was doing his best, but no food is average. Every food is digested in its own way.

Consider how vegetables vary in their digestibility. We eat the stems, leaves and roots of hundreds of different plants. The walls of plant cells in the stems and leaves of some species are much tougher than those in other species. Even within a single plant, the durability of cell walls can differ. Older leaves tend to have sturdier cell walls than young ones. Generally speaking, the weaker or more degraded the cell walls in the plant material we eat, the more calories we get from it. Cooking easily ruptures cells in, say, spinach and zucchini, but cassava (*Manihot esculenta*) or Chinese water chestnut (*Eleocharis dulcis*) is much more resistant. When cell walls hold strong, foods hoard their precious calories and pass through our body intact (think corn).

Some plant parts have evolved adaptations either to make themselves more appetizing to animals or to evade digestion altogether. Fruits and nuts first evolved in the Cretaceous (between 145 and 65 million years ago), not long after mammals were beginning to run between the legs of dinosaurs. Evolution favored fruits that were both tasty and easy to digest to better attract animals that could help plants scatter seeds. It also favored nuts and seeds that were hard to digest, however. After all, seeds and nuts need to survive the guts of birds, bats, rodents and monkeys to spread the genes they contain.

Studies suggest that peanuts, pistachios and almonds are less completely digested than other foods with similar levels of proteins, carbohydrates and fats, meaning they relinquish fewer calories than one would expect. A new study by Janet A. Novotny and her colleagues at the U.S. Department of Agriculture found that when people eat almonds, they receive just 129 calories per serving rather than the 170 calories reported on the label. They reached this conclusion by asking people to follow the same exact diets—except for the amount of almonds they ate—and measuring the unused calories in their feces and urine.

Even foods that have not evolved to survive digestion differ markedly in their digestibility. Proteins may require as much as five times more energy to digest as fats because our enzymes must unravel the tightly wound strings of amino acids from which proteins are built. Yet food labels do not account for this expenditure. Some foods such as honey are so readily used that our digestive system is hardly put to use. They break down in our stomach and slip quickly across the walls of our intestines into the bloodstream: game over.

Finally, some foods prompt the immune system to identify and deal with any hitchhiking pathogens. No one has seriously evaluated just how many calories this process involves, but it is probably quite a few. A somewhat raw piece of meat can harbor lots of potentially dangerous microbes. Even if our immune system does not attack any of the pathogens in our food, it still uses up energy to take the first step of distinguishing friend from foe. This is not to mention the potentially enormous calorie loss if a pathogen in uncooked meat leads to diarrhea.

WHAT'S COOKING?

PERHAPS THE BIGGEST PROBLEM with modern calorie labels is that they fail to account for an everyday activity that dramatically alters how much energy we get from food: the way we simmer, sizzle, sauté and otherwise process what we eat. When studying

IN BRIEF

Almost every packaged food today features calorie counts in its label. Most of these counts are inaccurate because they are based on a system of averages that ignores the complexity of digestion.

Recent research reveals that how many calories we extract from food depends on which species we eat, how we prepare our food, which bacteria are in our gut and how much energy we use to digest different foods.

Current calorie counts do not consider any of these factors. Digestion is so intricate that even if we try to improve calorie counts, we will likely never make them perfectly accurate.

the feeding behavior of wild chimpanzees, biologist Richard Wrangham, now at Harvard University, tried eating what the chimps ate. He went hungry and finally gave in to eating human foods. He has come to believe that learning to process food—cooking it with fire and pounding it with stones—was a milestone of human evolution. Emus do not process food; neither, to any real extent, do any of the apes. Yet every human culture in the world has technology for modifying its food. We grind, we heat, we ferment. When humans learned to cook food—particularly, meat—they would have dramatically increased the number of calories they extracted from that food. Wrangham proposes that getting more energy from food allowed humans to develop and nourish exceptionally large brains relative to body size. But no one had precisely investigated, in a controlled experiment, how processing food changes the energy it provides—until now.

Rachel N. Carmody, a former graduate student in Wrangham's lab, and her collaborators fed adult male mice either sweet potatoes or lean beef. She served these foods raw and whole, raw and pounded, cooked and whole, or cooked and pounded and allowed the mice to eat as much as they wanted for four days. Mice lost around four grams of weight on raw sweet potatoes but gained weight on cooked potatoes, pounded and whole. Similarly, the mice retained one gram more of body mass when consuming cooked meat rather than raw meat. This reaction makes biological sense. Heat hastens the unraveling, and thus the digestibility, of proteins, as well as killing bacteria, presumably reducing the energy the immune system must expend to battle any pathogens.

Carmody's findings also apply to industrial processing. In a 2010 study people who ate 600- or 800-calorie portions of whole-wheat bread with sunflower seeds, kernels of grain and cheddar cheese expended twice as much energy to digest that food as did individuals who consumed the same quantity of white bread and "processed cheese product." Consequently, people snacking on whole wheat obtained 10 percent fewer calories.

Even if two people eat the same sweet potato or piece of meat cooked the same way, they will not get the same number of calories out of it. Carmody and her colleagues studied inbred mice with highly similar genetics. Yet the mice still varied in terms of how much they grew or shrank on a given diet. People differ in nearly all traits, including inconspicuous features, such as the size of the gut. Measuring people's colons has not been popular for years, but when it was the craze among European scientists in the early 1900s, studies discovered that certain Russian populations had large intestines that were about 57 centimeters longer on average than those of certain Polish populations. Because the final stages of nutrient absorption occur in the large intestine, a Russian eating the same amount of food as a Pole is likely to get more calories from it. People also vary in the particular enzymes they produce. By some measures, most adults do not produce the enzyme lactase, which is necessary to break down lactose sugars in milk. As a result, one man's high-calorie latte is another's low-calorie case of the runs.

People differ immensely as well in what scientists have come to regard as an extra organ of the human body—the community of bacteria living in the intestines. In humans, two phyla of bacteria, Bacteroidetes and Firmicutes, dominate the gut. Researchers have found that obese people have more Firmicutes in their intestines and have proposed that some people are obese, in part, because the extra bacteria make them more efficient at metabolizing food: so instead of being lost as waste, more nutrients make their way into the circulation and, if they go unused, get stored as fat. Other microbes turn up only in specific peoples. Some Japanese individuals, for example, have a microbe in their intestines that is particularly good at breaking down seaweed. It turns out this intestinal bacterium stole the seaweed-digesting genes from a marine bacterium that lingered on raw seaweed salads.

Because many modern diets contain so many easily digestible processed foods, they may be reducing the populations of gut microbes that evolved to digest the more fibrous matter our own enzymes cannot. If we continue to make our gut a less friendly environment for such bacteria, we may get fewer calories from tough foods such as celery.

Few people have attempted to improve calorie counts on food labels based on our current understanding of human digestion. We could tweak the Atwater system to account for the special digestive challenges posed by nuts. We could even do so nut by nut or, more generally, food by food. Such changes (which have unsurprisingly been supported by the Almond Board of California, an advocacy group) would, however, require scientists to study each and every food the same way that Novotny and her colleagues investigated almonds, one bag of feces and jar of urine at a time. Judging by the FDA's regulations, the agency would be unlikely to prevent food sellers from adjusting calorie counts based on such new studies. The bigger challenge is modifying labels based on how items are processed; no one seems to have launched any efforts to make this larger change.

Even if we entirely revamped calorie counts, however, they would never be precisely accurate because the amount of calories we extract from food depends on such a complex interaction between food and the human body and its many microbes. In the end, we all want to know how to make the smartest choices at the supermarket. Merely counting calories based on food labels is an overly simplistic approach to eating a healthy diet—one that does not necessarily improve our health, even if it helps us lose weight. Instead we should think more carefully about the energy we get from our food in the context of human biology. Processed foods are so easily digested in the stomach and intestines that they give us a lot of energy for very little work. In contrast, veggies, nuts and whole grains make us sweat for our calories, generally offer far more vitamins and nutrients than processed items, and keep our gut bacteria happy. So it would be logical for people who want to eat healthier and cut calories to favor whole and raw foods over highly processed foods. You might call it the way of the emu. SA

MORE TO EXPLORE

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SCIENTIFIC AMERICAN ONLINE

To watch an Instand Egghead video about why traditional calorie counts are inaccurate, visit ScientificAmerican.com/sep2013/calories

PHYSIOLOGY

which one will make

Rigorously controlled studies may soon give us a definitive answer about

Photographs by Travis Rathbone

you fat?

what causes obesity—excessive calories or the wrong carbohydrates

By Gary Taubes

Gary Taubes is co-founder of the Nutrition Science Initiative and author of *Why We Get Fat: And What to Do about It* (Knopf, 2011).

WHY DO SO MANY OF US GET SO FAT? THE ANSWER appears obvious. "The fundamental cause of obesity and overweight," the World Health Organization says, "is an energy imbalance between calories consumed and calories expended." Put simply, we either eat too much or are too sedentary, or both. By this logic, any excess of calories—whether from protein, carbohydrate or fat (the three main components, or "macronutrients," in food)—will inevitably pack on the pounds. So the solution is also obvious: eat less, exercise more.

The reason to question this conventional thinking is equally self-evident. The eat less/move more prescription has been widely disseminated for 40 years, and yet the prevalence of obesity, or the accumulation of unhealthy amounts of body fat, has climbed to unprecedented levels. Today more than a third of Americans are considered obese—more than twice the proportion of 40 years ago. Worldwide, more than half a billion people are now obese.

Besides getting fatter, we are also developing more metabolic disorders, such as type 2 diabetes, which is marked by hormonal abnormalities in the processing and storage of nutrients and is far more common in obese individuals than in lean ones.

The dissonance of an ever worsening problem despite a seemingly well-accepted solution suggests two possibilities. One, our understanding of why people get fat is correct, but those who are obese—for genetic, environmental or behavioral reasons—are unable or unwilling to heal themselves. Two, our understanding is wrong and hence so is the ubiquitous advice about how to make things better.

If the second option is true, then maybe what makes us fat is not an energy imbalance but something more akin to a hormonal defect, an idea embraced by European researchers prior to World War II. If so, the prime suspect or environmental trigger of this defect would be the quantity and quality of the carbohydrates we consume. Under this scenario, one fundamental error we have made in our thinking about obesity is to assume that the energy content of foods—whether avocado, steak, bread or soda—is what makes them fattening, not the effects that these foods, carbohydrates in particular, have on the hormones that regulate fat accumulation.

Given how often researchers refer to obesity as a disorder of the energy balance, one might assume that the concept had been rigorously tested decades ago. But a proper scientific vetting never actually happened. The experiments were too difficult, if not too expensive, to do correctly. And investigators typically thought the answer was obvious—we eat too much—and so the experiments were not worth the effort. As a result, the scientific under-

IN BRIEF

Which is the more important cause of obesity: Eating too much food or eating the wrong kinds of food, especially easily digested carbohydrates?

Although nutrition researchers think they know the answer, investigators have never actually put the question to a rigorous, scientific test—until now.

Researchers sponsored by the Nutrition Science Initiative will soon address the question by precisely controlling food consumption by volunteers living in a test facility and then rigorously measuring energy expenditure and how it changes with differences in diet composition.

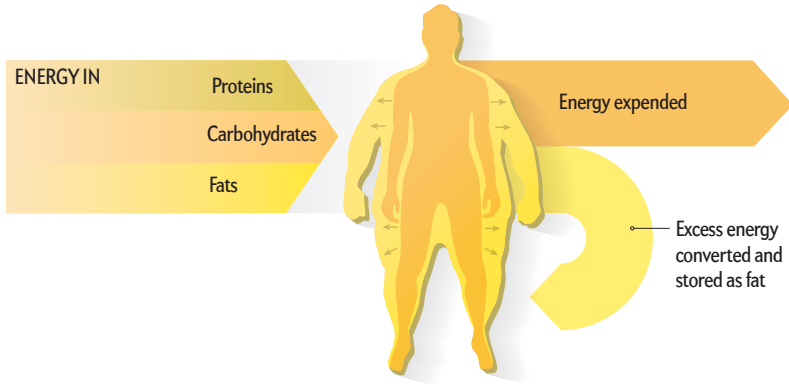
PRECEDING PAGES: FOOD STYLING BY LIZA JERNOW; SOURCE: KIRSTEN LARA GETCHELL

Calories vs. Carbohydrates

In the next couple of years, investigators funded by NuSI plan to test two competing hypotheses about the dietary causes of obesity under scientifically rigorous conditions that are designed to force one of the possibilities to emerge as the clear winner.

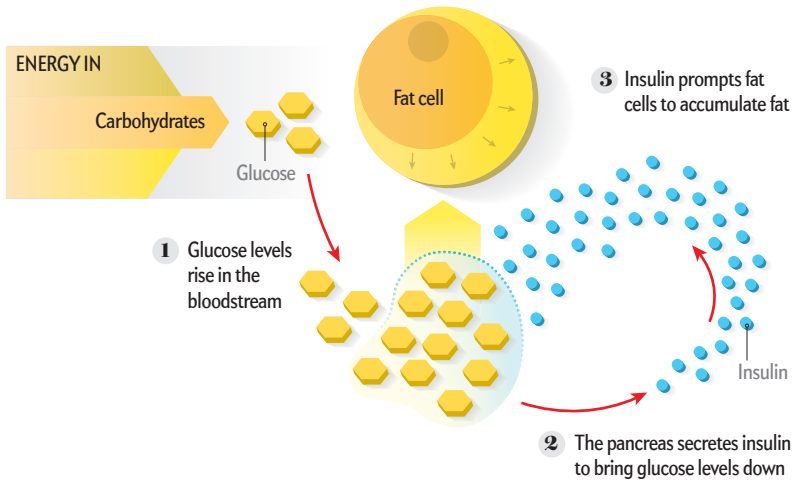
Energy Imbalance

The conventional explanation focuses on how the body regulates the intake and expenditure of energy (measured in calories). Consuming too much of anything—whether fats, carbohydrates or proteins—increases body fat. The only way to lose weight is to eat fewer calories or to expend more calories.



Hormone Imbalance

The alternative hypothesis focuses on the complex physiological regulation of fat cells. Consuming carbohydrates raises levels of sugar (glucose) in the blood, which in turn activates the release of the hormone insulin. Fat cells respond to insulin by holding on to their fat stores and even adding to them. Weight gain occurs when insulin levels—triggered by eating carbohydrates—remain elevated for long periods.



pinning of the most critical health issue of our era—the burgeoning rates of obesity and diabetes and their complications—remains very much an open question.

After a decade of studying the science and its history, I am convinced that meaningful progress against obesity will come only if we rethink and rigorously test our understanding of its cause. Last year, with Peter Attia, a former surgeon and cancer researcher, I co-founded a nonprofit organization, the Nutrition Science Initiative (NuSI), to address this lack of definitive evidence. With support from the Laura and John Arnold Foundation in Houston, Tex., we have recruited independent scientists to design and carry out the experiments that will meticulously test the competing hypotheses of obesity (and by extension, weight gain). The Arnold Foundation has committed to fund up to 60 percent of NuSI's current research budget and three years of operating expenses for a total of $40 million. The investigators will follow the evidence wherever it leads. If all works out as planned, we could have unambiguous evidence about the biological cause of obesity in the next half a dozen years.

THE HORMONE HYPOTHESIS

TO UNDERSTAND WHAT MAKES THE hormone hypothesis of obesity so intriguing, it helps to grasp where the energy-balance hypothesis falls short. The idea that obesity is caused by consuming more calories than we expend supposedly stems from the first law of thermodynamics, which merely states that energy can neither be created nor destroyed. As applied to biology, it means that energy consumed by an organism has to be either converted to a useful form (metabolized), excreted or stored. Thus, if we take in more calories than we expend or excrete, the excess has to be stored, which means that we get fatter and heavier. So far, so obvious. But this law tells us nothing about why we take in more calories than we expend, nor does it tell us why the excess gets stored as fat. And it is these "why" questions that need to be answered.

Specifically, why do fat cells accumulate fat molecules to excess? This is a biological question, not a physics one. Why are those fat molecules not metabolized instead to generate energy or heat? And why do fat cells take up excessive fat in some

Illustration by Jen Christiansen

areas of the body but not others? Saying that they do so because excess calories are consumed is not a meaningful answer.

Answering these questions leads to consideration of the role that hormones—insulin, in particular—play in stimulating fat accumulation in different cells. Insulin is secreted in response to a type of carbohydrate called glucose. When the amount of glucose rises in the blood—as happens after eating a carbohydrate-rich meal—the pancreas secretes more insulin, which works to keep the blood glucose level from getting dangerously high. Insulin tells muscle, organ and even fat cells to take up the glucose and use it for fuel. It also tells fat cells to store fat—including fat from the meal—for later use. As long as insulin levels remain high, fat cells retain fat, and the other cells preferentially burn glucose (and not fat) for energy.

The main dietary sources of glucose are starches, grains and sugars. (In the absence of carbohydrates, the liver will synthesize glucose from protein.) The more easily digestible the carbohydrates, the greater and quicker the rise in blood glucose. (Fiber and fat in foods slow the process.) Thus, a diet rich in refined grains and starches will prompt greater insulin secretion than a diet that is not. Sugars—such as sucrose and high-fructose corn syrup—may play a key role because they also contain significant amounts of a carbohydrate called fructose, which is metabolized mostly by liver cells. Though not definitive, research suggests that high amounts of fructose may be an important cause of "insulin resistance." When cells are insulin-resistant, more insulin is required to control blood glucose. The result, according to the hormone hypothesis, is an ever greater proportion of the day that insulin in the blood is elevated, causing fat to accumulate in fat cells rather than being used to fuel the body. As little as 10 or 20 calories stored as excess fat each day can lead over decades to obesity.

The hormone hypothesis suggests that the *only* way to prevent this downward spiral from happening, and to reverse it when it does, is to avoid the sugars and carbohydrates that work to raise insulin levels. Then the body will naturally tap its store of fat to burn for fuel. The switch from carbohydrate burning to fat burning, so the logic goes, might occur even if the total number of calories consumed remains unchanged. Cells burn the fat because hormones are effectively telling them to do so; the body's energy expenditure increases as a result. To lose excess body fat, according to this view, carbohydrates must be restricted and replaced, ideally with fat, which does not stimulate insulin secretion.

This alternative hypothesis of obesity implies that the ongoing worldwide epidemics of obesity and type 2 diabetes (which stems to great extent from insulin resistance) are largely driven by the grains and sugars in our diets. It also implies that the first step in solving these crises is to avoid sugars and limit consumption of starchy vegetables and grains, not worrying about how much we are eating and exercising.

FORGOTTEN HISTORY

CONVENTIONAL WISDOM did not always favor the energy-imbalance hypothesis that prevails today. Until World War II, the leading authorities on obesity (and most medical disciplines) worked in Europe and had concluded that obesity was, like any other growth disorder, caused by a hormonal and regulatory defect. Something was amiss, they believed, with the hormones and enzymes that influence the storage of fat in fat cells.

Gustav von Bergmann, a German internist, developed the original hypothesis more than a century ago. (Today the highest honor bestowed by the German Society of Internal Medicine is the Gustav von Bergmann Medal.) Bergmann evoked the term "lipophilia"—love of fat—to describe the affinity of different body tissues for amassing fat. Just as we grow hair in some places and not others, we store fat in some places and not oth-

Land of the Supersized

More than **72 MILLION** adults in the U.S. are obese

Colorado has the fewest proportion of obese adults in the U.S. **20.7%**

Annual medical costs average **$1,429** higher for obese persons than those of normal weight

Mississippi has the highest proportion **34.9%**

ers, and this "lipophilic tendency," he assumed, must be regulated by physiological factors.

The lipophilia concept vanished after World War II with the replacement of German with English as the scientific lingua franca. Meanwhile the technologies needed to understand the regulation of fat accumulation in fat cells and thus the biological basis of obesity—specifically, techniques to accurately measure fatty acids and hormone levels in the blood—were not invented until the late 1950s.

By the mid-1960s it was clear that insulin was the primary hormone regulating fat accumulation, but by then obesity was effectively considered an eating disorder to be treated by inducing or coercing obese subjects to eat fewer calories. Once studies linked the amount of cholesterol in the blood to the risk of heart disease and nutritionists targeted saturated fat as the primary dietary evil, authorities began recommending low-fat, *high*-carbohydrate diets. The idea that carbohydrates could cause obesity (or diabetes or heart disease) was swept aside.

Still, a few working physicians embraced the carbohydrate/insulin hypothesis and wrote diet books claiming that fat people could lose weight eating as much as they wanted, so long as they avoided carbohydrates. Because the most influential experts believed that people got fat to begin with *precisely* because they ate as much as they wanted, these diet books were perceived as con jobs. The most famous of these authors, Robert C. Atkins, did not help the cause by contending that saturated fat could be eaten to the heart's delight—lobster Newburg, double cheeseburgers—so long as carbohydrates were avoided—a suggestion that many considered tantamount to medical malpractice.

RIGOROUS EXPERIMENTS

IN THE PAST 20 YEARS significant evidence has accumulated to suggest that these diet doctors may have been right, that the hormone hypothesis is a viable explanation for why we get fat and that insulin resistance, driven perhaps by the sugars in the diet, is a fundamental defect not just in type 2 diabetes but in heart disease and even cancer. This makes rigorous testing of the roles of carbohydrates and insulin critically important. Because the ultimate goal is to identify the environmental triggers of obesity, experiments should, ideally, be directed at elucidating the processes that lead to the accumulation of excess fat. But obesity can take decades to develop, so any month-to-month fat gains may be too small to detect. Thus, the first step that NuSI-funded researchers will take is to test the competing hypotheses on weight loss, which can happen relatively quickly. These first results will then help determine what future experiments are needed to further clarify the mechanisms at work and which of these hypotheses is correct.

A key initial experiment will be carried out jointly by researchers at Columbia University, the National Institutes of Health, the Florida Hospital–Sanford-Burnham Translational Research Institute in Orlando, and the Pennington Biomedical Research Center in Baton Rouge, La. In this pilot study, 16 overweight and obese participants will be housed throughout the experiment in research facilities to ensure accurate assessments of calorie consumption and energy expenditure. In stage one, the participants will be fed a diet similar to that of the average American—50 percent carbohydrates (15 percent sugar), 35 percent fat and 15 percent protein. Researchers will carefully manipulate the calories consumed until it is clear the participants are neither gaining nor losing fat. In other words, the calories they take in will match the calories they expend, as measured in a device called a metabolic chamber. For stage two, the subjects will be fed a diet of precisely the same number of calories they had been consuming—distributed over the same number of meals and snacks—but the composition will change dramatically.

The total carbohydrate content of the new diet will be exceedingly low—on the order of 5 percent, which translates to only the carbohydrates that occur naturally in meat, fish, fowl, eggs, cheese, animal fat and vegetable oil, along with servings of green leafy vegetables. The protein content of this diet will match that of the diet the subjects ate initially—15 percent of calories. The remainder—80 percent of calories—will consist of fat from these real food sources. The idea is not to test whether this diet is healthy or sustainable for a lifetime but to use it to lower insulin levels by the greatest amount in the shortest time.

Meaningful scientific experiments ideally set up a situation in which competing hypotheses make different predictions about what will happen. In this case, if fat accumulation is primarily driven by an energy imbalance, these subjects should neither lose nor gain weight because they will be eating precisely as many calories as they are expending. Such a result would support the conventional wisdom—that a calorie is a calorie whether it comes from fat, carbohydrate or protein. If, on the other hand, the macronutrient composition affects fat accumulation, then these subjects should lose both weight and fat on the carbohydrate-restricted regime and their energy expenditure should increase, supporting the idea that a calorie of carbohydrate is more fattening than one from protein or fat, presumably because of the effect on insulin.

One drawback to this rigorous scientific approach is that it cannot be rushed without making unacceptable compromises. Even this pilot study will take the better part of a year. The more ambitious follow-up trials will probably take another three years. As we raise more funds, we hope to support more testing—including a closer look at the role that particular sugars and macronutrients have on other disorders, such as diabetes, cancer and neurological conditions. None of these experiments will be easy, but they are doable.

One ultimate goal is to assure the general public that whatever dietary advice it receives—for weight loss, overall health and prevention of obesity—is based on rigorous science, not preconceptions or blind consensus. Obesity and type 2 diabetes are not only serious burdens to afflicted individuals but are overwhelming our health care system and likely our economy as well. We desperately need the kind of unambiguous evidence that the NuSi experiments are designed to generate if we are going to combat and prevent these disorders. SA

MORE TO EXPLORE

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SCIENTIFIC AMERICAN ONLINE

For a video of Gary Taubes discussing some of his ideas with Stanford University nutrition expert Christopher Gardner, visit **ScientificAmerican.com/sep2013/taubes-video**

the first cookout

Nearly two million years ago our ancestors began to barbecue. And those hot meals, Richard Wrangham argues, are what made us human

Interview by Kate Wong

With our supersized brains and shrunken teeth and guts, we humans are bizarre primates. Richard Wrangham of Harvard University has long argued that these and other peculiar traits of our kind arose as humans turned to cooking to improve food quality—making it softer and easier to digest and thus a richer source of energy. Humans, unlike any other animal, cannot survive on raw food in the wild, he observes. "We need to have our food cooked."

Based on the anatomy of our fossil forebears, Wrangham thinks that Homo erectus *had mastered cooking with fire by 1.8 million years ago. Critics have countered that he lacks evidence to support the claim that cooking enhances digestibility and that the oldest known traces of fire are nowhere near as old as his hypothesis predicts. New findings, Wrangham says, lend support to his ideas.*

IN BRIEF

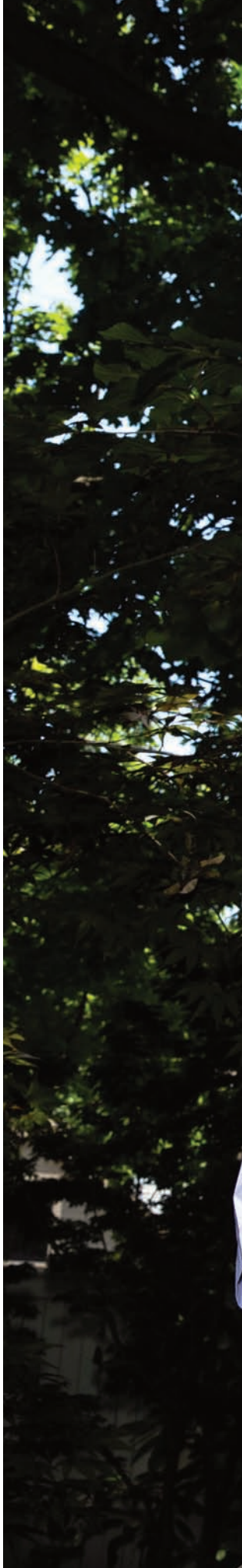
WHO
RICHARD WRANGHAM

VOCATION | AVOCATION
Anthropologist

WHERE
Harvard University

RESEARCH FOCUS
Chimpanzee behavior, ecology and physiology, which contribute to understanding human evolution

BIG PICTURE
Cooking made us human.



Photograph by Guido Vitti

COOKED FOOD provides more energy and requires less chewing than raw food does—benefits that may have fueled the evolution of key human traits, such as large brain size.

SCIENTIFIC AMERICAN: How did you come up with the cooking hypothesis?

WRANGHAM: I think of two strands. One is that I was trying to figure out what was responsible for the evolution of the human body form, and I was sensitive to the fact that humans everywhere use fire. I started thinking about how long ago you would have to go back before humans did not use fire. And that suggested to me the hypothesis that they always used it because they would not have survived without it. Humans as a genus [*Homo*] are committed to sleeping on the ground. I do not want to sleep on the ground in Africa without fire to keep the wild animals at bay.

The other strand is that I've studied chimpanzees and their feeding behavior for many years. I've eaten everything that I can get ahold of that chimpanzees eat. And I have been very much aware of the deeply unsatisfying nature of those foods because they are often quite fibrous, relatively dry, and contain little sugar, and they are often strong-tasting—in other words, really nasty. So here we are, two very closely related species with completely different dietary habits. It was an obvious hypothesis that cooking does something special for the food we find in nature. But I was astonished to discover that there was no systematic evidence showing what cooking does to the net energetic gain that we get from our foods.

For the past 14 years I've been focused on that question because to make a satisfactory claim about humans being adapted to cooked food, we have to produce some real evidence about what cooking does to food. Experiments conducted by Rachel N. Carmody of Harvard University have now given us the evidence: if we cook, we get more energy from our food.

Other researchers hold that increased access to meat allowed the teeth and gut to shrink. Why do you think cooking better explains these changes?

It's quite clear that humans began eating meat from large animals by 2.5 million years ago and have left a steady record of cut marks on bones since then. The cooking hypothesis does not deny the importance of meat eating. But there is a core difficulty with attributing changes in digestive anatomy to this shift.

Selection pressure on digestive anatomy is strongest when food is scarce. Under such conditions, animals have very little fat on them, and fat-poor meat is a very poor food because if you have more than about 30 percent protein in your diet, then your ability to get rid of ammonia fast enough is overwhelmed. Nowadays in surveys of hunters and gatherers, what you find is that during periods of food scarcity, there is always a substantial inclusion of plants. Very often it's tubers. To eat those raw, you would have to have the digestive apparatus to handle tough, fibrous, low-carbohydrate plant foods—that is, large teeth and a big gut.

So your idea is that by cooking those plant foods, our ancestors could evolve a smaller gut and teeth—and avoid overdosing on lean meat. Let's turn now to what happened when food was not so scarce and animals were good to eat. You have argued that cooking may have helped early humans eat more meat by freeing them up to hunt. What is your logic?

A primate the size of an early human would be expected to spend about half of its day chewing, as chimpanzees do. Modern humans spend less than an hour a day, whether you're American or living in various subsistence societies around the world. So you've got four or five hours a day freed by the fact that you're eating relatively soft food. In hunter-gatherer life, men tend to spend this time hunting.

That observation raises the question of how much hunting was possible until our ancestors were able to reduce the amount of time they chewed. Chimpanzees like to eat meat, but their average hunt is just 20 minutes, after which they go back to eating fruit. Hunting is risky. If you fail, then you need to be able to eat your ordinary food. If you hunt too long without success, you won't have enough time to process your usual, lower-quality fare. It seems to me that it was only after cooking enabled individuals to save time on chewing that they could increase the amount of time spent on an activity that, for all its potential benefits, might not yield any food.

You have also suggested that cooking allowed the brain to expand. How would cooking do that?

With regard to the brain, fossils indicate a fairly steady increase in cranial capacity, starting shortly before two million years ago. There are lots of ideas about why selection favored larger brains, but the question of how our ancestors could afford them has been a puzzle. The problem is that brains use a disproportionate amount of energy and can never be turned off.

I have extended the idea put forward by Leslie C. Aiello, now at the Wenner-Gren Foundation in New York City, and Peter Wheeler of Liverpool John Moores University in England that after cooking became obligatory, the increase in food quality contributed to reduced gut size. Their newly small guts were energetically cheaper, allowing calories to be diverted to the brain.

In 2012 Karina Fonseca-Azevedo and Suzana Herculano-Houzel of the Federal University of Rio de Janeiro added a new wrinkle. Their calculations showed that on a raw diet, the number of calories needed to support a human-sized brain would require too many hours eating every day. They argued that cooking allowed our ancestors the extra energy needed to support more neurons, allowing the increase in brain size.

Cooking is not the only way to make food easier to digest. How does it compare with other methods?

Simply reducing the size of food particles and the structural integrity of food—through pounding, for example—makes it easier to digest. Carmody did a study that looked at tubers and meat as representative types of food that hunter-gatherers eat and asked how well mice fared when eating each of these foods, either raw versus cooked or whole versus pounded. She very carefully controlled the amount of food that the mice received, along with the amount of energy they expended moving around, and assessed their net energetic gain through looking at body-mass changes. She found that pounding had relatively little effect, whereas cooking led to significant increases in body weight whether the food was tubers or meat.

This is incredibly exciting because, amazingly, this is the first study that has ever been done to show that animals get more net energy out of their food when it is cooked than when it's raw. Second, it showed that even if pounding has some positive effects on energy gain, cooking has much bigger effects. [*Editors' note:*

Wrangham was a co-author on the study, published in 2011.]

Is there any genetic evidence to support the cooking hypothesis?
There is essentially nothing published yet. But we're very aware that a really interesting question is going to be whether or not we can detect, in the human genome, evidence of selection for genes related to utilizing cooked food. They might be concerned with metabolism. They might be concerned with the immune system. They might be concerned somehow with responses to Maillard compounds, which are somewhat dangerous compounds produced by cooking. This is going to be a very exciting area in the future.

A really interesting question is going to be whether or not we can detect, in the human genome, evidence of selection for genes related to utilizing cooked food.

A central objection to the cooking hypothesis has been that there is no archaeological evidence of controlled fire as far back as the hypothesis predicts. Currently the oldest traces come from one-million-year-old deposits in Wonderwerk Cave in South Africa. But you have recently identified an independent line of evidence that humans tamed fire earlier than the archaeological record suggests. How does that work support your thinking?
Chimpanzees love honey, yet they eat very little of it because they get chased away by bees. African hunters and gatherers, in contrast, eat somewhere between 100 and 1,000 times as much honey as chimpanzees do because they use fire. Smoke interferes with the olfactory system of the bees, and under those conditions, the bees do not attack. The question is: How long have humans been using smoke to get honey? That's where the honeyguide comes in. The greater honeyguide is an African species of bird that is adapted to guiding humans to honey. The bird is attracted to human activity—sounds of chopping, whistling, shouting, banging and, nowadays, motor vehicles. On finding people, the bird starts fluttering in front of them and then leads them off with a special call and waits for them to follow. Honeyguides can lead humans a kilometer or more to a tree that has honey in it. The human then uses smoke to disarm the bees and opens the hive up with an ax to extract the honey inside. The bird gains access to the hive's wax, which it eats.

It used to be thought that the bird's guiding behavior [which is innate, not learned] originated in partnership with the honey badger and that humans moved in on this arrangement later. But in the past 30 years it has become very clear that honey badgers are rarely, if ever, led to honey by honeyguides. If there's no living species other than humans that has this symbiotic relationship with the bird, could there have been some extinct species of something that favored the honeyguide showing this behavior? Well, obviously, the most reasonable candidates are the extinct ancestors of humans. The argument points very strongly to our ancestors having used fire long enough for natural selection to enable this relationship to develop.

Claire Spottiswoode of the University of Cambridge discovered that there are two kinds of greater honeyguide females: those that lay their eggs in ground nests and those that lay in tree nests. Then she found that the two types of behavior are associated with different lineages of mitochondrial DNA [DNA that is found in the energy-producing components of cells and passed down from mother to offspring]. Based on a fairly conservative assessment of the rate of mutation, Spottiswoode and her colleagues determined that the two lineages had been separated for about three million years, [providing a minimum estimate for the age of the greater honeyguide species]. That doesn't necessarily mean that the guiding habit, which depends on humans using fire, is that old—it could be more recent—but at least it tells you that the species is old enough to allow for much evolutionary change.

If cooking was a driving force in human evolution, does this conclusion have implications for how people should eat today?
It does remind us that eating raw food is a very different proposition from eating cooked food. Because we don't think about the consequences of processing our food, we are getting a misunderstanding of the net energy gain from eating. One of the ways in which this can be quite serious is if people who are dedicated to a raw-food diet don't understand the consequences for their children. If you just say, "Well, animals eat their food raw, and humans are animals, then it should be fine for us to eat our food raw," and you bring your children up this way, you're putting them at very severe risk. We are a different species from every other. It's fine to eat raw food if you want to lose weight. But if you want to gain weight, as with a child or an adult who's too thin, then you don't want to eat a raw diet. SA

Kate Wong is a senior editor at Scientific American.

MORE TO EXPLORE

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Honey and Fire in Human Evolution. Richard Wrangham in *Casting the Net Wide: Papers in Honor of Glynn Isaac and His Approach to Human Origins Research*. Edited by Jeanne Sept and David Pilbeam. Oxbow Books, 2012.

Metabolic Constraint Imposes Tradeoff between Body Size and Number of Brain Neurons in Human Evolution. Karina Fonseca-Azevedo and Suzana Herculano-Houzel in *Proceedings of the National Academy of Sciences USA*, Vol. 109, No. 45, pages 18,571–18,576; November 6, 2012.

SCIENTIFIC AMERICAN ONLINE
Watch a video on why cooking makes food taste good at ScientificAmerican.com/sep2013/cooking

CONSERVATION

return of

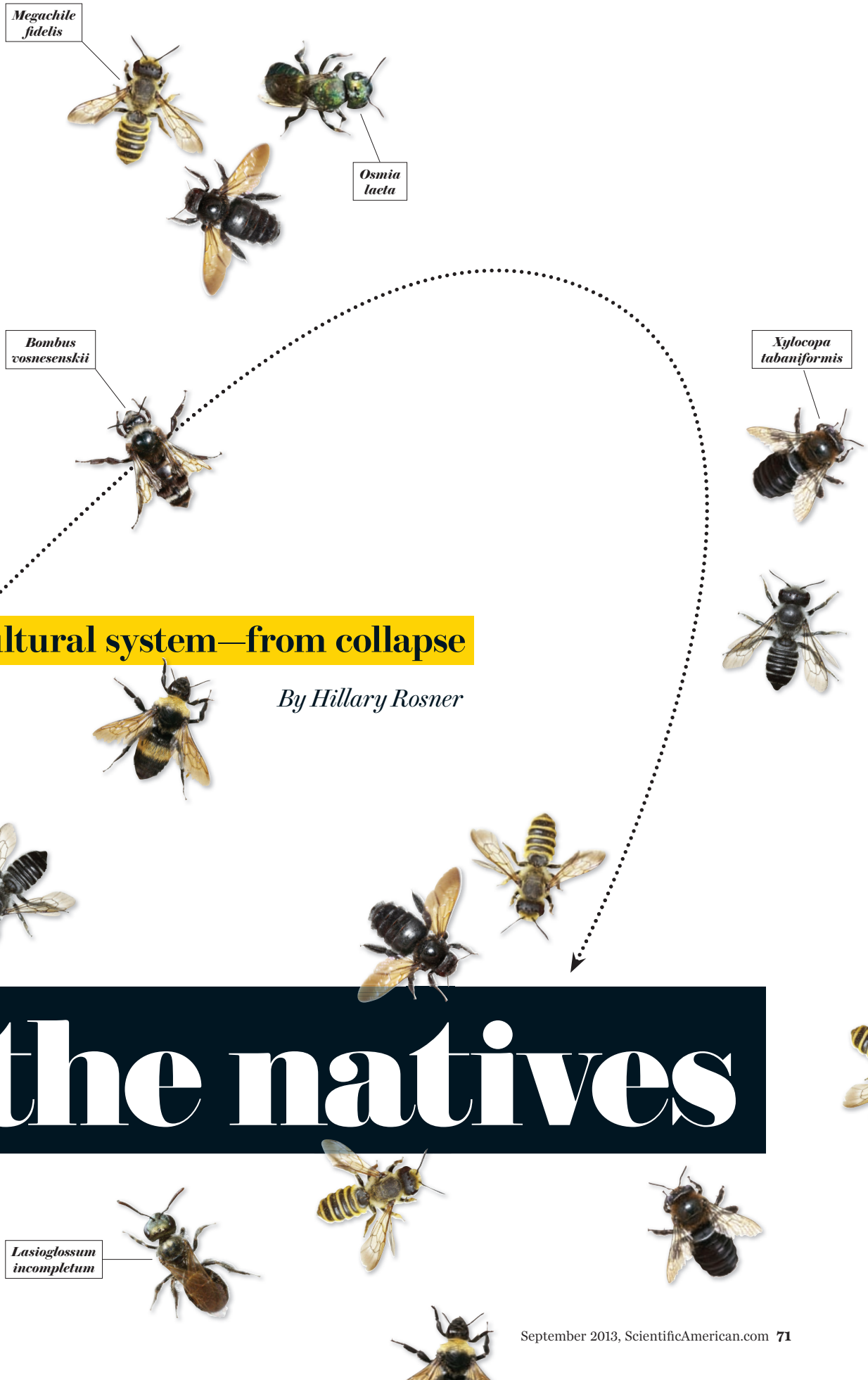
Reviving native bee species could save honeybees—and

IN BRIEF

The U.S. relies primarily on a single insect, the domesticated European honeybee, to pollinate one third of its food supply, including such delicious crops as apples, peaches, almonds, lettuces, broccoli, cranberries, squashes, melons and blueberries.

As colony collapse disorder and other maladies continue to devastate honeybee populations, researchers are turning their attention to alternative pollinators—the thousands of native bee species throughout the country—and are looking for ways to make croplands more attractive to these wild bees. **So far studies suggest** that restoring wild habitat near farms to welcome and nurture native bees not only increases crop yield but also makes honeybees themselves more efficient pollinators.

Photographs by David Liittschwager

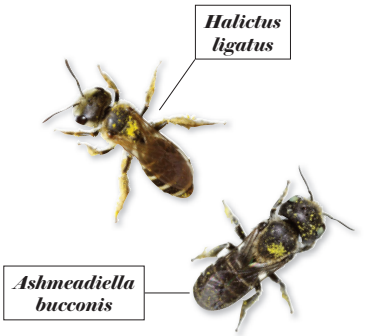


our agricultural system—from collapse

By Hillary Rosner

the natives

Hillary Rosner is a freelance writer based in Colorado. She has written for the *New York Times, Wired, Popular Science* and *Mother Jones,* among other publications.



FIELD BIOLOGISTS HAVE A STRANGE AFFINITY for spending countless hours in the hot sun scrutinizing tiny things. You might see a bee buzzing on a flower and think, "Oh, a bee." A biologist, though, will want to know: Is it a nonnative, domesticated honeybee? Or is it one of 4,000 bee species native to the U.S.—maybe an ultragreen sweat bee, a metallic-sheened creature that drinks human perspiration? Or perhaps a cuckoo bee, such as *Bombus suckleyi,* a type of bumblebee that sports yellow hair on its fourth abdominal segment, as opposed to the rare *B. occidentalis,* which has black or white hair in the same spot?

You also can probably name many reasons not to sit in a field counting grains of pollen, an activity that conservation biologist Claire Kremen thinks is a perfectly reasonable way to spend an afternoon. But then, you probably will not be the one to revamp the nation's food supply and rescue our agricultural system from looming collapse. Kremen, however, just might.

A decade ago, after years of work in Madagascar, she turned her attention to a problem brewing closer to home. Colony collapse disorder (CCD) had not yet been diagnosed or named, but already American beekeepers were reporting record deaths within their honeybee hives. A third of the U.S. food supply depends primarily on the honeybee for pollination—apples, almonds, peaches, lettuces, squashes, melons, berries and broccoli, to name a few crops. Kremen, now at the University of California, Berkeley, began to wonder about other kinds of bees. Could wild native bee species ease our dependence on honeybees by lessening their workload?

With Neal Williams, at the time a graduate student in her then Princeton University laboratory, and Robbin Thorp, a renowned bee taxonomist, Kremen studied watermelon pollination in California's Central Valley. Kremen and her team monitored how frequently each of 39 different bee species visited a flower and how much pollen each bee deposited. Based on previous studies, they knew that it takes around 1,000 grains of pollen to build a single juicy watermelon. Growers on organic farms surrounded by wild plants, it turned out, did not even bother hiring hives. The native bees did all the work, saving the farmers money year after year. In contrast, on conventional monoculture farms with large swaths of a single crop the wild bees barely made a dent. Without honeybees, those farmers would be looking for new jobs.

The resulting paper, published in 2002 in the *Proceedings of the National Academy of Sciences USA,* concluded that by restoring native bee habitat in their fields, farmers could "hedge their bets in the event of honey bee scarcity through partial replacement of honey bee by native bee services." In the decade since, other researchers have cited the paper—now seen as prescient—614 times. During that period, CCD has joined the already long list of maladies afflicting honeybee hives, and the economic fortunes of beekeepers have grown increasingly perilous. "Bees," says Kremen, who won a "genius" grant from the MacArthur Foundation in 2007, "are telling us something very fundamental about our agricultural system and how off-balance it is."

Kremen's work is now funded in part by the U.S. Army, which wants to safeguard the nation's food security. "It's a component of creating a resilient system," she says. Plants that require animal pollination contribute 98 percent of the total vitamin C supplied by major global crops, 70 percent of vitamin A, 55 percent of folic acid and 74 percent of lipids. "If all the pollinators went extinct, we probably wouldn't starve," Kremen says. "But we'd all have scurvy or some other vitamin-deficiency disorder."

The honeybee crisis underscores the tremendous risk we have unwittingly built into our farm system by relying on a single insect to pollinate so much of our food supply. As author Hannah Nordhaus put it in her book *The Beekeeper's Lament,* "Farmers expect bees to function like yet another farm machine—like shakers, sweepers, tillers and combines." But honeybees are living creatures, subject to the realities of biology. And despite 400 years of domestication, there are still many things about honeybee biology we cannot control—for instance, the insects' susceptibility to parasites, viruses and climatic conditions. They may be domesticated, but they do not exactly stay in a pen as cattle do.

There are other things we *can* control: namely, the environmental factors that govern the bees' life cycle. As it turns out, we have engineered an environment that, in some ways, could not be worse for the bees. "Our monoculture system," Kremen says, "is

creating a huge demand for an army of pollinators, and there's virtually no way to ensure that except for bringing in honeybees. If they're sick and having problems, what are we going to do?"

GHOST SHIPS

WHAT WE KNOW as the honeybee is more accurately called the European honeybee (*Apis mellifera*), which first arrived with early colonists on ships from England sometime around 1620. From the beginning, various pests and pathogens plagued hives, and beekeeping was a battle to stay a step or two ahead of the grim reaper's scythe. Wax moths, American foulbrood, drought, nosema disease: these are just a few of the things that have doomed both hives and beekeepers through the centuries.

In the fall of 2006 a now legendary beekeeper named Dave Hackenberg discovered that 360 out of his 400 hives in Florida were lifeless—no bees in sight. "They waited, fully stocked with pollen, honey, and larvae—like ghost ships—for their inhabitants to return," Nordhaus wrote. "But the bees never came back."

By the following winter some beekeepers had lost 90 percent of their hives; across the country a third of honeybee hives collapsed, many in this same mysterious way. Researchers named such disappearances "colony collapse disorder," although the term quickly became a metonym for all the maladies afflicting honeybees.

Scientists have failed to find a single culprit that is primarily responsible for CCD. A flurry of recent studies implicates neonicotinoids, or neonics, a widely used class of pesticides, but they probably do not deserve all the blame. The most likely scenario is that neonics are an indirect cause of bee declines, leaving colonies far more susceptible to pathogens such as the parasitic fungus that causes nosema disease and varroa mites—rust-colored parasites that suck out bees' vital fluids and spread crippling viral diseases. (In Australia, where neonics are heavily used but there are no varroa mites, honeybee colonies remain healthy.) Other contributing factors include fungicides, drought and an inadequately diverse diet.

The meta problem may be that our agricultural system is simultaneously dependent on honeybees and contributing to their demise. Relying on a single bee species to pollinate nearly 100 different crops is untenable. Every year beekeepers truck their hives around the country in the back of tractor-trailers, following the flowering of various crops: almonds to cherries to apples, and so on. Often, when no crops are in bloom, the bees do not have a lot to eat. Beekeepers supplement their diet with corn syrup or sugar water, which do not have nearly the nutritional value that natural pollen and nectar do. On top of that, during huge crop pollination events such as the almond bloom, around 1.5 million hives from around the country converge in California, creating near-perfect conditions for transmitting diseases. Imagine a giant gathering of kindergartners from every region of the nation, all intermingling their germs.

FLOWER POWER

ON A SUNNY DAY in early April, not long after the almond bloom has faded, I set out to see what Williams, now at U.C. Davis, and Kremen are up to. Next to a field of walnut trees near the university, a row of tall shrubs planted by the researchers stretches for several hundred yards: western redbud, coffeeberry, gum plant, sage, coyote brush. The bushes are in varying stages of bloom, and tiny, black bees fly from flower to flower. They are mason bees, known for building mud apartments inside wood dwellings.

Last year Kremen and her team recorded a total of 130 species of native bees lured to hedges neighboring 40 different farm fields. Based on historical records, California was once home to as many as 1,600 native bee species, although it is unclear how many of these persist today. A recent study published in the journal *Science* found that in a span of 120 years, Illinois lost half its wild bee species, largely because of diminished numbers of wild flowering plants. Another study concluded that four species of American bumblebees have lost up to 87 percent of their habitat, slashing their ranks by 96 percent.

Kremen is hoping to prove not just that her hedgerows attract bees, which is already clear, but also that they are increasing the overall number and diversity of bees in the area rather than siphoning bees from elsewhere. "It's possible that you plant this hedgerow and it sucks all the native bees from the landscape,"

The honeybee crisis underscores the tremendous risk we have built into our farm system by relying on a single insect to pollinate so much of our food supply.

says Leithen M'Gonigle, a postdoctoral researcher in Kremen's lab. "When your crop is flowering, you don't want the hedgerows to be more attractive." In other words, the architecture of restoration might matter a lot.

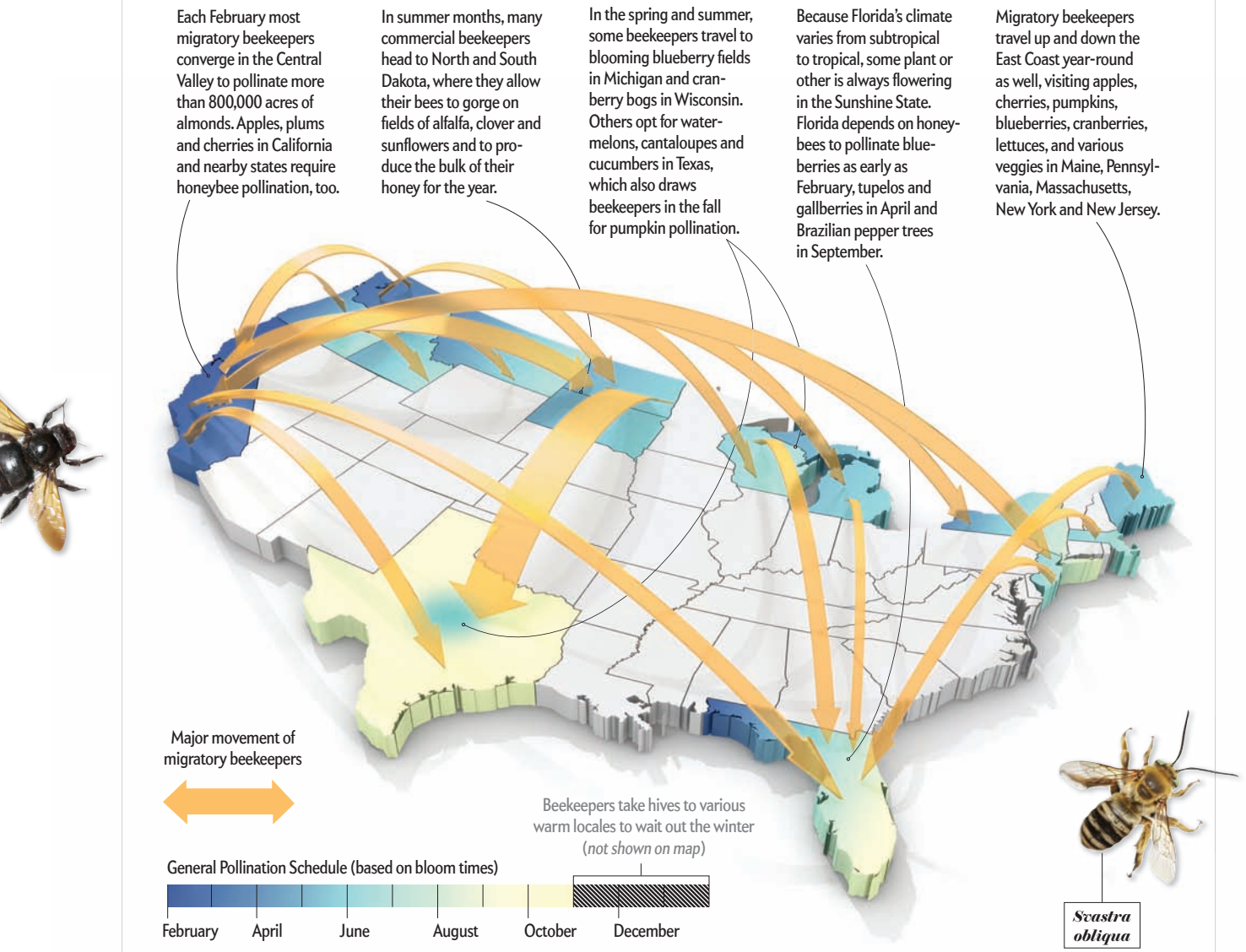
At a research farm owned by U.C. Davis, a giant bed of knee-high plants, some already budding and flowering, has taken root between neatly organized crop rows that run to the horizon. Here Williams is experimenting with forbs—perennial and annual flowering plants—that could appeal to farmers who do not want to deal with the hassle of woody plants on their fields. The nine plant species in Williams's current experimental mix are drought-tolerant, native, and selected to maintain diversity and abundance throughout the season.

Scientists also hope to learn more about how native bees and honeybees interact. In a study published this year researchers from Williams's and Kremen's labs found that honeybees became even more effective pollinators of almond trees in the presence of both various native species and blue orchard bees, a managed species. The more efficiently honeybees work, the fewer are needed to pollinate a given field. The investigators are now studying whether a specific chemical footprint left by the native bees in fact alerts the honeybees to extra competition.

Hedgerows and wildflowers sound like the province of mild-

Bees without Borders

In the U.S., many farmers cannot rely on native bees or even local honeybees to sufficiently pollinate their vast swaths of cropland. Rather they rent honeybee hives from the 1,600 or so migratory beekeepers who traverse the country between February and November. This annual migration mingles sick insects with healthy ones and deprives bees of proper nourishment when on the road.



mannered gardeners puttering about in floppy hats. Yet as mundane as the whole thing may seem, restoring native habitat to farmland could represent the start of an agricultural revolution—one that could make much of our food supply more sustainable. No existing technology can pollinate crops. In southwestern China, where a combination of habitat loss, wanton use of pesticides and overharvesting of honey has wiped out bees, workers pollinate apple and pear orchards by hand, transferring pollen from one flower to another with small brushes. Such a massive effort is far too labor-intensive for the U.S., where it would render fruit prohibitively expensive. Bees—not just honeybees but all bees—are our only hope.

One way scientists aim to jump-start this agricultural revolution is with a program called Integrated Crop Pollination, or ICP. Funded by the U.S. Department of Agriculture, ICP consists of a series of options and steps for supplementing honeybees—including expanding habitat, reducing pesticide use and adding in other managed pollinators. Currently several other bee species, such as the blue orchard variety, are commercially available and may help farmers supplement honeybee populations.

Illustration by Bryan Christie

ICP began as an idea flitting about the mind of Rufus Isaacs. As the resident blueberry entomologist at Michigan State University, he spends a lot of time among the fruiting shrubs. While researching ways to control Japanese beetles and other blueberry enemies, he began to notice all the bees. Honeybees, yes, but also Michigan natives such as plump *B. impatiens* bumblebees, hairy-shouldered *Andrena bees* and small, black *Ceratina* bees that nest in thin, hollow stems. Isaacs realized that no one really knew which bees, or how many kinds, were out there. So Julianna Tuell, then a graduate student in his lab, set about categorizing them. She found 112 species of native bees zipping through blueberry fields in bloom and an additional 54 species active before and after the flowering.

Most of the native bees were solitary varieties: individuals that make their own nests in the soil rather than living in social hives. The most common species was *Andrena carolina,* a medium-size brown bee that gathers pollen only from plants in the blueberry family, including cranberry, huckleberry and azalea. Overall, though, the bulk of the bee species were generalists, collecting pollen from a wide range of plants.

A few years ago Isaacs, like Kremen, decided to find out how much wild bees contribute to blueberry pollination. Researchers have estimated the value of wild bee crop pollination in the U.S. at $3.1 billion a year; honeybee pollination is worth roughly $15 billion. Isaacs discovered that in small fields of less than an acre, wild bees took care of 82 percent of pollination. In big fields—1.5 to 16 acres—wild bees accomplished only 11 percent of pollination. Because the bulk of Michigan's blueberries are grown on large farms, Isaacs estimated that wild bees provide just 12 percent of the state's blueberry pollination. That is nowhere near enough to serve as insurance against honeybee declines, he says.

Yet if farmers had an economic incentive to add habitat—on fallow fields or in areas that are frost-prone, have poor soil or are otherwise unfit for blueberries—the story could be different. A graduate student in Isaacs's lab investigated pollination in five blueberry fields of up to 10 acres, with up to two acres planted with native Michigan wildflowers in a mix that blooms from spring until early fall. The study, not yet published or peer-reviewed, showed that booming native bee populations increased blueberry yields to such an extent that farmers could recoup the cost of establishing habitat in three to four years. Setting up habitat costs around $600 per acre, Isaacs says, but the USDA's Natural Resource Conservation Service has programs that will cover between 50 and 90 percent of the expense.

Researchers continue to seek out the best ways to nurture native bees, but farmers can start improving crop pollination now. Gordon Frankie, a U.C. Berkeley bee biologist whose office sits directly above Kremen's, has spent more than a decade designing bee habitats for urban gardens, and now he has begun applying that knowledge to agriculture. "You can't have a one-size-fits-all approach," Frankie says. "Each farm will be different, with different needs. But the idea is that we'll be able to write a prescription for any farm—you need this, this and this." On four farms in Brentwood, Calif., about an hour outside Berkeley, he has planted a mix of shrubs and forbs near blackberry bushes and cherry trees. Frankie hopes to create a series of case studies—"an orchard cropper, a row cropper, 25 acres, 145 acres"—that he can use to reach out to similar types of farms.

Meanwhile, using data from Kremen, Williams and others, the Xerces Society has partnered with the USDA's Natural Resource Conservation Service to build a "pollinator-enhancement program." Since 2009 the group has trained more than 20,000 people—farmers, USDA representatives, cooperative extension agents—in the value of native bees. It has also developed a set of concrete guidelines for farmers, explaining how to plan a meadow to attract native bees and to minimize the effect of pesticides.

A farm set up to welcome native bees could, ultimately, be better off than one reliant on honeybees. More than 20,000 species of native bees are abuzz around the world; collectively, they are exceedingly more likely to recover from disease or extreme weather than any one species of pollinator. Kremen believes the hedgerows are only a first step. The real challenge will be scaling up to 1,000-acre farms, bringing pollinators back to massive monoculture operations. She envisions a system where farms are divided into blocks that bloom at different times, so there is always food for pollinators to eat.

It is a system some farmers are already embracing. In the Central Valley, Frank Muller and his two brothers farm a diverse assortment of conventional and organic crops for chain stores such as Safeway and Walmart, including canning tomatoes, pickling cucumbers, and everything from almonds to wine grapes to sunflowers. The Mullers have planted habitat to attract native bees and have started their own on-farm honeybee operation. "They can be in our crops all the way from February through August or September," he says. The farmers will also put in plants specifically chosen to provide nectar in the remaining months. "We're not going to lose our bees," Muller says of the crisis. "We just need to manage them differently."

For now the Mullers are still in a minority. Not all farmers are ready to upend their long-standing ways of doing things—or pay—to bring in more pollinators, at least not until the honeybee predicament directly harms them. As honeybees continue to suffer, though, more and more farmers may change their minds.

M'Gonigle thinks the honeybee crisis could be "a kind of blessing in disguise" because "it forces us to think, 'What are we going to do to keep our food production going?' In the long term, it might be that we look back and say, 'Wow, this was a good thing, a good way of getting us to reprioritize and start thinking about conservation of native species.'"

As I watch a mix of honeybees and their wild cousins dart among purple flowers in one of Kremen's hedgerows, it is easy to see what he means. Our entire modern-day agricultural system has grown up with honeybees, so we have never had to really consider the fact that relying on a single pollinator is probably not sustainable. This may be a window of opportunity—even if climbing through it could sting a little. SA

MORE TO EXPLORE

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Are Neonicotinoids Killing Bees? A Review of Research into the Effects of Neonicotinoid Insecticides on Bees, with Recommendations for Action. Jennifer Hopwood et al. Xerces Society for Invertebrate Conservation, 2012. www.xerces.org/neonicotinoids-and-bees

SCIENTIFIC AMERICAN ONLINE

To learn more about almond pollination in California and migratory beekeeping, visit ScientificAmerican.com/sep2013/migratory-beekeeping

Probiotics

MICROBIOLOGY

Enlisting bacteria and fungi from the soil to support crop plants is a promising alternative to the heavy use of fertilizer and pesticides

By Richard Conniff

TOMATOES FRESH FROM A ROADSIDE STAND, SLICED, GLISTENing, and served with nothing more than salt, pepper and a drizzle of olive oil—a sacred pleasure of summer. To die for? Possibly so.

Almost every year for the past decade or so, public health investigators on the East Coast have tracked down one or two *Salmonella* outbreaks and identified local tomatoes as the culprit. These outbreaks are typically small, affecting 10 to 100 people. Yet for the very old and very young, they can mean hospitalization and even death.

A few years ago Eric Brown, director of microbiology at the U.S. Food and Drug Administration's Center for Food Safety and Applied Nutrition, began to wonder: Why East Coast tomatoes? The *Salmonella* bug probably gets onto tomato fields from surface water and the droppings of seagulls, turtles, poultry, and other animals. So why aren't West Coast tomatoes contaminated, too?

The answer to Brown's question came from a close inspection of the community of bacteria, viruses and fungi living in and around all plants—what scientists call the microbiome. West Coast tomatoes, it turned out, grow in the company of soil bacteria that inhibit and even kill *Salmonella*. When researchers went to hunt for similar strains back East, they found them but in smaller numbers. Thus, in a pilot study in Virginia, the FDA has been brewing up populations of one of these local bacteria, *Paenibacillus,* spraying them onto tomato seedlings and getting the same anti-*Salmonella* effect on the crop. Brown expects to move the process out to commercial tomato growers in 2014 or 2015.

Adding bacteria to a crop to prevent human disease could be the start of a whole new path to food safety, possibly extending beyond tomatoes to cantaloupes, spinach, sprouts and other crops that have made *Salmonella* and *Escherichia coli* headlines. The tomato project fits into a far more dramatic shift in how we

Illustration by Don Foley

grow our food, based on a new understanding of microbes in the soil and of the many ways plants and microbes depend on one another.

It is almost the opposite of the green revolution, which dramatically boosted agricultural productivity in the mid-20th century with massive inputs of fertilizer, pesticides and water. The microbial revolution aims instead to take advantage of what is already there: as many as 40,000 microbe species in a gram of soil. Until recently, this microbial community—what might be called the "agribiome"—was largely a mystery. But over the past decade low-cost DNA sequencing and other technologies have opened up the secret world of microbes. Botanists can now identify every member of the microbial community that surrounds a plant. By doing so, they have begun to understand how various microbes behave in different seasons and soil environments and have even started devising ways to tweak them to help plants grow better.

Soil scientists must come to grips with so much new information, in fact, that Andrea Ottesen, the FDA microbiologist who cracked the tomato *Salmonella* case, describes it, with a sigh, as "kind of a huge rabbit's hole at this point." But sorting out that wealth of new information to help farmers grow better crops seems particularly urgent, given the vast challenges that agriculture now faces: the global water shortage; extreme and unpredictable weather events such as last summer's devastating drought in the U.S. corn belt; worries over the sustainability of nitrogen fertilizer produced with fossil fuels; and the prospect of having to feed an extra two billion people by midcentury.

New research suggests that microbes could provide an alternative to existing agricultural methods and genetic engineering in alleviating some of these problems. For instance, sunflowers and some other plants naturally produce the sugar trehalose, which helps to stabilize plant cell membranes and to reduce the damage from cycles of drying followed by rehydration. Other plants, including corn and potatoes, have been genetically engineered to manufacture trehalose. Yet molecular biologist Gabriel Iturriaga in Mexico hopes to eventually treat crops without any genetic modification by using the trehalose-producing bacterium *Rhizobium etli,* which is found around the roots of bean plants. An earlier experiment with a genetically altered version of the bacterium improved yields by 50 percent in normal conditions—and saved half the crop during a drought.

Microbial methods also give farmers more flexibility. One problem with plants that have been genetically engineered for drought resistance is that they do poorly in wet years. Thus, farmers have to try to predict the weather when they select seeds at the start of the growing season. But a cocktail of microbes may enable plants to adapt even when growing conditions suddenly shift.

Richard Conniff specializes in writing about human and animal behavior. His latest book is *The Species Seekers: Heroes, Fools, and the Mad Pursuit of Life on Earth* (W. W. Norton, 2010).

Russell Rodriguez and Regina Redman of Adaptive Symbiotic Technologies in Seattle have been working with a plant fungus that appears to make a range of food crops more tolerant of salinity, drought, and extreme heat or cold. The fungus thrives in panic grass, which survives soil temperatures as high as 70 degrees Celsius around thermal pools at Yellowstone National Park. The grass can stand the heat only if this particular fungus is present and only if the fungus contains a crucial virus that serves as a kind of on/off switch for heat tolerance. The researchers have gone on to collect root fungi in a range of high-stress environments, from sand dunes to alpine slopes. The ambition, Rodriguez says, is to achieve a blend that reliably boosts yields by 10 to 15 percent in an increasingly unpredictable range of conditions.

PHOSPHATE WARS

OTHER RESEARCHERS are tweaking the agribiome to help deliver crucial nutrients to plants. Farmers have, of course, recognized for thousands of years that soybeans, peanuts and other legumes have an almost magical power to fertilize the soil. Further, scientists have known for more than a century that it is not, in fact, the plants that manage the trick of pulling nitrogen out of the air, it is the rhizobial bacteria living in nodules on their roots.

Plants also require phosphate, which is exceptionally low in the soils of many tropical nations. Farmers in developing countries often depend entirely on the international market for phosphate fertilizer. In 2007 and 2008 prices for phosphate and other fertilizers spiked, contributing to food riots from Mexico to Bangladesh. In some countries, farmers now just skip phosphate fertilizer altogether and take their chances with starvation.

Yet researchers have known for decades of a possible remedy. Soil microbes called arbuscular mycorrhizal fungi form spores and filaments inside and around a plant's roots and help them acquire phosphate. There has never been a good way to mass-produce and deliver the stuff. Soil containing the spores that form new fungi can, in fact, be shipped from one country to another, but the environmental impact of introducing foreign fungi as exotic species remains uncertain. And the spores of the fungi are so thinly concentrated that a farmer planting a crop like cassava needs to apply the enriched soil at a rate of a metric ton per hectare.

With the help of new technologies, a few companies can now mass-produce the fungi in culture and market it in a highly con-

IN BRIEF

Microbes in and around food crops do not just cause human disease. In certain cases, they do exactly the opposite, acting as sentinels of food safety and furnishing an environmentally sound alternative to massive inputs of fertilizers and pesticides.

Spreading bacteria on crops became a strategy for researchers in Virginia who sprayed anti-*Salmonella* soil bacteria on tomato seedlings. The scientists hope the approach might prevent annual outbreaks of food poisoning from raw tomatoes grown on the East Coast.

Applying fungi to cassava plants, a project of researchers in Colombia, helps the roots acquire phosphate without the need for expensive fertilizers, a boon in tropical nations where the amount of nutrient that can be obtained from the soil is particularly low.

centrated gel. A farmer can carry enough to cover a hectare in a soda bottle. Research teams can collect local strains of the fungi, test which ones look most promising, then deliver them to a manufacturer for production. Ian R. Sanders of the University of Lausanne in Switzerland and Alia Rodriguez of the National University of Colombia began field studies last year focusing on cassava, a root crop that is a staple food for much of the developing world.

In the field, a farmer dilutes the gel in a bucket of water and dips a mesh sack of cassava stems in the bucket for a few seconds before planting them. In the first season of testing, that treatment cut phosphate use in half and boosted yields by 20 percent. Sanders and Rodriguez are now crossbreeding multiple fungus strains on the three or four common cassava varieties. They are also testing the strains in Africa and, if successful, will expand this program to half a dozen countries there, thus enabling the technology to benefit subsistence farmers.

Another promising path to agricultural symbiosis involves studying the chemical signals that microbes use to communicate with one another. Researchers monitor this everyday chitchat to identify which bacteria may be suited to the task of supplying plant nutrients or to find weaknesses in pathogens. This strategy has given rise to a potential weapon against *Xylella fastidiosa,* the bacterium that causes Pierce's disease, which is killing off vast swaths of California grapes. The bacterium is quiescent until its insect host (the glassy-winged sharpshooter) feeds on a grape plant. It wakes up inside the plant but later becomes quiescent again when it is time to be acquired by another insect.

"Basically, the lifestyle that it takes on to be transmitted by an insect is incompatible with its ability to move in the plant," says Steve Lindow of the University of California, Berkeley. Lindow took genes that the pathogen uses to signal quiescence and spliced them into the grape genome. When the pathogen arrives, the plant's transgenes tell it to behave as if it were about to be acquired by an insect, rendering it harmless.

UNMET PROMISES

IN THE PAST new microbial methods in agriculture have often failed to yield the promised results in the field, in part because of a lack of funding to translate basic research into practical applications. Molecular biologists also often lack the inclination to transfer their know-how to farmers. "It's a tale of two worlds," says Ken Giller of Wageningen University in the Netherlands, who works in Africa on improving use of rhizobial bacteria for nitrogen-fixing legumes. The molecular work on the genetics of nitrogen fixation has been "an absolutely fascinating story," he remarks. Meanwhile farmers continue to treat their plants with bacterial strains first isolated 30 years ago. "And it's largely because the scientists doing this are hell-bent on finding the next finer detail," Giller says. "A lot of interesting discoveries aren't being picked up and taken through to the point of application."

Many products that do make it into the field are ineffective because they have not been adequately tested or because they are manufactured carelessly, perhaps fraudulently. The International Institute of Tropical Agriculture (IITA) in Nigeria has tested 106 different farm products, most of them microbial. All but five failed because they did not contain the active ingredient on the label, they did not have enough of it, or they were not effective in greenhouse and field trials.

CHECKING THE CASSAVA: A student in Colombia inspects a plant treated with a fungi-laden gel that promotes the uptake of phosphate, an essential nutrient.

COURTESY OF IAN R. SANDERS *University of Lausanne*

Many of the flawed products come from Europe, the U.S. and Japan. Rather than taking on the manufacturers, IITA is training regulators in the target countries to do their own quality testing. The institute is also developing a seal of approval to let buyers know when a product meets reasonable standards. The program aims to help farmers understand not just which microbial products work but where and under what conditions.

Getting farmers to understand the new rules of the agribiome is "going to be incredibly complicated," says Ann Reid, director of the American Academy of Microbiology, but it will also be "very cool." It means convincing farmers that their work is not a simple business of inputs and outputs—some water here, some pesticides there. Instead it means waking up to what farming has always been—a collaboration with the vast community of microbes. If farmers and scientists together can get that right, we will have come a step closer to feeding a hungry world. SA

MORE TO EXPLORE

Trehalose Accumulation in *Azospirillum brasilense* Improves Drought Tolerance and Biomass in Maize Plants. Julieta Rodríguez-Salazar et al. in *FEMS Microbiology Letters,* Vol. 296, No. 1, pages 52–59; July 2009.

Microbes Helping to Improve Crop Productivity. Ann Reid in *Microbe Magazine,* Vol. 6, No. 10; October 2011. http://bit.ly/1aLQBDX

SCIENTIFIC AMERICAN ONLINE

Watch an animation showing how fungi can help a plant obtain an essential nutrient at ScientificAmerican.com/sep2013/microbial

BIOTECHNOLOGY

are engineered foods Evil?

Proponents of genetically modified crops say the technology is the only way to feed a warming, increasingly populous world. Critics say we tamper with nature at our peril. Who is right?

By David H. Freedman

ROBERT GOLDBERG SAGS INTO HIS DESK CHAIR AND GESTURES AT THE air. "Frankenstein monsters, things crawling out of the lab," he says. "This the most depressing thing I've ever dealt with."

Goldberg, a plant molecular biologist at the University of California, Los Angeles, is not battling psychosis. He is expressing despair at the relentless need to confront what he sees as bogus fears over the health risks of genetically modified (GM) crops. Particularly frustrating to him, he says, is that this debate should have ended decades ago, when researchers produced a stream of exonerating evidence: "Today we're facing the same objections we faced 40 years ago."

Across campus, David Williams, a cellular biologist who specializes in vision, has the opposite complaint. "A lot of naive science has been involved in pushing this technology," he says. "Thirty years ago we didn't know that when you throw any gene into a different genome, the genome reacts to it. But now anyone in this field knows the genome is not a static environment. Inserted genes can be transformed by several different means, and it can happen generations later." The result, he insists, could very well be potentially toxic plants slipping through testing.

Williams concedes that he is among a tiny minority of biologists raising sharp questions about the safety of GM crops. But he says this is only because

KEVIN VAN AELST

the field of plant molecular biology is protecting its interests. Funding, much of it from the companies that sell GM seeds, heavily favors researchers who are exploring ways to further the use of genetic modification in agriculture. He says that biologists who point out health or other risks associated with GM crops—who merely report or defend experimental findings that imply there may be risks—find themselves the focus of vicious attacks on their credibility, which leads scientists who see problems with GM foods to keep quiet.

Whether Williams is right or wrong, one thing is undeniable: despite overwhelming evidence that GM crops are safe to eat, the debate over their use continues to rage, and in some parts of the world, it is growing ever louder. Skeptics would argue that this contentiousness is a good thing—that we cannot be too cautious when tinkering with the genetic basis of the world's food supply. To researchers such as Goldberg, however, the persistence of fears about GM foods is nothing short of exasperating. "In spite of hundreds of millions of genetic experiments involving every type of organism on earth," he says, "and people eating billions of meals without a problem, we've gone back to being ignorant."

So who is right: advocates of GM or critics? When we look carefully at the evidence for both sides and weigh the risks and benefits, we find a surprisingly clear path out of this dilemma.

BENEFITS AND WORRIES

THE BULK OF THE SCIENCE on GM safety points in one direction. Take it from David Zilberman, a U.C. Berkeley agricultural and environmental economist and one of the few researchers considered credible by both agricultural chemical companies and their critics. He argues that the benefits of GM crops greatly outweigh the health risks, which so far remain theoretical. The use of GM crops "has lowered the price of food," Zilberman says. "It has increased farmer safety by allowing them to use less pesticide. It has raised the output of corn, cotton and soy by 20 to 30 percent, allowing some people to survive who would not have without it. If it were more widely adopted around the world, the price [of food] would go lower, and fewer people would die of hunger."

In the future, Zilberman says, those advantages will become all the more significant. The United Nations Food and Agriculture Organization estimates that the world will have to grow 70 percent more food by 2050 just to keep up with population growth. Climate change will make much of the world's arable land more difficult to farm. GM crops, Zilberman says, could produce higher yields, grow in dry and salty land, withstand high and low temperatures, and tolerate insects, disease and herbicides.

Despite such promise, much of the world has been busy banning, restricting and otherwise shunning GM foods. Nearly all the corn and soybeans grown in the U.S. are genetically modified, but only two GM crops, Monsanto's MON810 maize and BASF's Amflora potato, are accepted in the European Union. Eight E.U. nations have banned GM crops outright. Throughout Asia, including in India and China, governments have yet to approve most GM crops, including an insect-resistant rice that produces higher yields with less pesticide. In Africa, where millions go hungry, several nations have refused to import GM foods in spite of their lower costs (the result of higher yields and a reduced need for water and pesticides). Kenya has banned them altogether amid widespread malnutrition. No country has definite plans to grow Golden Rice, a crop engineered to deliver more vitamin A than spinach (rice normally has no vitamin A), even though vitamin A deficiency causes more than one million deaths annually and half a million cases of irreversible blindness in the developing world.

Globally, only a tenth of the world's cropland includes GM plants. Four countries—the U.S., Canada, Brazil and Argentina—grow 90 percent of the planet's GM crops. Other Latin American countries are pushing away from the plants. And even in the U.S., voices decrying genetically modified foods are becoming louder. At press time, at least 20 states are considering GM-labeling bills.

The fear fueling all this activity has a long history. The public has been worried about the safety of GM foods since scientists at the University of Washington developed the first genetically modified tobacco plants in the 1970s. In the mid-1990s, when the first GM crops reached the market, Greenpeace, the Sierra Club, Ralph Nader, Prince Charles and a number of celebrity chefs took highly visible stands against them. Consumers in Europe became particularly alarmed: a survey conducted in 1997, for example, found that 69 percent of the Austrian public saw serious risks in GM foods, compared with only 14 percent of Americans.

In Europe, skepticism about GM foods has long been bundled with other concerns, such as a resentment of American agribusiness. Whatever it is based on, however, the European attitude reverberates across the world, influencing policy in countries where GM crops could have tremendous benefits. "In Africa, they don't care what us savages in America are doing," Zilberman says. "They look to Europe and see countries there rejecting GM, so they don't use it." Forces fighting genetic modification in Europe have rallied support for "the precautionary principle," which holds that given the kind of catastrophe that would emerge from loosing a toxic, invasive GM crop on the world, GM efforts should be shut down until the technology is proved absolutely safe.

But as medical researchers know, nothing can really be "proved safe." One can only fail to turn up significant risk after trying hard to find it—as is the case with GM crops.

David H. Freedman has been covering science, business and technology for 30 years. His most recent book, *Wrong*, explores the forces that cause scientists and other experts to mislead us.

IN BRIEF

The vast majority of the research on genetically modified (GM) crops suggests that they are safe to eat and that they have the potential to feed millions of people worldwide who currently go hungry. **Yet not all criticisms** of GM are so easily rejected, and pro-GM scientists are often dismissive and even unscientific in their rejection of the counterevidence. **A careful analysis** of the risks and benefits argues for expanded deployment *and* safety testing of GM crops.

How to Build a Better Plant

Genetic modification and conventional plant breeding have much in common: both are ways of producing new crops by altering plant genomes. The differences have to do with the tools used, the number of genes swapped and the comfort level of the average consumer.

Conventional Crossbreeding

Since the earliest days of agriculture, humans have been selecting plants with desirable characteristics and crossbreeding them to make new, even more desirable specimens—in this case, a tomato plant that combines the blight resistance of one parent with the high fruit yield of another.

Genetic Modification

In the 1970s scientists began developing methods for quickly inserting specific genes from one plant into the DNA of another—a quick, targeted means to the same end as conventional crossbreeding. Early researchers used disarmed *Agrobacterium* to smuggle genes into plant cells. Later, scientists at Cornell University developed the DNA particle gun method, in which engineers shoot DNA-coated tungsten balls through the cell walls of the target plant by firing them out of a blank gunpowder cartridge.

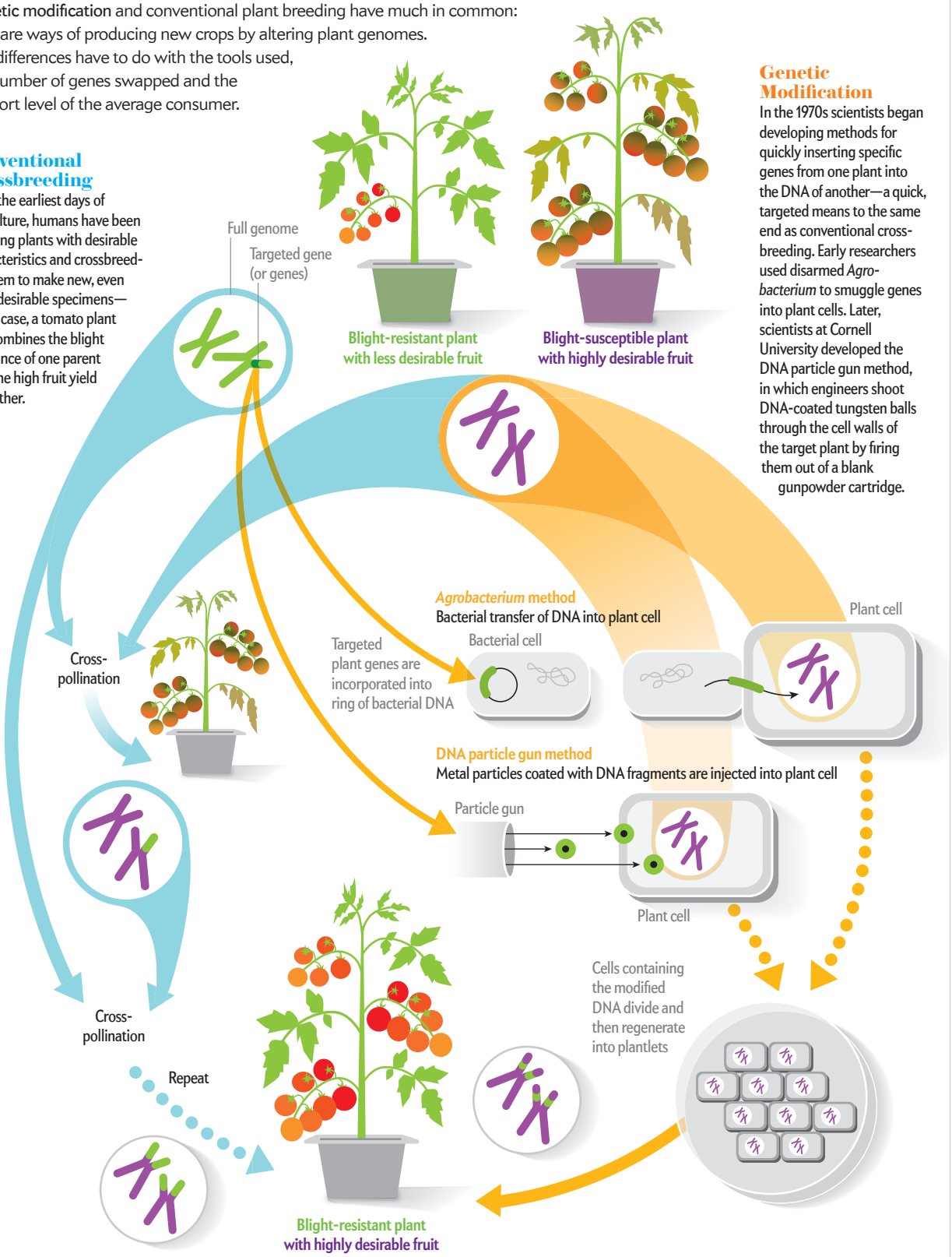


Illustration by Jen Christiansen

A CLEAN RECORD

THE HUMAN RACE has been selectively breeding crops, thus altering plants' genomes, for millennia. Ordinary wheat has long been strictly a human-engineered plant; it could not exist outside of farms, because its seeds do not scatter. For some 60 years scientists have been using "mutagenic" techniques to scramble the DNA of plants with radiation and chemicals, creating strains of wheat, rice, peanuts and pears that have become agricultural mainstays. The practice has inspired little objection from scientists or the public and has caused no known health problems.

The difference is that selective breeding or mutagenic techniques tend to result in large swaths of genes being swapped or altered. GM technology, in contrast, enables scientists to insert into a plant's genome a single gene (or a few of them) from another species of plant or even from a bacterium, virus or animal. Supporters argue that this precision makes the technology much less likely to produce surprises. Most plant molecular biologists also say that in the highly unlikely case that an unexpected health threat emerged from a new GM plant, scientists would quickly identify and eliminate it. "We know where the gene goes and can measure the activity of every single gene around it," Goldberg says. "We can show exactly which changes occur and which don't." [For more on how GM plants are analyzed for health safety, see "The Risks on the Table," by Karen Hopkin; SCIENTIFIC AMERICAN, April 2001.]

And although it might seem creepy to add virus DNA to a plant, doing so is, in fact, no big deal, proponents say. Viruses have been inserting their DNA into the genomes of crops, as well as humans and all other organisms, for millions of years. They often deliver the genes of other species while they are at it, which is why our own genome is loaded with genetic sequences that originated in viruses and nonhuman species. "When GM critics say that genes don't cross the species barrier in nature, that's just simple ignorance," says Alan McHughen, a plant molecular geneticist at U.C. Riverside. Pea aphids contain fungi genes. Triticale is a century-plus-old hybrid of wheat and rye found in some flours and breakfast cereals. Wheat itself, for that matter, is a cross-species hybrid. "Mother Nature does it all the time, and so do conventional plant breeders," McHughen says.

Could eating plants with altered genes allow new DNA to work its way into our own? It is theoretically possible but hugely improbable. Scientists have never found genetic material that could survive a trip through the human gut and make it into cells. Besides, we are routinely exposed to—we even consume—the viruses and bacteria whose genes end up in GM foods. The bacterium *B. thuringiensis,* for example, which produces proteins fatal to insects, is sometimes enlisted as a natural pesticide in organic farming. "We've been eating this stuff for thousands of years," Goldberg says.

In any case, proponents say, people have consumed as many as trillions of meals containing genetically modified ingredients over the past few decades. Not a single verified case of illness has ever been attributed to the genetic alterations. Mark Lynas, a prominent anti-GM activist who last year publicly switched to strongly supporting the technology, has pointed out that every single news-making food disaster on record has been attributed to non-GM crops, such as the *Escherichia coli*–infected organic bean sprouts that killed 53 people in Europe in 2011.

Critics often disparage U.S. research on the safety of genetically modified foods, which is often funded or even conducted by GM companies, such as Monsanto. But much research on the subject comes from the European Commission, the administrative body of the E.U., which cannot be so easily dismissed as an industry tool. The European Commission has funded 130 research projects, carried out by more than 500 independent teams, on the safety of GM crops. None of those studies found any special risks from GM crops.

Plenty of other credible groups have arrived at the same conclusion. Gregory Jaffe, director of biotechnology at the Center for Science in the Public Interest, a science-based consumer-watchdog group in Washington, D.C., takes pains to note that the center has no official stance, pro or con, with regard to genetically modifying food plants. Yet Jaffe insists the scientific record is clear. "Current GM crops are safe to eat and can be grown safely in the environment," he says. The American Association for the Advancement of Science, the American Medical Association and the National Academy of Sciences have all unreservedly backed GM crops. The U.S. Food and Drug Administration, along with its counterparts in several other countries, has repeatedly reviewed large bodies of research and concluded that GM crops pose no unique health threats. Dozens of review studies carried out by academic researchers have backed that view.

Opponents of genetically modified foods point to a handful of studies indicating possible safety problems. But reviewers have dismantled almost all of those reports. For example, a 1998 study by plant biochemist Árpád Pusztai, then at the Rowett Institute in Scotland, found that rats fed a GM potato suffered from stunted growth and immune system–related changes. But the potato was not intended for human consumption—it was, in fact, designed to be toxic for research purposes. The Rowett Institute later deemed the experiment so sloppy that it refuted the findings and charged Pusztai with misconduct.

Similar stories abound. Most recently, a team led by Gilles-Éric Séralini, a researcher at the University of Caen Lower Normandy in France, found that rats eating a common type of GM corn contracted cancer at an alarmingly high rate. But Séralini has long been an anti-GM campaigner, and critics charged that in his study, he relied on a strain of rat that too easily develops tumors, did not use enough rats, did not include proper control groups and failed to report many details of the experiment, including how the analysis was performed. After a review, the European Food Safety Authority dismissed the study's findings. Several other European agencies came to the same conclusion. "If GM corn were that toxic, someone would have noticed by now," McHughen says. "Séralini has been refuted by everyone who has cared to comment."

Some scientists say the objections to GM food stem from politics rather than science—that they are motivated by an objection to large multinational corporations having enormous influence over the food supply; invoking risks from genetic modification just provides a convenient way of whipping up the masses against industrial agriculture. "This has nothing to do with science," Goldberg says. "It's about ideology." Former anti-GM activist Lynas agrees. He recently went as far as labeling the anti-GM crowd "explicitly an antiscience movement."

PERSISTENT DOUBTS

NOT ALL OBJECTIONS to genetically modified foods are so easily dismissed, however. Long-term health effects can be subtle and nearly impossible to link to specific changes in the environment. Scien-

tists have long believed that Alzheimer's disease and many cancers have environmental components, but few would argue we have identified all of them.

And opponents say that it is not true that the GM process is less likely to cause problems simply because fewer, more clearly identified genes are switched. David Schubert, an Alzheimer's researcher who heads the Cellular Neurobiology Laboratory at the Salk Institute for Biological Studies in La Jolla, Calif., asserts that a single, well-characterized gene can still settle in the target plant's genome in many different ways. "It can go in forward, backward, at different locations, in multiple copies, and they all do different things," he says. And as U.C.L.A.'s Williams notes, a genome often continues to change in the successive generations after the insertion, leaving it with a different arrangement than the one intended and initially tested. There is also the phenomenon of "insertional mutagenesis," Williams adds, in which the insertion of a gene ends up quieting the activity of nearby genes.

True, the number of genes affected in a GM plant most likely will be far, far smaller than in conventional breeding techniques. Yet opponents maintain that because the wholesale swapping or alteration of entire packages of genes is a natural process that has been happening in plants for half a billion years, it tends to produce few scary surprises today. Changing a single gene, on the other hand, might turn out to be a more subversive action, with unexpected ripple effects, including the production of new proteins that might be toxins or allergens.

Opponents also point out that the kinds of alterations caused by the insertion of genes from other species might be more impactful, more complex or more subtle than those caused by the intraspecies gene swapping of conventional breeding. And just because there is no evidence to date that genetic material from an altered crop can make it into the genome of people who eat it does not mean such a transfer will never happen—or that it has not already happened and we have yet to spot it. These changes might be difficult to catch; their impact on the production of proteins might not even turn up in testing. "You'd certainly find out if the result is that the plant doesn't grow very well," Williams says. "But will you find the change if it results in the production of proteins with long-term effects on the health of the people eating it?"

It is also true that many pro-GM scientists in the field are unduly harsh—even unscientific—in their treatment of critics. GM proponents sometimes lump every scientist who raises safety questions together with activists and discredited researchers. And even Séralini, the scientist behind the study that found high cancer rates for GM-fed rats, has his defenders. Most of them are nonscientists, or retired researchers from obscure institutions, or nonbiologist scientists, but the Salk Institute's Schubert also insists the study was unfairly dismissed. He says that as someone who runs drug-safety studies, he is well versed on what constitutes a good-quality animal toxicology study and that Séralini's makes the grade. He insists that the breed of rat in the study is commonly used in respected drug studies, typically in numbers no greater than in Séralini's study; that the methodology was standard; and that the details of the data analysis are irrelevant because the results were so striking.

Schubert joins Williams as one of a handful of biologists from respected institutions who are willing to sharply challenge the GM-foods-are-safe majority. Both charge that more scientists would speak up against genetic modification if doing so did not invariably lead to being excoriated in journals and the media. These attacks, they argue, are motivated by the fear that airing doubts could lead to less funding for the field. Says Williams: "Whether it's conscious or not, it's in their interest to promote this field, and they're not objective."

Both scientists say that after publishing comments in respected journals questioning the safety of GM foods, they became the victims of coordinated attacks on their reputations. Schubert even charges that researchers who turn up results that might raise safety questions avoid publishing their findings out of fear of repercussions. "If it doesn't come out the right way," he says, "you're going to get trashed."

There is evidence to support that charge. In 2009 *Nature* detailed the backlash to a reasonably solid study published in the *Proceedings of the National Academy of Sciences USA* by researchers from Loyola University Chicago and the University of Notre Dame. The paper showed that GM corn seemed to be finding its way from farms into nearby streams and that it might pose a risk to some insects there because, according to the researchers' lab studies, caddis flies appeared to suffer on diets of pollen from GM corn. Many scientists immediately attacked the study, some of them suggesting the researchers were sloppy to the point of misconduct.

A WAY FORWARD

THERE IS A MIDDLE GROUND in this debate. Many moderate voices call for continuing the distribution of GM foods while maintaining or even stepping up safety testing on new GM crops. They advocate keeping a close eye on the health and environmental impact of existing ones. But they do not single out GM crops for special scrutiny, the Center for Science in the Public Interest's Jaffe notes: *all* crops could use more testing. "We should be doing a better job with food oversight altogether," he says.

Even Schubert agrees. In spite of his concerns, he believes future GM crops can be introduced safely if testing is improved. "Ninety percent of the scientists I talk to assume that new GM plants are safety-tested the same way new drugs are by the FDA," he says. "They absolutely aren't, and they absolutely should be."

Stepped-up testing would pose a burden for GM researchers, and it could slow down the introduction of new crops. "Even under the current testing standards for GM crops, most conventionally bred crops wouldn't have made it to market," McHughen says. "What's going to happen if we become even more strict?"

That is a fair question. But with governments and consumers increasingly coming down against GM crops altogether, additional testing may be the compromise that enables the human race to benefit from those crops' significant advantages. SA

MORE TO EXPLORE

Food, Inc.: Mendel to Monsanto—The Promises and Perils of the Biotech Harvest. Peter Pringle. Simon & Schuster, 2003.

Tough Lessons from Golden Rice. Martin Enserink in *Science,* Vol. 320, pages 468–471; April 25, 2008.

Case Studies: A Hard Look at GM Crops. Natasha Gilbert in *Nature,* Vol. 497, pages 24–26; May 2, 2013. www.nature.com/news/case-studies-a-hard-look-at-gm-crops-1.12907

SCIENTIFIC AMERICAN ONLINE

Watch a video on how genetically modified crops are made at ScientificAmerican.com/sep2013/gmo

Recommended by Lee Billings

Giraffe Reflections

by Dale Peterson and Karl Ammann. University of California Press, 2013 ($39.95)

Because the spindly-legged creatures had camel-like faces atop impossibly long, leopard-print necks, Greeks in Ptolemaic Egypt called them "camelopards." They were known as "tsu-la" to the Tang Dynasty Chinese and as "zarafa" to Arabs in the Middle Ages. Today we call them "giraffes," but our timeless fascination with these majestic animals remains unchanged. Peterson, a nature writer, has teamed with Ammann, a wildlife photographer, to present the natural and cultural history of giraffes in this elegant and comprehensive volume. In a series of lushly visual essays, the authors delve into the evolution of giraffes' strange anatomy and the intricacies of their behavior, as well as their possible futures alongside humans. Marvelously—and despite the book's encyclopedic presentation—giraffes become even more mysterious by the tome's end than they were at its beginning.

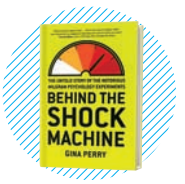
FROM *GIRAFFE REFLECTIONS*, UNIVERSITY OF CALIFORNIA PRESS, 2013

Countdown: Our Last, Best Hope for a Future on Earth?

by Alan Weisman. Little, Brown, 2013 ($28)

After penning his best seller *The World Without Us,* Weisman spent two years traveling the globe, investigating how we can survive in a world with entirely too many of us, set to brim with 10 billion humans later this century. The result is a frenzied barnstormer of a book. From Minneapolis to Mexico, from the Holy Land to Vatican City, Weisman presents the intermingled stories of the scientists, religious leaders and humble aid workers all striving for or against a sustainable human future. Ultimately, he finds few easy solutions. What emerges is a dismal picture of looming resource scarcities and rampant ecological destruction, brightened only by occasional success stories of countries and individuals mastering their fate. *Countdown* is a chaotic stew of big stories, bold ideas and conflicted characters, punctuated by moments of quiet grace—just like our people-packed planet.

Behind the Shock Machine: The Untold Story of the Notorious Milgram Psychology Experiments

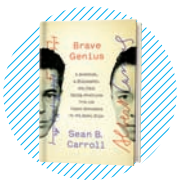


by Gina Perry. New Press, 2013 ($26.95)

Stanley Milgram's studies of human obedience to authority figures are arguably some of the most infamous psychology experiments ever. Until now, little has been written about how Milgram's subjects dealt with the experiments' aftermath. In *Behind the Shock Machine,* Perry, a professor at the University of Melbourne and a psychologist herself, tracks down some of the participants and explores how the study affected their lives. For many, the knowledge that they complied as they were urged by one of Milgram's actors to repeatedly "electrocute" a man in distress—a man who was actually only pretending to be in pain—turns out to be a terrible burden that even now elicits anger, confusion and self-doubt. Thanks to Perry's book, we gain more insight than ever before into Milgram's questionable practices and the scientific culture that allowed his experiment to take place.

—Arielle Duhaime-Ross

Brave Genius: A Scientist, a Philosopher, and Their Daring Adventures from the French Resistance to the Nobel Prize



by Sean B. Carroll. Crown, 2013 ($28)

Carroll, an evolutionary biologist, recounts the surprising tale of how two of France's most extraordinary 20th-century minds, biologist Jacques Monod and writer Albert Camus, each survived and rebelled against the Nazi occupation of France only to become close friends in the years leading up to their fame and receipt of Nobel Prizes. (Monod's Nobel was in medicine; Camus's was in literature.) Using a wealth of newly discovered letters and other documentation, Carroll beautifully encapsulates how two men seemingly so far apart in their philosophies and achievements both ended up sharing "exceptional lives" transformed by "exceptional events." *—Arielle Duhaime-Ross*

SCIENTIFIC AMERICAN ONLINE
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Michael Shermer is publisher of *Skeptic* magazine (www.skeptic.com). His book *The Believing Brain* is now out in paperback. Follow him on Twitter @michaelshermer

The Dangers of Keeping an Open Mind

Why great scientists make great mistakes

"Alien abductors have asked him to probe them." "Sasquatch has taken a photograph of him." The "him" is the "Most Interesting Man in the World," the faux character in the Dos Equis beer ad campaign, and these are my favorite skeptical lines from a litany of superfluities and braggadocios. ("In a past life, he was himself.")

My candidate for the most interesting scientist in history I'd like to have a beer with is Alfred Russel Wallace, the 19th-century naturalist and co-discoverer (with Charles Darwin) of natural selection, whose death centennial we will mark this November. As I document in my 2002 biography of him—*In Darwin's Shadow* (Oxford University Press)—Wallace was a grand synthesizer of biological data into a few core principles that revolutionized biogeography, zoology and evolutionary theory. He spent four years exploring the Amazon rain forest but lost most of his collections when his ship sank on his way home. His discovery of natural selection came during an eight-year expedition to the Malay Archipelago, where during a malaria-induced fever, it struck him that the best fit organisms are more likely to survive and reproduce.

Being open-minded enough to make great discoveries, however, can often lead scientists to make great blunders. Wallace, for example, was also a firm believer in phrenology, spiritualism and psychic phenomena, evidence for which he collected at séances over the objections of his more skeptical colleagues. Among them was Thomas Henry Huxley, who growled, "Better live a crossing-sweeper than die and be made to talk twaddle by a 'medium' hired at a guinea a séance."

Wallace's adventurous spirit led him to become ahead of his time in opposing eugenics and wasteful militarism and in defending women's rights and wildlife preservation. Yet he was on the wrong side when he led an antivaccination campaign. He was a first-class belletrist, but he fell for a scam over a "lost poem" that Edgar Allan Poe allegedly wrote to cover a hotel bill in California. Worst of all, he scientifically departed from Darwin over the evolution of the human brain, which Wallace could not conceive as being the product of natural selection alone (because other primates succeed with much smaller brains) and thus must have been designed by a higher power. Darwin snarled, "I hope you have not murdered too completely your own and my child."

Wallace is the prototype of what I call a "heretic scientist," someone whose mind is porous enough to let in both revolutionary and ridiculous ideas at the same time. Other such examples abound in astrophysicist Mario Livio's 2013 book, *Brilliant Blunders* (Simon & Schuster), in which he skillfully narrates the principle that "not only is the road to triumph paved with blunders, but the bigger the prize, the bigger the potential blunder." Livio's list includes Darwin's stumble in postulating the incorrect theory of pangenesis, based on the inheritance of particles he called gemmules that carried traits from parents to offspring; Lord Kelvin's gaffe of underestimating the age of the earth by almost 50 times, not because he ignored radioactivity, Livio argues, but because he dismissed the possibility of heat-transport mechanisms such as convection; Linus Pauling's misstep in building a DNA model as a triple helix inside out (because he rushed his research in the race against Francis Crick and James Watson); Fred Hoyle's bungle of siding with the steady state model of the universe over what he dismissively called the "big bang" model despite overwhelming evidence of the latter.

As for Albert Einstein's "biggest blunder" of adding a "cosmological constant" into his equations to account for the expanding universe, Livio claims Einstein never said it: instead Einstein applied the notion of "aesthetic simplicity" in his physical theories, which led him to reject the cosmological constant as an unnecessary complication to the equations.

How can we avoid such errors? Livio quotes Bertrand Russell: "Do not feel absolutely certain of anything." He then conveys a central principle of skepticism: "While doubt often comes across as a sign of weakness, it is also an effective defense mechanism, and it's an essential operating principle for science." SA

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Illustration by Ryan Inzana

The ongoing search for fundamental farces

Steve Mirsky has been writing the Anti Gravity column since a typical tectonic plate was about 34 inches from its current location. He also hosts the *Scientific American* podcast Science Talk.

Through a Glass, Obviously

Not every lawbreaker qualifies as a criminal mastermind

In a 2004 episode of *South Park,* the scamps think of themselves as warriors in an anime fantasy. Within that context, Cartman believes he has the power of invisibility—provided he removes all his clothing. He then tiptoes naked out of his constructed reality and into an auction before a large, shocked audience. His delusion is broken when the auctioneer says to him, "Kid, what the hell do you think you're doing?"

I recently discovered that a similar scene had played out in the real world two years earlier, when a man in Tehran hatched a really bad plan that sent him to the can. Like Cartman, our Iranian friend believed himself to be invisible. He was under the impression that he left no impression because he had paid about $500 to a local sorcerer, who in return provided him with spells to induce invisibility. That's according to the Iranian newspaper *Jam-e Jam,* which I'm appropriating as my hip-hop name. (The British newspaper *Metro* that picked up the story describes the charlatan as a "wizard imposter." Pro science tip: in this universe, all wizards are imposters.)

Our gullible friend entered a bank and confidently grabbed money from the customers, who, able to see him clearly, undoubtedly said the Farsi equivalent of "What the hell do you think you're doing?" before knocking him around a little.

I, too, have been convinced I was invisible, often while waiting in a line at an airport or bank, when people blithely walk directly in front of me. But I know that the real invisibility devices scientists have developed are too rudimentary to mask an entire human.

Sure, the Romulans and Klingons had cloaking devices that rendered their ships invisible. Harry Potter had an invisibility cloak that allowed him to vanish. But pretty much the best physicists can do right now is to sweep microwaves around a tiny object instead of letting the waves hit the thing and bounce back. The effect makes the object virtually invisible to any sensory equipment that detects only microwaves. The poor Iranian fellow did not even have one of these gizmos, you know, for moral support.

Such accounts of incompetent criminals have always intrigued me. So when the story of the visible man included a link to another promising example of ineptitude, I naturally pursued the lead. And thus discovered the story of a burglar in Germany who basically gave the local crime scene investigators the day off.

The thief in question, a teenager after a computer, scrupulously left the scene free of fingerprints. He did, however, leave behind one entire fingertip, which he sliced off negotiating with a broken window. The *Metro* quoted a local law-enforcement official: "We usually find fingerprints at the crime scene, but it's not every day that thieves leave the original there, too."

That original matched a print on record, and police swiftly arrested the young man. Now, a really good lawyer might have gotten him acquitted by arguing that the print on record no longer matched the rather minimalist version that the teenager now possessed. But the kid saved everyone time and effort by confessing when confronted with his former body part, having been both figuratively and literally fingered by the police.

Just a few days before this issue went to press in early July came a wonderful example of something dumb and possibly criminal, in terms of negligence. Turns out that a Russian rocket had its angular velocity sensors installed upside down. (Pro science tip: this is the opposite of good.) The technology news Web site Ars Technica reported that attempts to correct the flight trajectory based on sensor data (it's going down—make it go up!) actually turned the rocket back toward Earth, where it crashed 32 seconds after liftoff. The rocket was carrying satellites for the Russian GPS system, which does qualify as irony. An investigation is under way. Or possibly over way. SA

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Illustration by Matt Collins

50, 100 & 150 Years Ago compiled by Daniel C. Schlenoff

Innovation and discovery as chronicled in *Scientific American*

September 1963

Erich Fromm on Carl Jung

"Book Review: *Memories, Dreams, Reflections,* by Carl G. Jung. Pantheon Books ($7.50). Jung's life from childhood on was dominated by the quest for certainty. Was God real? Was he, Jung, real? Was evil real? Eventually he believed he had found an answer in the concept that his visions, dreams and fantasies were all manifestations of the unconscious and that he was the first to have discovered this ultimate reality, to have submitted to it in full awareness and so tamed it. His autobiography is illuminating and impressive. It would arouse deep compassion, at least in this reader, if it were not for the fact that Jung combined an incapacity to see the truth with such a degree of opportunism that as a tragic hero he often resembles the Pied Piper of Hamelin. —Erich Fromm"

September 1913

Disaster and Safety

"In the recent wreck on the New Haven Railroad, the heavy colliding engine and train split entirely apart the two rear wooden sleeping cars of the train ahead, scattering the wreckage and the helpless passengers to right and left as it crushed its way through. Over a score of people were killed outright. There is abundance of evidence, drawn from the behavior of steel cars under conditions practically as severe as these, to show that steel construction would have saved the lives of many, if not the greater part, of the occupants of these two rear cars."

To see a photograph album relating to disasters and safety from our archives of 1913, visit www.ScientificAmerican.com/sep2013/disaster-safety

HYDRAULIC LIFT LOCK: This image graced the cover of the September 6, 1913, issue, which featured articles on several canals. The lock shown here, at Peterborough on the Trent-Severn Waterway in Canada, carries barges 65 feet straight up and down. It is still in use today.

Igor Sikorsky, Flight Pioneer

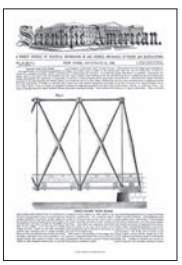
"The St. Petersburg correspondent of the Parisian sporting journal *Aero* telegraphs to his paper that Igor Sikorsky, a student at the technical high school of St. Petersburg, has built what is probably the biggest aeroplane which has thus far appeared. The span of the biplane is 27 meters. It is said that the machine actually made a flight with seven passengers of 90 kilometers lasting not quite two hours at an altitude of 500 meters, during which the pilots took turns in the pilot house and passengers walked about as if they were in a city apartment. Naturally these accounts of the machine's performances are received with considerable incredulity in France."

Sikorsky's Russky Vityaz *("Russian Knight") of 1913 was the world's first four-engine airplane. It was destroyed in a freak accident later that year.*

SCIENTIFIC AMERICAN, VOL. CIX, NO. 10, SEPTEMBER 6, 1913

Artificial Kidney

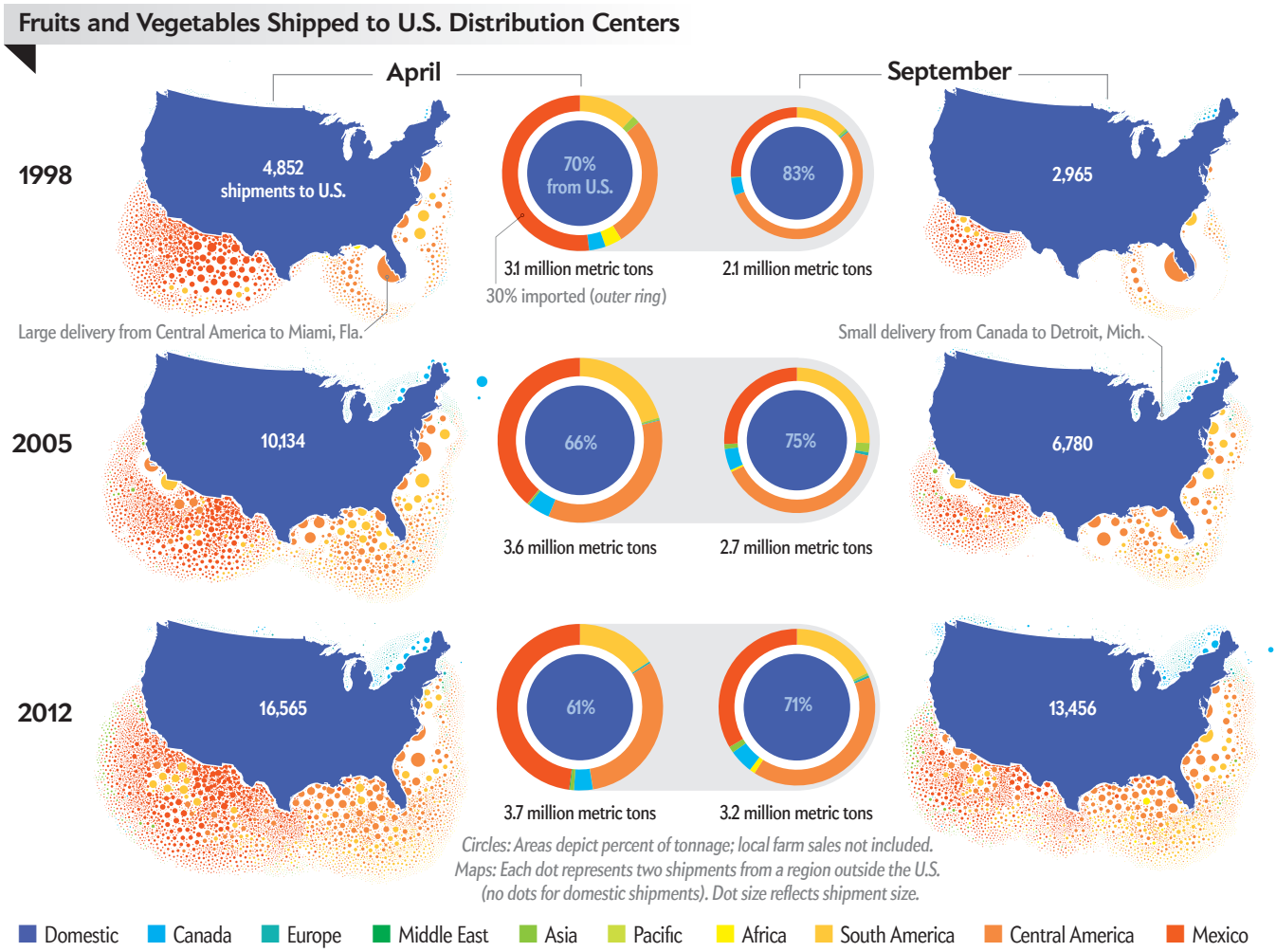
"The International Congress of Medicine, recently held in London, has this report from the London *Times:* 'A demonstration which excited great interest was that of Prof. [John Jacob] Abel of Baltimore. Prof. Abel presented a new and ingenious method of removing substances from the circulating blood, which can hardly fail to be of benefit in the study of some of the most complex problems. By means of a glass tube tied into the main artery of an anesthetized animal the blood is conducted through numerous celloidin tubes before being returned to the veins through a second glass tube. All diffusible substances circulating in the blood pass through the intervening layer of celloidin. In this way Prof. Abel has constructed what is practically an artificial kidney.' "



September 1863

Machine Breaking

"If Satan, in his hatred of mankind, should set himself to devise the best mode of lowering the rate of wages, he could find no plan more effectual than that of inducing mobs to destroy labor-saving machinery. Wealth is being constantly produced by labor, and the amount produced is in proportion to the quality and supply of the tools and machinery that the laborers have to work with. A man can produce something with his naked hands, more with the aid of an axe or hoe, more still with a horse and plow, and still more with a steam engine, or sawmill. When wealth is produced, it is divided between the laborer who does the work, and the man who owns the tools or machinery. The price of labor in England and the United States has multiplied several fold since the invention of the steam engine, the spinning jenny, the cotton gin and the power loom."

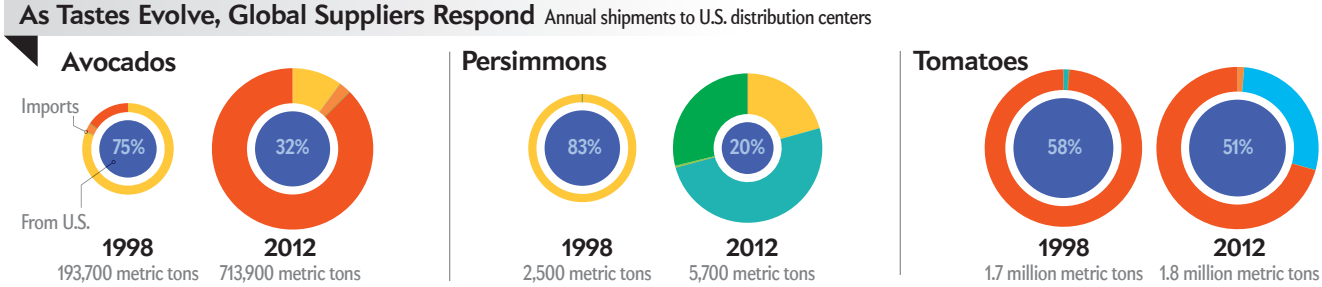


The 5,000-Mile Salad

Lettuce from Spain, avocados from Mexico, pomegranates from Israel—all arrive for your dining pleasure

Americans want fresh fruits and vegetables—year-round, not just at harvest time. U.S. farmers grow a lot of produce, but imports are meeting most of the increased demand, especially during off-season months such as April, thus capturing more of the total consumption (*six circles above*). Mexico, Central America and South America send the most produce to U.S. regional distribution centers (*maps above*). Some fruits and vegetables have recently become very popular, whereas others remain stable (*circles below*): avocados are way up, persimmons are on the rise and tomatoes are, well, tomatoes. —*Mark Fischetti*

SCIENTIFIC AMERICAN ONLINE For an interactive graphic showing shipments of specific fruits and vegetables, see ScientificAmerican.com/sep2013/graphic-science



Graphic by The Office for Creative Research