http://bbc.in/1GnZajy

Concern over hidden diesel pollutant

Atmospheric levels of a little known by-product from diesel engines are up 70 times higher than expected according to a study.

By Matt McGrath Environment correspondent, BBC News

Researchers found that long-chain hydrocarbons are significantly under-reported in car manufacturers' data. These hydrocarbons are a key component of two of the worst air pollutants, ozone and particulate matter. The authors believe these "hidden" emissions are having a large impact on air quality in cities like London.

The exhaust pipes of diesel-fuelled trucks and cars produce an array of emissions that have different impacts on the air that people breathe.

Complicated mixture

The nitrogen dioxide and particles that are emitted from burning diesel have a direct impact on human health in cities. But diesel also contains more complex, long-chain hydrocarbons, whose role in air pollution has been little understood until now. They can form dangerous air pollutants, especially ozone and particulate matter, which are emitted into the air as unburned fuel or diesel vapour.

Researchers from the University of York have been able to detect these complicated compounds in the London air, using sophisticated measuring technology.

"It's definitely been hidden until now," lead author Dr Jacqueline Hamilton told BBC News. "What we found is that there's actually a lot of this unburned material from diesel that we haven't seen before."

"That might be having a bigger impact on ozone and particle formation than petrol cars are, and historically no one has looked at these emissions at all."

The researchers found that close to 50% of the ozone production potential in London in winter was due to these diesel elements. In summertime, it was around 25%. The authors believe that these hydrocarbons are having a direct effect on health.

"I think it is having a large impact on air quality in our cities, the number of deaths associated with particle pollution are much higher than those from nitrogen dioxide, this is a route to increase particle pollution so it could have a major impact on human health."

The study also found that the scale of these hydrocarbons in the air was far in excess of the levels expected by government, which are based on data from car manufacturers' emissions tests. For some types of these diesel emissions, the real world samples were over 70 times greater during winter compared to the regulatory inventories.

The authors say these emissions are "massively under-predicted" but they are uncertain if this is a deliberate attempt by car manufacturers to conceal the scale of the problem or simply an omission through ignorance.

"Volkswagen have admitted they have deliberately turned off the emissions controls, and if these controls lower emissions of hydrocarbons, if you just turn that off, you are definitely gaming the system," said Dr Hamilton.

"If you look in the real atmosphere, compared to the test data, there's a lot of NO_x and a lot of these hydrocarbons as well."

Other researchers praised the study as a step forward in scientific understanding.

"It is science that has come up with this discovery, but it definitely has public policy implications," said Prof Paul Monks, from the University of Leicester, who is also the chair of the UK government's air quality expert group.

"It raises yet another question about diesel vehicles. They are implicated heavily in NO_2, they are implicated in toxic particulate matter, and this points to another deleterious environmental impact from diesels."

The authors are calling for a review of the way emissions from car and trucks are measured, with much more emphasis on real world testing and increased testing for a wider spectrum of substances coming out of the tail pipe. "We have a policy in the UK to look at these sorts of hydrocarbons from petrol cars, but we really have to start thinking more seriously about measuring these from diesel cars."

The research has been published in the journal Atmospheric Chemistry and Physics.

http://bit.ly/1Ocv1M4

Physics of falling says professional athletes are running wrong

Runners may be doing it all wrong. A slightly different posture could let runners and walkers get a gravity-driven boost – and potentially break world records.

To most runners and coaches, running is a series of jumps, says Svein Otto Kanstad, a physicist and former competitive runner based in Volda, Norway. Gravity isn't considered helpful, because its force is perpendicular to the direction a runner is moving. But this mindset neglects the concept of angular momentum, Kanstad says. Rather than thinking of running as a series of jumps – leaping off one foot and landing again on the other – runners should view their sport as a series of falls, aided by gravity, he says.

"We are falling forward, and our legs catch us," he says. With each footfall a runner's body actually rotates forward, pivoting on the foot in contact with the ground. "It is not a series of jumps, it is a series of rotations."

A hula hoop illustrates how this rotation provides angular momentum. If you simply throw a hula hoop vertically into the air, it will fall flat when it lands. But

if you spin the hoop as you launch it, it will roll away after it hits the ground because it has angular momentum.

"We are clever at using angular momentum without really knowing that's what we're using," he says. But for many runners there is room for improvement.

Best foot forward

As a runner's hips rotate to bring each leg forward, he or she gains angular momentum. But most runners don't make the best use of this. At the moment their leading leg hits the ground, the second leg is usually stretched out behind. In Kanstad's revised gait, the second leg will already have rotated forward again before the leading leg hits the ground. By doing this, the runner's centre of mass is tilted far forward allowing for more forward momentum, but the recovery leg is there to stop a fall.

It's tricky to do, but Kanstad has taught himself to run this way. He says that retired US sprinter Michael Johnson – who has held the world record in the men's 400 metres since 1999 – uses the same technique.

"The arms become very important as a counterbalance to the leg movement," says Kanstad. "You have to change to almost opposite the way you are used to using your arms and legs."

He trained distance runners from Tromsø to run using this technique on treadmills, while using straps that anchored them to the ceiling in case they fell. In one test, a male sprinter running at 14 kilometres per hour was making an energy saving of 10 per cent compared with his usual gait, as measured by his consumed oxygen volume per minute. As he ran, he shouted, "I'm flying!" Kanstad says.

Kanstad believes training distance runners and sprinters to run in this fashion would shave minutes off race times, resulting in a rash of new records.

"Gravity is there, and it drives us forward, but we immediately kill it by the way we run," he says. "Just by not being that killer, you can have 10 per cent more energy for free."

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http://bit.ly/1OcvZrH

Searching for Life in Martian Water Will Be Very, Very Tricky

The risk of microbial contamination could prevent humans and even robots from visiting the most promising parts of the Red Planet

By Lee Billings

NASA scientists announced today the best evidence yet that Mars, once thought dry, sterile and dead, may yet have life in it: Liquid water still flows on at least some parts of the Red Planet, seeping from slopes to accumulate in what might be life-nurturing pools at the bases of equatorial hills and craters. These remarkable sites on Mars may be the best locations in the solar system to search for extant extraterrestrial life—but doing so will be far from easy.

Examining potentially habitable regions of Mars for signs of life is arguably the primary scientific justification for sending humans there—but according to a new joint review from the National Academy of Sciences and the European Science Foundation, we are not presently prepared to do so.

The problem is not exploding rockets, shrinking budgets, political gamesmanship or fickle public support—all the usual explanations spaceflight advocates offer for the generations-spanning lapse in human voyages anywhere beyond low Earth orbit. Rather, the problem is life itself—specifically, the tenacity of Earthly microbes, and the potential fragility of Martian ones. The easiest way to find life on Mars, it turns out, may be to import bacteria from Cape Canaveral—contamination that could sabotage the search for native Martians. The need to protect any possible Martian biosphere from Earthly contamination, the review's authors wrote, could "prevent humans from landing in or entering areas" where Martian life might thrive. Although this sentiment is not new, its frank, formal acknowledgement in such an authoritative study is rare indeed. NASA is planning to send humans to Mars as soon as the 2030s; that such missions may unavoidably pose extreme contamination risks is understandably not something the agency is eager to highlight, even as it actively researches possible solutions to the problem.

Historically, in the context of Mars such "planetary protection" has primarily concerned robotic exploration. The risk of contamination is an issue even for machines, which, unlike humans, can endure being fried with radiation and bathed in harsh chemicals prelaunch to eradicate bacterial stowaways. Microbes that stubbornly refuse to die nonetheless turn up with regularity in NASA's supposedly sterile clean rooms for preparing interplanetary spacecraft. Apollo astronauts even found bacteria on the moon that had survived an almost total vacuum inside the robotic Surveyor 3 lander that had touched down more than two and a half years earlier. If terrestrial microbes could live in places like that, why not in some of the more habitable parts of Mars?

The United Nations Outer Space Treaty of 1967 forbids the "harmful contamination" of other worlds with Earth's biology, and an international organization called COSPAR (the Committee on Space Research) sets the planetary protection protocols for the U.S., Europe, Russia and other signatory spacefaring nations to follow. To protect Mars, since 2002 COSPAR has designated restricted "Special Regions" on the planet where conditions are warm and wet enough to possibly support extant Martian life—or to allow Earthly invaders to gain a flagella-hold. Because of rapid, ongoing progress in our knowledge of the Martian environment and the fundamental limits of Earthly

biology, the precise definitions for Special Regions remain works in progress that are officially revisited every two years. The new joint review, released last week, recommends revisions to the findings of a 2014 report on COSPAR's Special Regions from NASA's Mars Exploration Program Analysis Group.

The closer planetary scientists look at Mars, the more Special Regions they think they see. Special Regions pepper the planet's equator and mid-latitudes, in eroded gullies and in steep, rocky slopes of hills and craters, where new evidence published September 28 in Nature Geoscience indicates that briny water flows and pools from aquifers during Martian summers. Special Regions can also be found in caves, beneath the polar ice caps and in geothermal hotspots of seismic or volcanic activity. As little as five meters below the surface, where groundwater may persist as ice, vast areas of the planet could be considered a Special Region, just waiting to be transformed into a welcoming, watery microbial Eden by the heat from a new-formed impact crater or the operations of a recently arrived spacecraft. Special Regions should also exist, the new review notes, at the still-unknown sources of mysterious methane emissions recently detected on Mars. On Earth it is generated chiefly by microbes but detectable quantities of the gas could also arise on Mars from abiotic sources, although those lifeless production routes would also require liquid water.

But knowing for certain whether any of these places are actually special probably requires visiting them—something that is very difficult to do under current protocols. Before a spacecraft can visit a Special Region it must in part or in whole be stringently sterilized according to strict rules, potentially adding years of development time and many millions of dollars onto a mission's bottom line. Even then, the protocols may not be strict enough—current techniques are incapable of entirely cleansing a spacecraft of microbes, and no one really knows the threshold conditions for bacteria to create viable, self-sustaining colonies on Mars—or on Earth, for that matter.

The agency's first—and to date only—missions to Mars explicitly in search of life were the twin Viking landers, which landed on the Red Planet in 1976. All others since have focused on finding signs of life from Mars' ancient past rather than its present. If even sterilized robots cannot be trusted to venture into Special Regions, what about microbe-riddled humans? If astronauts shall only be allowed to visit subpar locales to search for life on Mars, can NASA or any other entity justify the tens to hundreds of billions of dollars required to send them there? If a human crew lands in an area thought unpromising for biology but discovers habitable conditions or something living there, would they have to immediately relocate, or even pack up their rocket and launch back to orbit? These and other unanswered questions show how in many ways, discovering a present-day Martian biosphere could be both the realization of NASA's wildest dream and its worst nightmare. They explain as nothing else can the otherwise inexplicable fact that in the quest for extant life on Mars NASA has been judiciously avoiding the very places where it may most likely be found.

Carl Sagan famously mused that if life is ever found on the fourth rock from the sun, "Mars then belongs to the Martians, even if the Martians are only microbes." In this view the planet would become a sacrosanct sanctuary, forever off-limits to encroaching humans. An alternate perspective holds that planetary protection efforts are futile, perhaps even naive: Thanks to likely contamination from earlier spacecraft, as well as ancient exchanges of material blasted between the planets by massive asteroid impacts, Mars has probably already experienced many waves of Earthly invaders—each of which could have been easily repulsed by any native, more adaptively fit biosphere.

Amid all the uncertainty, the new review notes, one thing is very clear: "The planetary protection implications of sending astronauts to Mars raises profound questions at the intersection of science, engineering, technology, project management and public policy." The statement's true meaning for NASA and other space agencies should be equally clear: Although inconvenient, the planetary protection issues associated with crewed missions to Mars are too severe to be dismissed, dodged or downplayed. Now is the time to begin addressing them. Otherwise, human voyages there may at best prove to be nonstarters and at worst become fiascos that forever extinguish hopes of studying pristine examples of Martian life.

http://bit.ly/1j5eb53

Millions of Americans Are Getting Lost in Translation During Hospital Visits

Rampant miscommunication in medicine due to language barriers compromises patient safety and quality of care while widening existing health disparities.

By Adam Hoffman

The two-year-old Latina girl arrived at a Massachusetts emergency room in 1999 with intense shoulder pain. "Se pegó, se pegó," her Spanish-speaking mother cried. To the attending resident, the phrase sounded like "she was hit," as in, she was struck by another person. X-rays revealed a fractured collarbone. Then the physician saw discharge papers from a previous hospital admission, which indicated the girl had broken her collarbone just two months earlier. Suspecting child abuse, the hospital contacted the Department of Social Services (DSS).

After questioning the family without an interpreter, the DSS caseworker concluded that the child was not safe at home. The little girl and her four-year-old brother were taken from their mother on the spot and placed in DSS custody. Two

hours later, the team interviewed the mother with the help of a trained Spanish interpreter and discovered that the child fell off her tricycle and accidentally struck her shoulder. After several days of red tape, the mother regained custody of her children.

This young girl's story is just one example of a growing problem across the United States, as the national health care system has struggled to adapt to the growing number of people who do not speak English as their primary language. According to U.S. Census data released earlier this month, over 63 million Americans speak a language other than English at home, and over 25 million self-identify as having limited English proficiency.

Rampant miscommunication compromises patient safety and quality of care while widening existing health disparities. Some technological solutions are on the rise, from videoconferencing sessions with interpreters to smartphone applications that act as digital translators, but these innovations have a ways to go before they can stand in for medically trained in-person aid.

“Good communication is essential for every medical encounter, whether you are talking about a visit for a rash or someone who is in the ICU,” says Glenn Flores, the distinguished chair of health policy research at the Medica Research Institute in Minneapolis.

“We know from extensive literature that language barriers affect access to care, health status, use of health services, patient/physician communication, satisfaction with care, quality and safety—it really spans the spectrum in terms of the impact,” he says.

Unbeknownst to many patients and physicians, individuals with limited English proficiency have been guaranteed language services under federal law for decades. Title VI of the Civil Rights Act of 1964 prevents discrimination based on race, color, religion, sex or national origin by any organization receiving federal funding.

And in Lau v. Nicols (1974), the Supreme Court set the precedent that language can be used as a proxy for national origin, specifically saying that schoolchildren who do not speak English as a first language must be given equal educational opportunities.

Because virtually all healthcare providers accept Medicare, Medicaid or some other form of federal funding, the rulings imply that providers cannot discriminate based on language and must supply an interpreter for limited English proficiency patients. These rights were reaffirmed in 2000, when President Bill Clinton issued an executive order that reiterated the requirements of Title VI and outlined expectations for healthcare providers.

“If you have someone who is limited English proficient who comes in for services, you need to ensure that they have meaningful access to your programs,” says Mara Youdelman, managing attorney at the National Health Law Program in Washington, D.C. “You can’t turn them away because they don’t speak English. You can’t say, 'Come back next Wednesday when my bilingual staff person is here.' You can’t make them bring their own interpreters. These patients should have the same access as an English speaking patient does.”

The trouble is that Title VI did not come with associated funding. “There is no requirement that either the federal government or the state pay for the language services in the providers' offices,” says Youdelman.

Only 13 states and Washington, D.C. have elected to specifically reimburse the costs of medical interpreters through Medicaid. The remaining states—including those with the largest non-English speaking populations, such as California and Florida—argue that the costs of language services are factored into existing reimbursement rates.

As a result, providers who are responsible for a higher percentage of the limited English proficiency population are forced to bear the costs of supplying interpreters on their own, which cuts into operating costs and puts the communities they serve at a disadvantage, Youdelman adds.

Meanwhile, Medicare and many private insurers refuse to pay for interpreters, despite the efforts of many policymakers to get Medicare reimbursement in the Affordable Care Act.

That was not the first time language issues had failed to get priority in health care policy. In 2000, the groundbreaking report “To Err is Human” highlighted many patient safety issues resulting from physician errors. But it failed to include language barriers as a significant threat to patient safety, despite the thousands of language-related cases that have been filed with the Department of Health and Human Services.

Without loud and clear announcements of the law, many health care providers remain unaware of their responsibilities, and enforcement of Title VI has been difficult.

“The way this is currently enforced is through administrative complaints,” says Youdelman. “So if a patient thinks that he or she was discriminated against, they can file a complaint with the Office for Civil Rights at the Department of Health and Human Services.”

But many of these individuals are likely to be unaware of their rights, or they might erroneously think that filing a complaint could affect their immigration status, says Youdelman. As a result, many remain silent.

If an incident is reported and the provider is found to be intentionally or unintentionally discriminating against someone, the consequences are rather feeble. Generally, the provider and the Office for Civil Rights simply come to an agreement as to what processes need to be fixed and what policies need to be implemented. In theory, the government could punish offenders by withdrawing federal funding, but that has never happened.

“There are two ways to get healthcare providers to follow the mandates,” says Francesca Gany, director of the Center for Immigrant Health and Cancer Disparities at Memorial Sloan Kettering Cancer Center. “One is to provide incentives to adhere, and the other is punishment if they don’t. And neither of those, the carrot or the stick, have seen much attention.”

Even in hospitals that have implemented language interpretation programs, many doctors elect to use their own skills or an ad hoc interpreter to save time. “Doctors often don’t call interpreters when they need to,” says Gany. “Given the time constraints that providers are under, if it takes one extra iota of time to use an interpreter, they will try and get by with their own rudimentary language skills.”

Being bilingual only gets you so far, says Youdelman. “Not many people who had high school or college language training or studied abroad would be able to translate specialized medical terminology like describing cancer treatment options. So there is definitely an overconfidence many providers have about their language skills.”

Part of the problem comes from a culture in medicine that says doctors should always have the answers, notes Wilma Alvarado-Little, a medical interpreter and former co-chair of the Board of the National Council on Interpreting in Health Care. “When physicians are constantly being put in situations where they need to know, saying 'I don't know' really isn’t the ideal response,” she says.

To assess physician language skills, Alvarado-Little often asks a series of pertinent questions: Who can respond to basic commands, who can navigate, who can joke in the language?

“But the last question, if they feel they are at the level that they can interpret, is ‘Do you feel your language skill can hold up in a court of law?’” she says. “Many people don’t realize that interpreters become part of the medical chart, which is a legal document. And so the communication has to be spot on.”

Having interpreters who are trained specifically for clinical settings is extremely important. In 2012, Flores led a study in emergency departments investigating the use of professional interpreters, untrained ad hoc interpreters or no interpreters. The study found that the use of trained interpreters resulted in 10 percent fewer errors with potential medical consequences than using untrained interpreters, and that using untrained interpreters could be just as dangerous as using no interpreters.

“When limited English proficiency patients do not have professional medical interpreters or bilingual providers available, they have to resort to the use of ad hoc interpreters, which are family members, friends, people from the waiting room or strangers pulled from the street,” says Flores. This can introduce a host of biases, such as when a family member withholds information to try and protect a loved one, or when a speaker uses slang or idioms unique to their country.

Such errors can lead to misdiagnoses, unnecessary tests and misinformed treatments that put a patient's health at risk.

In another high-profile case, a Florida teenager felt unwell while attending a high school sporting event. Before collapsing, he told his girlfriend, “Me siento intoxicado.” When the paramedics came, the girlfriend, who spoke limited English, repeated intoxicado, which the paramedics, who spoke minimal Spanish, interpreted as “intoxicated.”

They brought the teenager to the emergency room, where he was treated for drug abuse. But after the boy spent 48 hours in a coma, the hospital staff ordered a CT scan, which revealed that the teenager’s head had flooded with blood. It turns out that feeling intoxicado can also mean “sick to the stomach,” which is a symptom of a brain aneurysm. This communication breakdown led to a $71-million-dollar malpractice lawsuit.

So what can be done? Many experts believe that every aspect of the health care process—from initial appointment bookings to treatment protocols—needs to be reappraised to accommodate the language needs of the local population.

For example, a survey of pharmacies revealed that only half of them were able to print their prescriptions in a language other than English, while another study showed that limited English proficiency families were fundamentally unable to use hospital signage to navigate from the parking lot to the emergency department.

Advocates are calling for hospitals and other health care providers to begin routinely collecting data on the primary languages spoken by their patients and whether they have limited English proficiency, so that providers can be prepared with appropriate language services.

Hospitals could also screen doctors and nurses for non-English language skills to determine whether they are qualified to use those abilities in clinical interactions, and they should provide pay raises for suitably bilingual clinicians. “It is important to change the culture of the institution so that it is no longer OK for care providers to get by with rudimentary language skills,” adds Gany.

In the meantime, hospitals are beginning to use a variety of cost-effective technologies that can serve as alternatives to in-person interpretation.

"The technology is out there to connect well-trained interpreters with doctors, even if they are not in the same room," says Gany. Many companies offer phone interpretation services, where you can pay for remote access to speakers of hundreds of languages.

In one popular option called remote simultaneous medical interpreting, the clinician and patient each use a headset that is connected to an interpreter at a remote location. This approach, modeled after the UN interpreting system, allows for fast, reliable communication in a variety of languages.

"More and more hospitals are starting to use these services. It is better than it used to be, but it is still not nearly enough," says Gany.

Phone interpreters are sometimes limited because they cannot see non-verbal cues, so some care providers have also begun to incorporate videoconferencing with interpreters via tablets, laptops and smartphones—although these services can be expensive.

Other companies have engineered smartphone translation and interpretation applications that are specialized in common health care phrases and nomenclature. But such technologies are not perfect, and many physicians remain skeptical.

"Google Translate, Canopy and some of those phone apps are really dangerous, and they even have a disclaimer that they should not be used for safety-critical tasks," says Flores. For instance, Google Translate says that me siento intoxicado means "I feel intoxicated" and so would not have been much help to the paramedics in the Florida case.

Flores believes that a smartphone application could be developed that adequately serves as a reliable interpreter, but this is a long way off. He would instead prefer to see basic—and affordable—change come from state policy makers and hospital executives.

A 2002 report from the Office of Management and Budget found that it would cost an additional $4.04 per visit to provide all limited English proficiency patients in the U.S. with the appropriate language services. And states could be reimbursed for over 50 percent of these Medicaid costs through the Federal Medical Assistance Percentages program.

Without such actions, though, millions of Americans will remain lost in translation.

"I have seen what happens before and after we have implemented interpreter services," says Gany. "Patients are so grateful that they jump up and give you a hug, because it is the first time that they have felt understood in a doctor's office. And doctors have shared with me that it was the first time that they were able to diagnose depression in a patient or find out about their past history. It makes a huge difference."

http://bit.ly/1N8oOAD

NASA Unveils Giant Ice Cube With Wheels for Exploring Alien Oceans

An underwater rover might one day explore otherworldly seas

By Danny Lewis

From Europa to Enceladus, scientists have long agreed that the watery moons orbiting Jupiter and Saturn might be the best places to find life elsewhere in the solar system.

NASA has wrestled with figuring out how best to survey these moons, but thanks to a new prototype probe, researchers might soon gather information from rovers trundling about these extraterrestrial oceans.

Most rovers designed to explore other planets look kind of like cars, with big fat tires and treads for scrambling over rocky surfaces. But a new rover takes the opposite tact: designed for moons like Europa, which is covered in a thick sheet of ice. This Buoyant Rover for Under-Ice Exploration (BRUIE) might one day trundle along beneath the ice as its predecessors travelled along Mars' surface, Becky Ferreira writes for Vice Motherboard.

"We thought, 'oh well, we'll just invert the surface,'" Dan Berisfold, an astrobiologist and member of BRUIE's design team says in a new video by NASA's Jet Propulsion Laboratory. "Instead of having a rover that drives on the ground, we'll have a rover that drives on the ceiling [...] which is the ice surface."

As its name indicates, BRUIE is designed to float just under the surface level of the water, propelling itself underneath the ice with a pair of wheels while maintaining buoyancy.

While early prototypes were controlled via cable, future versions of the rover will send data back to Earth through a remote receiver on the ocean's surface, James O'Malley writes for Gizmodo.

While there are no current plans to send BRUIE on a mission into space, it has been busy gathering data on Earth's own oceans as its developers test it out in the methane-rich waters near Barrow, Alaska.

"Our research up in the Arctic has this win-win," astrobiologist Kevin Hand says in the video. "By studying the methane that's trapped in these lakes and coming out of the permafrost, we're helping to quantify the greenhouse gas emissions that are affecting climate change, while simultaneously building a vehicle and a scientific platform that serves as a precursor for something that may some day fly to Europa or Enceladus or one of the other moons that harbors an ocean."

BRUIE might just be a prototype, but with NASA planning a Europa-bound exploratory mission for 2020, its descendants might very soon be exploring alien oceans.

http://nyti.ms/1FLvwKu

A New Effort Has Doctors Turn Patients Into Donors

Doctors are being taught to identify wealthy patients who might be prospective donors to raise money for the medical center or their own research

By GINA KOLATA SEPT. 28, 2015

A well-to-do cancer patient is nearing the end of her treatments. During an office visit, she says to her doctor, "I can't thank you enough for the care you provided."

Should the doctor simply accept the patient's gratitude — or gently suggest a way for her to show it: "Perhaps you might consider making a donation?"

More and more these days, development offices at major cancer centers are teaching doctors to seize such opportunities to raise money for the medical center or for their own research.

In an unprecedented survey of more than 400 oncologists at 40 leading cancer centers, nearly half said they had been taught to identify wealthy patients who might be prospective donors. A third had been asked to directly solicit donations — and half of them refused. Three percent had been promised payments if a patient donated.

The study, which was published online Monday in The Journal of Clinical Oncology, was conducted by Dr. Reshma Jagsi, a radiation oncologist and ethicist at the University of Michigan, who had grown concerned about the practice and wanted to know more.

Dr. Jagsi said she had sat in on workshops, seminars, training sessions and department meetings that discussed how to identify good prospects for gifts, how to direct grateful patients to the development office, and how to ask them directly if they wanted to donate.

She was uncomfortable with the idea, but she also knew some patients want to donate and are grateful for guidance on how to do it. And she knew medical centers needed money now more than ever. What was the ethical way for doctors to help, she wondered? Or should they stay out of the donation business completely?

She searched the medical literature for studies on the subject and found pretty much nothing, so she decided to conduct her own research.

The issue is "extraordinarily important," said Arthur L. Caplan, head of the division of medical ethics at NYU Langone Medical Center, adding that he had never seen a paper that examined the issues as thoroughly as Dr. Jagsi's. "Hopefully, this paper will start a long overdue discussion," he said.

He ticked off some ethical pitfalls: "Patients may be emotionally vulnerable; doctors have very close ties to their patients, which can strain asking on both sides; and the fact that incentives to ask sometimes skew toward the doctor's own program rather than the most needy areas of the hospital."

Yet, the practice of doctors soliciting donations from patients "is something that is happening and all signs are that it is going to continue and that it will increase," said Dr. Joseph A. Carrese, a primary care doctor and bioethicist at Johns Hopkins. Patient donations, he added, are "an important source of resources when money is tight."

Dr. Carrese was concerned enough to join his colleagues in conducting an interview study of Hopkins doctors. He said he was reassured that the physicians recognized the ethical tightrope they were on. But some, he said, admitted to giving big donors special treatment.

"I'm more likely to arrange a special appointment time for those patients so we are not rushed," one doctor who was interviewed for the study said. Another said, "I'm asking them to go above and beyond their relationship with me as a patient so I feel like I have to go above and beyond."

Different medical centers have different policies. At the Harvard Dana-Farber Cancer Institute, the goal is to leave the doctor out of the equation, said the president and chief executive, Dr. Edward J. Benz Jr. If a patient asks how to donate, the doctor is supposed to direct the patient to the development office. At one point, administrators considered giving patients brochures on how to donate when their treatment ended, but then decided that would be inappropriate.

At the University of North Carolina, said Dr. Norman E. Sharpless, director of the Lineberger Comprehensive Cancer Center, oncologists are advised not to directly solicit patients but to notify a development officer when a patient seems able and willing to make a donation.

He explained how it often works: "A patient with financial capacity expresses an interest in helping. The doctor tells a development officer, who invites the patient and doctor to lunch. "When it comes time to discuss a donation, the doctor gets out of the way."

Dr. Sharpless said he has never seen people get special care because they are rich, but added that there are subtle advantages that can accrue to donors. "If you are a prospective donor, or a donor, the development people can visit you at your home, can take you to lunch. If you are having a problem, your Rolodex at U.N.C. is bigger. You can reach out to the development officer and say, 'I am having a problem.'"

For Tom and Nancy Chewning of Richmond, Va., the path to donation began when their daughter received what they considered extraordinary care at the Lineberger Center. On their own they made a generous gift in honor of their

daughter's oncologist, Lisa Carey. Then the development office asked if they might want to meet with Dr. Carey and discuss her needs.

So Mr. and Mrs. Chewning drove to Chapel Hill and sat down with Dr. Carey. When they asked what she needed, she said she could use money for her research and for helping patients, but she did not directly ask the Chewnings to contribute.

"I'm not very good at this," Dr. Carey said. Then Mr. Chewning asked her if she thought they could make a difference with a donation. He and his wife went home and made an even more generous donation, 10 times the original amount.

"It is something that says, 'I appreciate what you do,' " Mr. Chewning said. "I know it will be well spent."

Jack Hyer and his wife, Laura Jensen, who live just outside Chapel Hill, both were treated for cancer at the University of North Carolina and were so grateful for the care they received that they reconsidered their initial impulse to donate money to the university for athletic scholarships. After meeting with the head of the cancer center they ended up allocating about $2 million for research and for an endowed professorship in radiology.

"We committed our entire estate," Mr. Hyer said.

And Mr. Hyer made a training video for doctors to learn how to effectively ask for donations. "The video sort of alerted them to be aware of the role they might play in identifying someone who might want to give," Ms. Jensen said.

"They show that film regularly," Mr. Hyer added.

http://www.eurekalert.org/pub_releases/2015-09/kcl-air093015.php

Antipsychotics increase risk of death in people with Parkinson's disease psychosis

Antipsychotic drugs may increase the risk of death in people with Parkinson's disease psychosis

Antipsychotic drugs may increase the risk of death in people with Parkinson's disease psychosis (PDP), according to a new study led by researchers from the Institute of Psychiatry, Psychology & Neuroscience (IoPPN) at King's College London.

The study, published in JAMDA, found that people with PDP who were treated with antipsychotics were four times more likely to have died following three to six months of treatment than those who did not receive any antipsychotic medication. They were also more likely to experience serious health issues including cognitive decline, worsening of Parkinson's symptoms, stroke, infections and falls.

Parkinson's disease affects approximately 7-10 million people worldwide and is characterised by progressive loss of motor function, psychiatric symptoms and cognitive impairment. Psychosis is a common and distressing group of psychiatric symptoms affecting people with Parkinson's, usually manifesting as hallucinations and delusions.

PDP affects more than 50 per cent of people with Parkinson's at some point in their condition and antipsychotic drugs are often used to treat this psychosis, yet there is little evidence to support their use.

The researchers examined more than 400 people with PDP, who were taking part in a separate trial, to assess the impact of antipsychotic medications on their overall health and wellbeing. Participants were categorised into two groups - those receiving antipsychotics and those who did not take any antipsychotic medications at any time during the study.

Professor Clive Ballard from the Wolfson Centre for Age-Related Diseases at the IoPPN, King's College London, said: 'Our findings clearly indicate serious risks associated with antipsychotics and highlight the need for greater caution in treating psychosis in Parkinson's disease.

'Antipsychotics are known to be linked to serious harm in people with Alzheimer's Disease, and these findings show that a similar, although not identical, risk is seen in people with Parkinson's. Our findings therefore strongly suggest that doctors, patients and family members should consider these risks very carefully when considering potential treatments for psychosis and any other behavioural symptom in people with Parkinson's Disease, such as agitation or aggression.

'Further research is required to develop new, better treatments for psychosis and other behavioural symptoms.'

Professor Ballard added: 'For example, a study we published last year showed that a novel antipsychotic, pimavanserin, was effective and had far fewer side effects than traditional antipsychotics.'

http://www.bbc.com/news/health-34397794

Womb transplants given UK go-ahead

Doctors have been granted approval to carry out the UK's first 10 womb transplants, following the success of the procedure in Sweden.

The go-ahead has been given by the Health Research Authority - as part of a clinical trial - which launches in the spring. Around one in 7,000 women are born without a womb, while others lose their womb to cancer. If the trial is successful, the first UK baby could arrive in early 2018.

Dr Richard Smith, a consultant gynaecologist at the Queen Charlotte's and Chelsea Hospital in London who has been working on the project for 19 years, will lead the transplant team. He said childlessness could be a "disaster" for couples, but the technique would offer hope to those whose only other option is surrogacy or adoption.

How would the procedure work?

- ***The operation takes around six hours, with the organ coming from a donor who has died but whose heart has been kept beating***
- ***The recipient will need to take immunosuppressant drugs following the transplant and throughout any pregnancy to prevent the chance their body might reject the donor organ***
- ***The health of the woman will be monitored closely for a year and then an embryo will be implanted in the womb***
- ***This embryo would arise from a combination of the woman's own eggs and the partners' sperm - using an IVF procedure***
- ***If all goes well the baby will be delivered eight months later by caesarean section***
- ***Couples will be given the option of trying for two pregnancies before the womb is removed***
- ***Once it is no longer needed, the womb can be taken out by a team of surgeons. This would prevent the need for the woman to be on immunosuppressants for the rest of her life***

Dr Smith told the BBC Radio 4's Today programme: "Over the years I have quite a lot of crisis with this project... but when you meet the women who have been born without a uterus, or who have had their uterus removed for one reason or another, this is really heart-rending stuff and that is what has kept us going.

According to his team at Womb Transplant UK, each procedure costs around £50,000 to perform, but women will not have to pay for this themselves.

The project has so-far been self-funded and supported by public donations which researchers say will allow them to take on two procedures for now.

'To carry my own child would be amazing'

Sophie, 30, is one of the women hoping to be selected as one of the first 10 recipients of a womb transplant. She was 16 when she was diagnosed with Mayer-Rokitansky-Kuster-Hauser syndrome - a condition which meant her womb did not develop - and told she would not be able to give birth.

Sophie is now preparing to marry her long-term partner Tilden Lamb next year and says the desire to have children had increased as she has grown older.

She says: "To be able to carry my own child would be amazing."

'Specific criteria'

The women who will be selected for the trial must all meet criteria set out by Womb Transplant UK, which include being 38 or under, having a long-term partner and being a healthy weight. More than 300 women have approached the team, of whom 104 meet the criteria. Researchers plan to transplant wombs that have come from donors who are brain dead but whose hearts are still beating - unlike previous procedures in Sweden where live donors were involved.

Experts in the UK say a different decision has been reached here as the initial operation to remove the womb from the donor is complex and not without risk.

But details of how women could signal their wish to donate their wombs - for example through a donor card - still need to be ironed out.

The British Fertility Society welcomed developments in the UK. Chairman, Prof Adam Balen, said: "This opens up the possibility for these women to carry their own pregnancy rather than rely upon IVF with their eggs and surrogacy.

"The UK team have been working on this for many years and so it is very exciting that they have been given the go ahead to move into clinical practice."

In October last year a woman in Sweden became the first in the world to give birth to a baby after having a womb transplant, but from a living donor.

http://www.eurekalert.org/pub_releases/2015-09/uobc-fgb092515.php

Four gut bacteria decrease asthma risk in infants

New research finds that infants can be protected from getting asthma if they acquire four types of gut bacteria by three months of age

New research by scientists at UBC and BC Children's Hospital finds that infants can be protected from getting asthma if they acquire four types of gut bacteria by three months of age. More than 300 families from across Canada participated in this research through the Canadian Healthy Infant Longitudinal Development (CHILD) Study.

"This research supports the hygiene hypothesis that we're making our environment too clean. It shows that gut bacteria play a role in asthma, but it is early in life when the baby's immune system is being established," said the study's co-lead researcher B. Brett Finlay, Peter Wall Distinguished Professor in the Michael Smith Laboratories and the departments of microbiology & immunology and biochemistry and molecular biology at UBC.

Asthma rates have increased dramatically since the 1950s and now affect up to 20 per cent of children in western countries. The discovery opens the door to developing probiotic treatments for infants that prevent asthma. The finding could also be used to develop a test for predicting which children are at risk of developing asthma.

The researchers analyzed fecal samples from 319 children involved in the CHILD Study. Analysis of the gut bacteria from the samples revealed lower levels of four specific gut bacteria in three-month-old infants who were at an increased risk for asthma.

Most babies naturally acquire these four bacteria, nicknamed FLVR (Faecalibacterium, Lachnospira, Veillonella, Rothia), from their environments, but some do not, either because of the circumstances of their birth or other factors.

The researchers also found fewer differences in FLVR levels among one-year-old children, meaning the first three months are a critical time period for a baby's developing immune system.

The researchers confirmed these findings in mice and also discovered that newborn mice inoculated with the FLVR bacteria developed less severe asthma.

"This discovery gives us new potential ways to prevent this disease that is life-threatening for many children. It shows there's a short, maybe 100-day window for giving babies therapeutic interventions to protect against asthma," said co-lead researcher Dr. Stuart Turvey, pediatric immunologist, BC Children's Hospital, director of clinical research and senior clinician scientist at the Child & Family Research Institute, Aubrey J. Tingle Professor of Pediatric Immunology at UBC.

The researchers say that further study with a larger number of children is required to confirm these findings and reveal how these bacteria influence the development of asthma.

The study was published today in in Science Translational Medicine.

Watch a video with the researchers: https://youtu.be/rRlezDvY3Ew . Video files are available for download, please contact Heather Amos at 604.828.3867.

This research was supported by the Canadian Institutes of Health Research (CIHR). The CHILD Study is supported by the Allergy, Genes and Environment Network (AllerGen NCE Inc.), CIHR, Health Canada, Environment Canada, Canada Mortgage and Housing Corporation, and the Childhood Asthma Foundation. The researchers are supported by BC Children's Hospital Foundation, the University of British Columbia, Michael Smith Foundation for Health Research and Tula Foundation.

http://www.eurekalert.org/pub_releases/2015-09/nion-dvq092915.php

Dormant viral genes may awaken to cause ALS

NIH study may open an unexplored path for finding treatments

Scientists at the National Institutes of Health discovered that reactivation of ancient viral genes embedded in the human genome may cause the destruction of neurons in some forms of amyotrophic lateral sclerosis (ALS). The results, published in Science Translational Medicine, suggest a link between human endogenous retroviral genes (HERVs) and ALS. The findings also raise the question of whether antiretroviral drugs, similar to those used for suppressing HIV, may help some ALS patients.

For generations, humans have been passing on genetic remnants of HERV infections that may have happened millions of years ago. Although nearly eight percent of the normal human genome is made up of these genes, very little is known about their role in health and disease.

"People call the genes for these viruses junk DNA. Our results suggest they may become activated during ALS," said Avindra Nath, M.D., clinical director at the NIH's National Institute of Neurological Disorders and Stroke (NINDS) and a senior author of the study. "Ultimately we hope the results will lead to effective treatments for a heartbreaking disorder."

Currently, there is no effective treatment for the more than 12,000 Americans who live with ALS. This fatal disorder destroys neurons that control movements, including speaking, walking, breathing and swallowing. On rare occasions, HIV-infected, AIDS patients develop ALS-like symptoms. In many of these patients, the symptoms can be reversed by treatment with antiretroviral drugs. Previous studies found reverse transcriptase, a protein encoded by retroviral genes, in the blood of some ALS patients but its role in the disorder is unknown.

These observations prompted Dr. Nath and his team to explore the possible link between retroviruses and ALS. Unexpectedly they found that endogenous, or inherited, retroviruses may be involved with ALS.

Initially, they showed that brain samples from ALS patients had higher than normal levels of messenger RNA (mRNA) encoded by genes of the human endogenous retrovirus K (HERV-K). A protein encoded by a critical HERV-K gene, called env, was found in brain samples from ALS patients but not from healthy individuals or patients with Alzheimer's disease. They also showed that activation of HERV-K genes killed healthy human neurons grown in petri dishes.

To test the role of HERVs in ALS, the scientists genetically modified mice so that their neurons activated the HERV-K env gene. The mice died earlier than normal and had problems with balance and walking that progressively worsened with age. When the scientists inspected the brains, spinal cords and muscles of these mice they found that only motor neurons, the cells that control movements and die in ALS, were damaged. Cells in other parts of the nervous system remained healthy.

"We showed that motor neurons may be susceptible to activation of these genes during ALS," said Dr. Nath.

Finally the scientists showed that activation of HERV-K genes may be controlled by TDP-43, a gene-regulating protein that has been strongly linked to ALS and known to control HIV production. Genetically enhancing TDP-43 in human neurons increased the cells' production of HERV-K mRNA and proteins whereas genetically blocking TDP-43 in other cells reduced HERV-K reverse transcriptase activity.

Dr. Nath and his team are now collaborating with the ALS center at Johns Hopkins University to study whether antiretroviral treatments are effective at controlling HERV-K replication in a subset of patients with ALS.

"We may have discovered a precision medicine solution for treating a neurodegenerative disorder," said Dr. Nath.

This work was supported by intramural research programs at the NINDS, the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) and the Eunice Kennedy Shriver National Institute of Child Health and Development (NICHD).
Li et al. "Human endogenous retrovirus-K induces motor neuron disease," Science Translational Medicine, September 30, 2015. DOI: 10.1126/scitranslmed.aac8201

http://bit.ly/1FPIzKq

Chelyabinsk Meteor Played 4.5 Billion Years of Cosmic Pinball

When the Chelyabinsk meteor exploded in the morning skies of the southern Urals in February 2013 it prompted a flurry of interest in the vast population of small bodies that share our orbital turf around the Sun.

By Caleb A. Scharf | September 30, 2015

Now a study by a large group of scientists, published in the journal of Meteoritics & Planetary Science by Righter et al., suggests that the original Chelyabinsk object was already no stranger to cosmic collisions.

The researchers have analyzed and mapped the detailed properties of 3 pieces of the meteor that were recovered with minimal terrestrial contamination. That's a somewhat forgiving criterion: Two of these bits came from a sharp-eyed forester spotting holes in the snow and plucking out what are charmingly termed 'ice carrots' - the refrozen spikes after the meteor fragments ploughed into the surface. Nonetheless, combined with the analyses of other fragments, a dataset has been generated that offers a unique set of insights to the meteor's earlier history.

The new work is a tour de force of highly technical methods, deploying optical microscopy, electron microscopy, Raman spectroscopy, and the analysis of isotopic compositions with a range of sophisticated tools. Critically, the measurements reveal an extremely complex history for the Chelyabinsk material - recorded in isotopic ratios and mineralogical alterations.

In fact, some of the isotopic 'clocks' typically used to date rocky solar system material (such as Rubidium and Strontium ratios, and Argon isotope ratios) appear to have been partially or totally 'reset' at various points in the past. These resets can be caused by strong thermal changes altering isotopic ratios, and variations in exposure to cosmic radiation.

The conclusion is that before the Chelyabinsk body fell on the Earth it experienced at least 8 significant impacts, starting around 4.53 billion years ago, and again at 4.45 billion years, 3.73, 2.81, and 1.46 billion years ago, followed by 852 million years ago, 312, and most recently 27 million years ago.

Those impacts happened out in the wilds of interplanetary space, as asteroidal material collided with other asteroidal material in a great game of cosmic pinball. We can still see this kind of jostling taking place today. In 2010 the Hubble Space Telescope caught the aftermath of just such an impact, some 90 million miles from the Earth, and visible in this image:

Asteroids can hit asteroids **NASA/ESA and D. Jewitt UCLA**

Indeed, one other tantalizing result from the new study suggests that there could have been a collision event within the past million years or so (based on the rock's cosmic ray exposure). This indicates that what hit the Russian skies may have come from the quite recent breakup of a Near Earth Object.

The Chelyabinsk meteor carried all of this complicated and messy history with it.

Those earlier impacts led to thermal changes (metamorphism) and fragmentation. And that checquered past is even consistent with the trajectory of the meteor's fireball and the way in which it broke up in Earth's atmosphere - a last gasp before leaving its ancient secrets for humans to decipher.

http://www.eurekalert.org/pub_releases/2015-09/uop-pdm093015.php

Penn Dental Medicine study is proof-of-concept for low-cost drug made in lettuce

First time a group has shown the commercial viability of producing a low-cost drug made from whole plants

Biopharmaceuticals, or drugs that are based on whole proteins, are expensive to make and require refrigeration to store. Insulin, for example, is unaffordable and inaccessible to most of the global population.

At the University of Pennsylvania School of Dental Medicine, Henry Daniell and colleagues have been working to overcome these obstacles by using a plant-based system to make shelf-stable drugs. In a study published in the journal Biomaterials, the researchers confirmed the viability of their method for FDA approval and human use, producing an effective drug that promotes tolerance to clotting factors, which could be taken by hemophilia patients, using freeze-dried lettuce leaves.

This is the first time a group has shown the commercial viability of producing a low-cost drug made from whole plants.

"This is a milestone in our field, to make a fully functional drug in plants, produce it at a large scale and in quantities sufficient for human clinical trials," Daniell said.

Daniell, professor and interim chair in Penn Dental Medicine's Department of Biochemistry, is senior author on the study. Collaborators from the University of Florida led by Roland Herzog conducted animal studies and Fraunhofer USA's Steve Streatfield facilitated large-scale production of lettuce in the company's FDA-compliant facility.

The study builds on previous work by Daniell's group demonstrating an ability to use genetically modified plants to introduce a protein into the body that would teach the immune system to tolerate clotting factors that are given as a treatment for hemophilia.

Normally, 20 to 30 percent of people who get infusions of clotting factor develop antibodies against them that interfere with treatment. The earlier study, published in the journal Blood, successfully stopped and even reversed the production of these clotting factor inhibitors by feeding the plant-based drug to mice with hemophilia A.

That study used a tobacco plant platform to "grow" the drug. To take this approach into humans, however, Daniell's team knew they needed to use a different plant species.

They launched work with lettuce, which required using a completely different genetic vector to introduce the therapeutic gene into the plant cell's DNA, as the tobacco construct would not work in a different species. After identifying a compatible vector, they used a similar protocol to their previous work, bombarding lettuce leaves with a fusion of the therapeutic protein, coagulation factor IX, or FIX, with cholera toxin B subunit, which allows the protein to reach the immune system. They then evaluated the resulting plants for those that took it up and then grew those plants to maturity.

The next step was to ensure that the drug would be shelf stable. To do that, they freeze dried the plant material, ground it and analyzed the resulting fine powder for expression levels of the fusion protein to determine the appropriate dose and to evaluate its efficacy.

Similar to their previous experiments, Herzog's lab fed hemophilia B mice with a suspension of plant cell containing clotting factor IX twice a week for eight weeks and then gave them the same clotting factor that human hemophilia patients take to encourage blood clotting. As before, their product was a success: mice given the drug had greatly suppressed inhibitor formation compared to untreated animals, even when various doses of the drug were tested.

"One of the key findings of our study was that we found our drug was efficacious across at least a 10-fold dose range," Daniell said.

Such flexibility is important for translation of the drug to humans, as there may be individual variations in how a drug is metabolized in the gut as plant cells are broken down by commensal bacteria.

In the work, the researchers used two different growing systems. One was in the greenhouse on Penn's Pennovation Works campus, a high-tech facility that grows the plant in soil and uses natural light. The second was the Fraunhofer USA facility, which more closely replicates how a commercial pharmaceutical production facility would run, using a hydroponic system and artificial lighting.

"Despite the fact that plants in the greenhouse were receiving 50 times more light, the Fraunhofer yield was quite close to ours and quite good," Daniell said. "In 1,000 square feet, they could produce 36,000 doses."

A hydroponic system could also easily be scaled up by adding racks and thus using vertical space, which a traditional greenhouse could not do. The researchers were able to harvest a new batch of pharmaceutical-containing lettuce every four to six weeks.

With this study, which confirms the viability of a plant-based biopharmaceutical production on a commercial scale, the researchers have eliminated several expensive obstacles that hamper the development of affordable traditional protein drugs. The method requires no fermenter, no purification to ensure sterility and no cold chain to keep the drug refrigerated. In addition, the researchers found that their capsules remained potent and effective for two years, ensuring the product is shelf-stable and patients could theoretically take the drug from home.

"Not only did we show a truly translational result for helping hemophilia patients," Daniell said, "but this also changes the way we think about delivering protein-based drugs to human patients.

"Current treatments for inhibitor formation in hemophiliacs cost almost a million dollars and are not affordable for a significant segment of the patient population," he said, "but the new drug is dramatically cheaper and may offer even a better solution for treating hemophilia patients. Most important, developing a low cost platform for protein drug delivery will make these drugs affordable for a large majority of the global population."

Additional authors on the study included Aditya Kamesh from Penn Dental Medicine; co-lead author Liqing Zhu, Alexandra Sherman, Xiaomei Wang and Roland W. Herzog from UF; and Joey H. Norikane and Stephen J. Streatfield from Fraunhofer USA.

Large scale production was supported by Novo Nordisk, and basic science was supported by two National Institutes of Health grants. A short-term exchange student fellowship to Liqing Zhu was provided by the National Nature Science Foundation of China.

http://bit.ly/1OflOmm

Invasive herb could hamper East Africa's fight against malaria

GAINS made in the fight against malaria in East Africa could be set back by an invasive plant species, whose nectar could keep mosquitoes alive when blood isn't available.

American invader famine weed, or Santa Maria feverfew (*Parthenium hysterophorus*), is spreading rapidly across East Africa. The weed secretes a poisonous substance known as parthenin, which can cause dermatitis, hay fever and asthma. It is also harmful to livestock.

***A female* Anopheles *probes famine weed flowers* (Image: Robert Copeland/ICIPE)**

In recent years it has become clear that famine weed's flowers are attractive to the female *Anopheles gambiae* mosquito, which spreads malaria.

So Baldwyn Torto at the International Centre of Insect Physiology and Ecology in Nairobi, Kenya, and colleagues took day-old mosquitoes and raised them where they had access to one of three plants: famine weed, the castor oil plant (*Ricinus communis*) or *Bidens pilosa*, a Kenyan vegetable crop plant.

Survival rates after 14 days were about 45 per cent for the castor oil plant, about 30 per cent for famine weed and little more than 10 per cent for *B. pilosa*. This suggests that if famine weed continues to spread at the cost of *B. pilosa*, mosquitoes may find it slightly easier to survive between blood meals, potentially making the fight against malaria tougher (*PLoS One*, doi.org/7xh).

Torto and colleagues are now trying to find out the impact of parthenin on mosquitoes infected with the malaria parasite. For example, whether nectar from the weed increases biting frequency.

http://www.eurekalert.org/pub_releases/2015-10/aaos-lbm100115.php

Later bedtimes may lead to an increase in body mass index over time

Results highlight adolescent bedtimes as a potential target for weight management

DARIEN, IL - A new study suggests that going to bed late during the workweek from adolescence to adulthood is associated with an increase in body mass index over time. Results of hierarchal linear models involving a nationally representative sample of more than 3,000 participants show that going to bed during the workweek each additional hour later is associated with an increase in BMI of 2.1 kg/m2. Moreover, surprising to the researchers, the relationship between bedtime and BMI was not significantly changed or moderated by total sleep time, exercise frequency or screen time.

"The results are important because they highlight adolescent bedtimes, not just total sleep time, as a potential target for weight management concurrently and in the transition to adulthood," said first author Lauren Asarnow, a doctoral candidate at the University of California, Berkeley.

Study results are published in the October issue of the journal Sleep.

Along with Eleanor McGlinchey, PhD, and Allison G. Harvey, PhD, Asarnow analyzed three waves of data from the National Longitudinal Study of Adolescent Health (Add Health) to assess the bedtimes and BMI of 3,342 adolescents between 1994 and 2009. Sleep and circadian variables were determined via self-reported measures at all three waves. Investigators measured height and weight at each wave, from which BMI was calculated.

According to the authors, this is the first study to examine the longitudinal relationship between bedtimes and BMI in any age group in an observational study. The American Academy of Sleep Medicine recommends that adolescents get a little more than nine hours of nightly sleep for optimal health and daytime alertness during the critical transition from childhood to adulthood.

The study was supported by a National Science Foundation Graduate Research Fellowship and grants from the National Institute of Mental Health (NIMH) and the Eunice Kennedy Shriver National Institute of Child Health and Human Development.

http://www.bbc.com/news/health-34392522

'Good bacteria' key to stopping asthma

Being exposed to "good bacteria" early in life could prevent asthma developing, say Canadian scientists.

By James Gallagher Health editor, BBC News website

The team, reporting in Science Translational Medicine, were analysing the billions of bugs that naturally call the human body home. Their analysis of 319 children showed they were at higher risk of asthma if four types of bacteria were missing. Experts said the "right bugs at the right time" could be the best way of preventing allergies and asthma.

In the body, bacteria, fungi and viruses outnumber human cells 10 to one, and this "microbiome" is thought to have a huge impact on health. The team, at the University of British Columbia and the Children's Hospital in Vancouver, compared the microbiome at three months and at one year with asthma risk at the age of three. Children lacking four types of bacteria - Faecalibacterium,

Lachnospira, Veillonella, and Rothia (Flvr) - at three months were at high risk of developing asthma at the age of three, based on wheeze and skin allergy tests.

The same effect was not noticed in the microbiome of one-year-olds, suggesting that the first few months of life are crucial. Further experiments showed that giving the bacterial cocktail to previously germ-free mice reduced inflammation in the airways of their pups.

One of the researchers, Dr Stuart Turvey, said: "Our longer-term vision would be that children in early life could be supplemented with Flvr to look to prevent the ultimate development of asthma. "I want to emphasise that we are not ready for that yet, we know very little about these bacteria, [but] our ultimate vision of the future would be to prevent this disease."

Asthma is caused by airways that are more sensitive to irritation and inflammation. Cases have soared, and one in every 11 children in the UK is now diagnosed with asthma. One explanation for the rise in asthma and allergies is the "hygiene hypothesis", which suggests that children are no longer exposed to enough microbes to calibrate the immune system to tell the difference between friend and foe. Giving birth by Caesarean section and not breast-feeding both limit the bacteria that are passed to a newborn. Antibiotics taken by a pregnant woman or newborn child can also change the microbiome.

Dr Brett Finlay, another researcher in the project, said: "[I was] surprised to realise that faecal microbes may be influencing things. "What data's really starting to show these days is that the immune system gets itself set up in the gut and influences how it works everywhere else in the body."

'Right bugs, right time'

Dr Benjamin Marsland, from the University of Lausanne, in Switzerland, told the BBC: "For a number of years, exposure to microbes has been linked with protection against asthma, a classic example is growing up on a farm and drinking raw milk." His own research suggests a high-fibre diet reduces inflammation in the lungs and may ease asthma symptoms. He said previous research was showing a mounting role for diet, microbes and the first year of life being key.

He added: "This new study adds weight to these observations and supports the concept that there are certain developmental windows in early life, where it's really important to get the right signals. "A common factor in all studies so far has been the microbiota, in fact making sure babies have the right bugs, at the right time, might be the best step towards preventing asthma and allergies."

Dr Samantha Walker, from the charity Asthma UK, said: "Asthma is a complex condition, and this research suggests that the delicate balance of gut bacteria in our bodies affects our immune systems and may have a role to play in why some people go on to develop asthma.

"However, much more research is needed to help understand what these findings mean in terms of providing advice for new parents, developing treatments and ultimately a cure."

http://nyti.ms/1KTIqqU

A Breast Cancer Surgeon Who Keeps Challenging the Status Quo

One of only a few surgeons in the United States willing to put women with D.C.I.S. on active surveillance instead of performing biopsies, lumpectomies or mastectomies

By KATIE HAFNER

Late one afternoon this summer, Dr. Laura J. Esserman, a breast cancer surgeon at the University of California, San Francisco, sat in a darkened room scrutinizing a breast M.R.I. With a clutch of other clinicians at her side, she quickly homed in on a spot smaller than a pencil eraser.

She heard the words “six-millimeter mass.” Her response was swift:

“No.”

Meaning no biopsy.

Most doctors, including the radiologist seated next to her, would have said yes. But Dr. Esserman, who has dedicated much of her professional life to trying to get the medical establishment to think differently about breast cancer, foresaw only unnecessary anxiety for the patient, who had had several biopsies in the past — all benign.

Dr. Esserman, 58, is one of the most vocal proponents of the idea that breast cancer screening brings with it overdiagnosis and overtreatment. Her philosophy is controversial, to say the least. For decades, the specter of women dying for lack of intervention has made aggressive treatment a given.

But last month, her approach was given a boost by a long-term study published in the journal JAMA Oncology. The analysis of 20 years of patient data made the case for a less aggressive approach to treating a condition known as ductal carcinoma in situ, or D.C.I.S., for which the current practice is nearly always surgery, and often radiation. The results suggest that the form of treatment may make no difference in outcomes.

Dr. Esserman, who directs the Carol Franc Buck Breast Care Center, is one of only a few surgeons in the United States willing to put women with D.C.I.S. on active surveillance instead of performing biopsies, lumpectomies or mastectomies.

She and other critics of vigorous intervention point to the potential side effects and risks of sometimes disfiguring treatments for premalignant conditions that are unlikely to develop into life-threatening cancers.

She has also challenged the conventional wisdom surrounding screening, arguing that while mortality from breast cancer has decreased over the past three decades,

the approach to screening needs to change. She points out that the most lethal breast cancers appear between screens, while mammograms are finding more slow-growing cancers with a very low chance of metastasis. In addition, screening has revealed a reservoir of D.C.I.S., also known as Stage 0, which now accounts for 20 percent to 25 percent of all breast cancer diagnoses.

So convinced is Dr. Esserman that most patients will not benefit from early detection of such lesions that she has recommended to the National Cancer Institute that for many D.C.I.S. lesions, the ominous word "carcinoma" be dropped from the medical term for them and that they be renamed "indolent lesions of epithelial origin," or IDLEs.

Much of this unsettles cancer specialists, who believe that aggressive treatment is prudent given that D.C.I.S. can be a precursor to invasive cancer in some patients.

"What do you do if you hear a gunshot — duck or not?" asked Dr. Larry Norton, medical director of the Memorial Sloan Kettering Evelyn H. Lauder Breast Center, who nonetheless said he admired Dr. Esserman's professionalism and rigor.

In an era of 15-minute doctor visits, Dr. Esserman is known to spend hours with a patient (a practice that can be maddening to those in the waiting room) even if it means staying at the office until 10 p.m. She sends late-night text messages to patients and calls whenever she can.

One recent Sunday afternoon, she stood in the large, art-filled kitchen of her house in the Ashbury Heights district of San Francisco, rehearsing the song "Defying Gravity" from the musical "Wicked." It was a request from a patient.

For nearly two decades, Dr. Esserman has sung to her patients as they go under anesthesia. With enough notice, she takes requests.

"Ask for an aria and I might need a week, but most songs take about 15 minutes to learn," she said.

"Unlimited. My future is unli-mi-ted," she sang full-throated in her kitchen, for a visitor.

Then she stopped. "You see, that's the thing," she said, her gaze intense enough to double as a Vulcan mind meld. "You have to believe in the possible. The minute you think your future is limited, it is."

Slow to Gain Acceptance

Dr. Esserman received national attention five years ago with an innovative, adaptively randomized drug trial called I-SPY 2, aimed at reducing the cost and time required to test new medications for breast cancer. The trial matches drugs with patient subtypes, allowing drugs from different companies to be assessed simultaneously, and much earlier in the disease process, while quickly phasing out those that do not appear to be working.

Trials for drugs to treat other cancers, as well as Alzheimer's disease and Ebola, have adopted the design, said Donald Berry, a statistician at M.D. Anderson in Houston who designed I-SPY 2 with Dr. Esserman.

"The whole idea catapulted the concept of adaptive clinical trials and precision medicine," said Dr. Richard Schilsky, chief medical officer of the American Society of Clinical Oncology.

Dr. Esserman's approach to D.C.I.S. has been much slower to gain acceptance in the medical community.

"Laura is one of the people who's actively engaged in research in this area and will help us push the field forward to determine whether or not there is a group of people for whom surveillance will be appropriate," said Dr. Elisa Port, chief of breast surgery at Mount Sinai Hospital in New York and author of "The New Generation Breast Cancer Book." "But no one has these tools now to know whether or not it's safe, and the biggest factor is we know that when we do surgery on D.C.I.S., about 10 percent of the time, commingled with the D.C.I.S. is invasive cancer."

She added: "When we talk about watching and waiting with D.C.I.S., the question is, 'How do we know it's just D.C.I.S.?' The answer is that we don't."

But Dr. Esserman's minimally invasive approach is beginning to win some converts in clinical settings. One of the highest compliments she has received, she said, came recently from a colleague at U.C.S.F., Dr. Barbara Fowble, a radiation oncologist who has tended to favor more conventional treatment.

The two physicians were discussing a patient whose nodes showed no cancer after chemotherapy and surgery. Dr. Esserman said she assumed Dr. Fowble would favor radiating that region. To her surprise, Dr. Fowble said, "Absolutely not."

Dr. Esserman asked, "What happened to you?" Dr. Fowble's reply: "You happened to me."

When asked about the remark by a reporter, Dr. Fowble laughed. "Yes," she said. "I think we've both influenced each other. She was willing to do more surgery, and I was willing to back off on the radiation."

"She advances us forward," Dr. Fowble said of Dr. Esserman. "And you can either go with her or live in the past. I would rather go with her."

Challenging a Patient

One day last January, Ilene Katz, a registered nurse at the university, went to see Dr. Esserman. It was not about work. Ms. Katz had just learned that she had a 12-centimeter tumor in her breast. Frightened and in shock, she told Dr. Esserman she wanted to have both breasts removed.

The appointment lasted three hours. Scans showed that the tumor was self-contained.

That night Dr. Esserman called her new patient again, with pointed questions.

"She asked me why I was going to hurt my body when it wouldn't do any additional good," Ms. Katz, 45, recalled. "She asked me a bunch of questions that really made me think."

By the next day, she decided to have breast reduction surgery instead.

Several weeks later, Ms. Katz got a call from Dr. Esserman at 9:30 p.m. Dr. Esserman had just sat down to dinner.

"I could tell she couldn't stand knowing I was confused and scared," Ms. Katz said. "She wouldn't hang up until she was sure I felt better. She was talking between bites."

Peggy MacDonald, 51, is currently under Dr. Esserman's care, on a watch-and-wait course. She was diagnosed with D.C.I.S. in April 2013. "I didn't even know what it was," she recalled. "But there was cancer in the word and it was scary."

The first surgeons Ms. MacDonald saw in Portland, Ore., where she lives, immediately discussed surgery as a given. Then Ms. MacDonald heard about Dr. Esserman and flew to San Francisco for another opinion. Before the appointment, Dr. Esserman requested a few additional tests, including a high-resolution M.R.I. and blood tests to check hormone levels.

"She walked into the room and sat down and said, 'I don't think there's anything urgent going on here. We have time,' " Ms. MacDonald said.

Dr. Esserman put Ms. MacDonald on a course of ovarian suppression drugs and a hormonal agent. Last December, nearly two years after the diagnosis, Dr. Esserman told Ms. MacDonald that an M.R.I. showed no evidence of D.C.I.S.

Dr. Esserman is quick to point out that by no means should all cases of D.C.I.S. be treated with active surveillance. In contrast to Ms. MacDonald, she pointed to another patient, Courtney Hollander, 47, who received a diagnosis of D.C.I.S. last January. A surgeon Ms. Hollander saw in Los Angeles led her to believe she would need immediate surgery.

"I found myself being angry at having a double mastectomy for not having cancer," said Ms. Hollander. She sent Dr. Esserman an email and Dr. Esserman replied that night.

Ms. Hollander's D.C.I.S. did not respond to hormone therapy as hoped, and she is now planning to have a mastectomy.

"I didn't rush her into surgery," Dr. Esserman said. "And I think that's the essence of it. People don't want to think that a mastectomy is the first choice."

Making Noise at a Young Age

Dr. Esserman did not grow up around physicians. Her father was a car dealer in Miami, her mother a teacher. Gifted in science, she worked in a research lab at the University of Miami while still in high school, then went to Harvard.

Even then, her assertiveness was hard to miss. Dr. H. Gilbert Welch, a professor of medicine at Dartmouth, has known Dr. Esserman since they were undergraduates at Harvard, and he coached her on an intramural crew team.

"I cannot tell you who else I coached in that boat, but I never forgot coaching Laura," Dr. Welch said. "It was a lot of fun, but it wasn't quiet."

After Dr. Esserman finished her surgical training at Stanford University in 1991, she was recruited to the university's business school. She earned a master's in business while remaining part time on the School of Medicine surgical faculty and caring for her infant daughter, Marisa, the first of two children with her husband, Michael Endicott, a professional photographer and environmental activist.

Over the years, Dr. Esserman's philosophy has evolved away from the mainstream. "For years, I operated on people and felt that what I was doing was helpful," Dr. Esserman said, describing her treatment of D.C.I.S. "But the evidence started to show that we had made a mistake."

Over a decade, Dr. Esserman said, she saw the incidence of invasive breast cancer increase — in spite of the removal of some 60,000 D.C.I.S. lesions each year in the United States. "I had to be brutally honest, change my mind, and search for better answers," she said.

Pushing Forward While Cooking

"It makes no sense to keep arguing about this," Dr. Esserman was saying one night in her kitchen, pointing to the need for more robust and innovative clinical trials to determine the value of breast cancer screening. Gathered with her were a visiting biostatistician from Sweden and several local colleagues.

She made her point while painstakingly extricating the bones from a five-pound salmon she planned to grill for dinner, using a surgical instrument meant for suturing that resembled a pair of nail scissors.

Dr. Esserman frequently holds meetings in her kitchen, cooking for the group while hatching, say, a new idea for a paper. On this night, she was sautéing arugula, which seemed to vanish, only to resurface 30 minutes later in the potatoes she had mashed by hand.

The culinary magic occurred while she steered her colleagues through a gamut of complex topics, including the longstanding debate over breast cancer screening.

In 2009, the United States Preventive Services Task Force revised its breast cancer screening guidelines, recommending that women wait until age 50 to start regular screening, and that women 40 to 49 who were at a high risk for breast cancer discuss with their physicians the best time to start getting mammograms.

Dr. Esserman and others had been pushing for such changes for years, but the revised guidelines were met with outrage from breast cancer support groups, as well as some researchers and physicians, who argued that early detection had

saved millions of lives. (The American Cancer Society continues to recommend regular mammograms starting at age 40.)

Asserting the need for better evidence about the value of screening, Dr. Esserman paused briefly from her salmon deboning. “The only way to do better is to know better,” she said, crediting the poet Maya Angelou for that thought as she waved her small tool in the air. “The point is to try to move the field and do right by our patients.”

To this end, Dr. Esserman has embarked on an ambitious project — a multiyear trial involving some 100,000 participants. Called “Women Informed to Screen Depending on Measures of Risk,” or Wisdom, the five-year study will test participants for genetic markers and other factors that point to a risk of breast cancer, and screen those at risk more frequently than the current federal task force guidelines. Those deemed at less risk will receive fewer mammograms. A control group will receive annual mammograms.

Dr. Esserman is careful to point out that no one in the trial will receive screening that is less aggressive than the task force guidelines. “We’ll stay within the bounds,” she said, “but over time the goal is to learn what risk factors are the most important and how we can adapt screening accordingly.”

Even Dr. Esserman’s most outspoken critics respect her. “I think fundamentally she’s on the right track, and I’d be delighted to be disproved,” said Dr. Daniel Kopans, a professor of radiology at Harvard Medical School, who has long disagreed with Dr. Esserman about screening. Dr. Kopans, who specializes in breast imaging, cited studies showing that death rates of breast cancer patients who were not screened had declined at a much lower rate than those who were screened.

“Mammography isn’t the answer to breast cancer, by any means,” he said. “But don’t give up on mammography. And don’t stop screening because we haven’t figured out how to treat D.C.I.S. properly.”

As for her own screening, Dr. Esserman is aware that her risk for breast cancer is increasing with age. She said she planned to participate in the Wisdom trial. “I’m asking everyone else to be randomized, so I’ll probably be randomized,” she said. “I try to design trials that I would want to participate in.”

Dr. Esserman has received her share of angry letters, particularly from women with D.C.I.S. who chose to have mastectomies. “People have said, ‘How could you invalidate everything I’ve gone through?’ ” she said.

Every time she performs surgery, she hopes to help one more woman survive.

One morning earlier this summer, Dr. Esserman entered an operating room at UCSF Medical Center at Mount Zion, carrying a printout of the lyrics to the Beatles’ “With a Little Help From My Friends.” Ms. Katz had requested the song for a second surgery on her breast to remove any residual tumor cells.

As the anesthesiologist fit a mask to the patient’s face, Dr. Esserman cupped Ms. Katz’s hand tightly around her own, and together the physician and her frightened patient broke into song. Even after Ms. Katz had lost consciousness, Dr. Esserman kept singing, while stroking her patient’s cheek. She switched briefly to one of her favorites: “Sweet dreams that leave all worries behind you. But in your dreams, whatever they be, dream a little dream of me.”

Then she got to work.

http://www.eurekalert.org/pub_releases/2015-10/uob-tst100115.php

The solution to a 50-year-old riddle: Why certain cells repel one another

When cells from the connective tissue collide, they repel one another - this phenomenon was discovered more than 50 years ago.

It is only now, however, that researchers at the University of Basel have discovered the molecular basis for this process, as they report in the journal Developmental Cell. Their findings could have important implications for cancer research.

Fibroblasts are motile constituents of the connective tissue and also regulate its stiffness. Moreover, fibroblasts play an important role in malignant skin diseases such as melanoma. In research, they serve as a model system for studying cell migration.

Signaling pathway identified

In the early 1950s, the English researcher Michael Abercrombie discovered that colliding fibroblasts repel one another and, in the process, change their direction of motion. He called this phenomenon 'contact inhibition of locomotion'. Although individual proteins were identified as key factors in this process, the molecular basis of this reaction remained something of a puzzle. In particular, it was unclear which repulsion signals were involved in the process, how these signals entered the cells from the outside, and how they influenced the cytoskeleton, which in turn regulates the cell's movement.

Prof. Olivier Pertz's research group at the University of Basel has now precisely answered these questions. The group identified a coherent signaling axis consisting of three proteins called Slit2, Robo4, and srGAP2 which operates as follows:

The repulsion factor Slit2 binds to the receptor Robo4, whereupon the signal enters the cell's interior and activates srGAP2.

This molecule consequently inhibits the regulator Rac1, which coordinates the cytoskeleton.

The inactivation of Rac1 causes the cell to retract - such that the two cells repel one another.

If the function of Slit2, Robo4, or srGAP2 is deactivated, colliding cells will stick to one another and will not separate as easily.

A 'molecular bumper'

Intriguingly, the repulsion machinery is localized at the front - even in freely moving cells. By assembling this kind of a 'molecular bumper', the cell is prepared for collision with another cell. Where exactly this bumper must be positioned - namely, only in parts of the cell that are moving forwards - is determined by the cell's geometry, which in turn is deciphered by srGAP2.

The integration of membrane curvature and repulsion signals ensures that cell-cell repulsion takes place at the correct location. This repulsive reaction could play an important role in cancer metastasis. This is supported by the fact that the expression of Slit and Robo isoforms is deregulated in several tumor types.

http://nyti.ms/1Lbtl34

Crows May Learn Lessons From Death

A new study investigated what crows might understand about death

In recent years, a peculiar sort of public performance has taken place periodically on the sidewalks of Seattle.

It begins with a woman named Kaeli N. Swift sprinkling peanuts and cheese puffs on the ground. Crows swoop in to feed on the snacks. While Ms. Swift observes the birds from a distance, notebook in hand, another person walks up to the birds, wearing a latex mask and a sign that reads “UW CROW STUDY.” In the accomplice’s hands is a taxidermied crow, presented like a tray of hors d’oeuvres.

This performance is not surreal street theater, but an experiment designed to explore a deep biological question: What do crows understand about death?

Ms. Swift has been running this experiment as part of her doctoral research at the University of Washington, under the guidance of John M. Marzluff, a biologist. Dr. Marzluff and other experts on crow behavior have long been intrigued by the way the birds seem to congregate noisily around dead comrades. Dr. Marzluff has witnessed these gatherings many times himself, and has heard similar stories from other people.

“Whenever I give a talk about crows, there’s always someone who says, ‘Well, what about this?’ ” he said.

Dr. Marzluff and Ms. Swift decided to bring some scientific rigor to these stories. They wanted to determine whether a dead crow really does trigger a distinctive response from living crows and, if so, what the purpose of the large, noisy gatherings might be.

To run the experiment, Ms. Swift began by delivering food to a particular spot each day, so that the crows learned to congregate there to eat. Then one of her volunteers would approach the feast with a dead crow, and Ms. Swift observed how the birds reacted.

Almost every time, the crows mobbed the corpse-bearing volunteers. Ms. Swift is eternally grateful to her volunteers that they didn’t abandon the research at that point.

“If you’ve ever been divebombed by a crow, it’s really terrifying,” she said.

If the volunteer carried a dead pigeon, however, the crows mobbed the person only about 40 percent of the time. And if the volunteer stepped forward empty-handed, the crows just moved away until the coast was clear and then returned to the food.

Ms. Swift then ran more tests to see how much of an impression the dead crows made on the live ones. Because crows can tell individual humans apart by their faces, she had her volunteers wear latex masks. Even though she used a rotating crew of volunteers, each group of crows would see the same face throughout the trial. She had them return to the feeding site once a week to see how the crows responded.

“It’s a very Hannibal Lecter thing — it looks like you cut someone’s face off and are wearing it,” said Ms. Swift, who spent a lot of time reassuring Seattle residents that she was actually doing science. “A lot of people would say, ‘I don’t care what you say, I’m calling the police.’”

Up to six weeks later many birds still scolded the visitors even when they approached with nothing in their hands. Volunteers wearing unfamiliar masks, on the other hand, were scolded significantly less often.

Ms. Swift found more signs that dead crows left a strong impression on living ones. In the days after seeing a volunteer with a dead crow, birds took significantly longer to approach food. The sight of a dead pigeon had no such effect.

In their report, which appears in the November issue of Animal Behavior, Ms. Swift and Dr. Marzluff propose that crows pay careful attention to their dead as a way to gather information about threats to their own safety. “It’s a long-term learning opportunity,” said Ms. Swift. “Knowing that you need to be wary in a particular place — that’s valuable.”

The presence of a dead crow could tell other crows that a particular place is dangerous and should be visited only with caution. The loud calls the birds make could be a way to share information with the rest of their group.

"Work like this helps to remind us of the cognitive complexity that exists in animals other than humans," said Teresa Iglesias, an evolutionary biologist affiliated with Australian National University who was not involved in the study.

That's not to say every animal pays attention to its dead, however. In fact, the club is fairly exclusive, including species such as chimpanzees, elephants, dolphins and relatives of crows known as scrub jays.

"It's pretty consistently animals that live in social groups and are known for having more advanced cognitive skills," said Ms. Swift. "It's amazing to think a crow — a bird — is doing something like this that so few other animals are doing that we know."

http://www.eurekalert.org/pub_releases/2015-10/udq-qae092915.php

Glutamate, an essential food for the brain

Glutamate shown to be a source of energy for the brain

Glutamate is an amino acid with very different functions: in the pancreas, it modulates the activity of the pancreatic ß-cells responsible for insulin production, whereas in the brain it is the main excitatory neurotransmitter. In recent years, it has been suspected to play an additional role in the functioning of the brain. By discovering how the brain uses glutamate to produce energy, researchers at the University of Geneva (UNIGE) confirm this hypothesis and highlight unexpected links with the rest of the body. To read in Cell Reports.

Unlike other organs, the brain cannot draw its energy from lipids, an energy resource widely present in the body. The blood-brain barrier, which protects it from the pathogens and toxins circulating in the blood, indeed limits the passage of these lipids. Moreover, while most of the organs in the human body have the ability to store glucose by increasing their mass, the brain, prisoner of the cranial bones, cannot count on these variations in volume. Unable to store its food, it depends on sugar supplied in real-time by the rest of the body. This distribution of energy is controlled by the liver.

Pierre Maechler, professor at the Faculty of Medicine at UNIGE, and his team therefore decided to verify if glutamate was indeed an energy source for the brain. To do so, the researchers analyzed the role of the glutamate dehydrogenase enzyme in the brain. In mutant form, this enzyme, encoded by the Glud1 gene, is responsible for a congenital hyperinsulinism syndrome, a severe disease affecting at the same time the endocrine pancreas, the liver and the brain. Individuals affected by this syndrome suffer from intellectual disability and have a high risk of epilepsy. "We have suppressed the Glud1 gene in the brain of mice. In the absence of glutamate dehydrogenase, we observed that the brain was no longer able to convert glutamate into energy, even though the amino acid was present in the brain," explains Melis Karaca, first author of this study.

Priority to the brain

Devoid of the energy supplied by cerebral glutamate, the brain sends signals to the liver to requisition a compensatory proportion of glucose, at the expense of the rest of the body. This is why the transgenic mice also showed a growth deficit and muscle atrophy. "This clearly shows how the brain works in a just-in-time manner and that each percent of energy resources is essential for its proper functioning," highlights Professor Pierre Maechler. "If a part of this energy disappears, the brain serves itself first and the rest of the body suffers. The liver must then make more glucose by drawing upon muscle protein, resulting in loss of muscle mass. Knowing that the brain uses glutamate as an energy resource allows us to reflect on other ways to overcome a potential shortfall. "

Scientists also suspect a correlation between the Glud1 gene and some neurodevelopmental disorders, particularly epilepsy and schizophrenia. They are currently pursuing their research by introducing in mice the same Glud1 mutation detected in epileptic patients. At the same time, another group is working with schizophrenic patients to assess the way their brain uses glutamate.

http://bit.ly/1VyixNN

Researchers Devise Way to Determine Color from Fossils

Researchers now know how to tell what color an ancient animal was from its fossils

By Danny Lewis

Ancient fossils can reveal all kinds of information: what an animal might have eaten, what it looked like and how big it was, for instance. But for a long time, scientists have struggled to find a way to figure out what color an organism was from its fossilized remains. Now, a group of researchers have devised a method for determining the pigments in the fur of fossilized bats, revealing their true coloration.

Using this new technique they have confirmed what you may have suspected about the coloration of bats. "Well, the bats are brown," molecular paleobiologist Jakob Vinther of the University of Bristol tells Will Dunham for Reuters. "It might not be a big surprise, but that's what these 49-million-year-old bats are. So they looked perfectly like modern bats."

The bats may not have been rainbow-colored or electric green, but the method as described in a paper published in the Proceedings of the National Academy of Sciences last week could help scientists figure out what colors other ancient animals might have been. And it's all thanks to fossilized pigment. "Since so little is preserved in the fossil record, the color of extinct animals has always been left up to artists' interpretations, and important information regarding behavior has

been considered inaccessible," doctoral candidate in geological sciences at Virginia Tech and lead author Caitlin Colleary tells Dunham.

Traces of melanosomes, the organelles that produce the melanin that gives fur and skin their pigment, have been used in the past to figure out what color some dinosaurs and marine reptiles were. Melasomes are shaped differently depending on the color pigment they produce, which makes it easier for researchers to figure out what color a fossilized animal night have been, Dunham writes.

When Vinther first discovered remnants of melanosomes in a fossilized feather in 2008, skeptics argued that he had only identified bacterial remains that were lodged in the fossil. But in the new study, chemical analysis of their structures proved that the object were in fact melanosomes, Cari Romm writes for The Atlantic.

“People had questioned whether you could use the shape of the melanosome to tell anything about the color, because it’s been through a lot. Millions of years in the ground is obviously going to take a toll,” Colleary tells Romm. “So by finding traces of the chemical melanin in association with these structures, we’ve basically confirmed that you can use the shapes of the melanosomes themselves to tell what color something was.”

Thanks to melanosomes, researchers might finally be able to figure out one way or another whether dinosaurs were mottled green or living psychedelic showcases.

http://bit.ly/1Vyj3eO

Volcanoes plus asteroid might have finished off dinosaurs

Earthquakes set off by asteroid may havecaused increased volcanism

With their world in grave danger, the dinosaurs couldn’t catch a break. The famous asteroid or comet that hastened their demise touched down in the middle of a period of climate change caused by burbling volcanoes.

The resulting seismic shock may have then triggered even more eruptions, effectively meaning that a one-two punch killed them off. New evidence based on the most precise dating yet of lava from that time backs this idea.

This interpretation could help bring together two varying schools of thought on what caused one of the largest mass extinctions in our planet’s history at the end of the Cretaceous period, 66 million years ago.

The dominant theory is that the asteroid impact was chiefly responsible for wiping out the dinosaurs, among three-quarters of all species on Earth. The evidence for the impact at that time comes from a worldwide layer of debris and the Chicxulub crater, which is buried under Mexico’s Yucatan peninsula.

Another line of thought is that, regardless of the impact, climate change triggered by vast volcanic eruptions led to the downfall of the dinosaurs. Around that time, a region called the Deccan traps in modern-day India oozed more than a million cubic kilometres of lava over about 800,000 years, releasing sulphur dioxide and carbon dioxide and warming the atmosphere.

One factor supporting the Deccan-traps theory is that giant lava floods have also been pointed to as a likely culprit in the mass extinction at the end of the Permian, and between the Triassic and Jurassic periods. That makes them a popular explanation for global die-offs. Another point in favour of lava floods over the asteroid theory is that other impact craters as big as the Chicxulub one have been found, but they don’t seem to match with extinction events.

But assigning blame between Chicxulub and the Deccan traps has been difficult without precise dates of the lava flows in India relative to the timing of the asteroid impact.

The right timing

In a 2014 study, a team led by Blair Schoene of Princeton University, New Jersey, provided more insight on the timing of the impact in relation to the lava flows. The researchers found that the impact came about 250,000 years after the main Deccan-trap eruptions began and 500,000 years before they finished.

That overlap makes it look like both factors played a role in the downfall of the dinosaurs. “It’s pretty important to say that both did happen, and both alone have the potential to be pretty devastating,” says Schoene. A new study goes even further, arguing that the impact aggravated the volcanoes in the Deccan traps.

By using argon isotopes to date Deccan rocks, Paul Renne of Berkeley Geochronology Center in California and his team think that existing small lava flows became much worse after the Chicxulub impact. “Suddenly the lava flows erupting in the Deccan traps are much thicker and much more widely distributed, from Mumbai all the way to the Arabian coast,” Renne says. “Thousands of kilometres are affected.”

The post-impact lava seems to have erupted less frequently but in larger amounts.

According to a theory published earlier this year by Mark Richards, a colleague of Renne’s at Berkeley, the shock waves from Chicxulub – equivalent to a magnitude-11 earthquake – may have agitated a plume of mantle material that fuelled the Deccan traps from below. Continued eruptions for another half a million years may then have made it harder for ecosystems to bounce back.

“Various marine faunas and floras are suppressed until that changed volcanism dies off,” Renne says. “The recovery of life in the oceans, and actually on land, too, doesn’t begin in earnest until after the volcanism stops.”

Schoene isn’t yet convinced about the causal link between the impact and a second, more violent phase in the Deccan traps. A more detailed calculation of the rates of eruption over time might help. “I would want to see data down through the lava pile,” he says.

But the close relationship in time confirmed by Renne's new work may still help unite advocates and detractors of the impact theory. When trying to decide whether the Deccan traps or Chixculub impact led to the downfall of the dinosaurs, the safest bet for now might be both."There's been a tendency for real polarisation in this area," says Renne. "That kind of thinking just has to stop. Everything happened at the same time, and you can't tease it apart."

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http://bit.ly/1KZqBCi

Ape fossils put the origin of humanity at 10 million years ago

A new analysis of an ape that lived 12.5 million years ago suggests it is a type of gorilla.

If that's true, it means gorillas evolved much earlier than thought, and also pushes back the time when humans split from chimps by about 2 million years.

David Begun of the University of Toronto in Canada reanalysed fossils of *Dryopithecus* apes, which lived in what is now Europe as early as about 12.5 million years ago.

He says that the characteristics of the skull suggest that rather than evolving earlier than the great apes, as was previously thought, *Dryopithecus* was actually a great ape itself.

The angles at which bones in the skull connect, and the way the brain case is connected to the face all point to the conclusion that this was an early gorilla, Begun says.

Orang-utans are the earliest of the apes to have split from the human lineage, thought to be followed by *Dryopithecus*, then gorillas, then chimps.

But if *Dryopithecus* is in fact a gorilla, that puts the species closer to humans and chimps.

Dryopithecus lived about 12.5 million years ago **(Image: E. R. Degginger/SPL)**

Splitting up

The features suggest *Dryopithecus* split from the human lineage about 14 million years ago, Begun says. From that, he says, we can extrapolate that the human lineage split from chimps about 10 million years ago.

That's more than 2 million years earlier than the previous estimate based on the fossil record, but is actually close to recent estimates based on genetic analysis.

"What if *Dryopithecus* – that looks like a little gorilla – really was a little gorilla that had already branched off from humans?" Begun says.

We know the relative times of divergence between gorillas, chimps and humans, he says, so we can use the split of gorillas from the others to recalibrate the fossil clock.

Genetic comparisons can also indicate when species diverged from a common ancestor. They are based on the number of genetic differences between two species, which is proportional to the time that has passed since their last common ancestor was alive. Because of this they are also known as molecular clocks.

Molecular clocks

Current molecular clocks date the split between humans and chimps to at least 7 million years ago, matching the age of the oldest fossil thought to be in the human line, *Sahelanthropus*. But some reports quote molecular dates up to 13 million years.

Begun's figure falls in the middle. "We on the molecular side would be more comfortable with a split of the type he's talking about," says Aylwyn Scally of the University of Cambridge.

Calculating the amount of time elapsed between the last common ancestor and the appearance of two separate and co-existing species depends on the population of the original species: the more individuals, the more distant the last common ancestor.

If the initial population was large, as Scally says the genomes of humans and chimps show was the case with their last common ancestor, several million years could elapse before the two species finally separated, says Scally.

Interbreeding between subgroups, as is thought to have happened between modern humans and Neanderthals, also could delay speciation.

Begun will present his work at the annual meeting of the Society of Vertebrate Paleontology, in Dallas, Texas, later this month.

Palaeontologist Owen Lovejoy of Kent State University in Ohio is cautious about the results, noting Begun has not yet published his findings.

The real test will depend on finding fossils from 9 to 10 million years ago. But that won't be easy.

So far, finds have been few and very incomplete, and with early chimp and human ancestors likely to be very similar, Begun says, it's going to be difficult to decide which line they fall on.

http://www.eurekalert.org/pub_releases/2015-10/du-fr100215.php

Fusion reactors 'economically viable' say experts

Policy makers should start planning to build fusion reactors as a replacement for conventional nuclear power stations

Fusion reactors could become an economically viable means of generating electricity within a few decades, and policy makers should start planning to build them as a replacement for conventional nuclear power stations, according to new research.

Researchers at Durham University and Culham Centre for Fusion Energy in Oxfordshire, have re-examined the economics of fusion, taking account of recent advances in superconductor technology for the first time. Their analysis of building, running and decommissioning a fusion power station shows the financial feasibility of fusion energy in comparison to traditional fission nuclear power.

The research, published in the journal Fusion Engineering and Design, builds on earlier findings that a fusion power plant could generate electricity at a similar price to a fission plant and identifies new advantages in using the new superconductor technology.

Professor Damian Hampshire, of the Centre for Material Physics at Durham University, who led the study, said: "Obviously we have had to make assumptions, but what we can say is that our predictions suggest that fusion won't be vastly more expensive than fission."

Such findings support the possibility that, within a generation or two, fusion reactors could offer an almost unlimited supply of energy without contributing to global warming or producing hazardous products on a significant scale. Fusion reactors generate electricity by heating plasma to around 100 million degrees centigrade so that hydrogen atoms fuse together, releasing energy. This differs from fission reactors which work by splitting atoms at much lower temperatures.

The advantage of fusion reactors over current fission reactors is that they create almost no radioactive waste. Fusion reactors are safer as there is no high level radioactive material to potentially leak into the environment which means disasters like Chernobyl or Fukushima are impossible because plasma simply fizzles out if it escapes.

Fusion energy is also politically safer because a reactor would not produce weapons-grade products that proliferate nuclear arms. It is fuelled by deuterium, or heavy water, which is extracted from seawater, and tritium, which is created within the reactor, so there is no problem with security of supply either.

A test fusion reactor, the International Thermonuclear Experimental Reactor, is about 10 years away from operation in the South of France. Its aim is to prove the scientific and technological feasibility of fusion energy.

Professor Hampshire said he hoped that the analysis would help persuade policy-makers and the private sector to invest more heavily in fusion energy.

"Fission, fusion or fossil fuels are the only practical options for reliable large-scale base-load energy sources. Calculating the cost of a fusion reactor is complex, given the variations in the cost of raw materials and exchange rates. However, this work is a big step in the right direction" he said.

"We have known about the possibility of fusion reactors for many years but many people did not believe that they would ever be built because of the technological challenges that have had to be overcome and the uncertain costs."

"While there are still some technological challenges to overcome we have produced a strong argument, supported by the best available data, that fusion power stations could soon be economically viable. We hope this kick-starts investment to overcome the remaining technological challenges and speeds up the planning process for the possibility of a fusion-powered world."

The report, which was commissioned by Research Council UK's Energy Programme focuses on recent advances in high temperature superconductors. These materials could be used to construct the powerful magnets that keep the hot plasma in position inside the containing vessel, known as a tokamak, at the heart of a fusion reactor.

This advancing technology means that the superconducting magnets could be built in sections rather than in one piece. This would mean that maintenance, which is expensive in a radioactive environment, would be much cheaper because individual sections of the magnet could be withdrawn for repair or replacement, rather than the whole device.

While the analysis considers the cost of building, running and decommissioning a fusion power plant, it does not take into account the costs of disposing of radioactive waste that is associated with a fission plant. For a fusion plant, the only radioactive waste would be the tokamak, when decommissioned, which would have become mildly radioactive during its lifetime.

http://www.medscape.com/viewarticle/851321

Propensity Scores--Here, There, and Everywhere: But Are They Useful?

What Are Propensity Scores?

Aaron B. Holley, MD

I have been meaning to write about propensity scores for some time now. As a pulmonary and critical care physician in the United States, I dutifully read my *Chest* journal whenever possible. The editor sends emails to members of the *Chest* College that highlight important articles as they are published online.

This past week, two of the highlighted studies[1,2] used propensity scores to analyze their data.[3] The next email I received was from the pulmonary medicine fellowship director at my hospital. He had attached the article we will be discussing in our journal club this week. That's right, another study using a propensity score. I figured it was time to talk more about this technique.

Propensity scores are used to reduce confounding in observational studies.[4] When measuring the effect of an intervention using observational data, researchers need to "match" the patients with a population of control individuals who did not receive the intervention. Although it's possible to match for specific covariates (eg, age, race, and sex), two problems will inevitably arise: (1) If the controls are sampled from a comparable group of people who did not receive the intervention, there is a selection bias (unmeasured factor or factors that might cause clinicians to withhold the intervention and that can systemically influence group differences); and (2) depending on the size of the population being sampled, matching each patient to another using specific covariates may reduce the sample size. Only randomization can truly eliminate selection bias, but propensity matching offers a popular solution to both problems.

How Are Propensity Scores Used?

Generating a propensity score starts with identifying covariates that are associated with receiving the intervention being studied. These covariates are entered into a regression model, and a score is derived. The value of the score corresponds to the probability that a patient would receive the intervention. Then the score is used to adjust or match patients with controls before analyzing differences in the outcome under study.

We can take one of the studies published online in *Chest*[1] as an example. Using a large database, the investigators attempted to determine which drug—beta blockers (BBs), calcium channel blockers (CCBs), amiodarone, or digoxin—provides the best outcomes for treating atrial fibrillation (AF) in the presence of sepsis. Among other factors, treatment choice varied by year, geographic location, physician specialty, and hospital. It's not hard to imagine any one of these factors creating bias. What if internal medicine physicians use CCBs and intensivists use BBs to treat AF, but intensivists achieve better sepsis outcomes? Without some sort of matching or adjustment, one might erroneously conclude that BBs lead to better outcomes, when in fact it's the intensivist's care that is driving the difference. The same can be said about myriad other factors that drive prescribing practices and affect outcomes. Propensity scores allow researchers to eliminate all of these recognized biases. Unfortunately, only true randomization eliminates all selection bias, recognized or unrecognized. The volume of covariates used to derive the propensity score in this study is impressive, and the authors claim that their score accounts for 90% of the treatment variation seen in their data set.

Are propensity scores simply another statistical "weapon of mass destruction?" At quick glance and in my opinion, the answer is "no." They aren't nearly as "dirty" as meta-analyses can be. There is nuance, and certain areas deserve more study,[5,6] but propensity scores are powerful tools to help investigators properly interpret observational data. One need only to look at the three studies cited below and see how many covariates influence the selection of an intervention, thus creating bias, to see the value of propensity scores. I expect that we will see this statistical method often moving forward.

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Drug used to treat cancer appears to sharpen memory

Rutgers research provides clues to keeping brain cells alive in those with Alzheimer's

Can you imagine a drug that would make it easier to learn a language, sharpen your memory and help those with dementia and Alzheimer's disease by rewiring the brain and keeping neurons alive?

New Rutgers research published in the Journal of Neuroscience found that a drug - RGFP966 - administered to rats made them more attuned to what they were hearing, able to retain and remember more information, and develop new connections that allowed these memories to be transmitted between brain cells.

"Memory-making in neurological conditions like Alzheimer's disease is often poor or absent altogether once a person is in the advanced stages of the disease," said Kasia M. Bieszczad, lead author and assistant professor in Behavioral and

Systems Neuroscience in the Department of Psychology. "This drug could rescue the ability to make new memories that are rich in detail and content, even in the worst case scenarios."

What happens with dementias such as Alzheimer's is that brain cells shrink and die because the synapses that transfer information from one neuron to another are no longer strong and stable. There is no therapeutic treatment available that reverses this situation.

The drug being tested in this animal study is among a class known as HDAC inhibitors - now being used in cancer therapies to stop the activation of genes that turn normal cells into cancerous ones. In the brain, the drug makes the neurons more plastic, better able to make connections and create positive changes that enhance memory. Researchers found that laboratory rats, taught to listen to a certain sound in order to receive a reward, and given the drug after training, remembered what they learned and responded correctly to the tone at a greater rate than those not given the drug.

Scientists also found that the rodents were more "tuned in" to the relevant acoustic signals they heard during their training - an important finding Bieszczad said because setting up the brain to better process and store significant sounds is critical to human speech and language."People learning to speak again after a disease or injury as well as those undergoing cochlear implantation to reverse previous deafness, may be helped by this type of therapeutic treatment in the future," said Bieszczad "The application could even extend to people with delayed language learning abilities or people trying to learn a second language."

This hypersensitivity in processing auditory information enabled the neurons to reorganize and create new pathways - allowing more of the information they learned to become a long-term memory, said Bieszczad who collaborated with colleagues in the Department of Neurobiology and Behavior at the University of California Irvine. "People normally remember an experi nce with limited detail - not everything we see, hear and feel is remembered," she said. "What has happened here is that memory becomes closer to a snapshot of the actual experience instead of being sparse, limited or inaccurate."

http://www.eurekalert.org/pub_releases/2015-10/uoc--hds100115.php

High-fructose diet slows recovery from brain injury

UCLA study finds diet may predict ability to recover from mental deficits after head trauma

A diet high in processed fructose sabotages rat brains' ability to heal after head trauma, UCLA neuroscientists report. Revealing a link between nutrition and brain health, the finding offers implications for the 5.3 million Americans living with a traumatic brain injury, or TBI. According to the Centers for Disease Control and Prevention, an estimated 1.7 million people suffer a TBI each year, resulting in 52,000 annual deaths.

"Americans consume most of their fructose from processed foods sweetened with high-fructose corn syrup," said Fernando Gomez-Pinilla, a professor of neurosurgery and integrative biology and physiology at UCLA's David Geffen School of Medicine. "We found that processed fructose inflicts surprisingly harmful effects on the brain's ability to repair itself after a head trauma."

Fructose also occurs naturally in fruit, which contains antioxidants, fiber and other nutrients that prevent the same damage.

In the UCLA study, published today in the Journal of Cerebral Blood Flow and Metabolism, laboratory rats were fed standard rat chow and trained for five days to solve a maze. Then they were randomly assigned to a group that was fed plain water or a group that was fed fructose-infused water for six weeks. The fructose was crystallized from corn in a dose simulating a human diet high in foods and drinks sweetened with high-fructose corn syrup.

A week later, the rats were anesthetized and underwent a brief pulse of fluid to the head to reproduce aspects of human traumatic brain injury. After an additional six weeks, the researchers retested all the rats' ability to recall the route and escape the maze. The scientists discovered that the animals on the fructose diet took 30 percent longer to find the exit compared to those who drank plain water.

The UCLA team also found that fructose altered a wealth of biological processes in the animals' brains after trauma. The sweetener interfered with the ability of neurons to communicate with each other, rewire connections after injury, record memories and produce enough energy to fuel basic functions.

"Our findings suggest that fructose disrupts plasticity -- the creation of fresh pathways between brain cells that occurs when we learn or experience something new," said Gomez-Pinilla, a member of the UCLA Brain Injury Research Center. "That's a huge obstacle for anyone to overcome -- but especially for a TBI patient, who is often struggling to relearn daily routines and how to care for himself or herself."

Earlier research has revealed how fructose harms the body through its role in contributing to cancer, diabetes, obesity and fatty liver. Gomez-Pinilla's study is the latest in a UCLA body of work uncovering the effects of fructose on brain function. His team previously was the first to identify the negative impact fructose has on learning and memory.

"Our take-home message can be boiled down to this: reduce fructose in your diet if you want to protect your brain," Gomez-Pinilla stressed.

Sources of fructose in the western diet include honey, cane sugar (sucrose) and high-fructose corn syrup, an inexpensive liquid sweetener. Made from cornstarch,

the liquid syrup is widely added as a sweetener and preservative to processed foods, including soft drinks, condiments, applesauce and baby food.

The average American consumed roughly 27 pounds of high-fructose corn syrup in 2014 -- or just shy of eight teaspoons per day, according to the U.S. Department of Agriculture. That's a drop from a decade ago, when Americans consumed more than 36 pounds of the syrup per year.

Nonetheless, the USDA's Economic Research Service identifies the United States as the world's largest consumer of sweeteners, including high-fructose corn syrup. Though one of the largest global sugar producers, the United States is also among the largest sugar importers.

Gomez-Pinilla's coauthors included Rahul Agrawal, Emily Noble, Laurent Vergnes, Zhe Ying and Karen Reue, all from UCLA.

The work was funded by grants from the National Institute of Neurological Disorders and Stroke (NS050465) and the National Center for Research Resources (NCRR S10RR026744).

http://bit.ly/1M9qRp4

What Fingerprints Can Reveal About Ancestry

Clues as to whether people have European or African lineage may show up in the fine details of their fingers

By Marissa Fessenden

The unique pattern of ridges and dips on fingertips is a well known identifier for individuals, albiet a contested one. But researchers have found that fingerprints can reveal more than just an individual's identity, they can provide clues to a person's ancestral background, reports Michael Casey for *CBS News* (via *Science*).

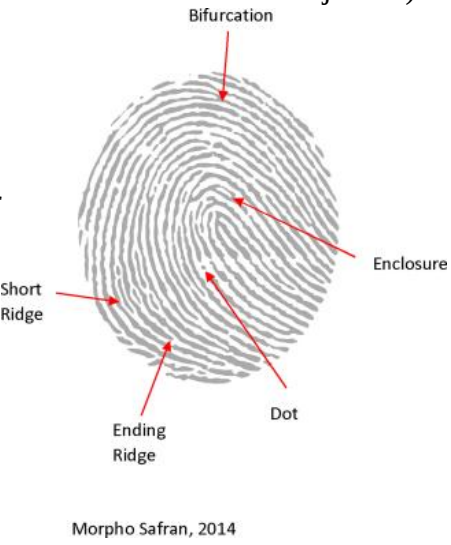
Pattern types.

Researchers at North Carolina State and Washington State universities cracked this code by analyzing the right index finger prints from 243 people who were either African American or European American.

The differences between men and women weren't significant, but the differences in ancestry were. But only when the researchers took into account the details. Experts can assess fingerprints at three levels. Level 1 includes the pattern (such as a whorl, loop or arch) and number of ridges. Level 2 includes finer detail such as bifurcations, or where a ridge splits (this is the level used in criminal justice). Level 3 peers all the way down to the pores.

It was that second level that revealed some differences. Bifurcations were the most significant difference between European Americans and African Americans, the researchers report in the *American Journal of Physical Anthropology*.

"This is the first study to look at this issue at this level of detail, and the findings are extremely promising," says co-author Ann Ross, a professor of anthropology at North Carolina State University, according to Casey. "But more work needs to be done. We need to look at a much larger sample size and evaluate individuals from more diverse ancestral backgrounds."



Minutiae types.

The National Research Council issued a call in 2009 for more rigor and science-backed methods in forensics. They signaled out fingerprint analysis as one of the areas that need work, so Ross says that this research could help. However, she also suspects the work would interest other experts. "[T]here's a level of variation in fingerprints that is of interest to anthropologists, particularly in the area of global population structures - we just need to start looking at the Level 2 fingerprint details," she says.

http://www.eurekalert.org/pub_releases/2015-10/teia-soa092815.php

Signs of ancient megatsunami could portend modern hazard

Evidence of an 800-foot wave in the Cape Verde Islands

Scientists working off west Africa in the Cape Verde Islands have found evidence that the sudden collapse of a volcano there tens of thousands of years ago generated an ocean tsunami that dwarfed anything ever seen by humans. The researchers say an 800-foot wave engulfed an island more than 30 miles away. The study could revive a simmering controversy over whether sudden giant collapses present a realistic hazard today around volcanic islands, or even along more distant continental coasts. The study appears today in the journal Science Advances.

"Our point is that flank collapses can happen extremely fast and catastrophically, and therefore are capable of triggering giant tsunamis," said lead author Ricardo Ramalho, who did the research as a postdoctoral associate at Columbia University's Lamont-Doherty Earth Observatory, where he is now an adjunct

scientist. "They probably don't happen very often. But we need to take this into account when we think about the hazard potential of these kinds of volcanic features."

The apparent collapse occurred some 73,000 years ago at the Fogo volcano, one of the world's largest and most active island volcanoes. Nowadays, it towers 2,829 meters (9,300 feet) above sea level, and erupts about every 20 years, most recently last fall. Santiago Island, where the wave apparently hit, is now home to some 250,000 people.

There is no dispute that volcanic flanks present a hazard; at least eight smaller collapses have occurred in Alaska, Japan and elsewhere in the last several hundred years, and some have generated deadly tsunamis. But many scientists doubt whether big volcanoes can collapse with the suddenness that the new study suggests. Rather, they envision landslides coming in gradual stages, generating multiple, smaller tsunamis. A 2011 French study also looked at the Fogo collapse, suggesting that it took place somewhere between 124,000-65,000 years ago; but that study says it involved more than one landslide. The French researchers estimate that the resulting multiple waves would have reached only 45 feet--even at that, enough to do plenty of harm today.

A handful of previous other studies have proposed much larger prehistoric collapses and resulting megatsunamis, in the Hawaiian islands, at Italy's Mt. Etna, and the Indian Ocean's Reunion Island. But critics have said these examples are too few and the evidence too thin. The new study adds a new possible example; it says the estimated 160 cubic kilometers (40 cubic miles) of rock that Fogo lost during the collapse was dropped all at once, resulting in the 800-foot wave. By comparison, the biggest known recent tsunamis, which devastated the Indian Ocean's coasts in 2004 and eastern Japan in 2011, reached only about 100 feet. (Like most other well documented tsunamis, these were generated by movements of undersea earthquake faults--not volcanic collapses.)

Santiago Island lies 55 kilometers (34 miles) from Fogo. Several years ago, Ramalho and colleagues were working on Santiago when they spotted unusual boulders lying as far as 2,000 feet inland and nearly 650 feet above sea level. Some are as big as delivery vans, and they are utterly unlike the young volcanic terrain on which they lie. Rather, they match marine-type rocks that ring the island's shoreline: limestones, conglomerates and submarine basalts. Some weigh up to 770 tons. The only realistic explanation the scientists could come up with: A gigantic wave must have ripped them from the shoreline and lofted them up. They derived the size of the wave by calculating the energy it would have taken to accomplish this feat.

To date the event, in the lab Ramalho and Lamont-Doherty geochemist Gisela Winckler measured isotopes of the element helium embedded near the boulders' surfaces. Such isotopes change depending on how long a rock has been lying in the open, exposed to cosmic rays. The analyses centered around 73,000 years--well within the earlier French estimate of a smaller event. The analysis "provides the link between the collapse and impact, which you can make only if you have both dates," said Winckler.

Tsunami expert Bill McGuire, a professor emeritus at University College London who was not involved in the research, said the study "provides robust evidence of megatsunami formation [and] confirms that when volcanoes collapse, they can do so extremely rapidly." Based on his own work, McGuire s says that such megatsunamis probably come only once every 10,000 years. "Nonetheless," he said, "the scale of such events, as the Fogo study testifies, and their potentially devastating impact, makes them a clear and serious hazard in ocean basins that host active volcanoes."

Ramalho cautions that the study should not be taken as a red flag that another big collapse is imminent here or elsewhere. "It doesn't mean every collapse happens catastrophically," he said. "But it's maybe not as rare as we thought."

In the early 2000s, other researchers started publishing evidence that the Cape Verdes could generate large tsunamis. Others have argued that Spain's Canary Islands have already done so. Simon Day, a senior researcher at University College London has sparked repeated controversy by warning that any future eruption of the Canary Islands' active Cumbre Vieja volcano could set off a flank collapse that might form an initial wave 3,000 feet high. This, he says, could erase more than nearby islands. Such a wave might still be 300 feet high when it reached west Africa an hour or so later he says, and would still be 150 feet high along the coasts of North and South America. So far, such studies have raised mainly tsunamis of publicity, and vigorous objections from other scientists that such events are improbable. A 2013 study of deep-sea sediments by the United Kingdom's National Oceanography Centre suggests that the Canaries have probably mostly seen gradual collapses.

Part of the controversy hangs not only on the physics of the collapses themselves, but on how efficiently resulting waves could travel. In 1792, part of Japan's Mount Unzen collapsed, hitting a series of nearby bays with waves as high as 300 feet, and killing some 15,000 people. On July 9, 1958, an earthquake shook 90 million tons of rock into Alaska's isolated Lituya Bay; this created an astounding 1,724-foot-high wave, the largest ever recorded. Two fishermen who happened to be in their boat that day were carried clear over a nearby forest; miraculously, they survived.

These events, however, occurred in confined spaces. In the open ocean, waves created by landslides are generally thought to lose energy quickly, and thus to pose mainly a regional hazard. However, this is based largely on modeling, not real-world experience, so no one really knows how fast a killer wave might decay into a harmless ripple. In any case, most scientists are more concerned with tsunamis generated by undersea earthquakes, which are more common. When seabed faults slip, as they did in 2004 and 2011, they shove massive amounts of water upward. In deep water, this shows up as a mere swell at the surface; but when the swell reaches shallower coastal areas, its energy concentrates into in a smaller volume of water, and it rears up dramatically. The 2004 Indian Ocean earthquake and tsunami killed 230,000 people in 14 countries; the 2011 Tohoku event killed nearly 20,000 in Japan, and has caused a long-term nuclear disaster.

James Hunt, a tsunami expert at the United Kingdom's National Oceanography Centre who was not involved in the study, said the research makes it clear that "even modest landslides could produce high-amplitude anomalous tsunami waves on opposing island coastlines." The question, he said, "is whether these translate into hazardous events in the far field, which is debatable."

When Fogo erupted last year, Ramalho and other geologists rushed in to observe. Lava flows (since calmed down) displaced some 1,200 people, and destroyed buildings including a new volcano visitors' center. "Right now, people in Cape Verde have a lot more to worry about, like rebuilding their livelihoods after the last eruption," said Ramalho. "But Fogo may collapse again one day, so we need to be vigilant."

In addition to his post at Lamont-Doherty, Ramalho is now based at the United Kingdom's University of Bristol. The study's other authors include Joerg M. Schaefer of Lamont-Doherty; José Madeira and Rui Quartau of the University of Lisbon; George Helfrich of the Tokyo Institute of Technology; Ana Hipólito of Portugal's Universidade dos Acores; and Katherine Adena of Bristol.

The paper, "Hazard Potential of Volcanic Flank Collapses Raised by New Megatsunami Evidence," is available from Science Advances: 202-326-6440

http://www.eurekalert.org/pub_releases/2015-10/bl-amt093015.php

Asthma medications taken during infancy linked to stunted growth

Infants given asthma medications during their first 2 years of age are likely to be stunted in later life

Infants given asthma medications during their first 2 years of age are likely to be stunted in later life, according to research presented today at the 54th Annual European Society for Paediatric Endocrinology Meeting. The findings highlight the importance of using these medicines in infants appropriately.

Inhaled corticosteroids (ICS) - medications used to treat conditions such as asthma - are frequently used in infants with recurrent wheezing. However, these medications may have harmful effects, for instance a reduced growth rate in development and a shorter height in adulthood.

In this study, researchers from Kuopio University Hospital and University of Eastern Finland analysed information on the height, weight and asthma medicine intake of 12,482 Finnish children aged 0-24 months. The researchers found that children who used inhaled corticosteroids during the first 2 years of life were too short for their age. This result was more evident in children taking the asthma medicine budesonide for more than 6 months.

Many factors that alter development in children, such as chronic illnesses and long-term use of oral corticosteroids, may cause a shorter than normal height in adulthood. "Previously, the impact of corticosteroids on growth was looked at in older children and was thought to alter growth only temporarily," said lead researcher Dr Antti Saari. "However, studies on inhaled corticosteroid use in infants are practically lacking and thus this has been questioned in the recent study. Our research shows a link between long-term treatment of ICS during infancy and stunted growth at or after the age of 2 in otherwise healthy children."

The group will next focus on assessing the impact of inhaled corticosteroids on growth in older children and observe them for longer time periods. "According to our research, we could only assess the impact of inhaled corticosteroids on growth in infancy until 2 to 3 years of age. The longitudinal impact of these medications is not clear and we would therefore like to investigate this further," said Dr Saari.

http://bit.ly/1VysHxS

Big Data Are Reducing Homicides in Cities across the Americas

City leaders across the Americas are exploiting science to reduce homicide

By Rodrigo Guerrero Velasco | Sep 15, 2015

In Brief

An epidemiological approach of data analysis can reveal the root causes of violence and the best steps to curtail it.

In Cali, Colombia, the method reduced homicides from 124 per 100,000 inhabitants to 86 in just three years. In Bogotá, the rate dropped from 80 to 20 over nine years.

Changes in gun and alcohol laws were crucial. So were increasing police presence and giving youth social activities and jobs.

Today numerous cities across the Americas hold regular meetings of all crime agencies to analyze data, plan interventions and evaluate them.

Violence is a big problem in modern society and in cities in particular. Homicides were rampant in my hometown of Cali, Colombia, when I became mayor in 1992. Few people saw murder as a pressing health problem, but I did—probably

because I had earned a Ph.D. in epidemiology at the Harvard School of Public Health. I decided to apply the statistical methods used by public health experts to identify the sources of homicide and to reveal social and policy changes that might make a difference.

At the beginning of my first term, the people of Cali and all of Colombia generally believed, mistakenly, that little could be done because we Colombians were "genetically violent." Other skeptics maintained that violent crime would not diminish unless profound changes were made on socioeconomic issues such as unemployment and educational levels. My administration and I proved all these people wrong.

We developed an epidemiological database about the many societal factors that significantly raised the risk that a homicide would happen. These included sometimes subtle aspects of human behavior, such as the desire to carry guns in certain places or the tendency to drink alcohol on certain days. This exhaustive and fine-grained information led to new laws and policies built on data, not politics.

The method worked. In 1994 annual homicides in my city, then home to nearly 1.8 million, dropped from 124 per 100,000 residents to 86 in just three years after the leading causes were found and policies were applied. An even larger decline took place over nine years in Bogotá, after our capital city adopted the same methods. And when I was elected mayor of Cali for a second time, in late 2011, after being out of office for almost 18 years, the same approach reduced homicide rates again. Let me tell you the story of how big data and scientific analysis can help solve entrenched social problems.

Pinpoint the root causes

When I began my first term, I did what epidemiologists generally do: plot cases on a map. I hung a big printout of Cali on my office wall and stuck color-coded pins in it at each location of a death, intentional injury, traffic accident, home burglary or other violent event. When a journalist saw the map, his local newspaper ran a headline that read: "Mayor Guerrero Intends to Curb Violence with Acupuncture."

Even to smart journalists, evidently, it was strange to look at homicide in a statistical way. But to me, it made perfect sense: if epidemiological methods could find the causes of medical diseases, they could find the causes of a societal disease.

Using statistics was crucial because Colombia had a long record of violence that left many misimpressions. Beginning in the late 1940s, La Violencia, a fierce struggle for power between the two main political parties, sparked over 200,000 killings across more than 10 years. Guerrilla warfare followed for decades. The cultural tolerance for violent responses to conflict was so high when I took office that quarrels between neighbors or drivers in traffic accidents frequently ended in homicide. In 1991 Medellín, the second-largest city in Colombia, had an annual homicide rate of 380 per 100,000. Around that time, Chile's rate was 2.9.

My epidemiological approach began with a definition of violence scripted by the World Health Organization: the use of force with the intention to cause harm or death. This definition does not include accidents or psychological or political violence.

Despite the media's preoccupation with domestic warfare, only 36 percent of the deaths in Colombia in 1991 were caused by guerrillas, mostly in rural areas. I thought drug dealers would arise as the culprits in the other 64 percent. As we investigated the who, where and when of each death in Cali, however, we found that homicide victims and aggressors were predominantly young, unemployed males who had low levels of education, came from the poorer sectors of the city and were frequently involved in gang fights. We also found that close to 80 percent of homicides were carried out with firearms. When we discovered that two thirds of homicides took place on weekends, we decided to chart blood alcohol levels in victims; more than half of them had been intoxicated. These facts pointed to social disintegration more than drug-related violence.

Drug traffic still had an effect, but it was not the direct cause of most homicides. As we analyzed the numbers, we realized that drug traffic was to society as HIV is to the human body: the virus attacks defense mechanisms, making the body vulnerable to other diseases. Likewise, drug dealers attack the police and the judiciary and political systems—the defense mechanisms of society. These weakened institutions arose as risk factors for violence. For example, the police identified a suspect in only 6 percent of homicides, and the judiciary system brought even fewer to trial.

Also, children were often exposed to violence and maltreatment, and violent content was prevalent in the media. In a culture of violence, economic inequality and ineffective public security, people killed and got killed, often under the influence of alcohol, over conflicts as simple as noisy neighbors or settling debts.

Change the culture

Our goal was to reveal risk factors we could control directly. Because firearms were used in a large proportion of homicides and alcohol was often associated with the deaths, in November 1993 I began to change gun and alcohol laws.

In my country, guns are manufactured and sold by the Colombian National Army, so military authorities opposed my idea of a permanent ban on weapon-carrying permits. But they agreed to our suspending the permits in public places on select dates identified by the data as posing a high risk, which was generally associated

with high alcohol consumption. These dates included New Year's Eve and (strangely) Mother's Day, as well as days when payments to employees, made on the 15th and 30th of each month in Colombia, coincided with a Friday.

I also restricted alcohol sales in public places after 2 A.M.—a measure my administration called the semidry law. Nightclub owners objected adamantly, so I proposed a deal: I would apply the law for three months, and if violent deaths and injuries did not diminish, I would drop it. After only two weeks, hospitals reported such a drastic reduction in violence-related emergencies that abandoning the measure was not an option. I enforced the semidry law until the end of my term.

An epidemiological strategy also calls for evaluating interventions. After several months, we found that when both alcohol sales and firearms permits were restricted, there was a 35 percent reduction in homicides versus days when neither were in force. The reduction was 14 percent when firearms alone were restricted.

Other interventions included adding more prosecutors, as well as putting more police on the streets and improving their equipment, such as surveillance cameras, cars and radios. To support these people in their challenging careers, we launched a privately funded program to help police officers become homeowners and gave computers and training to members of the judiciary. Crime prevention rose, and more suspects were brought to trial.

We also created two Houses of Justice—premises within violent neighborhoods on the outskirts of Cali in which all law-enforcement institutions operated 24 hours a day. Previously these services were available only downtown and during business hours. This change was particularly helpful in reducing domestic violence because investigations would begin immediately after forensic medical personnel certified a victim's injuries, which lessened the chance that women would withdraw their complaints under pressure from their husbands. In an effort to offer young males in poor districts greater opportunities for education, recreation, income and social connections, I launched DESEPAZ—a program to restore public safety by improving the cohesiveness of a neighborhood. As part of the program, we opened “youth houses” in several communities where people could socialize and gather around cultural and sports activities. City workers also trained youth who were involved with gangs to run small businesses. The city even hired one such business dedicated to manufacturing cobblestones to pave streets.

Improve the data

We realized early on that the data we were working with were not always cohesive. For example, in my first security council meeting in July 1992, it became clear that the police and judiciary used different definitions of homicide, which complicated our efforts to pin down causes of deaths. To fix the issue, I established weekly security meetings that involved officials from the police, judiciary and forensic authorities, members of the Institute for Research and Development in Violence Prevention and Promotion of Social Coexistence (CISALVA) at the University of Valle, cabinet members responsible for public safety, and the municipal statistics agency. Information was reported weekly to me and to police commanders. We held a security council meeting every week of my term. Slowly the data coalesced. The meetings evolved into “observatories of crime,” sometimes called “social observatories.” CISALVA, which is dedicated to studying violence prevention, has kept the observatory's weekly data running for 22 years—to my knowledge, the longest reliable set of information on violence in any Colombian city.

Based on the improved analysis of risk factors, we began interventions at the end of 1993 and widened them before my two-and-a-half-year period as mayor ended in December 1994. My successor continued them. The homicide rate in Cali dropped from 124 per 100,000 in 1994 to 112 in 1995, 100 in 1996, and 86 in 1997. It is difficult to say how much of the decline was a direct result of the interventions because the national government was also changing how police fought drug cartels. But evaluations in Cali and Bogotá confirm that the epidemiological approach played an important role. I believe that is true in part because the mayors who followed my successor did not keep in place unpopular measures such as the restriction of alcohol consumption, and the homicide rate climbed back up.

Experience in Bogotá, the country's largest city, backs up the data-intensive method. When Antanas Mockus became mayor there in January 1995, he applied and improved our strategy. His most important tactical interventions were increasing the police budget 10-fold, improving police education about violent crime, developing temporary detention centers for minor offenders and creating a government position of subsecretary of violence prevention. The social interventions included rebuilding dilapidated public spaces and tripling investment in health and education.

Mockus also implemented a semidry law and restrictions on firearms, which quickly reduced homicide rates as much as they had in Cali. In Bogotá, strict use of the epidemiological method spanned three administrations over nine years, from 1995 through 2003. Across that time, homicide rates dropped from 59 per 100,000 to 25. As in Cali, some of that improvement may have been helped by changes at the national level.

New tactics, 20 years later

In Colombia, mayors cannot be reelected consecutively (and I had other plans anyway). After I left office, I dedicated myself to spreading the word that urban violence could be controlled and to doing further research about that goal. I went to work at the Pan American Health Organization in Washington, D.C., was instrumental in actions that created the Inter-American Coalition for the Prevention of Violence and helped to garner approval of a loan from the Inter-American Development Bank to Cali, Medellín and Bogotá for deterring violence. After three years, I returned to Cali and helped to launch VallenPaz, an organization devoted to creating economic programs in rural southwestern Colombia as an alternative to the lure of money from guerrillas and illicit drug crops.

Years later, however, I found that there is no lifelong immunity to politics. I ran again for mayor of Cali.

When I took office on January 1, 2012, I found a different city. Cali had grown from 1.8 million inhabitants in 1994 to 2.4 million. Most of the additional people were migrants, primarily from Colombia's Pacific coast and neighboring rural areas. After years of incompetent administrations and one mayor ousted from office, collective self-esteem was low, and unemployment was up from 6.9 percent in 1994 to 13 percent in 2013. Although the large Colombian drug cartels were dismantled in the 1990s, they had fragmented into smaller cartels that worked rather independently in the nation's cities, particularly in Medellín and Cali. Drug dealing was still present, and new forms of crime had emerged, such as small “vaccine” payments required by gangs to protect local businesses and war over the territorial control of drug distribution and selling within cities.

The good news was that the Colombian police had become professional and trustworthy. The national homicide rate had dropped from 79 in 1991 to 36 in 2011. Yet Cali's homicide rate was around 80, compared with 22 in Bogotá and 70 in Medellín.

I immediately reinstated the weekly security council meetings. Soon our data analyses showed that the proportion of homicides resulting from interpersonal conflict such as quarrels and alcohol-related brawls had diminished compared with the period of 1992 to 1994. But killings that we classified as organized crime—those that were premeditated and involved sophisticated weapons such as machine guns—accounted for 67 percent of violent deaths in 2012. Data suggested that organized crime was playing a bigger role. The data also showed that social inequalities had gotten worse since my earlier term.

We presented our data to the national government and suggested it create specialized groups of criminal investigators, police and prosecutors to dismantle criminal bands. My administration also began a massive social investment plan in 11 districts that were home to a total of 800,000 people, 26 percent of them living in poverty and another 6.5 percent in extreme poverty.

The plan that resulted, called Territories of Inclusion and Opportunities, is still in effect today. It applies a geographical approach to fighting poverty, focusing interventions in impoverished areas and encouraging local residents to play big roles. Local and national officials work on raising incomes, extending school schedules, promoting cultural activities and sports, and improving housing, health facilities and public education. We also teach parenting skills and peaceful conflict resolution.

Together with the effort from the national government to fight organized crime, our interventions again reduced violence. Cali's homicide rate of 83 in 2012 dropped to 62 in 2014. This pattern has continued; the number of homicides in the first trimester of 2015 is less than in the same period in any of the past 12 years.

All these coordinated police and social actions help the crime interventions. A good example of the strategy is Comuna 6, a political district of Cali where 212,000 residents, most of whom are middle-income, live. We energetically implemented the coordinated police and social interventions, and homicides went down 44 percent within a year's time, from 160 in 2013 to 89 in 2014.

The epidemiological approach to reducing violence is passing the test in other cities in Colombia and across the Americas. Crime observatories—the evolution of our regular security council meetings—are essential to the approach. The Inter-American Development Bank, the U.S. Agency for International Development and the World Bank, among others, now recommend that cities or states create the observatories when seeking financial support for violence-prevention programs. Today four national and numerous municipal-level observatories are meeting systematically in 26 countries and cities in the Americas.

A study published in the International Journal of Injury Control and Safety Promotion found that homicides were significantly reduced in 22 Colombian cities in the three-year period after the observatories were implemented. Studies directly comparing cities in different countries are difficult, however, because countries have diverse definitions of crimes and varying criteria for collecting information. To improve the situation, the Inter-American Development Bank is supporting a project to standardize violence indicators across the Americas.

Political will is the top priority

Using an epidemiological strategy to help solve a social issue may seem straightforward, but it is not. The first lesson I can espouse is that such a move takes strong political will because the strategy frequently requires public officers to do things they would rather not do, such as making necessary but unpopular decisions to close bars or ban firearms. Making crime data public can also be

uncomfortable, but it is essential, just as economists releasing unemployment and gross domestic product numbers is essential to formulating economic strategy.

Data on social issues such as violence and education are now published periodically for various Colombian cities by nonprofit groups called Bogotá How Are We Doing, Cali How Are We Doing, and so on. The information makes public officials and mayors accountable in their communities.

The second lesson is that there is no one-size-fits-all approach in applying epidemiological methods to social issues because cities and countries have different risk factors. Data-driven observation is needed in each context to guide public officials.

The process also requires perseverance and patience. Certain risk factors can be controlled rapidly—for example, by banning firearms or restricting bar hours—but other measures, such as improving the reach of police and judiciary services, take longer. Steps such as correcting social inequalities or establishing healthy child-rearing practices need not only time and patience but also considerable resources.

Urban violence is socially regressive because it mostly affects the poor, and fighting crime devours a portion of the public budget, which could instead be invested to eradicate poverty. Violence prevention must therefore be a priority for humanity.

Rodrigo Guerrero Velasco has been mayor of Cali, Colombia, since 2012. He was also mayor from 1992 to 1994. After his first term, he worked for the Pan American Health Organization and helped to start ValleNPaz to create economic programs in guerrilla-infested and illicit drug–producing rural Colombia.

The Evaluation of a Surveillance System for Violent and Non-Intentional Injury Mortality in Colombian Cities. Maria Isabel Gutierrez-Martinez et al. in International Journal of Injury Control and Safety Promotion, Vol. 14, No. 2, pages 77–84; 2007.

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Human Brain Project Web site: www.humanbrainproject.eu

http://nyti.ms/1JMPQWz

The Folly of Big Science Awards

By emphasizing the importance of scientific breakthroughs, prizes diminish the way that great medical advances build on one another

ON Monday, the Nobel Prize in Physiology or Medicine will go to a few scientists for work that untangles the intricacies of the human body and may advance treatments for cancer, heart disease or other major illnesses. The prize comes with a sizable check and virtually ensures that the winners' research will be well funded for the rest of their careers.

Every recent recipient has undoubtedly deserved the honor. But that doesn't mean that prizes for medical research are a good idea.

The Nobel, along with the Dickson, Lasker-DeBakey, Canada Gairdner and other major awards, honors the scientists who are usually in the least need of recognition and funding, which squeezes out opportunities for other scientists.

More important, by emphasizing the importance of scientific breakthroughs — serendipitous occurrences that rely on decades of research — these prizes play down, and diminish, the way that great medical advances build on one another.

All scholarship is, to some extent, built on prior work — but this is especially true in scientific research. Consider James P. Allison, the winner of this year's Lasker-DeBakey prize in clinical medical research. His work helped clarify one way cancer cells hide from the immune system.

Around 1990, a team of scientists found a protein on the surface of immune cells and proposed that it stimulated the immune system. Dr. Allison's lab and a third group suggested that the protein put the brakes on immune responses. A fourth group confirmed that it halted the immune system, rather than stimulating it.

Dr. Allison later showed that blocking this protein with an antibody could unleash an immune response in animals that could lead not only to rejection of but also immunity to many kinds of cancers. A decade later, similar antibodies to this protein and other related ones were found to prevail against several types of human cancers.

Dr. Allison's work is surely impressive. But it occurred alongside and in dialogue with a number of related findings. Researchers analyzed the citations that led to Dr. Allison's drug and concluded that it relied on work conducted by 7,000 scientists at 5,700 institutions over a hundred-year period. Yet only he was recognized.

The prize industry contributes to a deeper problem in scientific research: We throw resources at a privileged few who have already achieved enormous fame.

One study that tracked funding for university professors and researchers over an eight-year period found that about 80 percent of research funds in basic medical sciences were concentrated among the top fifth of researchers.

This is bad for the long-term health of the discipline: After top scientists retire, who will replace them? We should be giving more support to midcareer scientists whose work will contribute to major advances in the future.

And there's yet another problem. By honoring breakthroughs, award committees reinforce the misconception that science is all about discoveries, when the

cornerstone of science is replication and corroboration of results, which ensure that a finding is real and not a false lead.

We especially need to dispel this myth now because the scientific community is in the midst of a replication crisis. Nearly all published medical papers report significant or positive results, but many efforts to duplicate the findings failed, putting subsequent research in doubt.

The regular occurrence of false leads also hints at the enormous role serendipity plays in discoveries, which some Nobel Prize winners have acknowledged in their acceptance speeches.

In one study of 101 basic science discoveries published in top journals that claimed a drug had promise, just five led to approved drugs. Even the most promising research may never translate into actual medicine. This means that a majority of creative, persistent and passionate scientists do not win awards, and may advance their fields only incrementally, if at all.

That's because science is hard. It's like exploring an unknown land; we'll never know whether over the next hill lies an expansive vista or just another hill. A finding that seems mundane or trivial may become immensely important years later when a parallel discovery contextualizes or clarifies its implications.

Medical research is even more elusive. We seek not only to understand the inner workings of human biology, but also to perfect the body and manipulate it to our desires. And, unlike physics, it can't be advanced by purely theoretical work, or by a single individual.

If we keep giving prizes, let's award them to experiments with rigorous methods — large sample sizes, representative populations, appropriate controls and blinded experiments that eliminate subconscious bias — instead of ones that achieve headline-grabbing results. Great scientists can control all these things, but they can't control the outcome.

Or we could break up big prizes and give out many smaller awards.

This may be more effective in supporting science, a view shared by Terence Tao, a mathematician who won $3 million from the inaugural Breakthrough Prize in Mathematics but tried to talk the man who gave it to him into spreading it around to more people.

Alternately, instead of giving out big science awards, let's use the prize money to study better ways to fund science.

All the winners of this year's Nobel Prizes deserve praise. But the most important scientists are the ones who demand better experimental design and pursue the truth, regardless of how things turn out.

http://www.wired.com/2015/10/battle-genome-editing-gets-science-wrong/

The Battle Over Genome Editing Gets Science All Wrong

CRISPR is currently at the heart of a bitter patent fight

Nobel prize speculation, gossip, and betting pools kick off every fall around the time Thomson Reuters releases its predictions for science's most prestigious prize. This year, one prediction was unusual: a genome-editing tool so hyped that it even got on the cover of WIRED.

(No, seriously, how often does molecular biology get to occupy the same space as *Star Wars* or Rashida Jones?)

The tool, Crispr/Cas9, is essentially a pair of molecular scissors for editing DNA, so precise and easy to use that it has taken biology by storm. Hundreds if not thousands of labs now use Crispr/Cas9 to do everything from making super-muscled pigs to snipping HIV genes out of infected cells to creating transgenic monkeys for neuroscience research. But the Nobel prediction stands out for two reasons: First, the highly-cited paper describing Crispr/Cas9 came out a mere three years ago, a blip in the timescale of science. Second, the technique is currently at the heart of a bitter patent fight.

Thomson Reuters bases its predictions on how often key papers get cited by other scientists. Here, the paper in question has as its authors Jennifer Doudna, a molecular biologist at UC Berkeley, and Emmanuelle Charpentier, a microbiologist now at the Max Planck Institute for Infection Biology. Missing is Feng Zhang (no relation to this writer), a molecular biologist at the Broad Institute and MIT, who actually owns the patents for CRISPR/Cas9 and says that he came up with the idea independently. So let's say Thomson Reuters gets it right. Could the patent for a discovery go to one scientist, and the Nobel prize for the discovery to someone else?

The two groups—or their patent lawyers, really—are in fact fighting over credit for CRISPR/Cas9. At stake are millions of dollars already poured into rival companies that have licensed patents from the two different groups.

But putting aside all the lawyers and all the money for a moment, obsessing over finding the one true origin of Crispr/Cas9 gets science all wrong. Casting the narrative as Doudna versus Zhang or Berkeley versus MIT is a misapprehension of history, creativity, and innovation. Discovery comes not from a singular stroke of genius, but an incremental body of research. "I'm not a great believer in the flash-of-genius theory. If you are a historian—" says Mario Biagioli, who is in fact a historian of science at UC San Diego—"you quickly will realize exactly how many times there are independent discoveries of the same thing." The dispute over credit for CRISPR/Cas9 is not the result of exceptional coincidence and disagreement. In fact, it illuminates how science always works.

The Other Crispr Scientist

The story of how Doudna, Charpentier, and Zhang came to discover Crispr/Cas9 has been told many times, including by WIRED. So I want to tell a different story—a largely forgotten one, about Crispr's early days.

Virginijus Siksnys is a molecular biologist at Vilnius University in Lithuania. He got interested in Crispr in 2007, when scientists working with yogurt bacteria first realized that odd repeats in their DNA—the "clustered regularly interspaced short palindromic repeats" that give Crispr its name—are in fact part of an ancient microbial immune system that fights viruses.The bits of DNA between the repeats were viral sequences, essentially mug shots for for the pathogens.

The bacteria also had Crispr-associated proteins (the "Cas" in "Cas9") that seemed to use these mug shots to cut up the genetic material of invading viruses.

"In my lab we didn't know how to make cheese or yogurt, but we know how to work with E. coli," says Siksnys.

So his lab took the Crispr and Cas sequences from yogurt bacteria and stuck them inside *E. coli* cells, which made those bacteria suddenly immune to some viruses. In *E. coli*, the researchers could delete the Cas genes one by one, and by 2012, Siksnys had honed in on one in particular, which coded for Cas9, solely responsible for snipping DNA.

In May, they submitted a paper detailing exactly how Cas9 cuts DNA to the *Proceedings of the National Academy of Sciences*. Peer reviewers came back with questions and that back and forth took a few months—typical of peer review.

Here is where the more famous narrative intersects. That June, a month after Siksnys' lab submitted their paper, Doudna and Charpentier's paper came out in *Science*—with many of the same findings as Siksnys'. (The key difference is that Doudna and Charpentier's paper shows that the two pieces of RNA that Cas9 needs to work can be fused into one chimeric segment.)

Science's editors, who obviously saw something big on their hands, fast-tracked the paper's review, and published it within a month of submission. The paper made a huge splash.

"Of course, we were disappointed," says Siksnys. His paper came out in *PNAS* in September to less fanfare. By then, Crispr/Cas9 was off to the races. Zhang and George Church of Harvard published papers in February of 2013 showing that Crispr/Cas9 could alter human cells in a dish; their work also further refined Cas9's DNA-editing abilities.

Then the US patent office awarded Zhang the patent, even though Doudna had filed first, sparking a fight between the University of California and the Broad and MIT. The US Patent and Trademark Office is trying to work it all out. (Doudna and Zhang declined to comment for this story.)

So while everyone is arguing about whether Doudna and Charpentier or Zhang deserve credit for discovering Crispr, popular accounts of the discovery—WIRED's included—have left out Siksnys' contribution. His paper also has received a fraction of the citations that Doudna's has. "Yes, I think of course my lab deserves credit because what we discovered was done independently in two labs," Siksnys says. "It's a very competitive field," he adds diplomatically. "It's part of the game."

Part of the Game

The eminent sociologist Robert Merton, who made a career out of studying scientists, writes about how every field of research builds upon an "accumulated cultural base." (Real catchy, I know.) What he means is that discoveries don't drop out of the air—they're products of their time.

Siksnys, Doudna, Charpentier, and Zhang all cracked Crispr/Cas9 around the same time because they all built on the same research from yet other scientists who figured what Crispr actually is. The 2007 paper kicked off a race. "People were working on the Crispr system," says Dana Carroll, a gene-editing expert at the University of Utah who was paid to write a technical analysis in support of Doudna's patent.

"They were kind of inching toward what the Doudna and Charpentier group finally demonstrated." Doudna and Charpentier published first, by a hair.

Dan Voytas, a gene-editing expert at the University of Minnesota, credits yet other researchers, like Carroll, who worked on earlier gene-editing systems that made the insight into Cas9 as a tool even possible. Figuring out that a DNA-cutting protein like Cas9 could be used to edit DNA is actually not a no-brainer. (You can only do so much with scissors and no glue.) Carroll and other researchers, working on another gene-editing technique called zinc-finger nucleases, found that when you cut DNA, one of two things can happen: The cell will 1) try to repair the cut by adding gibberish letters of DNA, rendering target gene useless or 2) insert a snippet of DNA chosen by the researcher.

That second one is way better. Without this work, no one would have been able to tell how useful Crispr/Cas9 could be.

By the early 2010s, the two lines of inquiry into Crispr and into gene-editing systems met. It was CRISPR/Cas9's time. Scientists had their accumulated cultural base. (Yeah, no, still not catchy.)

None of this history diminishes the hard work or intellectual acuity of individual scientists. Mentioning Siksnys' research does not diminish Doudna and Charpentier's. Mentioning Douda and Charpentier's research doesn't diminish Zhang's.

History is full of parallel discoveries: Isaac Newton and Gottfried Leibniz independently discovered calculus in the late 17th century and then spent years fighting over who got there first. Charles Darwin and Alfred Russel Wallace both came up with the theory of evolution through natural selection, though these two had a more amiable relationship.

Back in 1922, the sociologists William Ogburn and Dorothy Thomas catalogued 150 examples of independent discovery and invention. Merton even went so far as to say single discoveries are the real oddities.

Scientists naturally flock to the interesting scientific problems of their time, and again naturally, they use the tools of their time to solve them. No wonder they often come up with the same solutions.

The problem is, though, is that Nobel prizes go to a maximum of three people, and patents only to one group of inventors. Journalists want one good story rather than a tangle of characters. If you've found keeping track of all the names in this story difficult, well, yes.

The Negotiation

Ultimately the messy process of science gets reduced to a single moment. "A discovery is not always an absolute discrete moment, but something that has to be negotiated," says Nathaniel Comfort, a historian of medicine and science at Johns Hopkins University. People come to the table with different amounts of power. "That has a lot to do with ego, storytelling ability, and clout within the field. Who are the people who have the most power and get listened to?" says Comfort.

When asked why Doudna's paper has so eclipsed Siksnys', Carroll noted it was in fact published first. But also, "it may have something to do with the fact that Jennifer Doudna was very accomplished and known in the molecular biology community before this Crispr breakthrough."

Doudna may not be a household name among non-scientists, but she was already a big shot for earlier groundbreaking work on RNA. Zhang, on the other hand, has a reputation as a wunderkind, having worked on optogenetics (another discovery tipped to win a Nobel prize someday), an earlier genome-editing tool called TALENs, and now Crispr/Cas9—all before the age of 35.

Both researchers also have powerful institutional PR machines behind them. The Broad Institute has built an educational site with a timeline and a press release for a recent Crispr paper that other researchers have actually been criticized for minimizing Doudna's work.

But Berkeley's press releases about Doudna's work do not exactly give much credit to other groups either. Press releases are, by definition, self-promotion.

Passive-aggressive, aggrandizing moves like these are hardly unusual. In *Slate*, for example, Laura Helmuth wrote about the National Institutes of Health's curious decision to celebrate the anniversary of the sequencing of the human genome two years after the more widely agreed-upon date—all to play up the NIH over J. Craig Venter's Celera. And just a couple weeks ago, *Nature* reported on two rival groups fighting over the possible discovery of a protein that lets animals sense magnetic fields. Why such a big deal? Because it might win a Nobel prize, one of the researchers told the journal.

With few exceptions, though, most of the scientists I've ever spoken to have been happy to credit their predecessors and collaborators. Scientists are well aware that they, as Newton put it, stand on the shoulders of giants. That's why journal articles cite previous journal articles. But when the science meets patent law or the popular press or Nobel prizes, those nuances get lost.

Conventional wisdom says that it's probably way too early for Crispr/Cas9 to win a Nobel prize next week. Its true potential—in curing human diseases—is still just potential.

And last week, Zhang's lab reported finding another Crispr system that uses a different protein to cut DNA, which not only gives his lab its own free-and-clear discovery but, more importantly, suggests researchers might be able to find a whole library of editing proteins. As one scientist put it, the discoveries to date might just be "the tip of the iceberg."

The story of Crispr is only just beginning, but the scramble to write it is already well underway.

http://bit.ly/1FRP8fv

Altering how sperm develop could lead to a reversible male birth control

Blocking a certain protein in male mice prevented the rodents from impregnating any females

By Loren Grush on October 4, 2015 02:47 pm @lorengrush

It may be possible to create male birth control by altering how sperm develop, according to new research published this week in Science. Researchers blocked a protein that plays a role in sperm production — and that was enough to render male mice temporarily infertile. Since drugs that alter this protein are already on the market, development of male birth control could happen swiftly if the method works in people too.

In the study, scientists genetically altered mice so the rodents' reproductive cells didn't express certain genes. This caused the male mice's sperm to develop abnormally, preventing the sperm from fertilizing any eggs. Here's the important part: the effect could be recreated using certain drugs.

"Currently the strongest option for men is a vasectomy"

Nowadays, the burden of birth control is usually placed on women, for whom many forms of hormonal birth control already exist. Currently the strongest option for men is a vasectomy, which prevents sperm from leaving the testes. This option, in addition to requiring a surgery, is mostly permanent. Condoms can be an option for men, but they can break and can also decrease sensitivity. Another product in development is called Vasalgel, which is awaiting approval by the Food and Drug Administration; it's not permanent but does block sperm from passing through the penis for 10 years.

So an oral, reversible male contraceptive may be a much more attractive route for both men and women. It would also give couples more options to help decide which form of birth control works best for them."The development of new approaches that will enable couples to share birth control responsibilities ... has been an unmet need for a long time," said Lee Smith, chair of genetic endocrinology at the University of Edinburgh, who was not involved in the study.

This new method described in Science could help meet that need. An enzyme called calcineurin, which also plays a role in the immune system, is known to affect male fertility. Today's finding suggests that there's a sperm-specific version of the enzyme, found only in cells that produce the gamete, says study author Masahito Ikawa, a researcher at the Institute for Microbial Diseases at Osaka University.

"The mutant sperm had rigid tails"

The sperm-specific version has two crucial genes turned on. So scientists created a group of mice that didn't have the sperm-specific enzyme, by knocking out those genes. These mutant mice were healthy, and they were able to have sex and ejaculate normally. But the male mice couldn't impregnate any of the females. Using a high-speed camera, the researchers analyzed the mice's semen; the mutant sperm had rigid tails, making it harder for them to swim. The sperm were also unable to penetrate the egg's membrane.

In a second part of the study, normal mice were treated with medication that block calcineurin; these drugs are often taken as pills to treat rheumatoid arthritis. (They're also given to organ transplant patients to stave off rejection of the donor organ.) After four to five days of receiving the medications, the male mice became infertile. When the mice stopped receiving the drugs, they were fertile again a week later.

While these findings are promising, mice obviously aren't people — so the research may not hold up in clinical trials. Though medication to block calcineurin is already available, these drugs aren't designed to specifically target sperm production; the compounds would probably need to be altered to keep men healthy. But the research gives experts hope that a reversible — and less invasive — form of male birth control could be available soon. The fact that medications to block calcineurin are already on the market means the drug approval process could be relatively quick, according to Smith. "This should expedite development of a contraceptive," he said.

http://newsonjapan.com/html/newsdesk/article/113897.php

Japan to overhaul Fujita scale on tornado intensity

A revised version of the Fujita scale

The Japan Meteorological Agency has drafted a revised version of the Fujita scale, an international standard used to estimate the wind speeds of tornadoes from the extent of damage to property.

The draft, submitted to Friday's meeting of a study group of the agency, calls for adding data on damage to wooden houses, vending machines and other structures for such estimates.

The planned overhaul is intended to enable speedier and more precise estimates so that effective countermeasures are worked out, at a time when the number of tornadoes has been increasing in Japan.

The agency will formally adopt the revised Fujita scale at the study group's next meeting, slated for late December, at the earliest. It hopes to use the revised scale from fiscal 2016, which begins next April.

http://nyti.ms/1VAoDNN

Eye Treatment Closes In on Being First Gene Therapy Approved in U.S.

Success in a late-stage clinical trial in treating an inherited eye disease

By ANDREW POLLACK OCT. 5, 2015

What could become the first gene therapy to win approval in the United States moved closer to market on Monday, when its developer announced that the medicine had succeeded in a late-stage clinical trial in treating an inherited eye disease that can cause blindness.

The developer, Spark Therapeutics, said the treatment had allowed people with certain so-called inherited retinal dystrophies to more easily maneuver in dimmer light than they could before. The company said it planned to apply to the Food and Drug Administration next year for approval to sell the product.

"We saw substantial restoration of vision in patients who were progressing toward complete blindness," Dr. Albert M. Maguire, a professor of ophthalmology at the University of Pennsylvania and a lead researcher in the study, said in a news release being issued by Spark.

Dr. Katherine High, Spark's president and chief scientific officer, said this was the first successful randomized, controlled trial for any gene therapy aimed at an

inherited disease. “I’ve been working in gene therapy for most of my career,” she said. “It’s been a long time coming, and I’m delighted.”

Besides encouraging the once beleaguered field of gene therapy, the results — if interpreted positively by investors — could help lift biotechnology stocks, which have been battered recently by concerns over a backlash against high drug prices.

Still, much remains unknown. Spark did not provide the actual trial data, saying only that the treatment achieved the main goal of the study as well as two out of three of its secondary goals. It is also unclear what the F.D.A. will deem sufficient for approval of the product. Spark’s stock had slumped in the last two months as it changed how it would measure the results of the trial.

Gene therapy involves putting healthy genes into the body to take the place of mutated genes that cause disease. There have been hundreds of trials of gene therapy in humans since 1990, and none have resulted in a medicine winning approval from the F.D.A. One gene therapy for an extremely rare disease was approved in Europe in 2012, but there are questions about its effectiveness.

In the last few years, prospects and results have improved and several new gene therapy companies have gone public, including Spark. Even so, two gene therapy companies, Celladon and Avalanche Biotechnologies, had disappointing trial results recently.

Spark’s product, called SPK-RPE65, is aimed at retinal diseases caused by mutations in a gene called RPE65; this gene plays a role in maintaining the health of the photoreceptors in the eye. Spark estimates there are 3,500 people in the United States and five major European countries with these conditions, though the treatment would not be expected to help people whose disease had progressed past a certain point.

The 31 patients in the study, ranging in age from 4 to 44, have one type of Leber’s congenital amaurosis, which causes night blindness and an erosion of peripheral vision, and can eventually lead to total blindness.

Twenty-one of the participants were randomly assigned to have a virus carrying the RPE65 gene implanted into their eye via a surgical procedure. The 10 others, the control group, received no treatment.

The main measure of effectiveness was how much light participants needed to successfully navigate an obstacle course of sorts — following black arrows on a white tile floor, going up and down steps, and avoiding objects like wastebaskets. There were seven possible levels of illumination ranging from that of a moonless summer night to that of a brightly lit office.

One year later, those who had received the treatment improved by an average of two light levels, meaning they could complete the course in dimmer light than before, Dr. High said. That was better than those in the control group by a statistically significant amount, though she declined to provide the results for the control group. Two-thirds of those in the treated group were able to complete the course in the dimmest light, the level corresponding to a moonless summer night.

The company said there were no serious side effects or immune system reactions in the trial.

“It’s very exciting,” said Gordon Gund, chairman of the Foundation Fighting Blindness, which helped pay for the development of the therapy and some earlier studies of the therapy. He said that while the condition Spark is treating is rare, “this really provides us a platform for many other successful gene therapies.”

One question is how long the effect will last. Theoretically, gene therapy could provide a permanent fix. (Spark claimed the ticker symbol “ONCE” when it went public early this year.)

But some academic groups that tried a similar RPE65 gene therapy reported earlier this year that the effect wore off after one or more years.

Jeffrey D. Marrazzo, chief executive of Spark, said the effects from earlier, small trials of his company’s therapy had, so far, generally lasted for several years. Some of those earlier results were substantial: One boy, who had relied on canes and an aide in the classroom, became able to play baseball and read the blackboard.

In the new trial, the treatment did not achieve one of the secondary goals — improving visual acuity, as measured by reading letters on an eye chart, by a statistically significant amount.

Dr. Julia A. Haller, ophthalmologist in chief at the Wills Eye Hospital in Philadelphia and president of the Retina Society, said that visual acuity was “not an endpoint that would be expected to improve dramatically,” because the therapy was aimed at night vision and peripheral vision.

Dr. Haller, who was an unpaid consultant in the trial, said the presentation of data from the trial would be the “hottest thing” on the program at the Retina Society’s annual meeting in Paris this weekend. “To get to this point, for this to come to fruition, is huge,” she said.

The therapy was originally developed by Dr. High and her colleagues at the Children’s Hospital of Philadelphia. She co-founded Spark in 2013. In a somewhat unusual move, the hospital invested more than $30 million to help the company get started. The hospital now owns more than a third of the company, a stake worth close to $400 million.