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BEHAVIOR • BRAIN SCIENCE • INSIGHTS

The Power of an Open Mind

How it helps you see the world differently, get creative and think outside the box

The Science of Hot Sex • Mental Illness Everywhere • Chatting with Fido

FROM THE EDITOR

An Open Book

Generally speaking, psychology holds that five major factors, or traits, shape our personalities: openness to experience, conscientiousness (facility with planning ahead), extraversion (being sociable), agreeableness (including being considerate of others) and neuroticism (subject to worry). Each of these "big five" has half a dozen dimensions. If you're like me, you probably enjoy taking those free online tests to see where you fall on each.

It's perhaps immediately obvious that our personalities shape our responses to things that happen to us. Someone who is agreeable is going to make an effort when a surprise visitor pops by, for instance. But variations in our personality traits may also mean we may actually experience the world differently than others do.

Consider being "open-minded." In an experiment, Luke Smillie, a psychologist who is director of the Personality Processes Lab at the University of Melbourne in Australia, and his colleagues found that people who score higher on that character trait "may literally *see the world differently* from the average person." Their brains let in information that others filter out, helping to drive creative responses. Find out more here about the study in Smillie's feature article, "Openness to Experience: The Gates of the Mind."

More new ideas await in this issue: about "How Poverty Affects the Brain," the ways "Eyewitness Memory Is a Lot More Reliable Than You Think," and which 22 genes are associated with "Intelligence and the DNA Revolution." All you need to do is keep an open mind.

Mariette DiChristina
Editor in Chief

COVER BY BAONA; THIS PAGE: SPENCER HEYFRON

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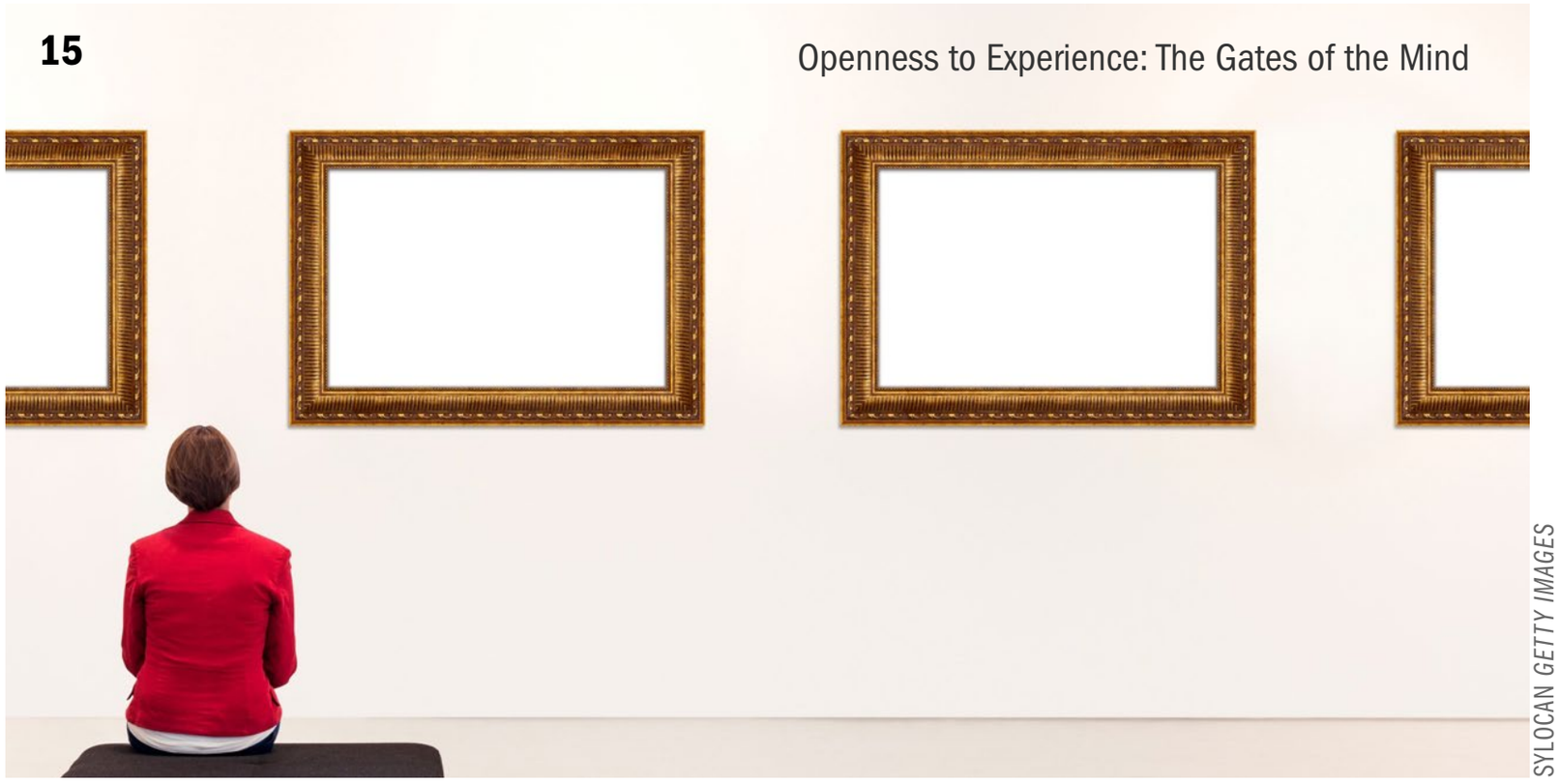
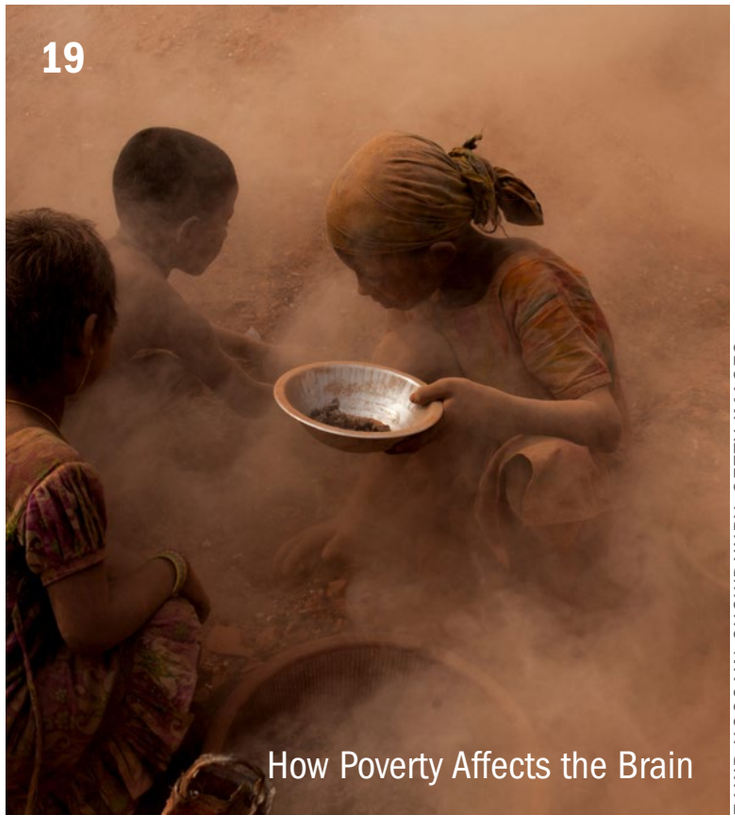


SYLOCAN *GETTY IMAGES*



ZAKIR HOSSAIN CHOWDHURY *GETTY IMAGES*

MICHELE BANKS

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ADAM GAULT *GETTY IMAGES*

NEWS

Brain’s Stem Cells Slow Aging in Mice

Transplanted cells offer middle-aged rodents an increased life span

Stem cells in the brain could be the key to extending life and slowing aging. These cells—which are located in the hypothalamus, a region that produces hormones and other signaling molecules—can reinvigorate declining brain function and muscle strength in middle-aged mice, according to a study published in August in *Nature*.

Previous studies have suggested that the hypothalamus is involved in aging, but the latest research shows that stem cells in this region can slow the process. That makes sense because the hypothalamus is involved in many bodily functions, including inflammation and appetite, says Dongsheng Cai, a neuroendocrinologist at the Albert Einstein College of Medicine and a co-author of the study.

In their study, Cai and his colleagues found that stem cells in the hypothalamus disappear as mice grow older. When the researchers injected their mice with viruses that destroy these cells, the animals seemed to grow older faster, experiencing declines in memory, muscle strength, endurance and coordination. They also died sooner than untreated mice of the same age.

Next, the team injected stem cells taken from the hypothalami of newborn mice into the brains of middle-aged mice. After four months, these animals had better cognitive and muscular function than untreated mice of the same age. They also lived about 10 percent longer, on average.

The researchers found that these stem cells release molecules called microRNAs, which help to regulate gene expression, into the cerebrospinal fluid. When the team injected these microRNAs into the brains of middle-aged mice, they found that the molecules slowed cognitive decline and muscle degeneration.

Forever Young

It is an interesting paper, says Leonard Guarente, a molecular biologist at the Massachusetts Institute of Technology, who studies aging and was not involved in the work. He adds that it could lead to various ways of developing antiaging therapies in people.

Stem cell therapies might enhance the ability of the hypothalamus to act as a master regulator, given that the latest results suggest it controls aging through signaling peptides such as hormones and microRNAs, Cai says. He adds that his team is trying to identify which of the thousands of types of microRNA produced are involved in aging and hopes to investigate whether similar mechanisms exist in nonhuman primates.

The findings represent a breakthrough in aging research, says Shin-ichiro Imai, who studies aging at Washington University in St. Louis and was not involved in the study. The next steps would be to link these stem cells with other physiological mechanisms of aging, he notes. For instance, these cells may have a role in regulating the neurons that release a hormone called gonadotropin-releasing hormone (GnRH), which is secreted by the hypothalamus and is associated with aging. Imai would also like to know whether the microRNAs from the cells can pass into the bloodstream, which would carry them throughout the body.

Cai suspects that antiaging therapies targeting the hypothalamus would need to be administered in middle age, before a person's muscles and metabolism have degenerated beyond a point that could be reversed.

It is unclear by how much such a therapy could extend a human life span, but Guarente says that slowing the effects of aging is the more important goal. "Living longer isn't important if you're not healthy," he says.

—Sara Reardon, *Nature* magazine

The primate brain has an uncanny ability to recognize faces. Now researchers are starting to crack that code.

GETTY IMAGES

NEWS

How We Save Face—Researchers Crack the Brain's Facial-Recognition Code

A Caltech team has deciphered the way we identify faces, re-creating what the brain sees from its electrical activity

The brain has evolved to recognize and remember many different faces. We can instantly identify a friend's countenance among dozens in a crowded restaurant or on a busy street. And a brief glance tells us whether that person is excited or angry, happy or sad.

Brain-imaging studies have revealed that several blueberry-size regions in the temporal lobe—the area under the temple—specialize in responding to faces. Neuroscientists call these areas "face patches." But neither brain scans nor clinical studies of patients with implanted electrodes explained exactly how the cells in these patches work.

Now, using a combination of brain imaging and single-neuron recording in macaques, biologist Doris Tsao and her colleagues at the California Institute of Technology appear to have finally cracked the neural code for primate face recognition. The researchers found the firing rate of each face patch cell corresponds to a separate facial feature. Like a set of dials, the cells can be fine-tuned to respond to bits of information, which they can then combine in various ways to create an image of every face the animal encounters. "This was mind-blowing," Tsao says. "The values of each dial are so predictable that we can re-create the face that a monkey sees by simply tracking the electrical activity of its face cells."

Previous studies had hinted at the specificity of these brain areas for encoding faces. In the early 2000s, when Tsao was a postdoctoral researcher at Harvard Medical School, she and electrophysiologist Winrich Freiwald showed that neurons in a monkey's face patches would fire electrical signals every time the animal saw pictures of a face. But the same brain cells showed little or no response to other objects, such as images of vegetables, radios or nonfacial body parts. Other experiments indicated that neurons in these regions could also distinguish among individual faces, even if they were cartoons.

In a famous set of experiments in human subjects in 2005, neuroscientist Rodrigo Quian Quiroga found that pictures of actor Jennifer Aniston activated a single brain cell in the hippocampus region—the so-called Jennifer Aniston neuron. A similar process was thought to occur elsewhere in the temporal lobe, where the prevailing theory held that each neuron in the face patches was sensitive to a few particular people, says Quian Quiroga, who is now at the University of Leicester in England and was not involved with the current work. But Tsao's recent study suggests that theory may be mistaken. "She has shown that neurons in face patches don't encode particular people at all; they just encode certain features," Quian Quiroga says. "That completely changes our understanding of how we recognize faces."

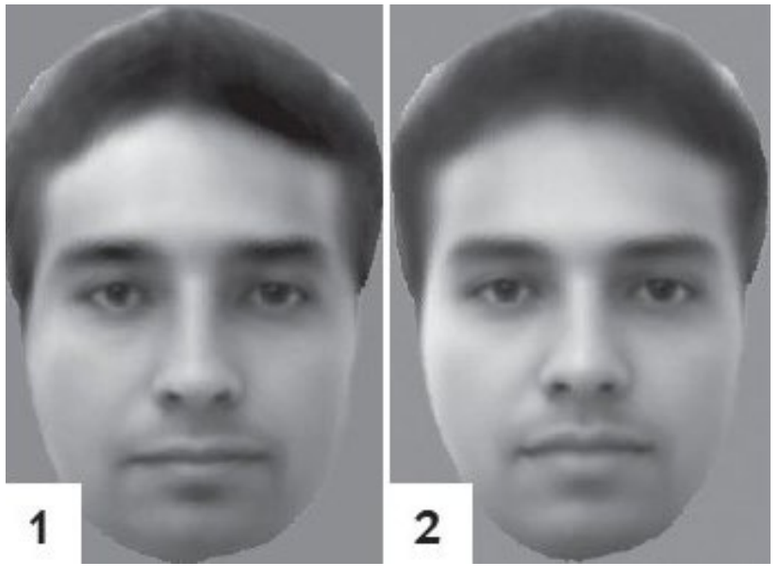
To decipher how cells perform this recognition task, Tsao and postdoc Steven Le Chang generated 2,000 human mug shots with variations in 50 features, including facial roundness, distance between the eyes, and skin tone and texture. They showed these images to two monkeys while recording electrical activity from individual neurons in three separate face patches in both animals.

Each neuron responded to only a single feature, the researchers found. Rather than encoding individuals' faces, like the Jennifer Aniston neuron in the hippocampus, the face patch neurons were dividing images into smaller regions and encoding specific features such as hairline width, Chang says. Moreover, the neurons in separate

face patches processed complementary information. Like factory workers, the various face patches had distinct jobs, cooperating, communicating and building on one another to provide a complete picture.

Once Chang and Tsao knew how this division of labor occurred, they could predict the neurons' responses to a completely novel face. They developed a mathematical model in which facial features were encoded by various neurons. Then they showed monkeys a previously unseen image of a human face (*1*). Using their algorithm for how various neurons would respond, the researchers were able to digitally re-create the visage that a monkey had viewed (*2*). "The re-creations were stunningly accurate," Tsao says. In fact, they were nearly indistinguishable from the actual pictures the monkeys saw.

Even more surprisingly, the researchers needed readings from only a relatively small set of neurons for the algorithm to accurately re-create the faces monkeys were seeing, Tsao says. Recordings from just 205 cells—106 in one patch and 99 in another—were enough. "It really speaks to how compact and efficient this feature-based neural code is," she says. It may also explain why primates are so good at facial recognition and how we can potentially distinguish among billions of different people without needing an equally massive number of face cells.



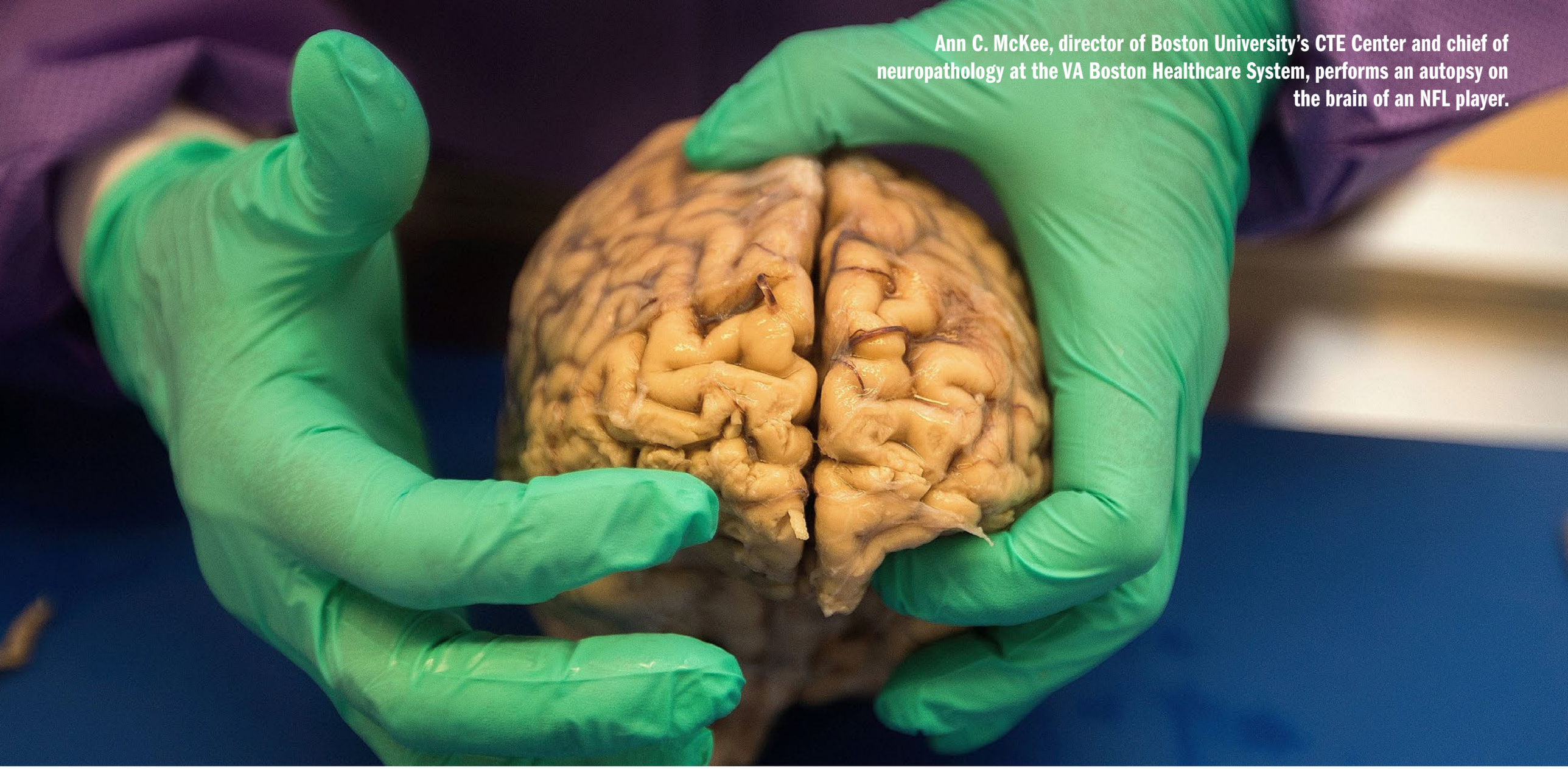
The original face (*1*) presented to a monkey and the face predicted by its brain activity (*2*).

The findings, which were published recently in *Cell*, provide scientists with a comprehensive, systematic model for how the brain perceives faces. This human cerebral machinery is very similar to that of monkeys, and we have face patches that respond like theirs to images in functional MRI studies, according to researchers. Yet the number of human face patches might differ.

Understanding the brain's facial code could help scientists study how face cells incorporate other identifying information, such as sex, age, race, emotional cues and names, says Adrian Nestor, a neuroscientist at the University of Toronto, who studies face patches in human subjects and did not participate in the research. It may even provide a framework for decoding how the brain processes nonfacial shapes. "Ultimately this puzzle is not just about faces," he explains. "The hope is that this neural code extends to object recognition as a whole."

—Knvul Sheikh

COURTESY OF STEVEN LE CHANG *CALIFORNIA INSTITUTE OF TECHNOLOGY*



Ann C. McKee, director of Boston University's CTE Center and chief of neuropathology at the VA Boston Healthcare System, performs an autopsy on the brain of an NFL player.

STAN GROSSFELD *GETTY IMAGES*

NEWS

Striking Evidence Linking Football to Brain Disease Sparks Calls for More Research

The biggest study of its kind offers the best evidence to date linking the sport to mood and cognitive impairments

The controversy began about 10 years ago, when it emerged that the National Football League had first tried to cover up evidence linking repetitive head injuries in players to chronic traumatic encephalopathy (CTE), a progressive neurodegenerative disorder, and then to discredit the scientists doing the work. Since then, evidence supporting this link has grown as an increasing number of players have come forward to report that they are suffering from depression, and some have committed suicide. And yet exactly how repetitive head injuries are linked to CTE development and the psychiatric symptoms associated with it is still a matter of debate.

The largest ever study of its kind has now given the most compelling evidence yet linking repetitive head impacts in football players to CTE. The study, published recently in *JAMA*, has notable limitations, however. It has also sparked calls for more research to measure the impact of head blows on players over the course of a lifetime.

The new work builds on findings from 2013: neuropathologist Ann C. McKee of Boston University and her colleagues published a postmortem report of 68 male athletes and military veterans with CTE, in which they described a spectrum of pathological signatures associated with the condition. McKee and her colleagues observed two distinct sets of clinical symptoms: one involving disturbances in mood and behavior, which was seen in the younger subjects, and the other including cognitive impairments, which developed at an older age.

Based on its findings, the team proposed a number of pathological criteria for the diagnosis of CTE, similar to those for Alzheimer's disease. Their proposal also identified stages by which these pathologies and the behavioral symptoms associated with them become more severe with time. According to the criteria set out by the researchers, early symptoms of CTE include headaches and attention deficits, progressing later to depression and short-term memory loss, then to cognitive impairments and finally to full-blown dementia and aggression.

In this latest study, the researchers examined the brains of 202 deceased football players, all of which had been donated to a brain bank created to investigate the long-term effects of repetitive head injuries in athletes, military personnel and victims of domestic abuse. They obtained detailed medical histories for all the subjects, measured the volume of their brain and then dissected the organ to look for CTE-related neuropathology.

Of the 202 participants examined in the study, 177 were diagnosed with CTE, based on the previously described pathological characteristics. They had an average of 15 years of experience with the game, at the high school, college or professional level. Their median age of death was 67 years, with the most common causes being neurodegenerative disease (39 percent), cardiovascular disease (19 percent) and suicide (10 percent). The severity of CTE pathologies was associated with the intensity of play, with all the former high school players exhibiting mild CTE pathology and more than half of the former college, semiprofessional and professional players exhibiting severe pathologies.

Behavioral and mood symptoms were common in all the former NFL players diagnosed with CTE, having occurred in almost all the 26 individuals with mild pathology and 75 of the 84 individuals with severe pathology. Other common symp-

toms included anxiety, depression and impulsivity. Verbal aggression, physical violence, suicidal thoughts and substance abuse had also occurred in the majority of those with mild CTE pathology, but post-traumatic stress disorder was uncommon, occurring in only three cases with mild pathology and nine cases of former players who were more severely affected.

Despite the high proportion of study participants exhibiting CTE pathology, the authors urged caution in interpreting their results. One important limitation of the study is the biased sample—brains that were donated for the specific purpose of examining links between head trauma and CTE. It does not necessarily follow that the frequency of CTE in the wider population of people exposed to repetitive head injuries is as high as that found in the study. Nor is the sample representative of the overall population of football players, most of whom play only on youth or high school teams. "Our sample has very clear ascertainment bias," says study co-author Bobby Abdolmohammadi, a research assistant in McKee's laboratory. "That is, families may have been motivated to donate the brains of their loved ones because they saw symptoms or were aware of the link between repetitive head trauma and football, [so] we can't jump to any conclusions about the frequency of the disease in the general population or among football players at this point."

Abdolmohammadi adds that determining the precise nature of the relationship between repetitive head injuries and CTE would require well-designed longitudinal studies. Such research may finally begin to determine the true impact of repetitive concussion on brain structure and function.

Still, experts say the new findings are striking. "This is an extremely important and convincing piece of work, [which] leaves no doubt that there is a serious issue in American football," says John Hardy, a professor of neuroscience at University College London, who was not involved with the study. "We now have very good evidence there is a problem…, which needs to be addressed immediately in terms of rule changes to reduce head contact if the sport is to survive."

Hardy acknowledges that the new results do not determine the extent of the problem, however. "As the authors are clear in pointing out, the cases were selected based on clinical suspicion of CTE," he says, adding that a broader effort is now needed. Hardy calls for more studies of both active and retired players that look for acute evidence of brain damage using scans and by measuring possible damage in blood and cerebrospinal fluid. "We need systematic data to understand how big the problem is," he says.

—Mo Costandi

An autoradiograph of a DNA strand, used to identify its base sequence.

ANDREW BROOKES *GETTY IMAGES*

NEWS

Intelligence and the DNA Revolution

Scientists identify 22 genes associated with intelligence

More than 60 years ago Francis Crick and James Watson discovered the double-helical structure of deoxyribonucleic acid—better known as DNA. Today, for the cost of a Netflix subscription, you can have your DNA sequenced to learn about your ancestry and proclivities. Yet while it is an irrefutable fact that the transmission of DNA from parents to offspring is the biological basis for heredity, we still know relatively little about the specific genes that make us who we are.

That is changing rapidly through genome-wide association studies—GWASs, for short. These studies search for differences in people's genetic makeup—their "genotypes"—that correlate with differences in their observable traits—their "phenotypes." In a GWAS recently published in *Nature Genetics*, a team of scientists from around the world analyzed the DNA sequences of 78,308 people for correlations with general intelligence, as measured by IQ tests.

The major goal of the study was to identify *single-nucleotide polymorphisms*—or SNPs—that correlate significantly with intelligence test scores. Found in most cells throughout the body, DNA is made up of four molecules called *nucleotides*, referred to by their organic bases: cytosine (C), thymine (T), adenine (A) and guanine (G). Within a cell, DNA is organized into structures called *chromosomes*. Humans normally have 23 pairs of chromosomes, with one in each pair inherited from each parent.

A SNP (pronounced "snip") is a nucleotide at a particular chromosomal region that can differ across people. For example, one person might have the nucleotide triplet TAC, whereas another person might have TCC, and this variation may contribute to differences between the people in a trait such as intelligence. Genes consist of much longer nucleotide sequences and act as instructions for making proteins—basic building blocks of life.

Of the more than 12 million SNPs analyzed, 336 correlated significantly with intelligence, implicating 22 different genes. One of the genes is involved in regulating the growth of neurons; another is associated with intellectual disability and cerebral malformation. Together the SNPs accounted for about 5 percent of the differences across people in intelligence—a nearly twofold increase over the last GWAS on intelligence. Examining larger patterns of SNPs, the researchers discovered an additional 30 genes related to intelligence.

As a check on the replicability of their results, the scientists then tested for correlations between the 336 SNPs and level of education—a variable known to be strongly correlated with intelligence—in an independent sample of nearly 200,000 people who had previously undergone DNA testing. Ninety-nine percent of the time, the SNPs correlated in the same direction with education as they did with intelligence. This finding helps to allay concerns that the SNPs associated with intelligence were false positives—in other words, caused by chance. More substantively, the finding adds to the case that some of the same processes underlie intelligence and learning. The authors concluded that the results "provide starting points for understanding the molecular neurobiological mechanisms underlying intelligence, one of the most investigated traits in humans."

As neuroscientist Richard J. Haier discusses in his excellent new book *The Neuroscience of Intelligence* (Cambridge Uni-

versity Press, 2017), other intelligence research is combining molecular genetic analyses and neuroimaging. In one study, using a sample of 1,583 adolescents, researchers discovered a SNP implicated in synaptic plasticity that was significantly related both to intelligence test scores and to cortical thickness, as measured by magnetic resonance imaging. In animal research, other researchers are using chemogenetic techniques to turn "on" and "off" neurons that may be important for intelligence.

Of course, intelligence is not solely the product of DNA—and no scientist studying intelligence thinks otherwise. The environment has a major impact on the development of intelligence or any other psychological trait. All the same, knowledge gained from molecular genetic research may one day be used to identify children at risk for developing serious intellectual deficits and those for whom certain types of interventions early in life may reduce that risk. This research is also providing a scientific foundation for thinking about how brain functioning might be manipulated to enhance intelligence.

The big picture to emerge from research on the neurobiological underpinnings of intelligence and other psychological traits is that the nature versus nurture debate is, once and for all, over. We are a product of both our genetic makeup and our environments, as well as the complex interplay between the two. Research aimed at better understanding this interplay will give scientists a richer understanding of the similarities and differences in our psychological makeup.

— Alexander P. Burgoyne,
David Z. Hambrick

Openness to Experience:
The Gates of the Mind

People who are “open to experience” literally see the world differently

By Luke Smillie

SYOLACAN *GETTY IMAGES*

What does it mean to be "open-minded"? Are some people genuinely more inclusive in their thinking, more expansive in how they process information? Experiments in personality psychology show that open-minded people do indeed process information in different ways and may literally *see the world differently* from the average person.

The personality trait that best reflects the lay concept of open-mindedness is called "openness to experience," or simply "openness." Open people tend to be intellectually curious, creative and imaginative. They are interested in art and are voracious consumers of music, books and other fruits of culture. They also tend to be politically liberal.

According to personality theorists, openness reflects a greater "breadth, depth, and permeability of consciousness" and propensity to "cognitively explore" both abstract information (ideas and arguments) and sensory information (sights and sounds). In other words, open people engage with the various percepts, patterns and perspectives that clamor for space in our mind—information is like catnip for their brain.

These abstract notions may well seem like academic hand waving, but they are anchored in concrete data from many research studies. For example, consider the superior performance by open people on tests of creativity called divergent thinking tasks. These require individuals to generate multiple, diverse solutions to a simple problem, such as: "How many uses can you think of for a brick?" Less open people typically generate fewer and more obvious answers to this question—building walls, building houses, building other stuff. But for highly open people, the possibilities flood in. A brick can be used as a weapon, a paperweight, a replacement leg for a broken sofa. Or it can be smashed up and mixed with water to make paint. Open people see more possibilities in even the most mundane of objects.

We see something similar in studies of latent inhibition, a process also known as learned irrelevance. Learning what to ignore is critical for effective psychological functioning—it would be simply overwhelming to process the full stream of information available to our senses as we make our way through the world. So we cull through this information for relevant details, screening out everything else. The problem is, the screened-out information might be useful later, but by then we are slow to realize its significance, to *unlearn* its irrelevance. This process can be modeled in the laboratory by preexposing participants to seemingly unimportant stimuli that later form the basis of a learning task. For the average person, this preexposure stifles subsequent learning—the critical stimulus has been rendered "irrelevant" and fails to penetrate awareness. Not so, however, for those high in openness, who are less susceptible to latent inhibition. This again demonstrates a more inclusive mode of thinking—a "leaky" cognitive system, if you will—that lets in information that others filter out.

These studies show that open people are less susceptible to the psychological "blind spots" that help us pare back the complexity of the world. And research shows that

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Luke Smillie is a senior lecturer in psychology at the University of Melbourne in Australia and has published numerous research papers on personality and individual differences. He was awarded his Ph.D. from the University of Queensland in Australia, has held positions at the University of London and is now director of the Personality Processes Lab at Melbourne.

this characterization is more than a metaphor: open people *literally see things differently* in terms of basic visual perception.

Consider inattentional blindness—the screening out of visual information beyond our attentional focus. You have experienced this if you have ever been so preoccupied with one thing that you failed to see something else right in front of your eyes. (Smartphone-jabbing pedestrians dawdling along the bike path, this means you.) In a classic study often dubbed the "Invisible Gorilla" test, researchers showed participants a film clip of several people passing a basketball back and forth and asked them to count the number of passes between players in white and to ignore the players in black. During the film, someone in a gorilla costume wandered in among the players. In full view, this hairy interloper looked into the camera, beat its chest and drifted off again. Amazingly, most participants in this study reported that they did not see anything unusual or surprising during the clip. Highly open people, on the other hand, are less susceptible to inattentional blindness: they tend to see the things that others block out.

My colleagues and I at the University of Melbourne in Australia have explored these ideas further. In one recent study, we examined links between openness and a perpetual phenomenon called binocular rivalry. This occurs when one image is presented to our left eye while a different image is presented to the right eye. Because the brain cannot extract a coherent picture from these incompatible percepts, the two images seem to flip back and forth in our mind's eye, each image rivaling the other for dominance. But sometimes both images do break through into conscious perception as a scrambled mash-up. In our study, we found that open people perceived this "mixed percept" for longer periods. It is as though the gates of perception are agape, allowing more visual information to flow into consciousness for open people.

We have also examined how these findings extend to a very different kind of experience called mixed emotions—the simultaneous experience of contrasting feeling states (bittersweetness, nervous excitement, and so on). Might open people also be susceptible to such experiences, to have seemingly incompatible *feelings* break through into conscious experience, analogous to the two percepts in binocular rivalry? Indeed, we found that such individuals do report experiencing mixed emotions more frequently in their lives. This may be another example of the "permeability of consciousness," in this case giving rise to complex emotional experiences.

What is happening in the brains of open people to produce these distinctive experiences? Here our knowledge is far murkier and less certain, the neuroscience of personality being a fraught and fledgling field. Some evidence implicates dopamine, a neurochemical that—among many other functions—signals the incentive value of information. This process might explain why open people seem to have more sensitive radars for detecting and processing all kinds of concepts, percepts and qualia. Another clue is an association between openness and activity in the "default network," a neural system that simulates various experiences such as mind wandering, mental time travel and imagining others' point of view. More research is needed to determine whether these neural processes underpin the flexible and inclusive cognition that characterizes open people.

As personality psychologists delve deeper into openness to experience, we push

back the boundaries of knowledge of this fascinating trait. Is it an advantage to be higher on openness, or are there downsides? Can we change our level of openness and, if so, how? Is openness a uniquely human trait? How did it evolve? As the answers to these questions unfold, we better understand what it means to be open-minded and how it shapes our experience of the world. M

How Poverty Affects the Brain

An unprecedented study in Bangladesh could reveal how malnutrition, poor sanitation and other challenges make their mark on child development

By Carina Storrs, *Nature* magazine

Children collect waste coal from a brickyard in Dhaka, Bangladesh. They sell what they find for about $4 per week.

ZAKIR HOSSAIN CHOWDHURY *GETTY IMAGES*

In the late 1960s a team of researchers began doling out a nutritional supplement to families with young children in rural Guatemala. They were testing the assumption that providing enough protein in the first few years of life would reduce the incidence of stunted growth.

It did. Children who got supplements grew one to two centimeters taller than those in a control group. But the benefits did not stop there. The children who received added nutrition went on to score higher on reading and knowledge tests as adolescents, and when researchers returned in the early 2000s, women who had received the supplements in the first three years of life completed more years of schooling and men had higher incomes.

"Had there not been these follow-ups, this study probably would have been largely forgotten," says Reynaldo Martorell, a specialist in maternal and child nutrition at Emory University, who led the follow-up studies. Instead, he notes, the findings made financial institutions such as the World Bank think of early nutritional interventions as long-term investments in human health.

Since the Guatemalan research, studies around the world—in Brazil, Peru, Jamaica, the Philippines, Kenya and Zimbabwe—have all associated poor or stunted growth in young children with lower cognitive test scores and worse school achievement.

A picture slowly emerged that being too short early in life is a sign of adverse conditions—such as poor diet and regular bouts of diarrheal disease—and a predictor for intellectual deficits and mortality. But not all stunted growth, which affects an estimated 160 million children worldwide, is connected with these bad outcomes. Now researchers are trying to untangle the links between growth and neurological development. Is bad nutrition alone the culprit? What about emotional neglect, infectious disease or other challenges?

Shahria Hafiz Kakon has been at the frontline of these issues, trying to answer these questions in the slums of Dhaka, Bangladesh, where about 40 percent of children have stunted growth by the age of two. As a medical officer at the International Center for Diarrheal Disease Research, Bangladesh, in Dhaka, she is leading the first ever brain-imaging study of children with stunted growth. "It is a very new idea in Bangladesh to do brain-imaging studies," Kakon says.

The research is innovative in other respects, too. Funded by the Bill & Melinda Gates Foundation, it is one of the first studies to look at how the brains of babies and toddlers in the developing world respond to adversity. And it promises to provide important baseline information about early childhood development and cognitive performance.

Kakon and her colleagues have run MRI tests on two- and three-month-old children and identified brain regions that are smaller in children with stunted growth than in others. They are also using other tests, such as electroencephalography.

"Brain imaging could potentially be really helpful" as a way to see what is going on in the brains of these young children, says Benjamin Crookston, a health scientist at Brigham Young University, who led studies in Peru and other lower-income countries that reported a link between poor growth and cognitive setbacks.

The Long Shadow of Stunting

In 2006 the World Health Organization

Carina Storrs is a freelance writer in New York City. The Pulitzer Center on Crisis Reporting provided travel support for this story.

(WHO) reported an extensive study to measure the heights and weights of children between birth and the age of five in Brazil, Ghana, India, Norway, Oman and the U.S. The results showed that healthy, well-fed children the world over follow a very similar growth trajectory, and it established benchmarks for atypical growth. Stunted growth, the WHO decided, is defined as two standard deviations below the median height for a particular age. Such a difference can seem subtle. At six months old, a girl would be considered to have stunted growth if she was 61 centimeters long, even though that is less than five centimeters short of the median.

The benchmarks helped to raise awareness about stunting. In many countries, more than 30 percent of children younger than five meet the definition; in Bangladesh, India, Guatemala and Nigeria, more than 40 percent do. In 2012 growing consensus about the effects of stunting motivated the WHO to pledge to reduce the number of children under five with stunted growth by 40 percent by 2025.

Even as officials started to take action, researchers realized there were serious gaps in protocols to identify the problems related to stunting. Many studies of brain development relied on tests of memory, speech and other cognitive functions that are ill suited to very young children. "Babies do not have much of a behavioral repertoire," says Michael Georgieff, a pediatrician and child psychologist at the University of Minnesota. And if parents and doctors have to wait until children are in school to notice any differences, it will probably be too late to intervene.

That is where Kakon's work fits in. At 1.63 meters, she is not tall by Western standards, but at the small apartment-building-turned-clinic in Dhaka where she works, she towers over most of her female colleagues. On a recent morning she was with a mother who had phoned her in the middle of the night: the woman's son had a fever. Before examining the boy, Kakon asked his mother how the family was and how he was doing at school, as she usually does. Many parents call Kakon *apa*—a Bengali word for "big sister."

About five years ago the Gates Foundation became interested in tracking brain development in young children living with adversity, especially stunted growth and poor nutrition. The foundation had been studying children's responses to vaccines at Kakon's clinic. The high rate of stunting, along with the team's strong bonds with participants, clinched the deal.

To get the study off the ground, the foundation connected the Dhaka team with Charles Nelson, a pediatric neuroscientist at Boston Children's Hospital and Harvard Medical School. He had expertise in brain imaging—and in childhood adversity. In 2000 he began a study tracking the brain development of children who had grown up in harsh Romanian orphanages. Though fed and sheltered, the children had almost no stimulation, social contact or emotional support. Many have experienced long-term cognitive problems.

Nelson's work revealed that the orphans' brains bear marks of neglect. MRIs showed that by the age of eight, they had smaller regions of gray and white matter associated with attention and language than did children raised by their biological families. Some children who had moved from the orphanages into foster homes as toddlers were spared some of the deficits.

The children in the Dhaka study have a completely different upbringing. They are surrounded by sights, sounds and extended

families, who often all live together in tight quarters. It is the "opposite of kids lying in a crib, staring at a white ceiling all day," Nelson says.

But the Bangladeshi children do deal with inadequate nutrition and sanitation. And researchers had not explored the impacts of such conditions on cerebral development. There are brain-imaging studies of children growing up in poverty—which, like stunting, could be a proxy for inadequate nutrition. But these have mostly focused on high-income areas, such as the U.S., Europe and Australia. No matter how poor the children there are, most have some nutritious food, clean water and plumbing, Nelson notes. Those in the Dhaka slums live and play around open canals of sewage. "There are many more kids like the kids in Dhaka around the world," he says. "And we knew nothing about them from a brain level."

The Marks of Adversity

By early 2015 Nelson's team and the Bangladeshi researchers had transformed the humble Dhaka clinic into a state-of-the-art laboratory. For their EEG equipment, they had to find a room with no wires in the walls and without air-conditioning units, both of which could interfere with the device's ability to detect activity in the brain.

One of the challenges of such studies is that researchers are still trying to work out what normal brain development looks like.

The researchers also set up a room for functional near-infrared spectroscopy (fNIRS), in which children wear a headband of sensors that measure blood flow in the brain. The technique gives information about brain activity similar to that from functional MRI but does not require a large machine, and the children do not have to remain motionless. FNIRS has been used in infants since the late 1990s and is now gaining traction in low-income settings.

The researchers are also performing MRIs at a hospital near the clinic. So far they have scanned 12 babies aged two to three months with stunted growth. Similar to the Romanian orphans and the children growing up in poverty in developed countries, these children have had smaller volumes of gray matter than a group of 20 nonstunted babies. It is "remarkably bad," Nelson says, to see these differences at such a young age. It is hard to tell which regions are affected in such young children, but having less gray matter was associated with worse scores on language and visual memory tests at six months old.

Some 130 children in the Dhaka study had fNIRS tests at 36 months old, and the researchers saw distinct patterns of brain activity in those with stunting and other adversity. The shorter the children were, the more brain activity they had in response to images and sounds of nonsocial stimuli, such as trucks. Taller children responded more to social stimuli, such as women's

faces. This could suggest delays in the process by which brain regions become specialized for certain tasks, Nelson observes.

EEG detected stronger electrical activity among children with stunted growth, along with a range of brain waves that reflect problem solving and communication between brain regions. That was a surprise to the researchers because studies in orphans and poor children have generally found dampened activity. The discrepancy could be related to the different types of adversity that youngsters in Dhaka face, including food insecurity, infections and mothers with high rates of depression.

Nelson's team is trying to parse out which forms of adversity seem to be most responsible for the differences in brain activity among the Dhaka children. The enhanced electrical signals in EEG tests are strongly linked to increases in inflammatory markers in the blood, which probably reflect greater exposure to gut pathogens.

If this correlation holds up as more children are tested, it could point to the importance of improving sanitation and reducing gastrointestinal infections. Or maternal depression could turn out to be strongly linked to brain development, in which case helping mothers could be just as crucial as making sure their babies have good nutrition. "We don't know the answers yet," Nelson says.

The participants tested at 36 months are now around five years old, and the team is getting ready to take some follow-up measurements. These results will give an idea of whether or not the children have continued on the same brain-development trajectory, Nelson says. The researchers will also give the five-year-olds IQ and school-readiness evaluations to gauge whether the earlier measurements were predictive of school performance.

A Better Baseline

One of the challenges of such studies is that researchers are still trying to work out what normal brain development looks like. A few years before the Dhaka study began, a team of British and Gambian researchers geared up to do EEG and fNIRS testing on children in rural Gambia during the first two years of life. They were also funded by the Gates Foundation.

Similar to the Dhaka study, the researchers are looking at how brain development is related to a range of measures, including nutrition and parent-child interaction. But along the way, they are trying to define a standard trajectory of brain function for children.

There is a big push at the Gates Foundation and the U.S. National Institutes of Health to nail down that picture of normal brain development, according to Daniel Marks, a pediatric neuroscientist at Oregon Health & Science University and a consultant for the foundation. "It is just a reflection of the urgency of the problem," he says.

One of the hopes for the Dhaka study, as well as the motivation for funding it, is that it will reveal distinct patterns in a baby's brain that are predictive of poor outcomes later in life and could be used to see whether interventions are working, says Jeff Murray, a deputy director of discovery and translational sciences at the Gates Foundation.

Any such intervention will probably have to include nutrition, Martorell says. He and his colleagues are doing yet another follow-up study of the Guatemalan villagers to see whether those who got protein supplements before the age of seven have lower rates of heart disease and diabetes 40 years later. But nutrition alone is unlikely to be enough—either to prevent stunting or to promote normal cognitive

development, Martorell asserts. So far the most successful nutritional interventions have helped overcome about one third of the typical height deficit. And such programs can be very expensive; in the Guatemalan study, for example, the researchers ran special centers to provide supplements.

Nevertheless, researchers are striving to improve interventions. A group involved in the vaccine study in Bangladesh is planning to test supplements in pregnant women in the hope of boosting babies' birth weight and keeping their growth on track in the crucial first two years of life. Tahmeed Ahmed, senior director of nutrition and clinical services at the Center for Diarrheal Disease Research, Bangladesh, is planning a trial of foods such as bananas and chickpeas to try to promote the growth of good gut bacteria in Bangladeshi children aged 12 to 18 months. A healthy bacterial community could make the gut less vulnerable to infections that interfere with nutrient absorption and that ramp up inflammation in the body.

Ultimately it is not about whether children have stunted growth or even what their brain looks like. It is about what their lives are like as they grow older. Studies such as the one in Dhaka strive to help determine whether interventions are working sooner rather than later. "If you have to wait until kids are 25 years old to see whether they are employed," Murray says, "it could take you 25 years to do every study."

M

MORE TO EXPLORE

WHO Child Growth Standards Based on Length/Height, Weight and Age. WHO Multicentre Growth Reference Study Group and Mercedes de Onis in *Acta Paediatrica*, Vol. 95, No. S450, pages 76–85; April 2006.

The Nutrition Intervention Improved Adult Human Capital and Economic Productivity. Reynaldo Martorell et al. in *Journal of Nutrition*, Vol. 140, No. 2, pages 411–414; February 2010.

Functional Near Infrared Spectroscopy (fNIRS) to Assess Cognitive Function in Infants in Rural Africa. Sarah Lloyd-Fox et al. in *Scientific Reports*, Vol. 4, Article No. 4740. Published online April 22, 2014.

Brain Imaging and Electrophysiology Biomarkers: Is There a Role in Poverty and Education Outcome Research? Alexandra E. Pavlakis et al. in *Pediatric Neurology*, Vol. 52, No. 4, pages 383–388; April 2015.

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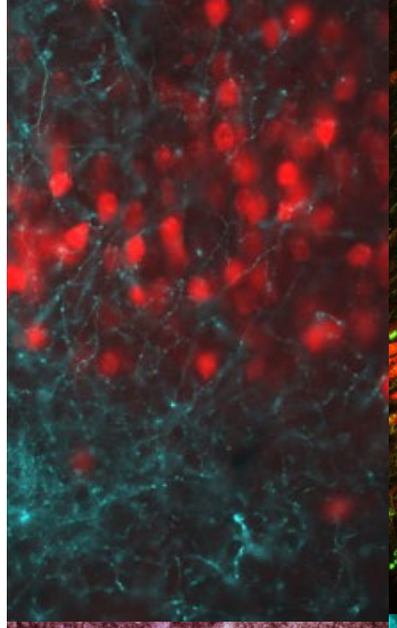
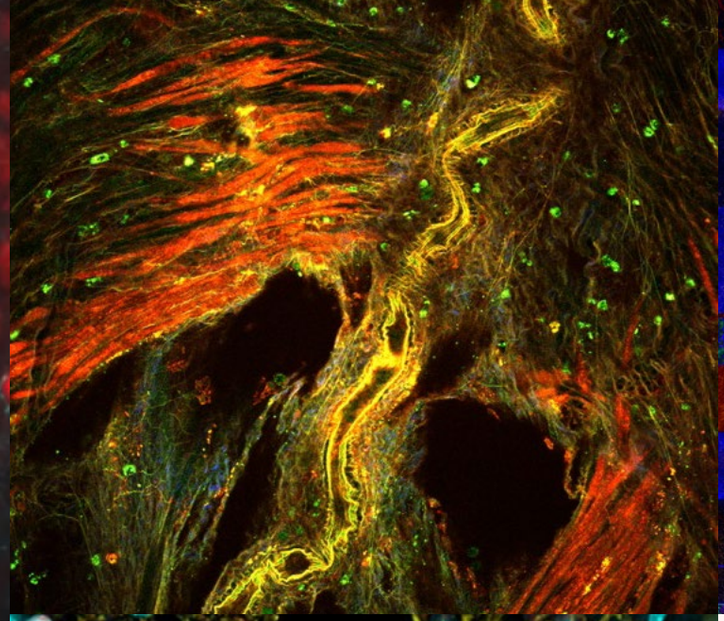
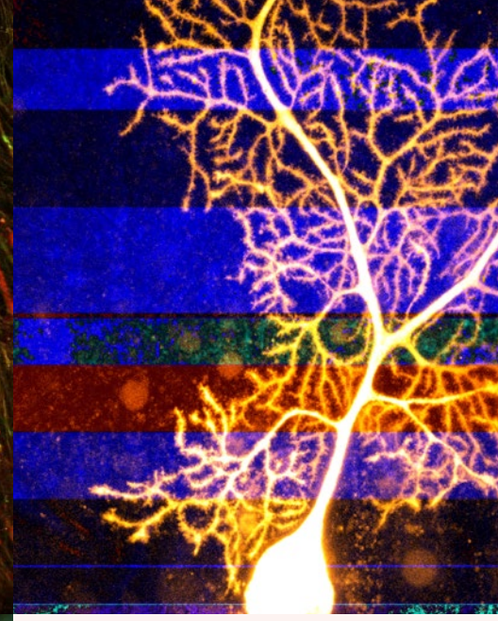
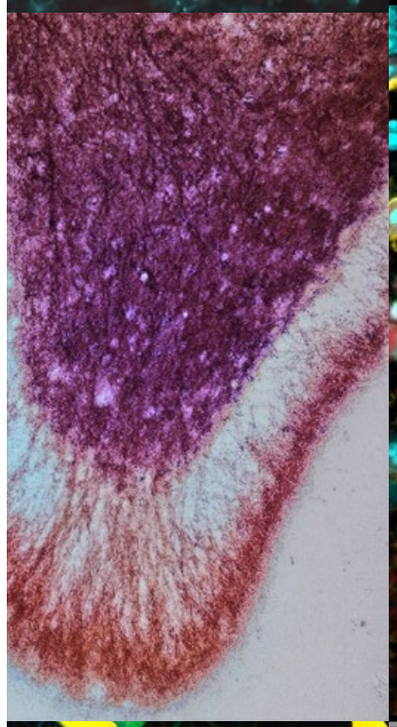
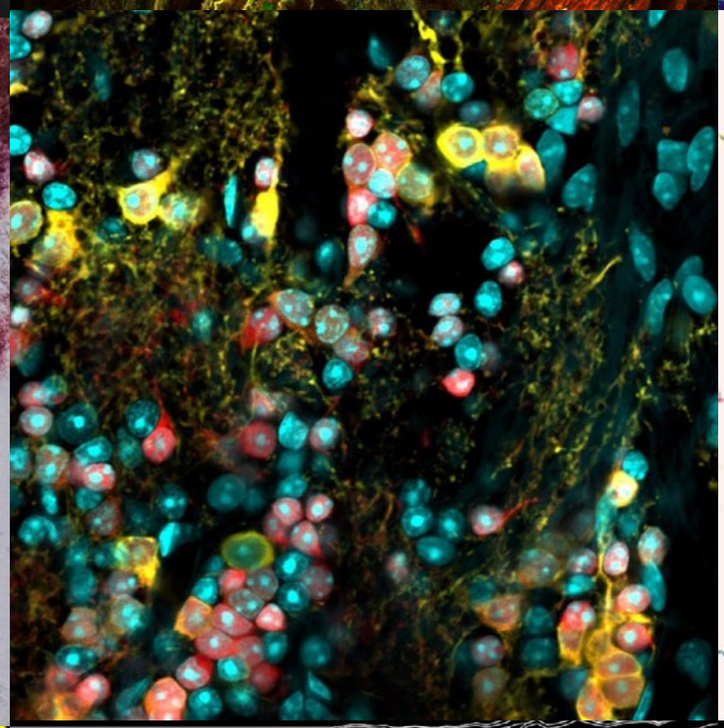
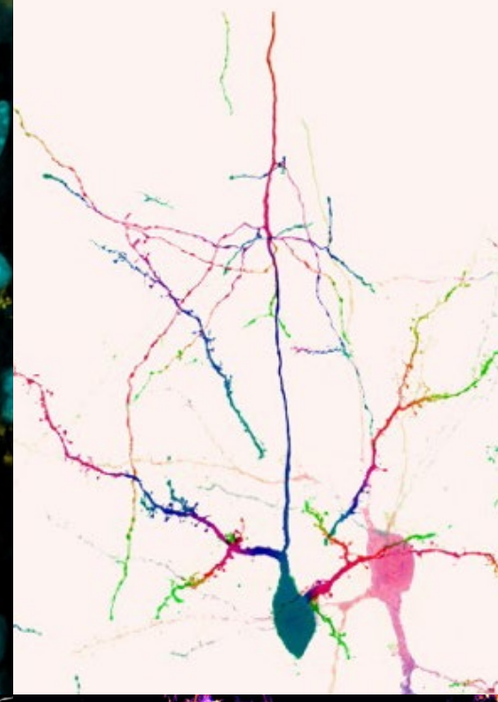
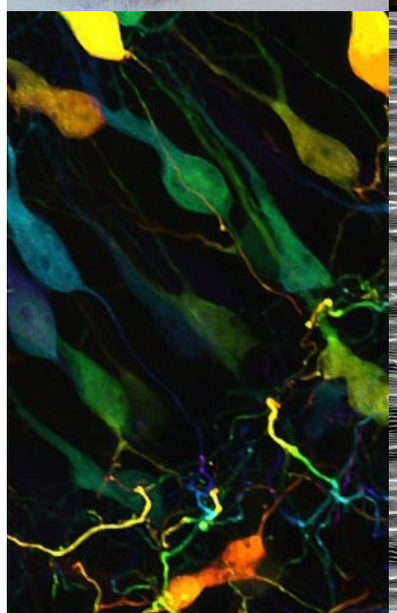
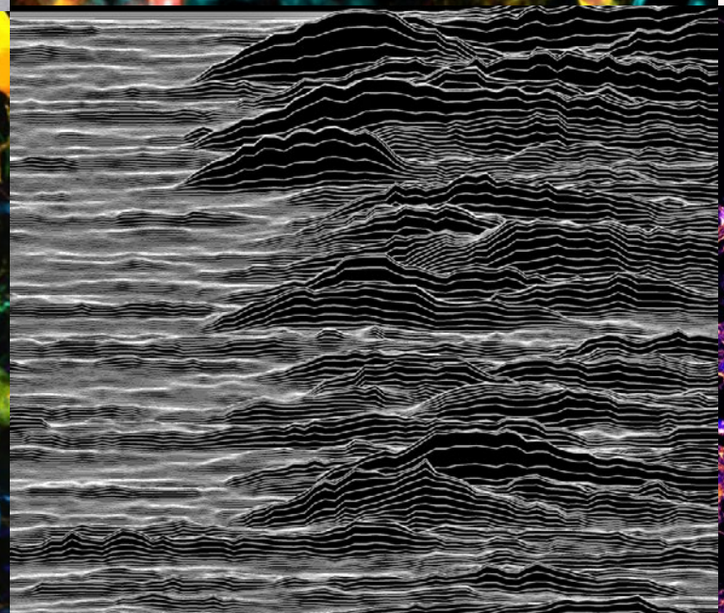
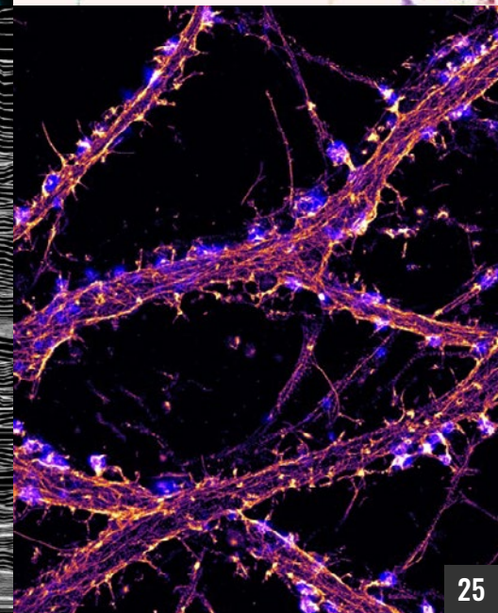
The Art of **Neuroscience**

The winners of an annual contest capture the brain at its most beautiful

By Leslie Nemo, Liz Tormes

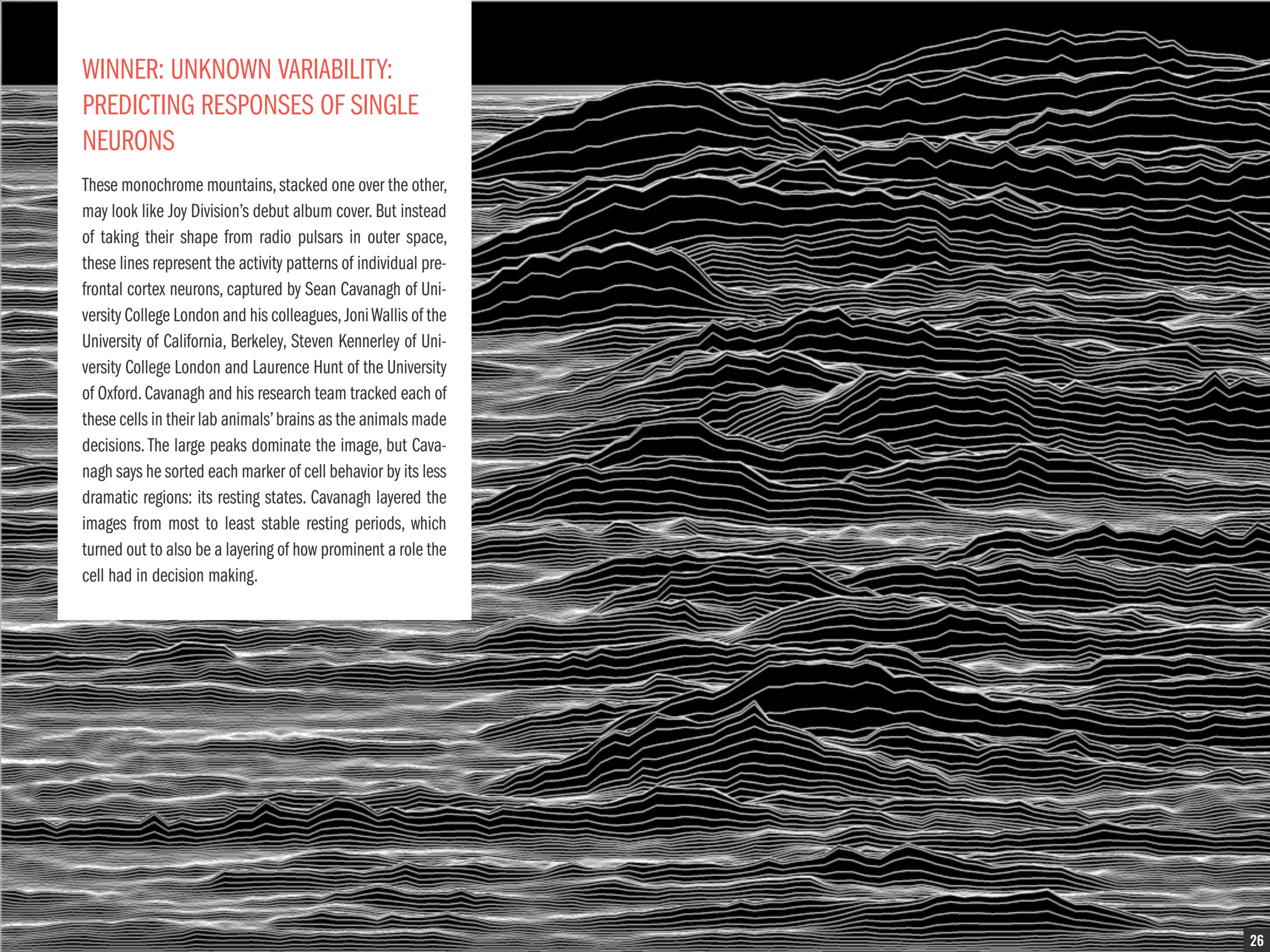
Gray, white and wet, an image of the brain by itself can repulse more often than inspire. But when researchers and artists look past its outward appearance, they can reveal thrilling images of the organ that the rest of us would otherwise never see. Although many of these images resulted from lab work and research into how our nervous system functions, they easily stand alone as art—clearly, a neuroscience degree is not necessary to appreciate the brain's intricacies.

For the seventh year in a row, the Art of Neuroscience competition out of the Netherlands Institute for Neuroscience in Amsterdam asked researchers and artists to submit their paintings, renderings, magnifications and videos of animal brains. The committee's winning entry and honorable mentions are presented below, along with a selection of *Scientific American* editors' favorites.

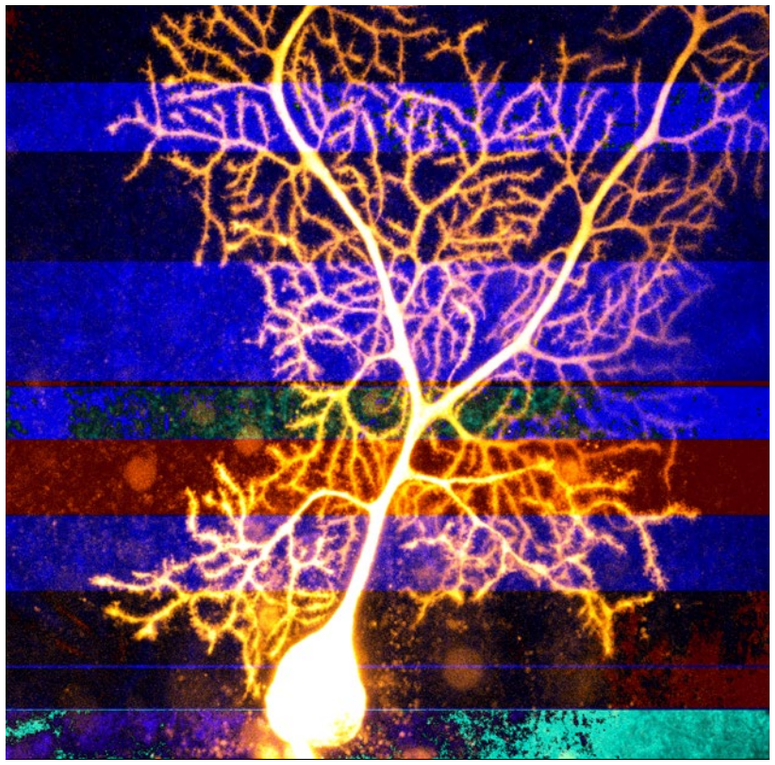










WINNER: UNKNOWN VARIABILITY: PREDICTING RESPONSES OF SINGLE NEURONS

These monochrome mountains, stacked one over the other, may look like Joy Division's debut album cover. But instead of taking their shape from radio pulsars in outer space, these lines represent the activity patterns of individual prefrontal cortex neurons, captured by Sean Cavanagh of University College London and his colleagues, Joni Wallis of the University of California, Berkeley, Steven Kennerley of University College London and Laurence Hunt of the University of Oxford. Cavanagh and his research team tracked each of these cells in their lab animals' brains as the animals made decisions. The large peaks dominate the image, but Cavanagh says he sorted each marker of cell behavior by its less dramatic regions: its resting states. Cavanagh layered the images from most to least stable resting periods, which turned out to also be a layering of how prominent a role the cell had in decision making.



HONORABLE MENTIONS



ANIMON

Often forgotten for its roles outside of bone structure, calcium is key for Purkinje cells. These neurons, which help to monitor balance and posture, rely on the element for synaptic neurotransmitter movement and cell communication. In an effort to understand why individuals on the autism spectrum sometimes struggle with movement, Ph.D. candidate Dana Simmons of the University of Chicago investigates calcium behavior in the minuscule nodules called spines sticking off the dendrites. The entire depth of the neuron is imaged here in 2-D with a series of scans. A single photograph can capture only one segment of the cell, so Simmons compiled them into one picture, creating the striped background.

Leslie Nemo was formerly an editorial intern for *Scientific American*.

Liz Tormes is assistant photo editor for *Scientific American*.

ROOT AND BRANCH BRAIN

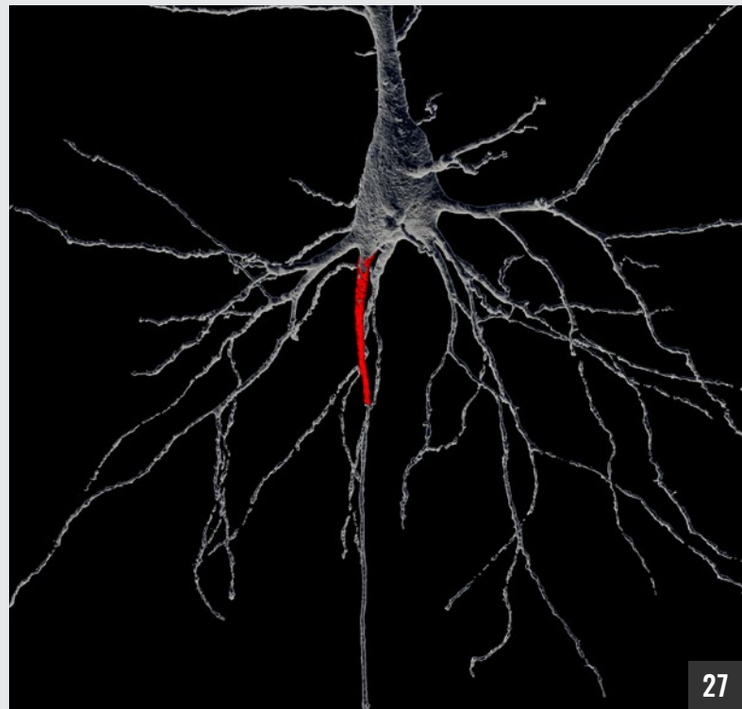
Looking at Michele Banks's *Root and Branch Brain* watercolor, it's easy to see a canopied tree with roots below—and that's no coincidence. Banks, who has focused on interpreting science with art since 2010, marvels at how much of nature forms branching networks, whether it's the brain, a treetop, a water system or blood vessels. The color palette of blues and greens, synonymous with the outdoors, emphasizes the connections our own neurology has with the rest of nature.

POINT OF CHANGE

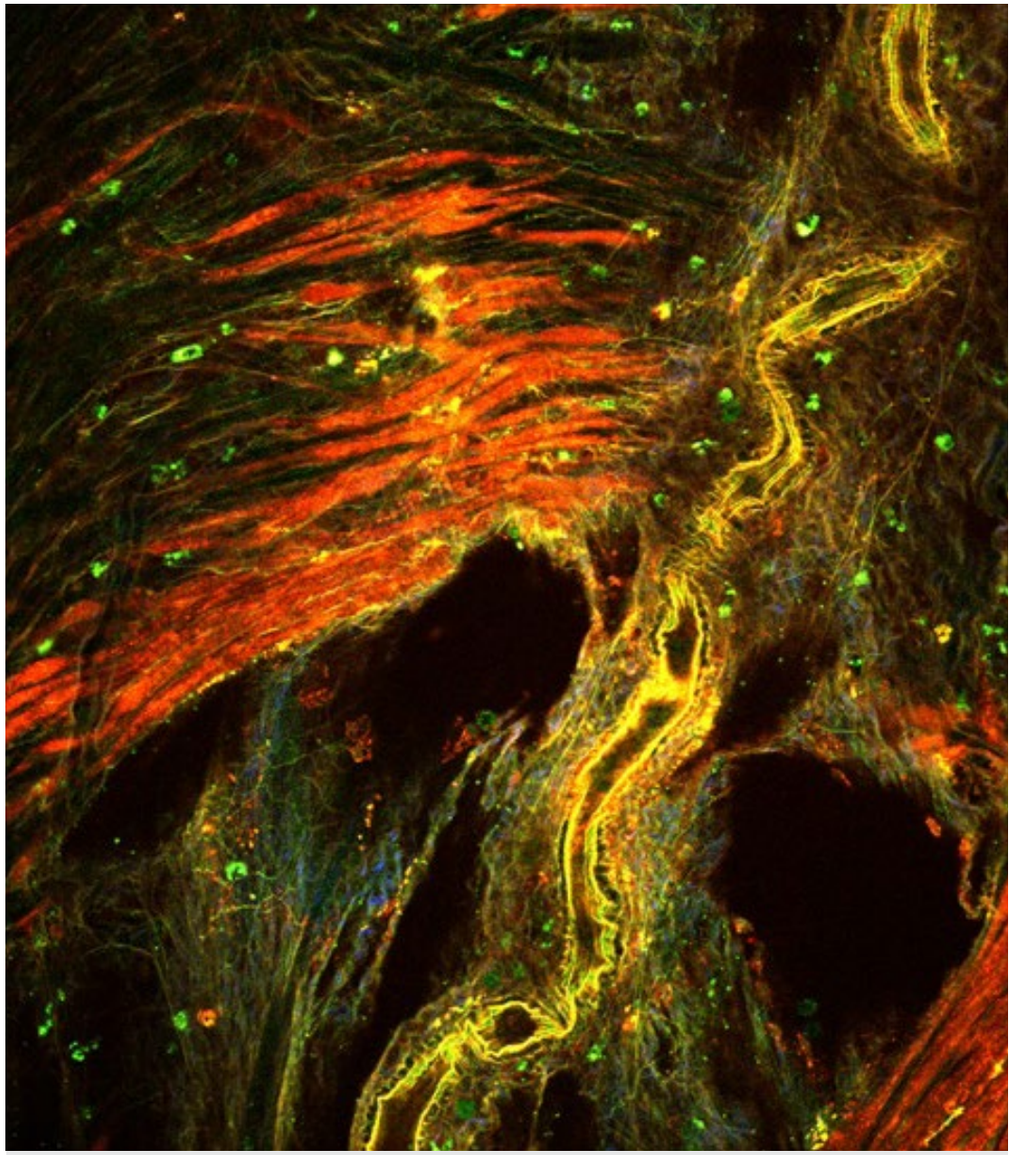
A dancer by training, Daniel Barkan wondered if the concept of neurofeedback—using electroencephalographs (EEGs) to inform individuals of their changing brain-wave activity—could be applied in improvisatory dance. While the dancers move to create the shape of a brain with rope, their EEG-measured brain waves project as music. In response, the dancers change their movement. Barkan, who received a bachelor's degree in dance at the ArtEZ University of of the Arts in Arnhem, the Netherlands, hopes to expand on this feedback-based performative process in the future.

EXCITATION LOCUS

In this stark image, Mustafa S. Hamada of the Netherlands Institute for Neuroscience isolates one thick-tufted layer 5 pyramidal neuron, one of the dominant neuron varieties in the somatosensory cortex. While some initial branching is visible here, the dendrites extend even further, hence the "thick-tufted" description. In contrast to the far-reaching cellular extensions is the neuron's axon (*highlighted in red*). This short segment—responsible for generating all action potentials in the cell—consumed all of Hamada's Ph.D. research. Hamada generated four academic publications out of this one small but complex region.

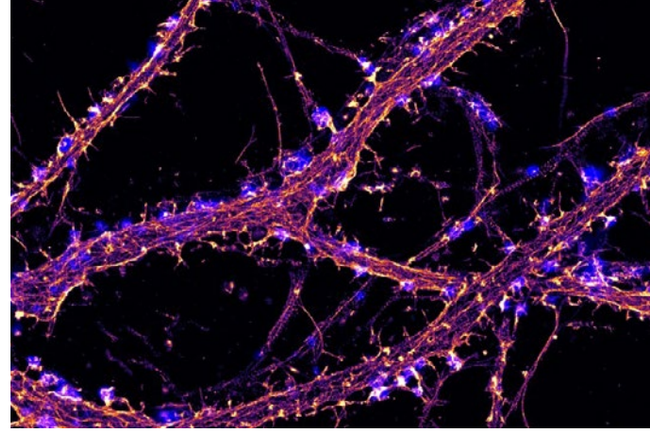


EDITORS' PICKS



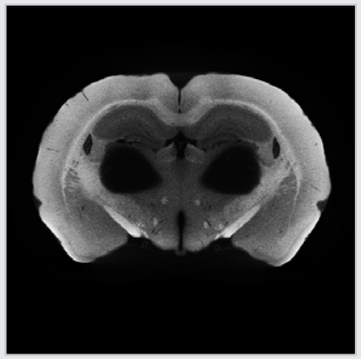
BEAUTIFUL BLADDER

Maybe they aren't the most glamorous organ, but Anna Schüth of Maastricht University in the Netherlands thinks bladders are spectacular. She makes a good point in this image of the nerves, blood vessels and muscles of the bladder wall. And whereas the red waves are stained to be visible, the green streaks of nerves glow because of their own fluorescent chemicals, something Schueth discovered while taking this image for her research. Schueth learned this while examining the cells with two-photon microscopy, which only made the bladder more beautiful in her eyes.



SYNAPTIC ACTIN

In this splattering of glowing neurons, Christophe Leterrier's lab at the French National Center for Scientific Research studies actin filament on the axonal side of synapses. These fibers are not so easy to see. While actin (*orange strings*) is abundant on the dendritic cords that dominate the image, it is less plentiful on the other end of the synaptic connections (*blue flashes*). Sparse does not mean less important, however, and these actin fibers are ripe for further investigation by Leterrier and his team.

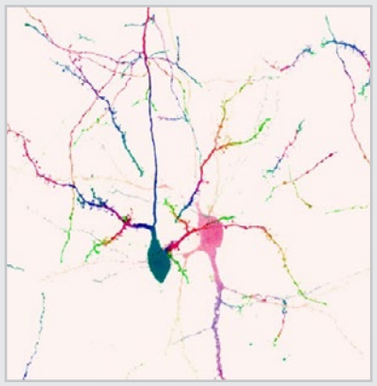


CORONAL SLICES

This ghostly silhouette marks a scientific failure. Javier Masis of Harvard University was supposed to produce a 3-D image of a rat brain by saturating the tissue with osmium. Instead the heavy metal failed to infiltrate the brain's core, leaving blank holes in the virtual reconstruction. The view here is of a cross section of the brain, oriented as if you and the rat are looking eye to eye.

SPIKY GLIA

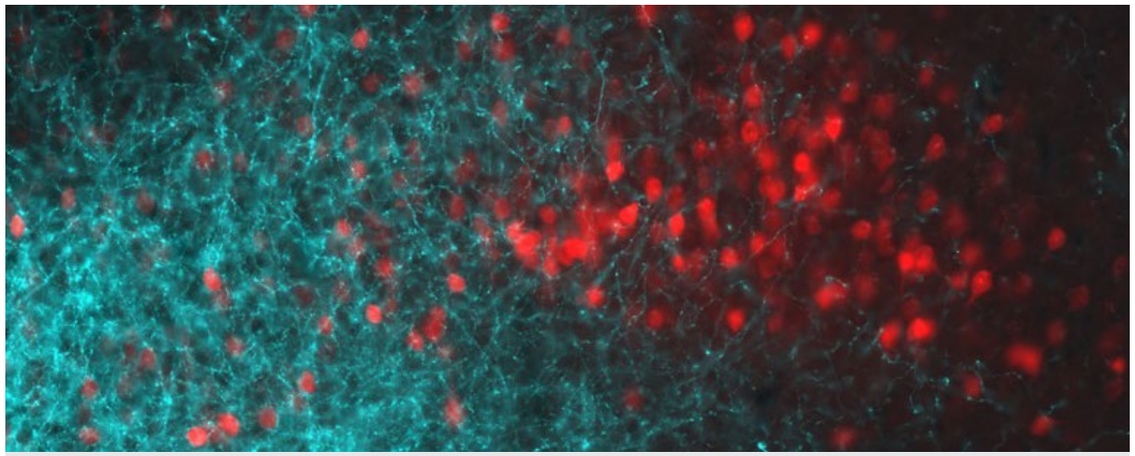
Passing by and forgetting something beautiful is difficult, so Leterrier and Sofia Yousfi, also at the French National Center for Scientific Research, chose to image this glial cell even though the focus of their work is on a specific component housed in actual neurons—lignin. These support cells still grow in culture along with typical neurons and serve as test subjects when the team practices isolating and coloring actin (*dark gray*).



CLOSER

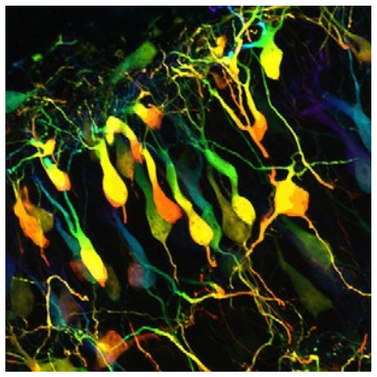
This image highlights the intertwined relationship of just two neurons, disentangled from their fellow cells. Their meandering dendrites are approximately 100th of the width of a human hair. Luke Hammond of Columbia University captured these ultrafine tendrils with the help of 3-D imaging techniques and fluorescent dyes, which change color according to how close or far the branch is from the viewer's perspective.

EDITORS' PICKS



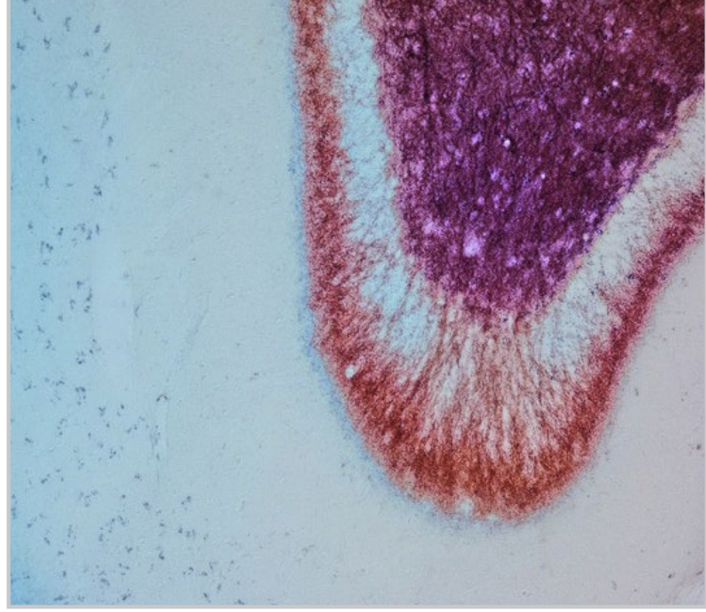
ORBITING AROUND THE FRONTAL INPUTS AND OUTPUTS

The mechanisms behind "monkey see, monkey do"—or in this case, "rodent see, rodent do"—come to life in this image. The turquoise static of axons makes up the primary visual cortex, which receives information from the retina. The visual information proceeds from the blue region into the secondary motor cortex (*red*), where a movement response to the original sight initiates. It was not until Tuce Tombaz of the Center for Neural Computation at the Norwegian University of Science and Technology sliced the brain and stained each set of neurons that she realized the turquoise axons wash over the red cells, literally and visually inundating the motor output cells with commands.



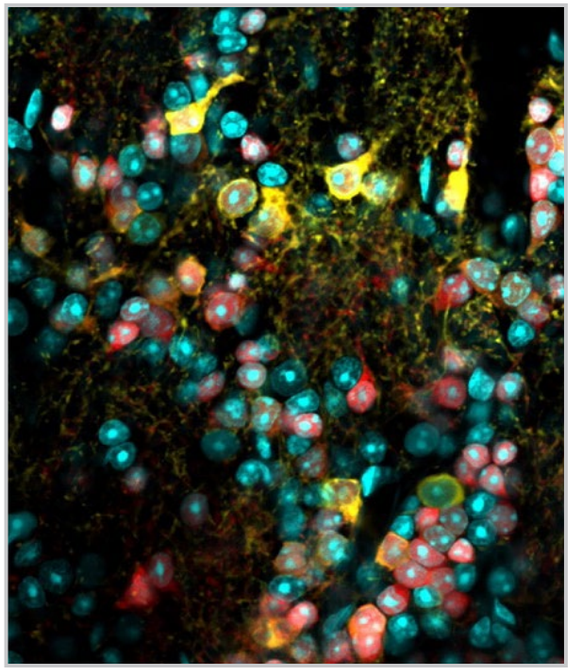
WITHIN THE IN-BETWEEN

Drippy and brooding where Hammond's other image was light and sparse, this perspective of brain neurons was captured with similar 3-D and dye methods. Here Hammond puts the interwoven nature of the human mind's structural elements on full display.



MOSSY FIBER SPROUTING AND ASTROGLIOSIS IN EPILEPTOGENESIS

The exact cause of epilepsy still eludes researchers, but one of the potential causes is imaged here. In a healthy hippocampus, the dentate gyrus, edited to appear fuchsia (*right*), lacks the fuzzy, reddish ring. But if a brain injury damages granular cells in this external region, the cells regrow synapses on top of one another, forming nets of fibers that connect each other and the dentate gyrus into a giant feedback circuit—one that allows an electrical excitation to keep going, as happens during a seizure. Derek Chan captured this neurological regrowth in the brain of an epileptic rat at the University of Amsterdam. Typically a dentate gyrus stain shows up in shades of black and brown, so he digitally retouched the core and external ring for a more vibrant effect.



KLIMT'S GLOMERULI

In this image, the background players—interneruons—take center stage. Pictured in glowing, geometric clusters, these cells relay impulses from neuron to neuron. And although it looks like their ends taper into nothing, the dark regions are actually dense networks of axons and synapses called glomeruli. These black depths wrap around the olfactory bulb and serve as the first neural response to smells. Oliver Braubach of the Center for Functional Connectomics at the Korea Institute of Science and Technology captured these interneurons by developing his own staining technique: cyan for cell nuclei, red for the GABA neurotransmitter and yellow for dopamine.

SEAN CAVANAGH *UNIVERSITY COLLEGE LONDON*, JONI WALLIS *UNIVERSITY OF CALIFORNIA, BERKELEY*, STEVEN KENNERLEY *UNIVERSITY COLLEGE LONDON AND LAURENCE HUNT UNIVERSITY OF OXFORD* (*UNKNOWN VARIABILITY: PREDICTING RESPONSES OF SINGLE NEURONS*); DANA SIMMONS *CHRISTIAN HANSEL LABORATORY, UNIVERSITY OF CHICAGO* (*ANIMON*); MICHELE BANKS (*ROOT AND BRANCH BRAIN*); MUSTAFA S. HAMADA *KOLE RESEARCH GROUP, NETHERLANDS INSTITUTE FOR NEUROSCIENCE* (*EXCITATION LOCUS*); DANIEL BARKAN *ARTEZ UNIVERSITY OF THE ARTS* (*POINT OF CHANGE*); ANNA SCHÜTH *MAASTRICHT UNIVERSITY* (*BEAUTIFUL BLADDER*); CHRISTOPHE LETERRIER *NEUROBIOLOGY OF CELLULAR INTERACTIONS AND NEUROPHYSIOPATHOLOGY, FRENCH NATIONAL CENTER FOR SCIENTIFIC RESEARCH AND AIX-MARSEILLE UNIVERSITY* (*SYNAPTIC ACTIN*); CHRISTOPHE LETERRIER *NEUROBIOLOGY OF CELLULAR INTERACTIONS AND NEUROPHYSIOPATHOLOGY, FRENCH NATIONAL CENTER FOR SCIENTIFIC RESEARCH AND AIX-MARSEILLE UNIVERSITY* (*SPIKY GLIA*); JAVIER MASIS *HARVARD UNIVERSITY* (*CORONAL SLICES*); DEREK CHAN AND CATO DRION *UNIVERSITY OF AMSTERDAM* (*MOSSY FIBER SPROUTING AND ASTROGLIOSIS IN EPILEPTOGENESIS*); TUCE TOMBAZ *KAVLI INSTITUTE FOR SYSTEMS NEUROSCIENCE, CENTER FOR NEURAL COMPUTATION, NORWEGIAN UNIVERSITY OF SCIENCE AND TECHNOLOGY* (*ORBITING AROUND THE FRONTAL INPUTS AND OUTPUTS*); LUKE HAMMOND, *ZUCKERMAN INSTITUTE, COLUMBIA UNIVERSITY; CAPTURED AT THE QUEENSLAND BRAIN INSTITUTE, UNIVERSITY OF QUEENSLAND* (*CLOSER*); OLIVER BRAUBACH *CENTER FOR FUNCTIONAL CONNECTOMICS, KOREA INSTITUTE OF SCIENCE AND TECHNOLOGY* (*KLIMT'S GLOMERULI*); LUKE HAMMOND *ZUCKERMAN INSTITUTE, COLUMBIA UNIVERSITY; CAPTURED AT THE QUEENSLAND BRAIN INSTITUTE, UNIVERSITY OF QUEENSLAND* (*WITHIN THE IN-BETWEEN*)

HOUNDS SEARCH FOR NOVELIST

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Was Agatha Christie's Mysterious Amnesia Real or Revenge on Her Cheating Spouse?

Ninety years ago, she stayed in a hotel for 11 days under an assumed name, supposedly because she had suffered from a loss of memory. How plausible is her story?

By Stefania de Vito, Sergio Della Sala

GETTY IMAGES

On Saturday, December 4, 1926, a green Morris Cowley motorcar stood abandoned in a roadside ditch near the city of Guildford, England. The car belonged to the renowned author Agatha Christie, who had apparently disappeared without a trace. But after missing for 11 days, she turned up in a hotel in Harrogate, a spa town in Yorkshire 200 miles north of Guildford. Christie was unable to explain what had transpired during the intervening time period, nor is this mysterious episode mentioned in her autobiography. Unlike those in her many books, this mystery remains unsolved.

Is it possible that Christie suffered from what is called retrograde amnesia as a result of an automobile accident and was no longer capable of remembering the event? Was she, by disappearing, perhaps exacting revenge on her unfaithful husband? Or was this just a clever public relations ploy aimed at promoting her latest novel?

Stefania de Vito is a psychologist and neuroscientist at the University of East London.

Sergio Della Sala is a professor of cognitive neuroscience at the University of Edinburgh in Scotland.

The drama began in April 1926, when Christie's mother died. According to Christie's biographer Janet Morgan, the death hit her very hard. At the time her husband, Colonel Archibald Christie, known as Archie, was on a business trip. On returning, he informed his psychologically fragile wife that he had fallen in love with a woman named Nancy Neele. For a while the Christies stayed together for their daughter's sake, even moving together to Styles, her house in Sunningdale, in the county of Berkshire. All the while, however, Archie maintained his affair with Neele.

On the morning of December 3, Agatha and Archie had a domestic argument. She drove off in the car that was found the next day near Guildford. Christie, meanwhile, had checked into the hotel in Harrogate—under the name Neele—and listed her place of residence as Cape Town, South Africa. While news of her disappearance sped around the globe and newspapers everywhere featured her photograph, Christie, alias Neele, took the cure, relaxing in the atmosphere of the spa. By all appearances, she seemed happy and content, not at all fearful of being discovered by the mob of journalists who were hot on her trail.

"I Do Not Think She Knows Who She Is"

In fact, two other esteemed writers, Sir Arthur Conan Doyle and Dorothy Sayers, joined in the search. One evening Christie was recognized in the hotel by a local musician, Bob Tappin. He notified the police, and two days later Archie confirmed that the lady in question was indeed his wife. Archie was also the one who first raised the question of a loss of memory. In a newspaper interview on December 15, 1926, he declared, "She has suffered from the most complete loss of memory, and I do not think she knows who she is. She does not know me, and she does not know where she is. I am hoping that rest and quiet will restore her. I am hoping to take her to London tomorrow to see a doctor and specialist."

In her novel *The Lost Days of Agatha Christie*, American therapist Carole Owens doubts this was a case of amnesia because the author clearly remembered the name of her husband's lover, at least to the extent that she used it in Harrogate. This argument, however, is less than cogent because we know today that partial amnesia can occur and that it can affect certain memory systems such as episodic memory (of events) while leaving semantic (factual)

memory intact. In other words, the crime novelist could well have lost all recollection of how she got from Guildford to Harrogate while nonetheless retaining the name of her rival. In addition, as an eyewitness reported, she had no trouble playing cards with other guests in the hotel lobby and even dancing. This means her procedural memory, which enables people to perform actions such as dancing or cycling automatically, was apparently unaffected.

In addition, short-term memory lapses such as transient global amnesia lasting no more than 24 hours are known to occur. Although affected persons may be as attentive and awake as ever, their access to previously acquired memories may be disturbed (retrograde amnesia). Nor are they able to store new information (anterograde amnesia). This type of amnesia may be caused by severe emotional stress, but in such cases, people almost always retain knowledge of who they are and of those with whom they are close. This suggests Christie probably did not suffer from this syndrome.

Writing in 2003 in *Practical Neurology*, psychologists Mireia Pujol and Michael Kopelman, both then at King's College London, discussed the possibility that Christie may have experienced psychogenic amnesia secondary to trauma. This memory disorder, also called dissociative amnesia, generally lasts for a few hours, days or even months and affects primarily memories of those events that occurred immediately before the traumatic experience. Such amnesia may be triggered by catastrophic news, a fight, financial ruin or war.

In 1935 two psychiatrists writing in *Ar-*

GETTY IMAGES

Writer Agatha Christie and her second husband, Max E. L. Mallowan, pose in March 1946 on the grounds of their home, Greenway House, in the county of Devon in England. This marriage lasted until Christie's death in 1976.

chives of Neurology and Psychiatry described the case of a woman who left her husband for another man. After a week, she decided to return home as if nothing had happened. The sheer impossibility of this endeavor caused her to consider suicide, just before she developed amnesia.

Fragments of Memories

Such a psychogenic "flight syndrome" exhibits some striking overlap with organic amnesia. In both types, for example, fragments of prior memories may remain but are covered by a blanket of forgetting. In neurological memory disorders, however, memories from the more distant past tend to be retained, whereas newly absorbed information is lost. In syndromes caused by psychological stress, this pattern is generally reversed: the memory of recently experienced events is retained, whereas older memories disappear.

This presumably has neurophysiological causes: Although freshly formed memory traces are largely limited to the hippocampus in the temporal lobes, information that has been repeatedly called up over time is activated by links with other regions of the cortex, such as the frontal lobe. The hippocampus is often damaged during brain injuries caused by an accident or by organic causes. When linked regions are intact, however, shards of older memories may make their way into consciousness. Acute stress, on the other hand, decreases neural processing in the frontal lobe, which is involved in retrieving episodic, autobiographical and factual knowledge. Because, among other things, certain regions of the frontal cortex are responsible for inhibiting memory, stress-related changes in the activity of the frontal lobe may more easily impair personal memories and semantic information acquired long ago than does damage to the hippocampus.

According to Kopelman, cases of total memory loss of the kind often seen in movies is practically unknown. Whether amnesia is caused by an accident or stress, the idea that patients would cease to have any knowledge of who they are, where they are or the identity of their spouse is largely a fiction. But because this cliché has been so popularized by the media, experts are easily able to distinguish simulated amnesia when they see it.

Was It All Just Made Up?

Gwen Robyns, author of *The Mystery of*

Diagnostic Criteria: Dissociative Amnesia

According to the World Health Organization, the main feature of this condition is "loss of memory, usually of important recent events, that is not due to organic mental disorder, and is too great to be explained by ordinary forgetfulness or fatigue. The amnesia is usually centered on traumatic events, such as accidents or unexpected bereavements, and is usually partial and selective. Complete and generalized amnesia is rare, and is usually part of a fugue.... If this is the case, the disorder should be classified as such. The diagnosis should not be made in the presence of organic brain disorders, intoxication or excessive fatigue."

In other words, Agatha Christie could have suffered from a dissociative fugue, or state of psychological flight. Persons with this disorder exhibit all the symptoms of dissociative amnesia. Additionally, a person suffering from this disorder may seek to move well beyond his or her usual sphere of travel. And although the individual may suffer amnesia during a fugue state, behavior may seem completely normal to outsiders.

Source: *International Statistical Classification of Diseases and Related Health Problems*, 10th Revision. 2016 Version. World Health Organization, 2016

Agatha Christie, believes Christie was in full control of her memory during the entire episode of her disappearance. As Robyns sees it, Christie was intent on exacting revenge on her husband by prolonging the separation. Another Christie expert, journalist Jared Cade, believes it is entirely plausible that Christie simply intended to make her husband's liaison with Neele as stressful as possible. Cade asserts that Agatha later admitted to her husband that she had staged the entire disappearance. Edgar Wallace, another writer colleague of Christie's who worked for the British tabloid the *Daily Mail*, claimed that this was a typical case of "the betrayed wife's revenge."

Her disappearance became a major public cause célèbre and, as Cade avers in his book, had political implications as well. During a parliamentary debate, Labor Party MP William Lunn asked a question that amounted to heresy: How much money had the state spent searching for this star author? Home Secretary William Joynson-Hicks estimated police costs at less than £13—about $1,000 in today's dollars. To which Lunn responded, "Who is going to compensate the thousands of people who were deliberately misled by this cruel hoax?" Joynson-Hicks declined to answer.

But feigning the kind of amnesia from which Christie apparently suffered is much more difficult than it might appear. Most people have no idea what symptoms they should or should not display. A simple test of episodic, semantic and procedural memory would quickly diagnose fakery.

Given the many questions that are unresolved 90 years after Christie's disappearance, it remains a mystery. Nor is it at all clear whether her amnesia was staged or was in fact caused by stress or some organic disorder. With regard to human memory, in which reality and fiction so easily become intermingled in an inextricable mélange, matters are never as straightforward as Christie, speaking of her mysteries, would have you believe: "The simplest explanation is usually the right one." M

Editors' Note: This story is a translation of an article from Scientific American's *Italian-language sister publication,* Mente&Cervello.

Eyewitness Memory Is a Lot More Reliable Than You Think

What law enforcement—and the public—needs to know

By John Wixted, Laura Mickes

GETTY IMAGES

The ability of a person who witnesses a crime to later pick the perpetrator out of a lineup is atrocious—right? The answer seems like a resounding "yes" if you consider some well-known and rather disconcerting information. Eyewitness misidentifications are known to have played a role in 70 percent of the 349 wrongful convictions that have been overturned based on DNA evidence (so far). Psychologists have learned a lot about why such errors happen. With surprising ease, for example, participants in a memory experiment can be led to believe that they saw a stop sign when they actually saw a yield sign or that they became lost in a shopping mall as a child when no such experience actually occurred. In much the same way, an eyewitness can be led to falsely remember someone committing a crime that was actually committed by someone else.

But there is more to the story. Consider the important, and often overlooked, distinction between malleability and reliability. Just because memory is malleable—for example, it can be contaminated with the trace of an innocent person—does not mean that it has to be unreliable. What it means is that the malleability of memory can harm reliability. Once this fact is appreciated, then proper testing protocols can be put in place to minimize the likelihood that the original memory trace is contaminated.

Current procedures for collecting and assessing evidence from eyewitnesses are often not designed to minimize contamination. This problem does not apply to other kinds of forensic evidence. Imagine if police let unauthorized people have willy-nilly access to a crime scene that is under investigation. What would that mean for the reliability of the blood or fingerprint evidence, for example, collected at that scene? Blood and fingerprint evidence, per se, would not be deemed unreliable. Instead evidence collected at the contaminated crime scene would probably be declared inadmissible. Not so with eyewitness memory.

Any evidence can potentially be contaminated, including what is considered to be the gold standard of forensic evidence: DNA. Like eyewitness memory, DNA evidence can be contaminated with the trace of an innocent person. If the contaminated evidence is relied on to establish guilt versus innocence at a trial, the risk of a wrongful conviction is high. Consider, for example, the case of Gary Leiterman, who, in 2005, was convicted of murder and sentenced to life in prison following a cold case investigation in which his DNA was found on the clothing of a woman named Jane Mixer, who was murdered in 1969. Many believe that this conviction was based on contaminated DNA evidence.

What facts gave rise to the belief that Leiterman may have been wrongfully convicted based on contaminated evidence? First, another DNA profile—one belonging to John Ruelas, who was just a four-year-old preschooler at the time of the murder—was found on a blood spot taken from the victim's left hand. Even though no connection between Leiterman and Ruelas was ever established, the prosecution theorized that all three must have been together after midnight at the murder scene in 1969, with the preschooler bleeding on the victim for some unknown reason while Leiterman killed her. According to the same theory, 33 years later, in 2002, in an almost in-

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conceivable coincidence, evidence samples from Mixer, Ruelas and Leiterman just happened to be together again in the Michigan State Police Laboratory. The murder victim's cold case evidence was there because the case had recently been reopened; the 1969 preschooler's DNA sample was there as part of an active murder investigation, and Leiterman's DNA sample was there because he had recently been arrested for forging a prescription. The nearly simultaneous analyses of evidence from these three cases in the same crime lab in 2002—a reunion, of sorts, among Mixer, Ruelas and Leiterman, who were ostensibly last together on the night of the murder in 1969—was either an incredible coincidence or the Mixer evidence was contaminated with DNA from both Leiterman and Ruelas.

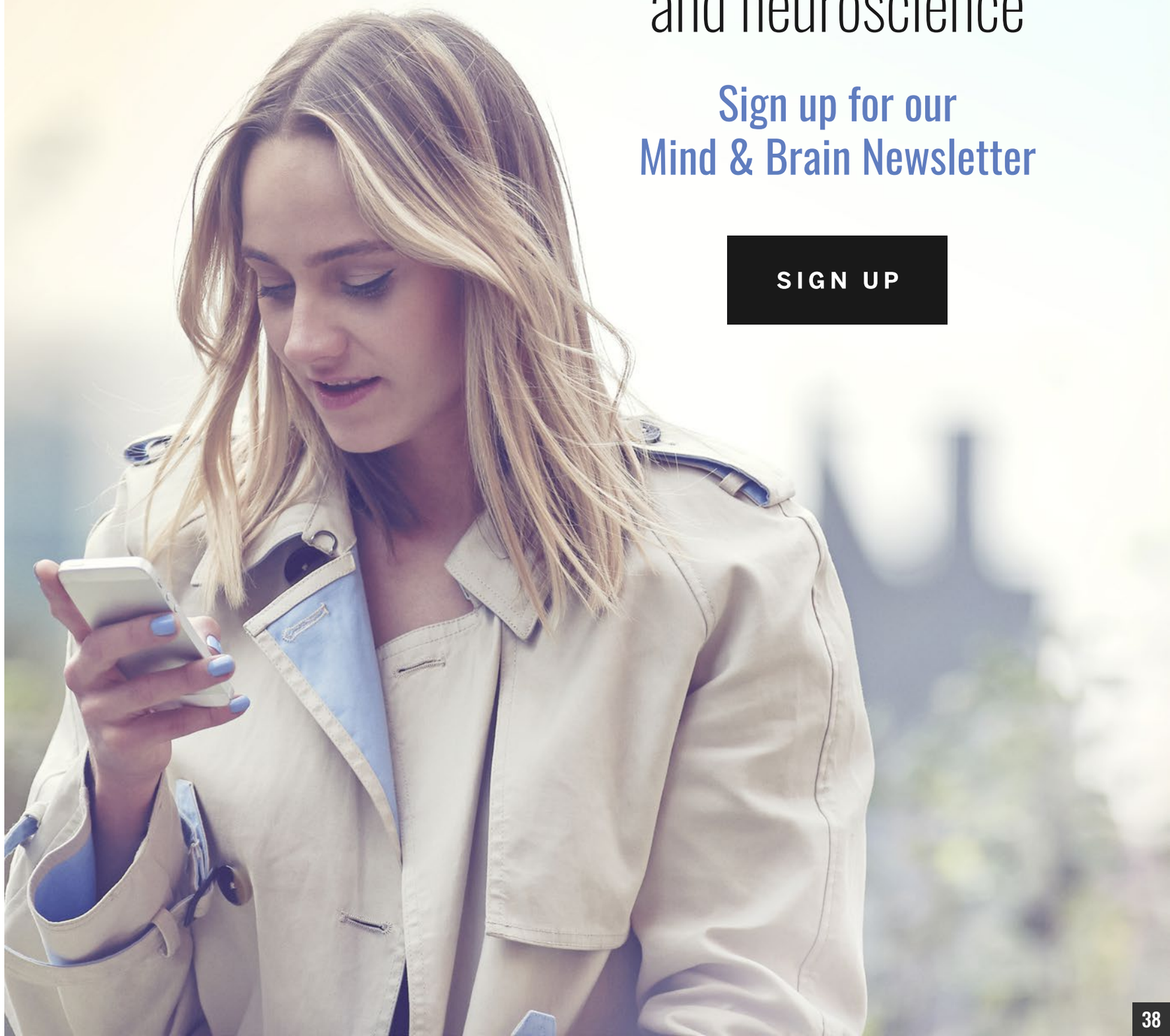
Instead of concluding that DNA evidence is inherently unreliable because of the contamination that apparently occurred in this case, a more reasonable conclusion would be that for DNA testing to be reliable, proper protocols must be followed. Few would doubt that under such conditions, DNA evidence is highly reliable. The same is true of eyewitness memory: memory can be contaminated with the trace of an innocent person, but under proper testing conditions, eyewitness evidence is highly reliable. As with DNA evidence, eyewitness evidence needs to be safeguarded against contamination.

To do this, proper testing protocols that reduce chances of contamination need to be followed. Some elements include the following: First, and most important of all, because the test itself contaminates memory, only the *initial* memory test provides uncontaminated results. Subsequent memory tests, including the dramatic one that occurs in court in front of the jury, constitute contaminated evidence. Second, the police lineup has to be fair (that is, the suspect should not stand out). And third, the confidence expressed by the eyewitness following an identification of someone from the lineup must be recorded. Assessing confidence is critical because it provides direct information about the trustworthiness of the uncontaminated ID. An initial eyewitness identification made with low confidence indicates that even though memory was not contaminated, the ID is untrustworthy (that is, by indicating low confidence, the eyewitness is effectively saying, "There's a good chance that I'm making an error"). In contrast, a high-confidence ID is highly accurate, a surprising fact that has only recently come to be appreciated by experimental psychologists. In a recent review of the literature, the authors reported across 15 experiments, suspect identifications made with high confidence were, on average, 97 percent accurate!

Although the high accuracy of an initial ID made with high confidence is important to appreciate, the low accuracy of an ID made with low confidence may be even more important to appreciate. To see why, let us revisit those DNA exoneration cases that so often involve eyewitness misidentification. University of Virginia law professor Brandon Garrett analyzed trial materials for 161 DNA exonerated individuals who had been misidentified with high confidence by one or more eyewitnesses in a court of law. By itself, that fact only shows that contaminated memory is unreliable (just as contaminated DNA evidence is). But in 57 percent of those cases, it was possible to determine what happened on the initial (uncontaminated) memory test. For every one of those tests, the eyewitnesses were, at best, *uncertain*.

Attributing that error to the unreliabili-

ty of eyewitness memory is, in our view, pointing the finger of blame in the wrong direction. Eyewitness memory is reliable when initially tested using proper procedures, but the legal system nonetheless habitually relies on unreliable (contaminated) eyewitness evidence from later IDs. The sooner police, prosecutors and judges understand that fact, the better off we all will be—including you, if you are ever incorrectly fingered by an eyewitness. M



GARY WATERS *GETTY IMAGES*

OPINION

Mental Illness Is Far More Common Than We Knew

New research suggests that nearly everyone will develop a psychological disorder at some point in their life—but for most, it's temporary

By Aaron Reuben, Jonathan Schaefer

Most of us know at least one person who has struggled with a bout of debilitating mental illness. Despite their familiarity, however, these kinds of episodes are typically considered unusual and even shameful.

New research, from our laboratory and from others around the world, however, suggests mental illnesses are so common that almost everyone will develop at least one diagnosable mental disorder at some point in their life. Most of these people will never receive treatment, and their relationships, job performance and life satisfaction will likely suffer. Meanwhile the few individuals who never seem to develop a disorder may offer psychology a new avenue of study, allowing researchers to ask what it takes to be abnormally, enduringly mentally *well*.

Epidemiologists have long known that at any given time, roughly 20 to 25 percent of the population suffers from a mental illness, which means they experience psychological distress severe enough to impair functioning at work, at school or in their relationships. Extensive national surveys, conducted from the mid-1990s through the early 2000s, had suggested that a much higher percentage, close to half the population, would experience a mental illness at *some point*.

These surveys were large, involving thousands of participants representative of the U.S. in age, sex, social class and ethnicity. They were also, however, *retrospective*, which means they relied on survey respondents' accurate recollection of feelings and behaviors months, years and even decades in the past. Human memory is fallible, and modern science has demonstrated that people are notoriously inconsistent reporters about their own mental health history, leaving the final accuracy of these studies up for debate. Of further concern, up to a third of individuals contacted by the national surveys failed to enroll in the studies. Other tests suggested that these "nonresponders" tended to have worse mental health.

A new study by one of us (Schaefer), published earlier this year in the *Journal of Abnormal Psychology* (whose very name suggests an outdated understanding of the prevalence of mental illness), took a different approach to estimating disease burden. Rather than asking people to think back many years, Schaefer and his colleagues instead closely followed one generation of New Zealanders, all born in the same town, from birth to midlife. They conducted in-depth check-ins every few years to assess any evidence of mental illness having occurred during the preceding year.

They found that if you follow people over time and screen them regularly using simple, evidence-based tools, the percentage of those who develop a diagnosable mental illness jumps to well more than 80 percent. In the cohort only 17 percent of study members did not develop a disorder, at least briefly, by middle age. Because Schaefer's team could not be certain that these individuals remained disorder-free in the years between assessments, the true proportion that never experienced a mental illness may be even smaller.

Put another way, the study shows that you are more likely to experience a bout of mental illness than you are to acquire diabetes, heart disease or any kind of cancer

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whatsoever. These findings have been corroborated by data from similar cohorts in New Zealand, Switzerland and the U.S.

If you ever develop a psychological disorder, many assume you will have it for life. The newest research suggests that for the most common psychological complaints, this is simply not true. "A substantial component of what we describe as disorder is often short-lived, of lesser severity or self-limiting," says John Horwood, a psychiatric epidemiologist and director of the longitudinal Christchurch Health and Development Study in New Zealand. (Horwood has found that close to 85 percent of the Christchurch study members have a diagnosable mental illness by midlife.)

This may be a useful message to spread. According to Jason Siegel, a professor of social psychology at Claremont Graduate University, people tend to be more sympathetic and helpful when they believe that a friend or co-worker's health problems are temporary.

And individuals with mental illness need support. Even short-lived or self-limiting disorders can wreak havoc on a person's life. To be classified as having a disorder, "an individual typically has to meet fairly stringent symptom criteria," Horwood says. "There needs to be substantial impairment of functioning."

To some, though, the new statistics on mental illness rates can sound a lot like the overmedicalization of "normal" human experience. Advocates for people with mental health concerns tend to disagree with this perspective. "I'm not at all surprised by these findings," says Paul Gionfriddo, president of Mental Health America, a national advocacy group. His organization views mental illnesses as common, "though not always enduring." Three years ago Mental Health America launched a Web-based tool to allow individuals to discreetly screen themselves for possible psychological disorders. Since then, the tool has been used for more than 1.5 million screenings, with more than 3,000 screens a day now used to determine if people may have a condition that could benefit from treatment.

The widespread nature of mental illness, unearthed by careful longitudinal research, holds some implications for the way we study and treat disease in this country. To Gionfriddo, a former lawmaker who watched his son end up homeless and incarcerated following undiagnosed childhood schizophrenia, "one implication of these new studies is that we as a society will get tremendous benefit out of ubiquitous mental health screening."

Although the U.S. Preventive Services Task Force currently recommends mental health screening on a regular basis for everyone older than 11 years, such screening is still far from routine. "At a time when we have recognized the importance of early intervention for cancer or for diabetes or heart disease, why would we say, 'Okay, for mental illness, we aren't going to screen or do early intervention'?" Gionfriddo says. "This should be as common for adults as blood pressure screening. Putting our head in the sand and waiting for a catastrophe is not a health care plan."

Another implication stems from the fact that individuals who never develop a mental illness—those who experience what we call "enduring mental health"—are actually quite remarkable. These people may be the mental health equivalents of healthy centenarians: individuals who somehow manage to beat the odds and enjoy good health for much longer than we would expect. It is possible that studying the mentally robust more closely could provide insight into how we can help more people to enjoy lives like theirs.

Who are these extraordinary people? In Schaefer's New Zealand cohort, his team found that those with enduring mental health tended to have two things going for them: First, they had little to no family history of mental illness. Second, they tended to have what the researchers call "advantageous" personalities. As early as age five, individuals who would make it to midlife without an episode of mental disorder tended to display fewer negative emotions, get along better with their peers and have greater self-control. Perhaps just as striking, the team found that these individuals were *not* any richer, smarter or physically healthier than their peers, at least in childhood.

Ultimately the most important suggestion from the newest research is that mental health concerns may be nearly universal. As a result, society should begin to view mental illnesses like bone breaks, kidney stones or common colds—as part of the normal wear and tear of life. Acknowledging this universality may allow us to finally devote adequate resources to screening, treating and preventing mental illnesses. It may also help us go easier on ourselves and our loved ones when we inevitably hit our own rough patches in the road. M

OPINION

The Science of Passionate Sex

How to have hot sex, according to science

By Scott Barry Kaufman

NOVIEMBRE ANITA VELA *GETTY IMAGES*

Our culture is obsessed with sex. Everywhere you look is another article on how to have hot sex, harder erections, mind-bending orgasms and ejaculations that go on for days. What people seldom realize, though—and which the latest science backs up—is that *this is exactly the problem*.

There's nothing wrong with desiring sex. I'm extremely sex-positive. Rather I believe it's the obsessive focus on the pragmatics and mechanization of sex—in isolation from the rest of the person—that is making us actually less satisfied with sex. We aren't integrating our sexual desires into the totality of our being, and our whole selves are suffering as a result.

In a series of clever studies, psychologists Frédérick L. Philippe and Robert J. Vallerand and their colleagues studied a concept they refer to as harmonious sexual passion: passion for sex that is well integrated and in harmony with other aspects of the self, creating minimal conflict with other areas of life. Harmonious integration of one's sexual desires frees one up to fully engage in and enjoy sexual activity in an open, spontaneous and nondefensive manner. Items measuring harmonious sexual passion include "sex is in harmony with the other things that are part of me," "sex is well integrated in my life" and "sex is in harmony with the other activities in my life."

In contrast, those who have "obsessive sexual passion" have not integrated their sexuality well into the totality of their being. Their sexual desires remain detached from other areas of their self as well as other domains in life. This leads to more narrow goals, such as immediate sexual gratification (for example, orgasm) and to an urgent feeling of sex as a goal, compelling us to perform, instead of us being in control of our sexuality. Such behavior can significantly limit the full enjoyment of sex as well as life. Items measuring obsessive sexual passion include: "I have almost an obsessive feeling for sex," "sex is the only thing that really turns me on" and "I have the impression that sex controls me."

Across a number of studies, the researchers found that these two forms of sexual passion—obsessive and harmonious—differ remarkably in how sexual information is processed and how sexual activities are experienced. During sexual activities, obsessive sexual passion was related to negative emotions. And outside of sexual intercourse, it was related to intrusive thoughts about sex, conflict with other goals, attention to alternative partners and difficulty concentrating on a current goal when unconsciously viewing pictures of sexually attractive people.

Obsessive sexual passion was also related to the biased processing of information. Those scoring higher for this trait were more likely to perceive sexual intent in ambiguous social interactions, as well as to perceive sexuality in words that did not explicitly have a sexual connotation (for example, "nurse," "heels," "uniform"). Obsessive sexual passion was also related to violent actions under threat of romantic rejection, as well as greater dissolution of romantic relationships over time.

In contrast, harmonious sexual passion showed much greater integration with more loving aspects of the self, as well as

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other life domains. For instance, participants were asked to list as many words as they could in one minute related to the word "sex." Those scoring higher in harmonious sexual passion were still sexually passionate beings: they listed quite a number of sexually related words. But they had a more balanced profile of purely sexual representations (for example, "penis," "breasts," "vibrator") and sexual-relational representations ("intimate," "caress," "intercourse"). In fact, the magic number seemed to be a ratio of two: once the number of sexual words outweighed the number of sexual-relational words by a factor of two, there was a substantial increase in obsessive sexual passion and a marked decrease in harmonious sexual passion.

Those scoring high in harmonious sexual passion also showed greater control over their sexual drive. Whenever a sexual stimulus was subconsciously encountered (for example, a beautiful person), they were able to remain on task—which was to identify natural versus artificial objects. Harmonious sexual passion was also related to less sexually intrusive thoughts and was unrelated to attentiveness to alternative partners. This greater integration and absence of conflict led to higher relationship quality over time.

It is important to note that obsessive sexual passion is not the same thing as sexual compulsivity or even sex addiction (although some still debate whether sexual addiction actually exists). Even though obsessive sexual passion was correlated with negative emotions during sexual activity, it did not lead to greater feelings of distress. Also, both harmonious and obsessive sexual passion were related to loving and enjoying sex-related activities.

In fact, both harmonious and obsessive sexual passion were equally correlated with sexual desire. This is a really important finding because we have a tendency to stigmatize those with greater sociosexuality in our society. Those with a more unrestricted sociosexual orientation are more willing to engage in casual sex and report increased sexual desire and frequency of fantasizing about sex. These results suggest that sociosexuality itself is not the problem; rather it is how your sociosexuality is integrated into your identity and other areas of your life that matters.

Perhaps instead of fixating on our cultural obsession with sexual performance, we should shift more toward helping people accept and feel comfortable with their sexuality, embrace sexual passion and harness that passion in ways that bring joy, vitality and openness to all areas of their life. M

OPINION

Yes, We Can Communicate with Animals

But only in limited ways because our brains are so fundamentally different

By Denise D. Cummins

TIM ROBBERTS *GETTY IMAGES*

Looking directly at the camera, Adam Cole, host of NPR's Web series *Skunk Bear*, laments, *"It's pretty clear that I'll never get to have a real human-style conversation with an ape."*

In his short and very entertaining video, Cole summarizes decades of research aimed at teaching apes human language, all of which, we are to understand, came to naught. But what the video actually shows us is how little the average person (and many scientists) understands about language. At one point, Cole tells his dog to sit, and the dog sits. This, he tells us, is not evidence that the dog knows English.

But actually it is.

The dog's behavior shows us that he is capable of understanding the simple concept of sitting, that he is capable of distinguishing the verbal signal "sit" from other verbal signals and that he is capable of connecting the two. This isn't rocket science, it isn't magic and it isn't anthropomorphizing. It is just the way word learning works.

In studies conducted at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany, a border collie named Rico was taught the meanings of 200 words. He could even use the process of elimination to figure out unfamiliar words: if he already knew the word "ball," and his trainer showed him a ball and a stick and told him to get the "stick," he would bring the stick. He could remember new words even after a month of not hearing them.

More recently, another border collie named Chaser learned a whopping 1,022 words, as reported in February 2011 in the peer-reviewed science journal *Behavioural Processes*. Kanzi, the bonobo trained by psychologist Sue Savage-Rumbaugh, has amply and repeatedly demonstrated his knowledge of more than 3,000 lexigrams (word symbols). In a long-term study of chimpanzee gestural communication in the wild in Uganda, researchers at the University of St. Andrews in Scotland discovered that the apes communicated with one another through a repertoire of about 66 different gestures. Many of these gestures had been documented from other chimpanzee sites both in captivity and in the wild.

But I assure you, no dog (or ape) will ever learn words, lexigrams or gestures for "bacteria," "economy" or "atom." They may be able to hear or see the differences among them, but the concepts they represent are beyond their conceptual capacity. You can't learn words for things you can't understand.

Where the Rubber Hits the Road

But what Cole has in mind is having a conversation with an ape in the way humans converse with one another—with sentences. He points out that the longest "sentence" signed by a chimpanzee named Nim Chimpsky was *"Give orange me give eat orange me orange me eat give me you."*

What Nim's sentence lacks is not just conceptual complexity but grammatical orderliness. And this is what sets human languages apart from communicative systems of other species. Our languages consist of word categories such as nouns, verbs, adjectives, adverbs, prepositions, and so on. We modify word order and word endings to create different tenses so that we can describe events

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from the past or imaginary ones from the future. This grammatical complexity emerges quite early in child development, beginning in the second year of life and exploding with full force in the third year of life. No nonhuman animal to date has demonstrated the ability to construct sentences with the level of grammatical complexity typical of a three-year-old human child.

There are two reasons why humans are capable of understanding complex concepts and generating grammatically complex utterances. The first is our extraordinarily large brain. To appreciate just how large the human brain is, consider how our so-called encephalization quotient (EQ) compares with that of other species. Cephalization is the tendency for neural tissue to be located in the front (head) of an organism. It usually corresponds to brain size. EQ is an estimate of the possible intelligence of an animal.

An EQ of 1.0, for example, means that the species has (on average) the brain size that would be expected given its body size: we would expect a whale's brain to be bigger than a mouse's simply because a whale's body is so much more massive. An EQ of 2.0 means that the species has a brain twice as large as would be expected in an animal that size. Dogs have an EQ of about 1.0—their brain is about as big as you would expect it to be. Chimpanzees have an EQ of 2.5; dolphins have an EQ of 5.3. And humans? We have an EQ of about 7.5. Our brain is seven times larger than it should be given our body size. That is a very large brain.

It isn't just intelligence that matters. Even individuals with low IQ, such as those with Down syndrome or Williams syndrome, can master the complexity of human language just fine. The key is the way the human brain is genetically wired for communication.

The *FOXP2* gene is present in most species, from reptiles to humans. Its primary function appears to be directing neural wiring that impacts communication. Mice that are genetically altered to have only one functional copy of the *FOXP2* gene have significantly reduced vocalizations as pups. Altering the *FOXP2* genes of songbirds impairs their ability to learn and imitate songs.

About 200,000 years ago a mutation of the *FOXP2* gene appeared in hominins. This genetic mutation entirely replaced more primitive versions of the gene within 500 to 1,000 human generations—a mere 10,000 to 20,000 years, which is an eyeblink in evolutionary time. This is also the period when anatomically modern humans appeared. The consensus among scientists is that the *FOXP2* gene has been the target of heavy selection during recent human evolution because it changed the way our brain was wired for communication.

It is not reasonable to expect other species that have the more ancient form of this gene to master the grammatical complexity of human language. And it is not reasonable to expect other species with a smaller EQ to grasp the abstract concepts that humans readily grasp. But you can expect to communicate with them about concepts that are well within their mental capacity, using simple language.

What would that be for apes? In a 2007 paper primatologist Joan Silk put it this way:

Primates are endowed with cognitive abilities that are especially well suited to tracking social information. For example, primates are able to recognize individuals; identify kin; compute the value of resources and services; keep track of past interactions with group members; make transitive inferences; discriminate between cooperators and defec-

tors; and assess the qualities of prospective rivals, mates and allies.

If you want to communicate with an ape, try communicating about these topics. Just remember to keep it simple. You may be surprised. Boyce Rensberger, a former science writer for the *Washington Post*, learned American Sign Language (ASL) from his parents, who could neither speak nor hear, although he could do both. When interacting with a chimp who had mastered a bit of ASL, he said, "Suddenly, I realized I was conversing with a member of another species in my native tongue." M

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