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DOES INTELLIGENCE MATTER?

Past a certain age, expertise, wisdom and purpose may be more valuable than cognitive power

WITH COVERAGE FROM nature

PLUS

HOW OUR LANGUAGE SHAPES OUR PERCEPTION

THE EVOLUTIONARY ADVANTAGES OF MENTAL ILLNESS

IS ONLY-CHILD SYNDROME REAL?

FROM THE EDITOR

LIZ TORMES

Outsmarting Our Obsession with Smartness

If the nearly 180,000 parenting guides being offered on Amazon are any indication, people are eager to raise good kids. But more important, we want our children to be smart. After all, studies have shown that the more intelligent you are, the more money you'll make, the further you'll go in your career, and the better "luck" you'll have in life. So it's no wonder that we're all eager to improve our child's brainpower (and ours as well). Many research dollars and hours have been spent on how to trigger a cognitive boost and on how to measure it. Yet, as Scott Barry Kaufman asks in these pages, what if we're looking at this the wrong way? Rather than obsessing over something as fluid and mercurial as raw intelligence, perhaps we should be nurturing overall well-being, combined with a sense of purpose and meaning, in ourselves and in our children. Studies have shown that such factors are linked to longevity, reduced occurrence of heart trouble and stroke, and many other factors (see "When Does Intelligence Peak?").

Elsewhere in this issue, Catherine L. Caldwell-Harris describes the new data that show that, contrary to older hypotheses, our language can actually influence our visual perception (see "Our Language Affects What We See"). And Corinna Hartmann digs into the evidence for so-called only-child syndrome—are they really more self-focused and spoiled (see "Is Only-Child Syndrome Real?")? As the eldest child in my own family, I will refrain from commenting on this further, as I have biased opinions. But I hope you enjoy the read!

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On the Cover

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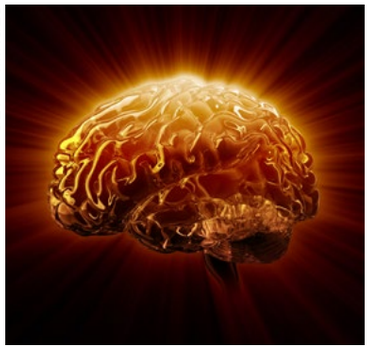

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WHAT'S INSIDE

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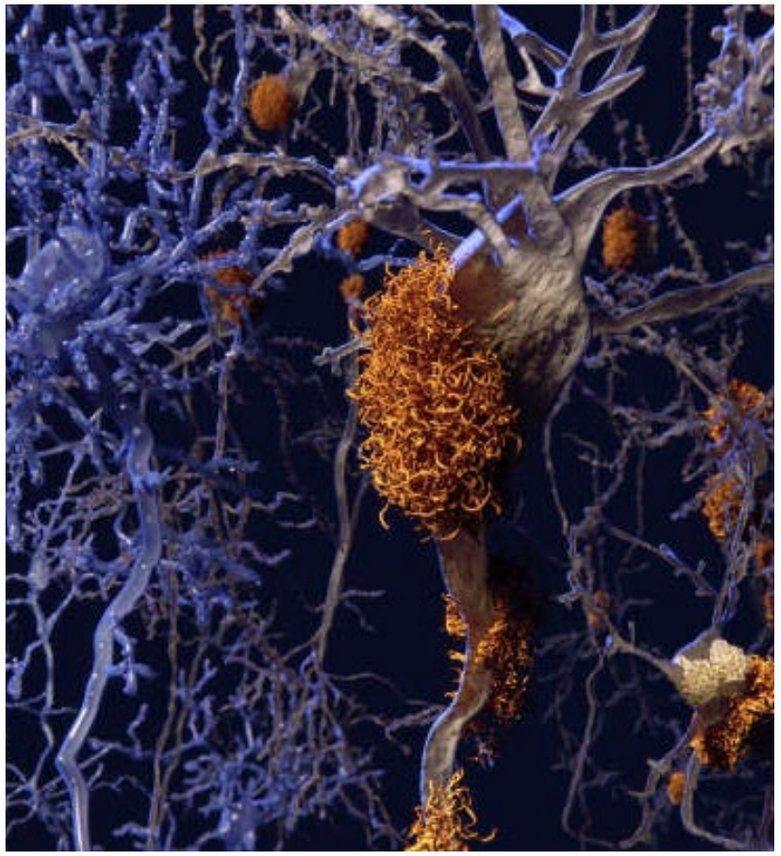

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Why the Secrets You Keep Are Hurting You

It may not be what you think

JUAN JIMENEZ *GETTY IMAGES*

IT HURTS TO KEEP secrets. Secrecy is associated with lower well-being, worse health and less satisfying relationships. Research has linked secrecy to increased anxiety, depression, symptoms of poor health and even the more rapid progression of disease. There is a seemingly obvious explanation for these harms: Hiding secrets is hard work. You have to watch what you say. If asked about something related to the secret, you must be careful not to slip up. This could require evasion or even deception. Constant vigilance and concealment can be exhausting.

New research, however, suggests that the harm of secrets doesn't really come from the hiding after all. The real problem with keeping a secret is not that you have to hide it, but that you have to live with it, and think about it.

The concept of secrecy might evoke an image of two people in conversation, with one person actively concealing from the other. Yet such concealment is actually uncommon. It is far more common to ruminate on our secrets. It is our tendency to mind-wander to our secrets that seems most harmful to well-being. Simply thinking about a secret can make us feel inauthentic. Having a secret return to mind, time and time again, can be tiring. When we think of a secret, it can make us feel isolated and alone.

To better understand the harms of secrecy, my colleagues and I first set out to understand what secrets people keep, and how often they

keep them. We found that 97 percent of people have at least one secret at any given moment, and people have, on average, 13 secrets. A survey of more than 5,000 people found that common secrets include preferences, desires, issues surrounding relationships and sex, cheating, infidelity and violations of others' trust.

Across several studies, we asked participants to estimate how frequently they concealed their secret during conversations with others, and also how frequently they thought about the secret outside of social interactions. We found that the more frequently people simply thought about their secrets, the lower their well-being. The frequency of active concealment when interacting with others, however, had no relationship to well-being.

Following up this research, a new paper reveals why thinking about secrets is so harmful. Turning the question around, we examined the consequences of confiding secrets. We found that when a person confides a secret to a third party, it does not reduce how often they have to conceal the secret from others who are still kept in the dark. Rather, it reduces how often their mind wanders toward the secret in irrelevant moments.

The act of confiding a secret can feel cathartic and relieving. But mere catharsis is not enough. When confiding a secret, what is actually helpful is the conversation that follows. People report that when sharing a secret with another person, they often receive emotional support, useful guidance and helpful advice. These forms of support make people feel more confident and capable in coping with the secret. When people find a healthier way of thinking about their secret, they ruminate less on it, and have improved well-being. Our studies suggest that what is important is talking to another person about a secret. A single conversation can lead to a healthier outlook and mind.

This new science of secrecy brings both good and bad news. The bad news is that even when we are not hiding our secrets, they are still very much with us, and can still hurt us. The good news is that even when we choose to still keep something secret, talking to another person can make the world of difference. Secrets don't have to hurt as much as they do.

—Michael Slepian

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The Creativity of ADHD

More insights on a positive side of a "disorder"

ATTENTION-DEFICIT/HYPERACTIVITY disorder (ADHD) is typically described by the problems it presents. It is known as a neurological disorder, marked by distractibility, impulsivity and hyperactivity, which begins in childhood and persists in adults. And, indeed, ADHD may have negative consequences for academic achievement, employment performance and social relationships.

But ADHD may also bring with it an advantage: the ability to think more creatively. Three aspects of creative cognition are divergent thinking, conceptual expansion and overcoming knowledge constraints. Divergent thinking, or the ability to think of many ideas from a single starting point, is a critical part of creative thinking.

Previous research has established that individuals with ADHD are exceptionally good at divergent thinking tasks, such as inventing creative new uses for everyday objects, and brainstorming new features for an innovative cell phone device. In a new study, college students with ADHD scored higher than non-ADHD peers on two tasks that tapped conceptual expansion and the ability to overcome knowledge constraints. Together with previous research, these new findings link ADHD to all three elements of the creative cognition trio.

Prior knowledge can be an obstacle to creativity. When we look to a prior model or example for inspiration, we may actually become stuck: designers refer to this as "fixation." In creative generation research, when participants are given examples before a task that requires them to invent something new, such as a new toy, their inventions tend to incorporate aspects of the examples—and thus are less novel. The ability to overcome recently presented information is therefore essential to creative thinking.

Similarly, knowledge of the world can stunt one's ability to imagine it differently. For instance, if asked to invent an animal or fruit that might exist on another planet, most people would start by thinking of a typical animal or fruit on Earth and then modify it somewhat into an "alien" version. Research suggests that when people invent alien creations based on specific Earth examples, the creations are rated as less original compared to those not inspired by specific examples. The key to being creative under these conditions is conceptual expansion, or the ability to loosen the boundaries of concepts. For instance, a paperclip is designed to hold papers together. By conceptual expansion, one might think beyond this definition and imagine a paperclip as something else—such as a tool to pry open the battery compartment of a wristwatch. In creating an alien animal, one might assume that the animal would need to be bilaterally symmetrical, as most Earth animals possess this attribute. Conceptual expansion might allow one to imagine an animal with an asymmetrical form, which differs from a typical Earth animal.

Evidence suggests that ADHD may offer some protection from the constraining effects of knowledge. In a study of adolescents, a group with ADHD was compared to a group of non-ADHD peers on a toy invention task. Participants were first shown a set of example toys that shared specific features (e.g., a ball), then asked to invent new toys that were very different from any existing toys. The toys invented by the ADHD group included fewer elements of the task examples compared to toys created by the non-ADHD group. In the same study, there were no differences between the ADHD and non-ADHD groups on a conceptual expansion task. However, a study of college students found that, compared to non-ADHD peers, ADHD students showed a broader scope of semantic activation—which is the "turning on" of concepts and ideas that are stored in memory and is correlated with conceptual expansion in other research. Given evidence that linked ADHD to higher divergent thinking and ability to overcome the constraining effect of task examples, it seemed intuitive to look more closely at the relationship between ADHD and the third element of creative thinking, conceptual expansion.

The innovative, original thinking style of people with ADHD may be a great fit for innovative fields where it's an advantage to be on the cutting edge.

I compared college students with and without ADHD on two tasks. In the first, I told participants to imagine they worked for an advertising agency and that they'd been asked to invent names for new products in three categories (pasta, nuclear elements and pain relievers). For each category, six examples were provided that shared certain endings (e.g., pain relievers ending in –ol, such as Midol, Tylenol, and Panadol). I then instructed participants to invent a name for a new product in each category without using any aspects of the examples provided. And then, to explore conceptual expansion, I asked participants to draw and describe a fruit that might exist on another

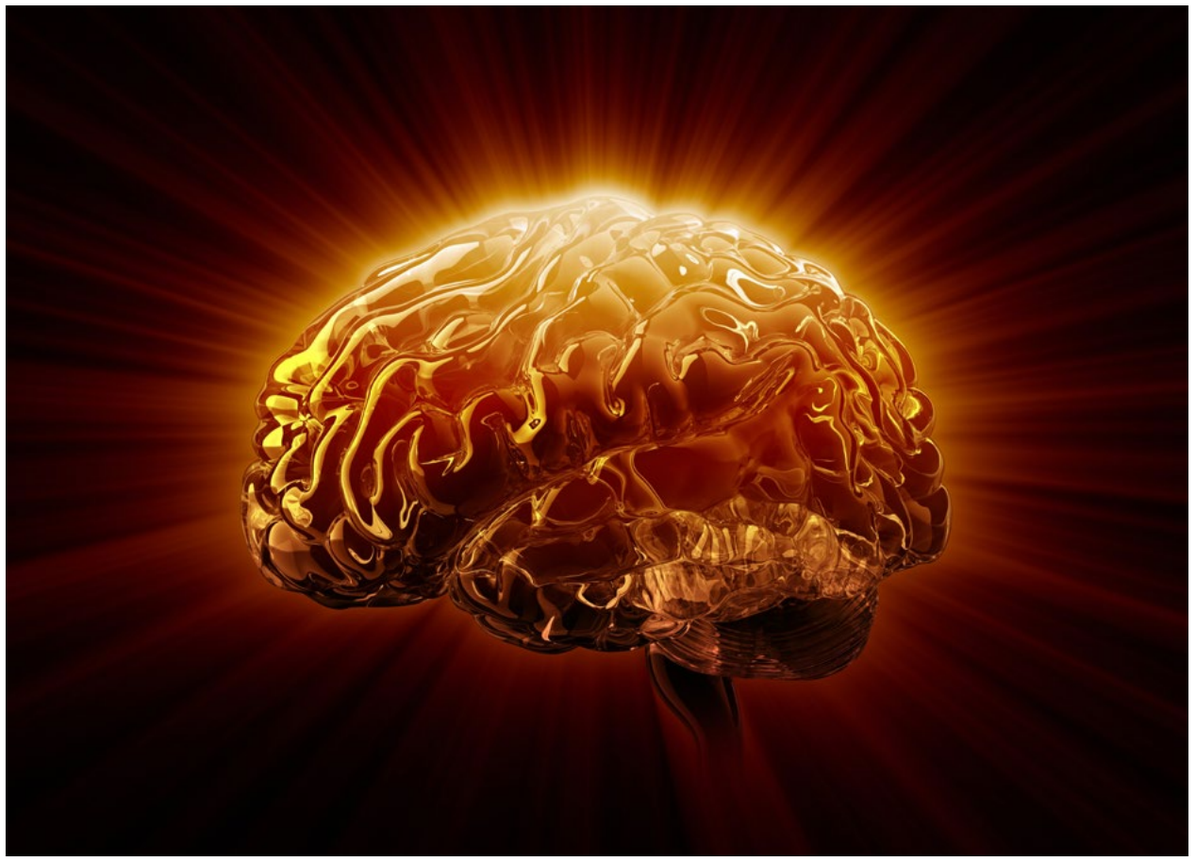
planet very different from Earth. The creations were to be as creative as possible and not duplicate any fruit that existed on Earth.

As expected, the ADHD students were less constrained by task examples on the product label invention task; compared to non-ADHD peers, ADHD students were less likely to include the example endings, yet invented labels that were equally descriptive of the product category. On the alien fruit task, the ADHD students invented fruits that were rated as more original and less representative of Earth fruit, compared to non-ADHD students. And while the groups were comparable in their inclusion of typical fruit features, such as seeds and stems, the ADHD students were more likely to include atypical features such as antenna, tongues, straws and hammers. The ADHD students also demonstrated higher conceptual expansion by violating conventional boundaries of the fruit category—for instance, by making the fruit poisonous or adding properties of nonliving things such as tools. Similar results have been reported for gifted individuals in a non-ADHD population.

At first glance, nonconformity and conceptual expansion may not sound very impressive. But in the context of creative innovation, a small change may unlock a breakthrough. Take the sewing needle, for example. The basic design (eye on the blunt end for threading) dates back to our Denisovan ancestors, at least 50,000 years ago. Then, in the early 1800s, inventor Balthasar Krems flipped that design upside-down to create the world's first eye-pointed needle—which paved the way for the sewing machine.

ADHD may create difficulties for individuals in many contexts that require focused, sustained attention—such as school, where students are expected to sit still and pay attention. On the other hand, the same distractibility and chaotic mind can give people with ADHD an edge when it comes to creative, original thinking. This new study suggests that ADHD may be especially beneficial when the goal is to create or invent something new without being locked into—and constrained by—old models or conventions. The innovative, original thinking style of people with ADHD may be a great fit for innovative fields where it's an advantage to be on the cutting edge.

—Holly White



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For Alzheimer's Sufferers, Brain Inflammation Ignites a Neuron-Killing "Forest Fire"

And it could also be the kindling sparking Parkinson's and other neurodegenerative maladies

FOR DECADES researchers have focused their attacks against Alzheimer's on two proteins, amyloid beta and tau. Their buildup in the brain often serves as a defining indicator of the disease. Get rid of the amyloid and tau, and patients should do better, the thinking goes.

But drug trial after drug trial has failed to improve patients' memory, agitation and anxiety. One trial of a drug that removes amyloid even seemed to make some patients

worse. The failures suggest researchers were missing something. A series of observations and recently published research findings have hinted at a somewhat different path for progression of Alzheimer's, offering new ways to attack a disease that robs memories and devastates the lives of 5.7 million Americans and their families.

One clue hinting at the need to look further afield was a close inspection of the 1918 worldwide flu pandemic, which left survivors with a higher chance of later developing Alzheimer's or Parkinson's. A second inkling came from the discovery that the amyloid of Alzheimer's and the alpha-synuclein protein that characterizes Parkinson's are antimicrobials, which help the immune system fight off invaders. The third piece of evidence was the finding in recent years, as more genes involved in Alzheimer's have been identified, that traces nearly all of them to the immune system. Finally, neuroscientists have paid attention to cells that had been seen as ancillary—"helper" or "nursemaid" cells. They have come to recognize these brain cells, called microglia and astrocytes, play a central role in brain function—and one intimately related to the immune system.

All of these hints are pointing toward the conclusion that both Alzheimer's and Parkinson's may be the results of neuroinflammation—in which the brain's immune system has gotten out of whack. "The accumulating evidence that inflammation is a driver of this disease is enormous," says Paul Morgan, a professor of immunology and a member of the Systems Immunity Research Institute at Cardiff University in Wales. "It makes very good biological sense."

The exact process remains unclear. In some cases the spark that starts the disease process might be some kind of insult—perhaps a passing virus, gut microbe or long-dormant infection. Or maybe in some people, simply getting older—adding some pounds or suffering too much stress could trigger inflammation that starts a cascade of harmful events.

This theory also would explain one of the biggest mysteries about Alzheimer's: why some people can have brains clogged with amyloid plaques and tau tangles and still think and behave perfectly normally. "What made those people resilient was lack of neuroinflammation," says Rudolph Tanzi, a professor of neurology at Harvard Medical School and one of the leaders behind this new view of Alzheimer's. Their immune systems kept functioning normally, so although the spark was lit, the forest fire never took off, he says. In Tanzi's fire analogy, the infection or insult sparks the amyloid match, triggering a brush fire. As amyloid and tau accumulate, they start interfering with the brain's activities and killing neurons, leading to a raging inflammatory state that impairs memory and other cognitive capacities. The implication, he says, is that it is not enough to just treat the amyloid plaques, as most previous drug trials have done. "If you try to just treat plaques in those people, it's like trying to put out a forest fire by blowing out a match."

"The accumulating evidence that inflammation is a driver of this disease is enormous."

—Paul Morgan

LIGHTING THE FIRE

One study published earlier this year found gum disease might be the match that triggers this neuroinflammatory conflagration—but Tanzi is not yet convinced. The study was too small to be conclusive, he says. Plus, he has tried to find a link himself and found nothing. Other research has suggested the herpes virus could start this downward spiral, and he is currently investigating whether air pollution might as well. He used to think amyloid took years to develop, but he co-authored a companion paper to the herpes one last year, showing amyloid plaques can literally appear overnight.

It is not clear whether the microbes—say for herpes or gum disease—enter the brain or whether inflammation elsewhere in the body triggers the pathology, says Jessica Teeling, a professor of experimental neuroimmunology at the University of Southampton in England. If microbes can have an impact without entering the brain or spinal cord—staying in what's called the peripheral nervous system—it may be possible to treat Alzheimer's without having to cross the blood-brain barrier, Teeling says.

Genetics clearly play a role in Alzheimer's, too. Rare cases of

Alzheimer's occurring at a relatively young age result from inheriting a single dominant gene. Another variant of a gene that transports fats in brain cells, *APOE4*, increases risk for more typical, later-onset disease. Over the last five years or so large studies of tens of thousands of people have looked across the human genome for other genetic risk factors. About 30 genes have jumped out, according to Alison Goate, a professor of neurogenetics and director of the Loeb Center for Alzheimer's Disease at Icahn School of Medicine at Mount Sinai in New York City. Goate, who has been involved in some of those studies, says those genes are all involved in how the body responds to tissue debris—clearing out the gunk left behind after infections, cell death and similar insults. So, perhaps people with high genetic risk cannot cope as well with the debris that builds up in the brain after an infection or other insult, leading to a quicker spiral into Alzheimer's. "Whatever the trigger is, the tissue-level response to that trigger is genetically regulated and seems to be at the heart of genetic risk for Alzheimer's disease," she says. When microglia—immune cells in the brain—are activated in response to tissue damage, these genes and *APOE* get activated. "How microglia respond to this tissue damage—that is at the heart of the genetic regulation of risk for Alzheimer's," she says.

"How microglia respond to this tissue damage—that is at the heart of the genetic regulation of risk for Alzheimer's."

—Alison Goate

But *APOE4* and other genes are part of the genome for life, so why do Alzheimer's and Parkinson's mainly strike older people? says Joel Dudley, a professor of genetics and genomics, also at Mount Sinai. He thinks the answer is likely to be inflammation, not from a single cause for everyone but from different immune triggers in different individuals.

Newer technologies that allow researchers to examine a person's aggregate immune activity should help provide some of those answers, he says. Cardiff's Morgan is developing a panel of inflammatory markers found in the blood to predict the onset of Alzheimer's before much damage is done in the brain, a possible diagnostic that could point to the need for anti-inflammatory therapy.

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A similar inflammatory process is probably also at play in Parkinson's disease, says Ole Isacson, a professor of neurology at Harvard Medical School. Isacson points to another early clue about the role of inflammation in Parkinson's: people who regularly took anti-inflammatory drugs like ibuprofen developed the disease one to two years later than average. Whereas other researchers focused exclusively on genetics, Isacson found the evidence suggested the environment had a substantial impact on who got Parkinson's.

In 2008 and 2009, Isacson worked with a postdoctoral student on an experiment trying to figure out which comes first in the disease process: inflammation or the death of dopamine-producing neurons, which make the brain chemical involved in transmitting signals among nerve cells. The student first triggered inflammation in the brains of some rodents with molecules from gram-negative bacteria and then damaged the neurons that produce dopamine. In another group of rodents, he damaged the neurons first and then introduced inflammation. When inflammation came first, the cells died en masse, just as they do in Parkinson's disease. Blocking inflammation prevented their demise, they reported in the *Journal of Neuroscience*.

Other neurodegenerative diseases also have immune connections. In multiple sclerosis, which usually strikes young people, the body's immune system attacks the insulation around nerve cells, slowing the transmission of signals in the body and brain.

The spinal fluid of people with MS include antibodies and high levels of white blood cells, indicating the immune system is revved up—although it is not clear whether that immune system activation is the cause or result of MS, says Mitchell Wallin, who directs the Veterans Affairs Multiple Sclerosis Centers of Excellence. People with antibodies to the Epstein-Barr virus in their

systems, especially if they caught the virus in late adolescence or early adulthood run a higher risk of developing MS—supporting the idea that an infection plays a role in MS.

Thanks to newer medications and improvements in fighting infections, people with MS are now living longer. This increased longevity puts them at risk for neurological diseases of aging, including Alzheimer's and Parkinson's, Wallin says. Lack of data has left it unclear whether people with MS are at the same, higher or lower risk for these diseases than the general population. "How common it is, we're just starting to explore right now," Wallin says.

COMING SOON?

It will be years before the concept of a neuroinflammatory can be fully tested, but there are already some relevant drugs in development. One start-up, California-based INmune Bio, recently received a $1-million grant from the Alzheimer's Association to advance XPro1595, a drug that targets neuroinflammation. The company is beginning its first clinical trial this spring, treating 18 patients with mild to moderate-stage Alzheimer's who also show signs of inflammation. The company plans to test blood, breath by-products and cerebral spinal fluid as well as conduct brain scans to look for changes in inflammatory markers. That first trial will just explore if XPro1595 can safely bring down inflammation and change behaviors such as depression and sleep disorders. Company CEO and co-founder Raymond Tesi says he expects to see those indicators improve, even in a short, three-month trial.

The best way to avoid Alzheimer's is to prevent it from ever starting, which might require keeping brain inflammation to a minimum, particularly in later life. Preventative measures are already well known: eat healthy foods, sleep well, exercise regularly, minimize stress and avoid smoking and heavy drinking.

You can't do anything about your genetics but living a healthy lifestyle will help control your inheritance, says Tanzi, who, along with Deepak Chopra, wrote a book on the topic, *The Healing Self: A Revolutionary New Plan to Supercharge Your Immunity and Stay Well for Life.* "It's important to get that set point as high as possible."

—Karen Weintraub

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The Kids (Who Use Tech) Seem to Be All Right

A rigorous new paper uses a new scientific approach that shows the panic over teen screen time is likely overstated

SOCIAL MEDIA is linked to depression—or not. First-person shooter video games are good for cognition—or they encourage violence. Young people are either more connected—or more isolated than ever.

Such are the conflicting messages about the effects of technology on children's well-being. Negative findings receive far more attention and have fueled panic among parents

and educators. This state of affairs reflects a heated debate among scientists. Studies showing statistically significant negative effects are followed by others revealing positive effects or none at all—sometimes using the same data set.

A new paper by scientists at the University of Oxford, published in January in *Nature Human Behaviour*, should help clear up the confusion. It reveals the pitfalls of the statistical methods scientists have employed and offers a more rigorous alternative. And most important, it uses data on more than 350,000 adolescents to show persuasively that, at a population level, technology use has a nearly negligible effect on adolescent psychological well-being, measured in a range of questions addressing depressive symptoms, suicidal ideation, prosocial behavior, peer-relationship problems and the like. Technology use tilts the needle less than half a percent away from feeling emotionally sound. For context, eating potatoes is associated with nearly the same degree of effect, and wearing glasses has a more negative impact on adolescent mental health.

"This is an incredibly important paper," says Candice Odgers, a psychologist studying adolescent health and technology at the University of California, Irvine, who wasn't involved in the research. "It provides a sophisticated set of analyses and is one of the most comprehensive and careful accountings of the associations between digital technologies and well-being to date. And the message from the paper is painstakingly clear: The size of the association documented across these studies is not sufficient or measurable enough to warrant the current levels of panic and fear around this issue."

To date, most of the evidence suggesting digital technologies negatively impact young people's psychological well-being comes from analysis of large, publicly available data sets. Those are valuable resources but susceptible to researcher bias, say Andrew Przybylski, an experimental psychologist at Oxford and his graduate student Amy Orben, co-authors of the new paper. To prove their point, they found over 600 million possible ways to analyze the data contained in the three data sets in their study. "Unfortunately, the large number of participants in these designs means that small effects are easily publishable and, if positive, garner outsized press and policy attention," they wrote.

"It's about setting a standard. This kind of data exploration needs to be systematic."

—Andrew Przybylski

This type of research intends to modify the status quo. "We're trying to move from this mindset of cherry-picking one result to a more holistic picture of the data set," Przybylski says. "A key part of that is being able to put these extremely minuscule effects of screens on young people in a real-world context."

That context is illuminating. Whereas their study found digital technology use was associated with 0.4 percent of the variation that disrupts adolescent well-being, the effects of smoking marijuana and bullying had much larger negative associations for mental health (at 2.7 and 4.3, respectively in one of the data sets). And some positive behaviors such as getting enough sleep and regularly eating breakfast were much more strongly associated with well-being than the average impact of technology use.

Strikingly, one of the data sets Przybylski and Orben used was "Monitoring the Future," an ongoing study run by researchers at the University of Michigan that tracks drug use among young people. The alarming 2017 book and article by psychologist Jean Twenge claiming that smartphones have destroyed a generation of teenagers also relied on the data from "Monitoring the Future." When the same statistics Twenge used are put into the larger context Przybylski and Orben employ, the effect of phone use on teen mental health turns out to be tiny.

The method the Oxford researchers used in their analysis is called Specification Curve Analysis, a tool that examines the full range of possible correlations and maps "the sum of analytical decisions that could be made when analyzing quantitative data." Rather than reporting a handful of results, researchers using SCA report all of them. It is the statistical equivalent of seeing the forest for the

trees. "It's about setting a standard," Przybylski says. "This kind of data exploration needs to be systematic."

All of this is not to say there is no danger whatsoever in digital technology use. In a previous paper, Przybylski and colleague Netta Weinstein demonstrated a "Goldilocks" effect showing moderate use of technology—about one to two hours per day on weekdays and slightly more on weekends—was "not intrinsically harmful," but higher levels of indulgence could be. And in a 2015 paper Odgers and a colleague reviewed the science addressing parents' top fears about technology and found two important things: First, most of what happens online is mirrored offline. Second, effects really do depend on the user; benefits are conferred on some, whereas risks are exacerbated for others, such as children who already suffer from mental health problems.

"We're all looking in the wrong direction," Odgers says. "The real threat isn't smartphones. It's this campaign of misinformation and the generation of fear among parents and educators."

—Lydia Denworth

A Touch to Remember

The sense of touch generates surprisingly powerful and long-lasting memories

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TOUCH IS PERHAPS the most intimate of the senses. When you grasp or brush against an object—anything from an outstretched hand to a leather-bound book—you are physically as close to it as you can possibly be. At that moment, specialized skin cells convey a wealth of information, such as shape, texture, size and weight. Yet when you stop touching that object, much of that information appears to fade away rather quickly. After a few days, you may only be able to bring a vague impression to mind. It would seem then that the sense of touch is largely useful in the moment, and not much after that.

Over the decades, there has been surprisingly little research to test that assumption. Yet a common perspective is that the sense of touch is, by far, of limited use over the long term, and especially when compared to the visual system. However, a new study by Fabian Hutmacher and Christof Kuhbandner, researchers at the University of Regensburg, provides the strongest challenge yet to that perspective. Their finding: the sense of touch generates memories that are far more complex and long-lasting than previously thought.

In the study's first experiment, blindfolded participants "haptically explored" over 150 household objects for one hour. This involved them picking up and touching a series of kitchen utensils, stationery goods and other items. They studied each object with their hands for 10 seconds each. Next, while remaining blindfolded, each participant completed a memory test. On this test, two nearly identical versions of each object were successively held (for instance, two dinner spoons). Only one of each had been presented before, and participants had to determine which.

When the memory test occurred just after the study period, participants chose the correct object 94 percent of the time. Just briefly touching an object enabled them to distinguish it with almost perfect accuracy. Given the challenge of memorizing the many details that may

differentiate an object from another (such as the curve of a spoon handle or its overall length), and the fact that hundreds of items were touched in a short period of time, that outcome is no small feat. Yet human beings can accomplish this with relative ease.

Just as impressively, when the memory test occurred a week later, very little had been forgotten. The average accuracy rate was 85 percent. Thus, not only does touch generate memories that are highly detailed and precise, but those memories can endure over the long term.

A second experiment was even more startling. This time, a new group of blindfolded participants explored the same objects by touch. Rather than carefully study, they simply rated how pleasant each object felt. There was no intentional effort to memorize. A surprise memory test, occurring one week later, was designed to be more difficult: participants were no longer blindfolded and had to *visually* identify which of two nearly identical objects they had touched before—and without having previously seen either or having another opportunity to touch. Yet the accuracy rate remained high (averaging 73 percent). Even when participants felt unsure and had to guess, they still identified the correct object more often than not.

It would appear then that the cognitive capacities of touch, which was among the first of the sensory systems to evolve, have long been underestimated. Contrary to the view that it is only useful in real time, touch leaves a memory trace that persists long after the physical sensation is gone. Moreover, information appears to be stored without much conscious awareness. As a result, those memories can manifest in interesting ways. For instance, you may not be able to verbalize how something felt, but you will be able to recognize it by grasping it or looking at it.

The finding that touch generates memories of how an unseen object should look—an almost magical ability—remains to be fully explained. However, neuroimaging studies have found that touch not only activates the somatosensory cortex (the brain region that processes the sense of touch) but can also activate regions that are involved in processing visual signals. Thus, it could be that when you touch an unseen object, your brain forms a mental image of its probable appearance. This may be especially likely when that object resembles a familiar item that you have come into contact with before. When you do see that object for the first time, you can recognize it.

Typically, the sense of touch is so omnipresent, and its effects so seemingly ephemeral, that it is often taken for granted or overlooked. We now know that its effects linger in the brain long after the sensory experience ends, and often without our being directly aware of it. Surprisingly large amounts of information are preserved as well. As such, a single touch has a far greater impact on the mind than one might have ever imagined—which makes the act of doing so all the more powerful.

—Steven C. Pan

Drunk Witnesses Remember a Surprising Amount

Interviewing an inebriated person at the scene may be more accurate than waiting until he or she is sober

POLICE OFFICERS investigating a crime may hesitate to interview drunk witnesses. But waiting until they sober up may not be the best strategy; people remember more while they are still inebriated than they do a week later, a new study finds.

Malin Hildebrand Karlén, a senior psychology lecturer at Sweden's University of Gothenburg, and her colleagues recruited 136 people and gave half of them vodka mixed with orange juice. The others drank only juice. In 15 minutes women in the alcohol group consumed 0.75 gram of alcohol per kilogram of body weight, and men drank 0.8 gram (that is equivalent to 3.75 glasses of wine for a 70-kilogram woman or four glasses for a man of the same weight, Hildebrand Karlén says). All participants then watched a short film depicting a verbal and physical altercation between a man and a woman. The researchers next asked half the people in each group to freely recall what they remembered from the film. The remaining participants were sent home and interviewed a week later.

The investigators found that both the inebriated and sober people who were interviewed immediately demonstrated better recollection of the film events than their drunk or sober counterparts who were questioned later. The effect held even for people with blood alcohol concentrations of 0.08 or higher—the legal limit for driving in most of the U.S. (Intoxication levels varied because different people metabolize alcohol at different speeds.) The results suggest that intoxicated witnesses should be interviewed sooner rather than later, according to the study, which was published online last October in *Psychology, Crime & Law*.

The findings are in line with previous research, says Jacqueline Evans, an assistant professor of psychology at Florida International University, who was not involved in the new work. Evans co-authored and published a 2017 study in *Law and Human Behavior* that found similar results for moderately drunk witnesses. "Any effect of intoxication is not as big as the effect of waiting a week to question somebody," she says.

KRIS KESIAK *GETTY IMAGES*

The new study also found that some aspects of the drunk people's recollections were not that different from those of the sober participants. For instance, both groups seemed particularly attuned to the details of the physical aggression portrayed in the film. "This research should at least make us more interested in what intoxicated witnesses have to say," Hildebrand Karlén says, "and perhaps take them a bit more seriously."

—Agata Boxe

A new look at "the Russian blues" demonstrates the power of words to shape perception

By Catherine L. Caldwell-Harris

Our Language Affects What We See

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Does the language you speak influence how you think?

Catherine Caldwell-Harris, associate professor at Boston University, directs the Psycholinguistics Laboratory in the department of psychological and brain sciences. She brings her cognitive science training to a range of interdisciplinary questions, including cross-cultural psychology, foreign language learning, immigration, using technology to facilitate language learning in autistic children, understanding humor in another language and why people believe or do not believe in God.

This is the question behind the famous linguistic relativity hypothesis, that the grammar or vocabulary of a language imposes on its speakers a particular way of thinking about the world.

The strongest form of the hypothesis is that language determines thought. This version has been rejected by most scholars. A weak form is now thought to be obviously true, which is that if one language has a specific vocabulary item for a concept but another language does not, then speaking about the concept may happen more frequently or more easily. For example, if someone explained to you, an English speaker, the meaning for the German term *Schadenfreude*, you could recognize the concept, but you may not have used the concept as regularly as a comparable German speaker.

Scholars are now interested in whether having a vocabulary item for a concept influences thought in domains far from language, such as visual perception. Consider the case of the "Russian blues." While English has a single word for blue, Russian has two words, *goluboy* for light blue and *siniy* for dark blue. These are considered "basic level" terms, like green and purple, since no adjective is needed to distinguish them. Lera Boroditsky and her colleagues displayed two shades of blue on a computer screen and asked Russian speakers to determine, as quickly as possible, whether the two blue colors were different from each other or the same as each other. The fastest discriminations were when the displayed colors were *goluboy* and *siniy*, rather than two shades of *goluboy* or two shades of *siniy*. The reaction time advantage for lexically distinct blue colors was strongest when the blue hues were perceptually similar.

To determine if words were being automatically (and perhaps unconsciously) activated, the researchers added the following twist: they asked their Russian participants to perform a verbal task at the same time as making their perceptual discrimination. This condition eliminated the reaction time advantage of contrasting *goluboy* and *siniy*. However, a nonverbal task (a spatial task) could be done at the same time while retaining the *goluboy*/*siniy* advantage. The dual task variants indicated that the task of discriminating color patches was aided by silent activation of verbal categories. English speakers tested on the identical discrimination tasks showed no advantage for the light blue/dark blue trials.

Recently the Russian blues have been used again to investigate how language influences thought. In the journal *Psychological Science*, Martin Maier and Rasha Abdel Rahman investigated whether the color distinction in the Russian blues would help the brain become consciously aware of a stimulus that might otherwise go unnoticed. Would salience help a light blue color or a dark blue color be noticed (i.e., enter conscious awareness) in a situation in which attention is overloaded and not all stimuli can be noticed?

The task selected to investigate this is the "attentional blink." This is an experimental paradigm frequently used to test whether a stimuli is consciously noticed. Research participants are asked to monitor a sequence of stimuli, displayed at high speeds (typically at least 10 per second), and to press a button every time they see a certain item. The searched-for item can be a letter amid a sequence of numbers, or that target can be, for example, an emotion word in a sequence of neutral words. Participants are very good at detecting the first target they see, but if a second target follows immediately after the first, or with a lag of two to three items, the second target can be missed. It is as if the brain's attentional system "blinked." The reason for the missed item can be understood intuitively: the

brain was busy processing the first target and didn't have attentional resources to spare to detect the second target.

In the decades since it was discovered, the attentional blink has been used in myriad ways to document what stimuli have an advantage in capturing attention. For example, imagine that you are asked to monitor for instances of proper names in a stream of rapidly displayed nouns. You do not miss your own name even if it occurs after a prior target. Researchers conclude that the salience of your own name protects it from the attentional blink.

Would the salience of a blue color contrast, using the Russian blues, protect a stimulus from the attentional blink? The authors tested whether colored triangles could be detected more easily when the triangles were made visually salient by being positioned against a contrasting color. For example, a dark green color against a light green background is harder to see than a dark green color against a dark blue background. Green against blue is easier to see because of the strong color contrast between dark blue and dark green provided by linguistic categorization. What if the colors were *goluboy* and *siniy*? For Russians speakers, contrasting light and dark blue should be as salient as the contrast between dark green and dark blue (always being careful to keep perceptual similarity between contrasting stimuli comparable).

Maier and Rahman designed stimuli that were geometric shapes positioned against a light blue circle. The task of research participants was to press a button when they saw either a semicircle or a triangle, ignoring stars, squares, diamonds and other shapes. Distractor shapes were plain gray shapes against a light blue background. As noted, the targets, which were triangles or semicircles, were colored in ways that allowed their visual distinctiveness to be precisely varied. The least salient triangle was a light green triangle against a dark green background. This was not salient because the two green colors are in the same linguistic category. A highly salient stimulus was a green (either light or dark green) triangle against a blue (either light or dark blue) background, because the colors were in different linguistic categories. A stimulus that would also be highly salient for Russian speakers was a light or dark blue triangle positioned against a circle with the differing blue color.

While English has a single word for blue, Russian has two words, *goluboy* for light blue and *siniy* for dark blue.

The attentional blink task contained a sequence of two to six stimuli to be ignored (nontarget shapes), then a colored semicircle (target 1), and then, followed by a lag of either three or seven items, the second target, a triangle. At lag 3, when participants' brains were busy processing target 1, how difficult would it be to detect the green triangle?

The results supported the hypothesis that the linguistic distinction of the Russian blues helps stimuli enter conscious awareness. That is, the least salient targets, green triangles on green backgrounds, were missed the most. The easiest target to detect was the blue/green contrast. But more important, the contrast between *goluboy* (light blue) and *siniy* (dark blue) was a stimulus that grabbed the brain's attention centers more than the light green/dark green contrast. Interestingly, these results were also found in a study of Greek speakers, as Greek resembles Russian in having separate lexical items for light and dark blue. German was used as the "control" language since like English, it has only one word for blue. For German speakers, detection rates of the blue/blue and green/green trials were identical.

What is occurring in the brain during this visual task? The authors monitored scalp potentials during the attentional blink task. When blue contrasts were detected (meaning the blink was avoided), an event related potential occurred that is known to accompany the stage of early visual processing. This neural signature was not present for the light green/dark green stimulus, indicating that the brain processes the light blue/dark blue differently, for speakers whose language makes a lexical distinction.

The current study is an important advance in documenting how linguistic categories influence perception. Consider how this updates the original Russian blues study, in which observers pressed a button to indicate whether two shades of blue were the same or different. In that study, it seems likely that observers silently labeled colors in order to make fast decisions. It is less likely that labeling was used during the attentional blink task, because paying attention to color is not required and indeed was irrelevant to the task. All observers had to do is try to detect a triangle in a rapid sequence of diverse shapes. It is thus a powerful finding that the incidental contrast of dark blue triangle against a light blue background helped push the triangle into conscious awareness.

What arenas of perceptual-linguistic interaction remain to be conquered? The current finding indicates that linguistic knowledge can influence perception, contradicting the traditional view that perception is processed independently from other aspects of cognition, including language. This is most famously seen in the case of visual illusions, which are mostly impervious to knowledge about the illusion. Hmm. One wonders: Could the Russian blues be recruited in altering a visual illusion that depends on color shades?

Why Do We Crave Sweets When We're Stressed?

A brain researcher explains our desire for chocolate and other carbs during tough times

By Achim Peters

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Achim Peters is a brain researcher and diabetologist. He leads the Selfish Brain clinical research group at the University of Lübeck and has authored two books on how the selfish brain influences weight under chronic stress.

Although our brain accounts for just 2 percent of our body weight, the organ consumes half of our daily carbohydrate requirements—and glucose is its most important fuel. Under acute stress the brain requires some 12 percent more energy, leading many to reach for sugary snacks.

Carbohydrates provide the body with the quickest source of energy. In fact, in cognitive tests subjects who were stressed performed poorly prior to eating. Their performance, however, went back to normal after consuming food.

When we are hungry, a whole network of brain regions activates. At the center are the ventromedial hypothalamus and the lateral hypothalamus. These two regions in the upper brain stem are involved in regulating metabolism, feeding behavior and digestive functions. There is, however, an upstream gatekeeper, the nucleus arcuatus in the hypothalamus. If it registers that the brain itself lacks glucose, this gatekeeper blocks information from the rest of the body. That's why we resort to carbohydrates as soon as the brain indicates a need for energy, even if the rest of the body is well supplied.

To further understand the relationship between the brain and carbohydrates, we examined 40 subjects over two sessions. In one, we asked study participants to give a 10-minute speech in front of strangers. In the other session they were not required to give a speech. At the end of each session, we measured the concentrations of stress hormones cortisol and adrenaline in participants' blood. We also provided them with a food buffet for an hour. When the participants gave a speech before the buffet, they were more stressed, and on average consumed an additional 34 grams of carbohydrates, than when they did not give a speech.

So what about that chocolate, then? If a person craves chocolate in the afternoon, I advise him or her to eat chocolate to stay fit and keep his or her spirits up. That's because at work people are often stressed and the brain has an increased need for energy. If one doesn't eat anything, it's possible the brain will use glucose from the body, intended for fat and muscle cell use, and in turn secrete more stress hormones. Not only does this make one miserable, it can also increase the risk of heart attacks, stroke or depression in the long run. Alternatively, the brain can save on other functions, but that reduces concentration and performance.

In order to meet the increased needs of the brain, one can either eat more of everything, as the stressed subjects did in our experiment, or make it easy for the body and just consume sweet foods. Even babies have a pronounced preference for sweets. Because their brain is extremely large compared with their tiny body, babies require a lot of energy. They get that energy via breast milk, which contains a lot of sugar. Over time, our preference for sweets decreases but never completely disappears, even as we become adults. The extent to which that preference is preserved varies from person to person and seems to depend, among other things, on living conditions. Studies suggest people who experience a lot of stress in childhood have a stronger preference for sweets later in life.

For some, the brain cannot get its energy from the body's reserves, even if there are enough fat deposits. The most important cause of this is chronic stress. To ensure their brain is not undersupplied, these people must always eat enough. Often the only way out of such eating habits is to leave a permanently stressful environment. So although many tend to be hard on themselves for eating too many sweets or carbs, the reasons behind such craving aren't always due to a lack of self-control and might require a deeper look into lifestyle and stressful situations—past and present. Once the root cause of stress is addressed, eating habits could ultimately resolve themselves.

Susceptibility to Mental Illness May Have Helped Humans Adapt over the Millennia

Psychiatrist Randolph Nesse, one of the founders of evolutionary medicine, explains why natural selection did not rid our species of onerous psychiatric disorders

By Dana G. Smith

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NEARLY ONE IN FIVE AMERICANS CURRENTLY suffers from a mental illness, and roughly half of us will be diagnosed with one at some point in our lives. Yet these occurrences may have nothing to do with a genetic flaw or a traumatic event.

Randolph Nesse, a professor of life sciences at Arizona State University, attributes high rates of psychiatric disorders to natural selection operating on our genes without paying heed to our emotional well-being. What's more, the selective processes took place thousands of years before the unique stresses of modern urban existence, leading to a mismatch between our current environment and the one for which we were adapted.

In his new book, *Good Reasons for Bad Feelings: Insights from the Frontier of Evolutionary Psychiatry*, Nesse recruits the framework of evolutionary medicine to make a case for why psychiatric disorders persist despite their debilitating consequences. Some conditions, like depression and anxiety, may have developed from normal, advantageous emotions. Others, such as schizophrenia or bipolar disorder, result from genetic mutations that may have been beneficial in less extreme manifestations of a trait. *Scientific American* spoke to Nesse about viewing psychiatry through an evolutionary lens to help both patients and clinicians.

[*An edited transcript of the interview follows.*]

A big part of your thesis is that some traits of mental disorders can be advantageous or adaptive—a depressed mood, for instance, might be beneficial for us. Where do you draw the line between the normal spectrum of emotion and pathology?

You can't decide what's normal and what's abnormal until you understand the ordinary function of any trait—whether it's vomiting or cough or fever or nausea. You start with its normal function and in what situation it gives selected advantages. But there are a lot of places where natural selection has shaped mechanisms that express these defenses when they're not needed, and very often that emotional response is painful and unnecessary in that instance. Then there's a category of emotions that make us feel bad but benefit our genes. A lot of sexual longings [extramarital affairs or unrequited love], for instance, don't do us any good at all, but they might potentially benefit our genes in the long run.

So it's not saying that these emotions are useful all the time. It's the capacity for these emotions that is useful. And the regulation systems [that control emotion] were shaped by natural selection—so sometimes they're useful for us, sometimes they're useful for our genes, sometimes it's false alarms in the system and sometimes the brain is just broken. We shouldn't try to make any global generalizations; we should examine every patient individually and try to understand what's going on.

In the book you suggest that low mood could be advantageous for two very separate reasons. One of the motivators is to shift strategies to escape a situation, and the other is to have people stop striving and conserve energy. How do you reconcile these opposing theories?

It's intuitively obvious that when an organism, not just a human, is wasting energy trying to pursue a goal and not making progress, it's best to wait and slow down and not waste energy. Then if nothing works—even when you try to find a new strategy—to give up that goal completely.

Of course for we humans, it's not always seeking out nuts and fruits and berries. We're trying to garner social resources, and that creates inordinate complexity and competition. And it's not so easy to give up looking for a marital partner or give up looking for a job; we can't just do that. These moods are guiding us to try to put effort into things that are going to work instead of things that are not going to work. That doesn't mean we should just follow them, but it does mean respecting them more and trying to figure out what they might be telling us about the things we're trying to do in life.

Could treating someone with antidepressants be disadvantageous, then, if low mood is a normal coping mechanism?

Dana G. Smith is a freelance science writer specializing in brains and bodies. She has written for *Scientific American*, the *Atlantic*, the *Guardian*, NPR, *Discover*, and *Fast Company*, among other outlets. In a previous life, she earned a Ph.D. in experimental psychology from the University of Cambridge.

Evolutionary psychology I see very much as a subset of evolutionary medicine in general. And one of the most practically useful insights of evolutionary medicine is that we should be analyzing the costs and benefits of blocking every single defensive response, whether it's fever or pain or nausea or vomiting or cough or fatigue. Usually because of the "smoke detector" principle you can block these things safely. [The principle is Nesse's theory that an overactive fight-or-flight response that causes false alarms—and potentially an anxiety disorder—is better than an underactive system that fails to alert you to danger and could result in death.]

Some people have said that because I say low mood can be useful, I think we shouldn't treat it with medications. I say exactly the opposite. Once you know that low mood is usually not helpful even though it's normal, you go ahead and relieve it however you can.

You talk a lot about genes in the book, but also how we've come up short in looking for genes for depression or schizophrenia. What role do you think genes play in the evolutionary model of the mental illness?

First of all, there are two very different categories of illness that should be kept separate. One is the emotional disorders, which are potentially normal, useful responses to situations. And in all such responses, variability and sensitivity are influenced by lots of different genes.

There are also mental disorders that are the most severe ones that are just plain old genetic diseases: bipolar disease and autism and schizophrenia. They're genetic diseases, and whether you get them or not is overwhelmingly dependent on what genes you have. But why would a strong, inheritable trait that cuts fitness by half not be selected against? I think this is one of the deepest mysteries in psychiatry.

What could be some of the potential benefit of these latter conditions or other uses for these genes?

For bipolar disease, the reduction in the number of offspring is not very great at all, so it might be that there's not much selection acting there. And what if a tendency to be bipolar resulted in having even more children? What would happen then? It [the gene] would become universal, even though it caused bipolar disease. Maybe something like this has already happened. Maybe many of us have tendencies to grand ambitions and mood swings that probably aren't good for us but might lend to grand successes on occasion, and that might lead to great reproductive success.

Then there's the "cliff edge" effect, which is the possibility that some traits are pushed very far toward a peak that's close to a place where fitness collapses for a few percent of the population. This could be a new way of looking at all of these diseases in which we have many genes with small effects. It might be that what we should be looking for is the fitness landscape and not assume that the genes involved are abnormal.

What do you hope patients or clinicians can gain from reading your book?

I find many of my patients feel like they're abnormal if they are told, "You have an anxiety disorder; you have a depressive disorder." I talk with them a little bit about the fact that there are advantages to anxiety and that low moods might have meaning. It might not just be something that's broken in you—it might be that your emotions are trying to tell you something. I think that makes many people feel less like they're defective.

Is Only-Child Syndrome Real?

Children without siblings have long been thought of as spoiled and selfish. Are the claims true?

By Corinna Hartmann

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Corinna Hartmann has a bachelor's degree in psychology and works as a science journalist in Saarbrücken, Germany.

ONLY CHILDREN always want to get their way, can't share and are generally selfish—or so the long-held prejudice goes. According to recent research, however, these claims are overstated. So where did these biases come from?

In *A Study of Peculiar and Exceptional Children*, published in the 19th century, E. W. Bohannon of Clark University in Massachusetts detailed the results of a questionnaire—a new form of data collection at the time—filled out by 200 test subjects. In it he had asked respondents about the peculiarities of any only children they knew. In 196 cases, participants described children without siblings as excessively spoiled.

Bohannon's colleagues agreed with the results, and the idea took hold. The widespread skepticism toward only children was further strengthened by the fact middle-class families were having fewer children and society's privileged class feared growth of the population's "inferior strata." Furthermore, in the early 20th century, some were concerned that growing up without siblings causes children to become hypersensitive: If the parents concentrated all their worries and fears on one offspring, that child would become overly sensitive and eventually a hypochondriac with weak nerves.

According to data compiled in the 21st century, however, these notions are nonsense, and only children show no serious deficits. Toni Falbo, a psychologist at the University of Texas at Austin, and an only child, opposes the idea you need brothers and sisters to grow into a decent person. In her 1986 survey, for which she examined more than 200 studies on the subject, she concluded the characteristics of children with and without siblings do not differ. The only difference, she found, was that only children seemed to have stronger bonds with their parents compared with children who had siblings.

This idea was later confirmed by a 2018 study in which Andreas Klocke and Sven Stadtmüller of the Frankfurt University of Applied Sciences used longitudinal data from around 10,000 German schoolchildren to track down the peculiarities of firstborns, only children and those with siblings. Among other things, they looked at the quality of the parent-child relationship, a metric measured by how easy it was for a child to speak with their parents about important matters.

Twenty-five percent of only children considered their relationship with their parents positive. Just under 24 percent of firstborns, 20 percent of middle children and 18 percent of youngest children also reported very good relationships with their parents.

Despite having strong bonds with their parents, only children often regret having grown up without siblings. In 2001 Lisen Roberts of Western Carolina University and Priscilla Blanton of the University of Tennessee Knoxville asked young adults to look back on their childhoods. Many found it particularly unfortunate they did not have a trusted playmate as those with siblings had. In fact, preschool-aged only children often developed imaginary friends with whom they could be allies and share everyday things. But there's no reason for concern—creative play with imaginary companions promotes social development and the ability to communicate.

There are, however, indications only children are less willing to come to terms with others. In new findings from China, where the one-child policy dictated family planning for nearly four decades, researchers led by psychologist Jiang Qiu of Southwest University, Chongqing, examined 126 students without siblings and 177 with siblings in terms of thinking ability and personality. In one survey only children achieved lower scores in terms of how tolerant they were. According to the five-factor model, a model of personality dimensions, particularly tolerant people are altruistic, helpful, compassionate and cooperative. Intolerant individuals are often characterized as quarrelsome, distrustful, egocentric and more competitive.

The students were also asked to master a creativity test known as the Torrance Tests of Creative Thinking. For example, they had to come up with as many original uses for an everyday object, such as a tin can. As it turns out, only children seem to be better lateral thinkers, meaning they could solve problems more creatively, especially in the category of flexible thinking. This, the authors explain, could be because without siblings only children often had

to rely on themselves and were thus forced to become inventive and resourceful at an early age.

But that is not all. MRI tests revealed differences in brain structure. In the supramarginal gyrus, a cortical area associated with creativity and imagination, researchers found more gray matter (linked to intelligence) among only children. Researchers, however, discovered fewer gray cells in the frontal brain, more precisely in the medial prefrontal cortex, of only children than those with siblings. This deficit was accompanied by lower tolerance. Earlier studies also attributed important functions to this brain region when it comes to processing emotional information, including the ability to attribute feelings to others and regulating one's own emotions.

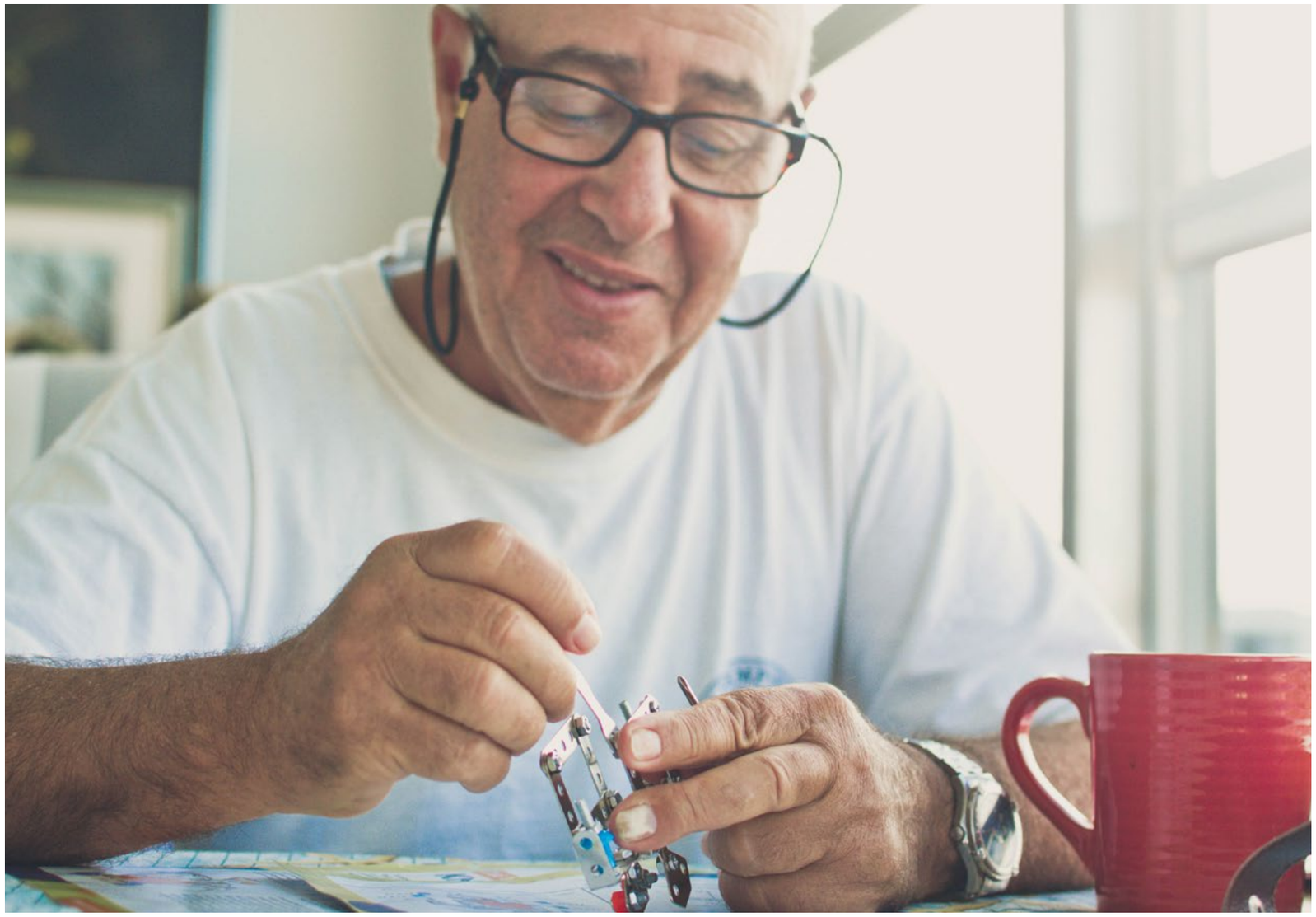
How much influence the effect of being an only child has is questionable. It may depend on how many other opportunities an only child regularly has to develop his or her social and cognitive abilities. After all, only children are by no means cut off from social settings—contacts in kindergarten, for example, offer a varied interpersonal training ground. Parents likely have to work harder at teaching their only kids social skills and engineering opportunities where children would have to share their toys, books and parental attention. Otherwise, creating a loving and calm environment seems more important than the number of children in a household.

Scott Barry Kaufman is a psychologist at Barnard College, Columbia University, exploring intelligence, creativity, personality and well-being. He hosts The Psychology Podcast, and is author and/or editor of eight books, including *Wired to Create: Unravelling the Mysteries of the Creative Mind* (with Carolyn Gregoire) and *Ungifted: Intelligence Redefined*.

BEAUTIFUL MINDS

When Does Intelligence Peak?

Maybe that's not even the right question



FRAN POLITO *GETTY IMAGES*

When does cognitive functioning peak? As we get older, we certainly feel as though our intelligence is rapidly declining. (Well, at least I do!) However, the nitty-gritty research on the topic suggests some really interesting nuance. As a recent paper notes, "Not only is there no age at which humans are performing at peak on all cognitive tasks, there may not be an age at which humans perform at peak on most cognitive tasks."

In one large series of studies, Joshua Hartshorne and Laura Germine presented evidence from 48,537 people from standardized IQ and memory tests. The results revealed that processing speed and short-term memory for family pictures and stories peak and begin to decline around high school graduation; some visual-spatial and abstract reasoning abilities plateau in early adulthood, beginning to decline in the 30s; and still other cognitive functions such as vocabulary and general information do not peak until people reach their 40s or later.

THE DARK MATTER OF INTELLIGENCE

The picture gets even more complicated, however, once we take into account the "dark matter" of intelligence. As Phillip Ackerman of the Georgia Instutite of Technology points out, should we really be judging adult intelligence by the same standard we judge childhood intelligence? At what point does the cognitive potential of youth morph into the specialized expertise of adulthood?

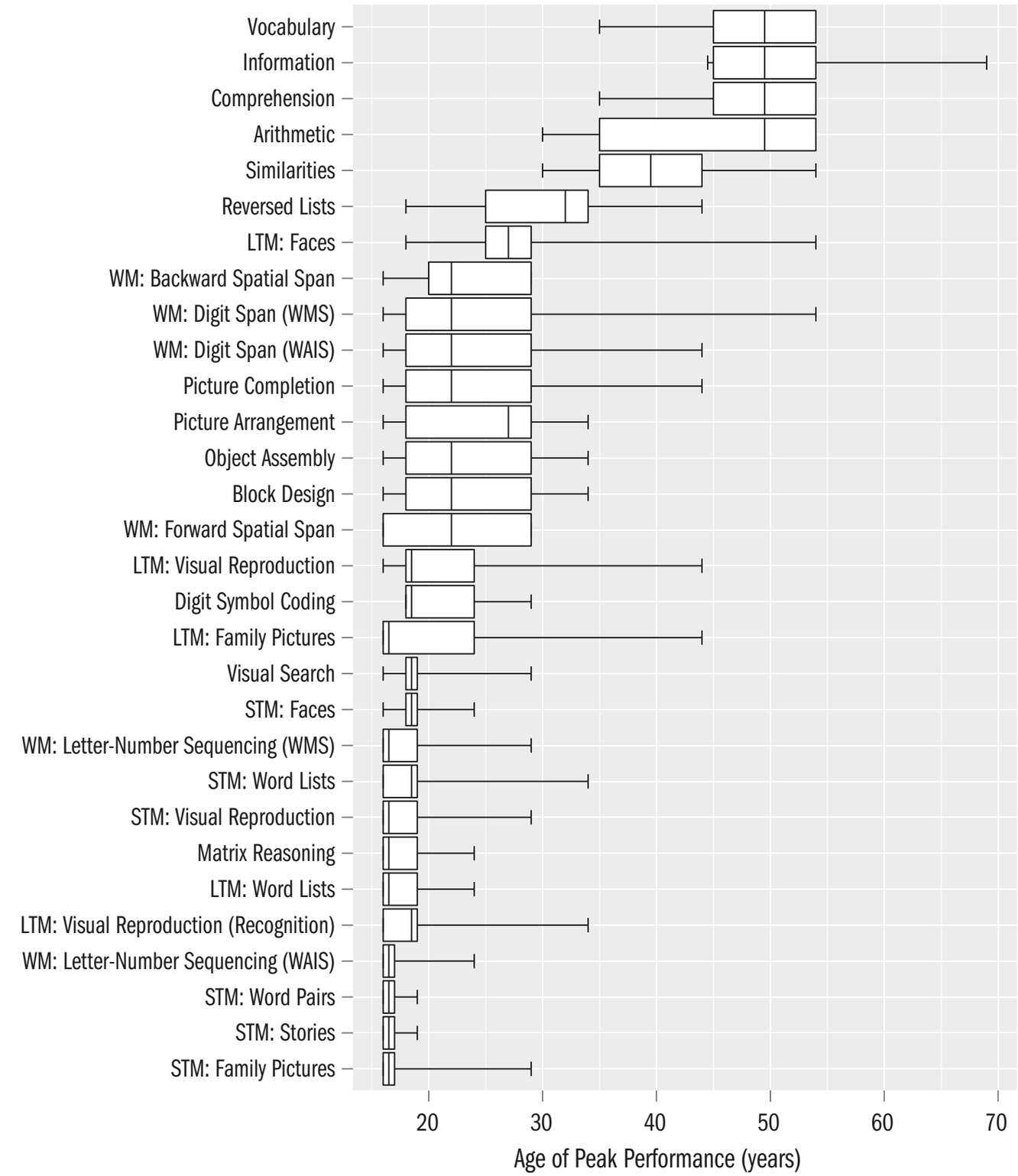
In the intelligence field, there is a distinction between "fluid" intelligence (indexed by tests of abstract reasoning and pattern detection) and

"crystallized" intelligence (indexed by measures of vocabulary and general knowledge). But domain-specific expertise—the dark matter of intelligence—is not identical to either fluid or crystallized intelligence. Most IQ tests, which were only ever designed for testing schoolchildren, don't include the rich depth of knowledge we acquire only after extensive immersion in a field. Sure, measured by the standards of youth, middle-aged adults might not be as intelligent as young adults, on average. But perhaps once dark matter is taken into account, middle-aged adults are up to par.

To dive deeper into this question, Ackerman administered a wide variety of domain-specific knowledge tests to 288 educated adults between the ages of 21 and 62. Domains included art, music, world literature, biology, physics, psychology, technology, law, astronomy and electronics. Ackerman found that in general, middle-aged adults are more knowledgeable in many domains compared with younger adults. As for the implications of this finding, I love this quote from the paper:

"*[M]any intellectually demanding tasks in the real world cannot be accomplished without a vast repertoire of declarative knowledge and procedural skills. The brightest (in terms of IQ) novice would not be expected to fare well when performing cardiovascular surgery in comparison to the middle-aged expert, just as the best entering college student cannot be expected to deliver a flawless doctoral thesis defense, in comparison to the same student after several years of academic study and empirical research experience. In this view, knowledge does not compensate for a declining adult intelligence; it is intelligence!*"

There was an important exception to Ackerman's finding, however. All three science-related tests (chemistry, physics and biology) were *negatively* associated with age. Tellingly, these three tests were most strongly correlated with fluid intelligence. This might explain why scientific



WM = working memory (immediate test after each trial); STM = short-term memory (test soon after stimulus presentation); LTM = long-term memory (test 20 to 30 minutes after stimulus presentation)

"WHEN DOES COGNITIVE FUNCTIONING PEAK? THE ASYNCHRONOUS RISE AND FALL OF DIFFERENT COGNITIVE ABILITIES ACROSS THE LIFE SPAN," BY JOSHUA K. HARTSHORNE AND LAURA T. GERMINE, IN *PSYCHOLOGICAL SCIENCE*; MARCH 13, 2015

genius tends to peak early.

Nevertheless, on the whole, these results should be considered good news for older adults. Unless you're trying to win the Nobel Prize in Physics at a very old age, there are a lot of domains of knowledge that you can continue to learn about throughout your life. What's more, Ackerman found that certain measures of personality, such as intellectual curiosity, were related to domain-specific knowledge above and beyond the effects of standard measures of intelligence.

And even if you do want to maintain your fluid intelligence as long as possible, there is recent research suggesting that having a greater purpose in life can help protect against cognitive decline among older adults. Giyeon Kim of Chung-Ang University in South Korea and colleagues combined seven items looking at various aspects of purpose, including plans for the future, importance of daily activities, dedication to ensure plans made are actualized in the future, a good sense of what one wishes to accomplish in life, whether one has accomplished all one wishes to accomplish in life, whether one cares about the future, and whether one has a sense of direction and purpose in one's life. They found that after adjusting for covariates, purpose in life acted as a protective factor against cognitive decline.* The researchers argue that purpose in life could be used as a treatment technique for cognitive decline in clinical settings.

Their research adds to a growing literature showing the many benefits of maintaining a purpose in life for health and well-being. Greater purpose in life has been linked to reduced all-cause mortality and cardiovascular problems, increased longevity, maintenance of general physical functioning, reduced risk of stroke and reduced incidence of sleep disturbances. One longitudinal study over a 10-year period found that increased meaning in life was associated with lower allostatic load (the "wear and tear on the body"). This is important considering that allostatic load has also been positively linked with increased risk of diseases, mortality and cognitive decline.

The good news for older adults is that not only can we continue to acquire domain-specific knowledge into older age, but purpose in life is also modifiable. It seems that the question "When does intelligence peak?" is actually a rather meaningless question. Not only do our various cognitive functions peak at different times, but past a certain age it might make more sense to view adult intelligence not through the lens of youthful general processing speed and reasoning, but through the lens of expertise, wisdom and purpose.

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*Interestingly, the results were particularly pronounced among older people and those who are black, and they did not find any effect based on sex.

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OBSERVATIONS

The Emotional Toll of Grad School

Mental health disorders and depression are far more likely for grad students than they are for the average American

A recent Harvard study concluded that graduate students are over three times more likely than the average American to experience mental health disorders and depression. The study, which surveyed over 500 economics students from eight elite universities, also concluded that one in 10 students experienced suicidal thoughts over a two-week period, a result consistent with other recent reports. While these findings are alarming to some, as a current graduate student myself, I regard them as hardly surprising. But to understand the struggles graduate students face, you have to understand the structure of graduate school itself.

Most people probably lump doctoral students into the same category as undergrads or students in professional schools such as law or medicine. The reality is their lifestyle and the nature of their work are fundamentally different. In the STEM fields where I have personal experience, as well as many other fields, graduate students are really hardly students at all. For most of their programs, which last over six years on average, they aren't preparing for written exams, taking courses or doing any of the tasks usually associated with student life. Instead they are dedicating often over 60 hours a week toward performing cutting-edge research and writing journal articles that will be used to garner millions of dollars in university research funding.

While graduate students are compensated for their work by a supervising professor, their salaries substantially lag what the open job market would offer to people with their qualifications, which often include both master's and bachelor's degrees. For example, graduate student salaries are typically around $30,000 a year for those in STEM—and can be substantially lower for those in other fields.

Further, unlike many professional school stu-

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dents, doctoral students do not leave their program with job security or even optimistic financial prospects. In fact, according to a study in 2016, nearly 40 percent of doctoral students do not have a job lined up at the time of graduation. Even for those who do snag a job, mid-career salaries can be significantly less than those for individuals who graduated from other professional programs.

So if doctoral students are underpaid and overworked, why do over 100,000 students—more than the number for dentistry, medical and law schools combined—complete these programs every year?

There are many answers to this question, and they vary from department to department, individual to individual. For some, graduate school is a convenient next step, a way to inch toward adulthood while keeping your career options open and remaining in a familiar university environment. For others, graduate school offers something they simply cannot get elsewhere. These students enter graduate school because they are extremely passionate about their field—passionate enough that they are willing to dedicate over six years to studying off-the-wall research ideas in excruciating detail.

Universities, with a commitment to intellectual freedom, are one of the few environments capable of providing the funding and resources necessary for this type of work. So, we put up with the hours, put up with the pay, and put up with the dwindling career prospects in the hope that we can pursue research we are passionate about—and then we cross our fingers and hope the rest will work out.

Unfortunately, as the study pointed out, it often does not work out. Mistaking casual interest for passion, many students realize halfway through their degree that they aren't as enthusiastic as they thought about their research. Still several years away from graduating, they have to deliberate between grinding through the remainder of their program or exiting early and entering the job market in an awkward position: underqualified compared to other doctoral graduates and inexperienced compared to others who joined the workforce directly after college.

Even those who are interested in their work have to grapple with seemingly infinitely postponed graduation dates. Unlike other programs, there is no "units threshold" you have to meet in order to graduate—instead your graduation date is overwhelmingly determined by the amount of novel research you perform. No matter how hard you may work, no results will likely mean no degree. Even the best researchers can see years slip by without any significant results as a result of factors completely out of their hands such as faulty equipment, dwindling research budgets or pursuing research ideas that simply just don't work.

Even for students who are lucky enough to produce results, frustratingly, individual professors have their own standards for what constitutes "enough research" to graduate. Is it four first-author research articles? What about one review paper and a few conference presentations? The answers you hear will vary widely, and ultimately, a student's supervising professor usually has sole power in determining when a student graduates. At best, this creates a confusing system where students perform substantially different amounts of work for the same degree. At worst, it fosters a perverse power dynamic where students feel powerless to speak out against professors who create toxic working conditions, even resulting in cases of sexual exploitation.

Then there's always the existential, "what even is my purpose?" mental black hole that many graduate students fall into. Yes, research has historically produced innovations that have revolutionized society. But for every breakthrough there are many other results without any clear social application, and given the slow, painstaking process of research, you may not be able to tell which is which for decades. As a student, it can be easy to doubt whether you're pursuing work that will ever be useful, producing a sense of meaninglessness for some that can facilitate depression.

Clearly, if nearly 10 percent of the graduate population is experiencing suicidal thoughts, something is not working right in the system. Still, progress on these issues has been slow, largely because the people who are most affected—graduate students—are often the ones with the least agency to spur change. As a student, by the time you've seen the cracks in the academic infrastructure, you'll likely only have a few more years until graduation. Do you really want to dedicate time toward fixing a system you're leaving soon when you could be performing career-vaulting research instead? Are you willing to risk upsetting professors whose recommendation letters will dictate your employment prospects? For many, the an-

swer is no.

Granted, the issues surrounding graduate student mental health are much easier to describe than to solve. But if academia is good at anything, it's tackling complex, multifaceted problems exactly like these, and there are a number of starting points for both students and administrators to push forward. For example, universities could require multiple advisers within a student's field to evaluate degree time lines, preventing labor exploitation by a single professor with vested interests in prolonging graduation dates.

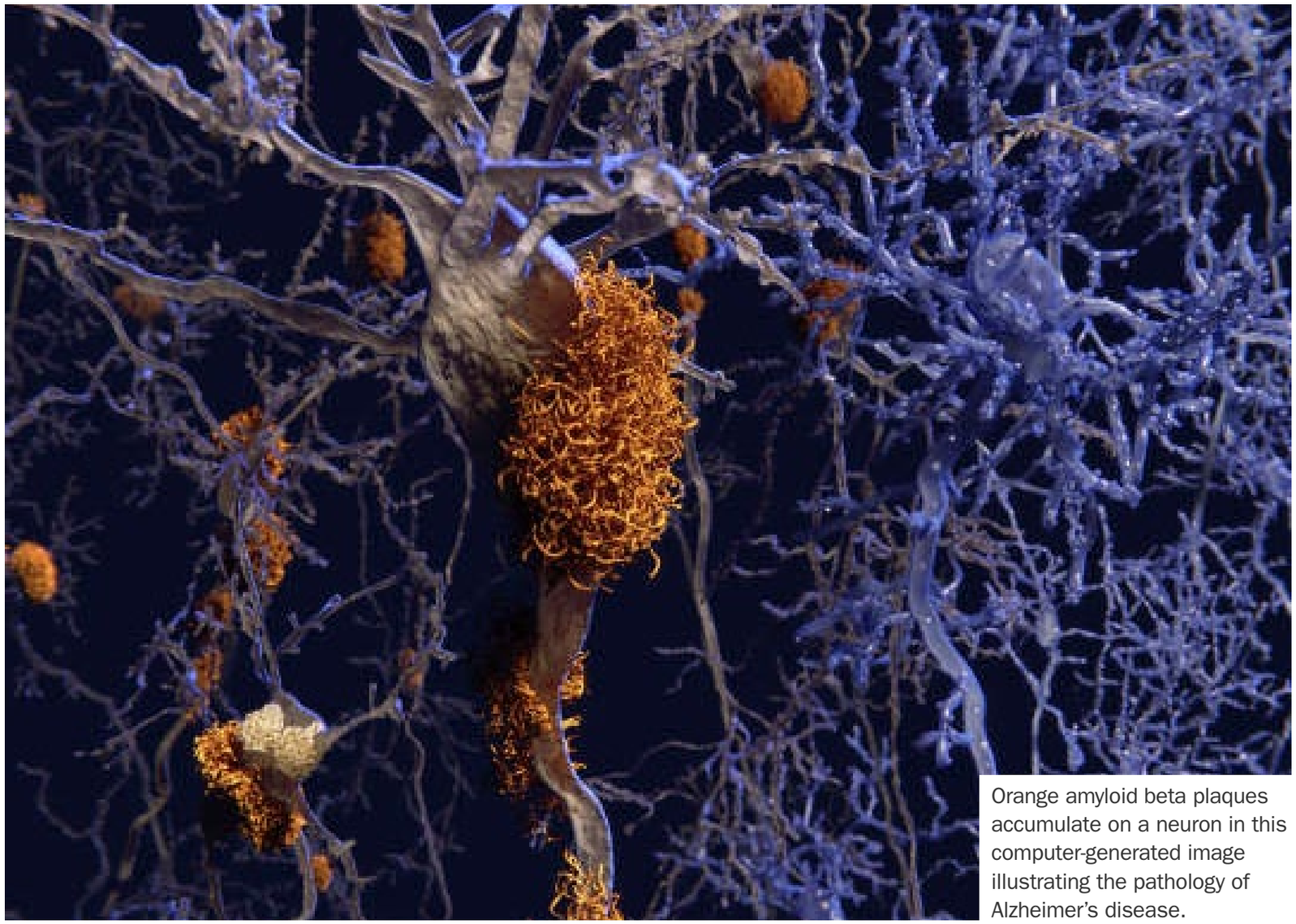
Departments could also streamline their graduation criteria to reduce disparities in student workload among different research groups and to increase transparency of degree requirements. Further, administrators could increase funding for popular student mental health services and subsidized housing that help graduate students offset cost-of-living expenses. Some universities have already adopted these policies in earnest and others only in name, but the point is academic institutions need to be making a concerted effort to improve the graduate student experience. For all the research they have done, graduate students deserve to start seeing some results.

Jennifer Frazer is an AAAS Science Journalism Award-winning science writer. She has degrees in biology, plant pathology/mycology and science writing, and has spent many happy hours studying life in situ.

THE ARTFUL AMOEBA

The Case for Transmissible Alzheimer's Grows

What separates a lethal prion from a dementia-associated amyloid plaque? Maybe not much



Orange amyloid beta plaques accumulate on a neuron in this computer-generated image illustrating the pathology of Alzheimer's disease.

JUAN GAERTNER *GETTY IMAGES*

The unsettling evidence that Alzheimer's disease may be transmissible under limited—but definitely nonzero—circumstances keeps growing.

Last December I wrote about research that revealed that infectious, lethal proteins called prions have the potential to be transmitted on optical medical equipment because they are present throughout the eyes of victims.

This was all the more disturbing in light of a study I had also recently written about that suggested that peptide aggregates—essentially sticky, self-propagating clumps of misfolded protein bits collectively referred to as amyloid—found in the brains of Alzheimer's patients may be transmissible in the same ways that prions are.

Then, just a few days after I wrote about the prion eye hazard, a new paper appeared in *Nature* that seemed to take the evidence for the transmissibility of Alzheimer's peptides from "circumstantial" to "experimentally produced." It is fascinating, if unsettling, news, that further blurs the line between amyloid and prions.

Human prion diseases are rare. Prions usually form spontaneously or are inherited via faulty

genes, but sometimes find their way into humans through consumption of contaminated brain or spinal cord tissue. In the case of mad cow disease, it happened via contaminated beef.

In rare cases (so far as we know), human prion transmission has happened when surgical instruments used on an infected patient were cleaned and reused on an uninfected one. Prions stick to steel like glue, are stable for decades at room temperature, and survive a bombardment of chemical and physical cleaning assaults that are more than sufficient to obliterate other pathogens. Prions are survivors.

In the original Alzheimer's transmissibility study, scientists examined the brains of eight patients treated with prion-contaminated human growth hormone as children who decades later died from prion disease (out of over 30,000 people so treated, more than 200 died this way).

The hormone had become contaminated with prions because it had been extracted from cadavers—one or a few of whom presumably died of prion disease—and processed in such a way that the prions remained. Of course, prions are not the only misfolded proteins that potentially lurk in the brains of cadavers.

The researchers discovered the brains of seven of the eight contained, in addition to prions, peptide aggregates called amyloid beta (Aβ for short). Aβ is a collection of misfolded peptides whose correctly folded versions are present in the human brain and perform a variety of mid-level tasks. When the misfolded versions form, they behave like prions, catalyzing the conversion of healthy forms into diseased ones and accumulating in clumps called plaques. Indeed, past experiments have shown that injecting small amounts of human Aβ into the brains of primates or of mice bred to express a humanized form of the Aβ precursor protein generates Aβ plaques in these animals.

Plaques are characteristic of and possibly the instigators of Alzheimer's disease when they accumulate around neurons in the brain. However, the seven brains did not have plaques. The Aβ in these brains had built up in the walls of blood vessels, where such accumulations can cause bleeding and dementia. This condition is called cerebral amyloid angiopathy, and it co-occurs with most Alzheimer's disease but can also strike on its own.

The eight victims had all still been young enough that their brains would not be expected to show any signs of Alzheimer's or cerebral amyloid angiopathy unless they had genetic risk factors. Understandably, given the implications, the scientists who studied their brains were concerned.

The December *Nature* study was authored by this same team. In it, they revealed that they had managed to get their hands on original vials of prion-contaminated growth hormone that had been helpfully squirreled away for decades by Public Health England.

They tested the samples for both Aβ peptides and tau, another protein that builds up in the brains of Alzheimer's patients and causes its other brain pathology: tangles. Indeed, two types of Aβ and tau were still present in the vials, even after more than *three decades of room-temperature storage*. Aβ and tau, at least, are survivors, too.

This team took its study a step further by injecting a tiny sample of these vintage vials into the brains of mice engineered to be susceptible to human Alzheimer's. The mice developed both Aβ plaques and cerebral amyloid angiopathy, although they showed no signs of tau. Aβ peptides had not only managed to survive decades of room-temperature storage, they were also still transmissible. This is concerning.

It is important—imperative—to emphasize that transmissible does not equal contagious. There is absolutely no evidence that people with dementia can spread their disease casually to people around them. Even donated blood appears to be safe, as no association with blood transfusions and Alzheimer's disease has ever been detected.

Rather, in the course of some neurological surgeries—and perhaps certain kinds of medical exams—prions may become lodged on equipment. And there is a chance this equipment could transmit the disease. Organ donation protocols may also warrant some review. It was already known that donations of dura mater, a tough brain covering, have transmitted Aβ to young people in the past.

And I wonder. Since Alzheimer's disease is so common, and we have not (to my knowledge) been looking for Alzheimer's caused by surgical or other medical procedures that access eye or neural tissue—particularly in patients for whom the appearance of Alzheimer's would not be surprising—is it possible that we are underestimating the transmission potential of this disease, and that such events are less rare than we would guess?

Alzheimer's is not the only neurodegenerative

disease in which aggregating misfolded host proteins—a class referred to as amyloid—seem to propagate and wreak havoc either. In Parkinson's disease, misfolded alpha-synuclein proteins spread through the brain, and in amyotrophic lateral sclerosis (Lou Gehrig's disease), the misfolded, accumulating protein is TDP-43. We should investigate the transmission potential of these diseases as well.

The only thing that seemed to separate these conditions from classic prion diseases was transmissibility. But now that that barrier has been breached for at least one, I also wonder: What is the difference between amyloid and prions? Are they part of a spectrum? Are they one and the same? If not, what is the difference? Can what we've learned about the biology of prions help our efforts to fight amyloid dementias? Of course, since we still can't cure prion diseases, it may not be much help even if so.

The realization that the peptides involved in some of the most common and feared dementias on Earth may be transmissible under even limited conditions is a sobering and humbling reminder of how very little we still understand about them. Given what we know about prions, I think we would be wise not to underestimate their abilities.

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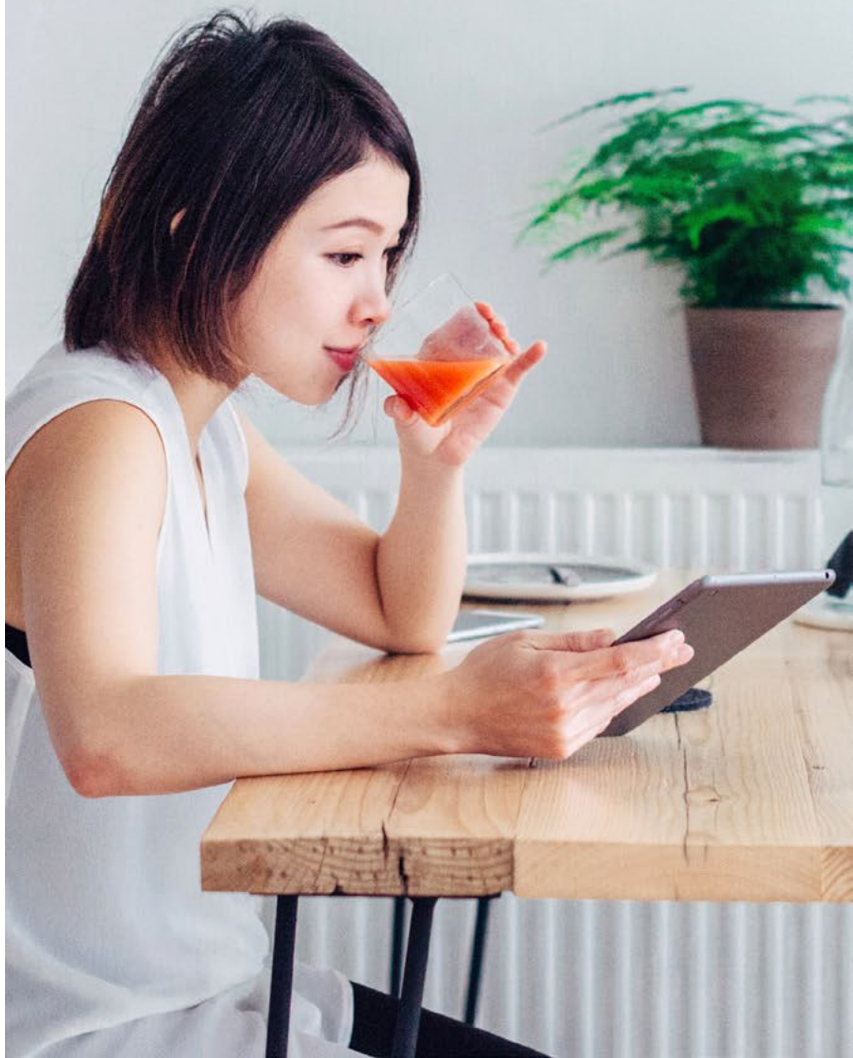

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