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SCIENTIFIC AMERICAN
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BEHAVIOR • BRAIN SCIENCE • INSIGHTS

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SCIENTIFIC AMERICAN MIND
Volume 20, Number 5, September/October 2009

COVER STORY

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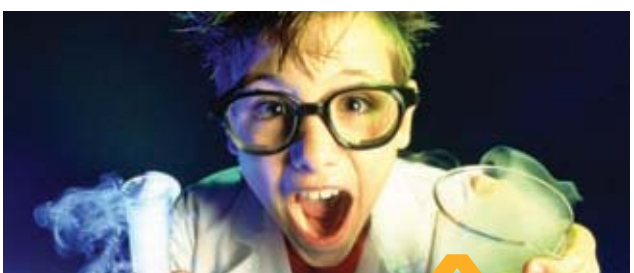
MIND ON PAIN—A SPECIAL SECTION



DEPARTMENTS

Scientific American Mind (ISSN 1555-2284), Volume 20, Number 5, September/October 2009, published bimonthly by Scientific American, Inc., 75 Varick Street, 9th Floor, New York, NY 10013.

Periodicals postage paid at New York, NY, and additional mailing offices. Canada Post International Publications Mail (Canadian Distribution) Sales Agreement No. 40012504. Canadian BN No. 127387652RT; QST No. Q1015332537. Publication Mail Agreement #40012504. Canada Post: Return undeliverables to 2835 Kew Dr., Windsor, ON N8T 3B7. Subscription rates: one year (six issues), $19.95; elsewhere, $30 USD. Postmaster: Send address changes to Scientific American Mind, 75 Varick Street, 9th Floor, New York, NY 10013. To purchase additional quantities: U.S., $10.95 each; elsewhere, $13.95 each. Send payment to SA Mind, PO Box 4002812, Des Moines, IA 50340. **For subscription inquiries, call (888) 262-5144. To purchase back issues, call (800) 925-0788.** Printed in U.S.A.

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MIND
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PHOTOILLUSTRATION BY AARON GOODMAN

Social Saviors

An elegant presence in a dark suit with tie neatly knotted, he reclined with eyes closed, hands clasped. His face was still boyishly handsome at 57 under the sweep of silver hair. My father rested in his open casket, and as I stood alone in the funeral home room, I at last understood the cliché of the crushing weight of grief.

Moments later face after friendly face poured into the room, smiling encouragingly at me, touching my arm, murmuring words of support, sharing memories. Physically I felt as if I had been lying, flattened by sorrow, on a bedsheet, and all the friends and relatives around me had grabbed the edges and lifted me up. As the days and weeks passed, members of my healing human network—at home, at holiday gatherings, during the commute, at work, at the gym—bolstered my spirits.

And so it is with all of us, as social psychologist Jolanda Jetten and her colleagues reveal in our cover story, "The Social Cure," which begins on page 26. As studies show, being part of many social groups fosters resilience, giving us the strength to get through hardships such as job loss, a move or other challenging life events. What is more, social groups promote better cognition and physical health. So go ahead and take the time for your bridge club, golf foursome, lunch date or other seemingly guilty social pleasure—and know that you're actually making yourself mentally healthier.

For the millions who suffer from chronic pain, a return to full health can feel like an elusive dream. While an ache normally serves the useful purpose of warning us away from further injury, chronic pain doesn't ebb when the wound is gone. Now new insights into the mechanisms behind the condition may at last help us control that formidable tormentor. In a special section of three feature articles, starting on page 34, you will learn how pain can become lasting, how psychology influences our perception of it and why some people are more (or less) susceptible. As the articles show, a feeling of reward can have an analgesic effect. Does that mean the pleasure gained from learning about how the mind works can count? It doesn't hurt to try.

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(letters) april/may/june 2009 issue

UNINTENTIONAL COMEDY

I have been really enjoying your magazine since I started reading it last year.

In the "Humor in the Brain" sidebar in "Laughing Matters," by Steve Ayan, the picture of the brain with eyeballs actually looks pretty hilarious. I keep visualizing Slinky-style springs behind the eyeballs going "Sproing!"

Anyway, keep up the good work.

Meghan O'Connell
via e-mail

RISKS AND REWARDS

I feel that "Knowing Your Chances," by Gerd Gigerenzer, Wolfgang Gaissmaier, Elke Kurz-Milcke, Lisa M. Schwartz and Steven Woloshin, is a very important article. As a physician, I know it is often difficult to follow through with scientific recommendations. This difficulty results in part from a fear—justified or not—that runs through the medical community: if you do not do everything possible for a patient, no matter how small the benefit, you will be sued. The saying is, "No one is ever sued for overtreatment." As acknowledged in the article, patients want certainty. They want to feel like everything that can be done has been done. This may not be the best physical approach, but it can be mentally reassuring to a patient and the family.

"akaamd"
adapted from a comment at www.ScientificAmerican.com/Mind-and-Brain

"Knowing Your Chances" is a very good article. Although the authors touched on drug efficacy, I am surprised they did not invoke the concept of "number needed to treat," or NNT. This statistic indicates how many people would have to take a particular drug to achieve the desired results in one individual.

For instance, note the NNT of 35 for statins (cholesterol-lowering drugs) in the primary prevention (avoiding a first-time event) of any bad thing (such as a

Knowing Your Chances

When might a positive HIV test be wrong? Are your chances of surviving cancer better in the U.S. or in England? Learn how to put aside unjustified fears and hopes and how to weigh your real risk of illness—or likelihood of recovery

By Gerd Gigerenzer, Wolfgang Gaissmaier, Elke Kurz-Milcke, Lisa M. Schwartz and Steven Woloshin

In a 2007 campaign advertisement, former New York City mayor Rudy Giuliani said, "I had prostate cancer, five, six years ago. My chances of surviving prostate cancer—and thank God, I was cured of it—in the United States? Eighty-two percent. My chances of surviving prostate cancer in England? Only 44 percent under socialized medicine." Giuliani used these statistics to argue that he was lucky to be living in New York and not in York. This statement was big news. As we will explain, it was also a big mistake.

In 1938 in *World Brain* (Methuen & Co.), English writer H. G. Wells predicted that for an educated citizenship in a modern democracy, statistical thinking would be as indispensable as reading and writing. At the beginning of the 21st century, nearly everyone living in an industrial society has been taught reading and writing but not statistical thinking—how to understand information about risks and uncertainties in our technological world. That lack of understanding is shared by many physicians, journalists and politicians such as Giuliani who, as a result, spread misconceptions to the public.

Statistical illiteracy is not rooted in inherent intellectual deficits—say, in the lack of a "math gene"—but rather in societal and emotional forces. These influences include the paternalistic nature of the doctor-patient relationship, the illusion of certainty in medicine, and the practice of presenting health information in opaque forms that erroneously suggest big benefits and small harms from interventions. When citizens do not understand the numbers, they are susceptible to political and commercial manipulation of their anxieties and hopes. The result can be serious damage to physical health and emotional well-being.

We show you how to spot three types of statistical manipulation and confusion in medicine, to translate opaque figures into ones that make sense and to use that information to make better medical decisions. To avoid such misunderstandings in the first place, we argue that medical journals, the media and others should communicate risk in more easily understood forms. In addition, we recommend introducing young children to statistical thinking and teaching statistics as a way of solving real-world problems rather than as a purely mathematical discipline.

Trust Your Doctor?

Medicine has held a long-standing antagonism toward statistics. For centuries, treatment was based on an ethic of personal trust as opposed to quantitative facts, which were dismissed as impersonal or irrelevant to the individual. Even today many doctors think of themselves as artists, relying more on intuition and faith in their own judgment than on numbers. For their part, many patients prefer to trust their doctors rather than even asking for data to analyze. For example, in a 2008 unpublished survey by one of us (Gigerenzer) and his colleagues, two thirds of more than 100 American economists said they had not weighed any pros and cons of getting a prostate cancer screening test but simply followed their doctor's recommendation.

PANELS FROM LEFT: AGE FOTOSTOCK; GETTY IMAGES; LIGHTSCAPES PHOTOGRAPHY, INC./CORBIS; GETTY IMAGES; DICE: LEV MEL /iStockphoto

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heart attack), according to a table provided by the journal *Bandolier,* found at http://tinyurl.com/mrxngz. [*Editor's note: the URL has been shortened to make it easier to type into a browser.*] Your doctor may advise you to take a statin if your cholesterol is slightly elevated, but he or she probably will not tell you that out of 35 people taking the drug for four years, only one person will actually benefit from it in terms of avoiding a coronary event or another bad outcome. I wonder how many people with slightly elevated cholesterol would feel this rather minimal risk reduction to be worth the cost and the potentially bad side effects of taking this type of drug for the rest of their lives.

"MikeB"
adapted from a comment at
www.ScientificAmerican.com/
Mind-and-Brain

NOTES ON DEPRESSION

As a biological anthropologist and someone who has been through postpartum depression, it worries me to see too much eagerness to slap an adaptive explanation onto PPD, as anthropologist Edward H. Hagan does in "Ask the Brains." As scientists, it is our responsibility to acknowledge that we cannot explain everything and that not everything has a purpose.

Sometimes things are coincidental—and sometimes they are the result of maladaptive traits being tagged onto adaptive ones. Perhaps the hormonal shifts themselves are highly adaptive, but their ability to completely throw a new mother's mind out of whack is not.

Assuming that everything is an adaptation—or failing to present the possibility that there are good scientific explanations besides adaptive ones for certain phenomena—ultimately undermines our credibility with those who do not wish to believe the adaptive explanations for which we *do* have good evidence. The scientific community (including myself, Hagan and *Scientific American Mind*) has a responsibility to the public to avoid simply providing stimulating or fascinating potential explanations using evolutionary theory. We also need to provide sound, proven explanations for phenomena that are only potentially evolutionary in origin.

"beak3chimps"
adapted from a comment at
www.ScientificAmerican.com/
Mind-and-Brain

A few years ago I described the basic symptoms of PPD to a group of physicians but altered one important fact: I said that the sufferer was male. The response was a relatively bored and quite immediate: "protein deficiency." I would respectfully suggest that in at least some cases of PPD, simple dietary modification to include higher levels of quality protein would moderate many of the symptoms.

Given the protein price paid by the mother for construction and maintenance of a growing baby, anything more than a short-term deficit in protein intake must result in a clinical deficiency.

I have applied this knowledge to my own practice and have observed, among those who have responded (approximately 30 to 35 percent of sufferers), an excellent resolution of their "PPD" symptoms. Those who failed to respond to dietary protein also reported a great many co-factors, and they required pharmaceutical or psychological support (or a combination of both).

"Ashmore Health Centre"
adapted from a comment at
www.ScientificAmerican.com/
Mind-and-Brain

MIND CONTROL

Regarding "Building around the Mind," by Emily Anthes, I had a good firsthand brush with this topic a couple of years back. My wife and I were on a tour of Frank Lloyd Wright's "Fallingwater" home in Pennsylvania. Upon reaching the master bedroom, half of the group headed straight to the balcony without looking to either side or paying any attention to the room. They stopped and apologized to the tour guide, who laughed and said, "That happens on every tour, and there's a reason for that. This room is specifically designed to draw you out to the balcony. You did exactly as you were supposed to do."

Throughout the rest of the tour, I learned that Wright had built even more such behavior-influencing "tricks" into the building. I left even more in awe of his talents than I had been before arriving.

"SpoonmanWoS"
adapted from
a comment at
www.ScientificAmerican.
com/Mind-and-Brain


Building around the Mind

Brain research can help us craft spaces that relax, inspire, awaken, comfort and heal

By Emily Anthes

In the 1950s prizewinning biologist and doctor Jonas Salk was working on a cure for polio in a dark basement laboratory in Pittsburgh. Progress was slow, so to clear his head, Salk traveled to Assisi, Italy, where he spent time in a 13th-century monastery, ambling amid its columns and cloistered courtyards. Suddenly, Salk found himself awash in new insights, including the one that would lead to his successful polio vaccine. Salk was convinced he had drawn his inspiration from the contemplative setting. He came to believe so strongly in architecture's ability to influence the mind that he teamed up with renowned architect Louis Kahn to build the Salk Institute in La Jolla, Calif., as a scientific facility that would stimulate breakthroughs and encourage creativity.

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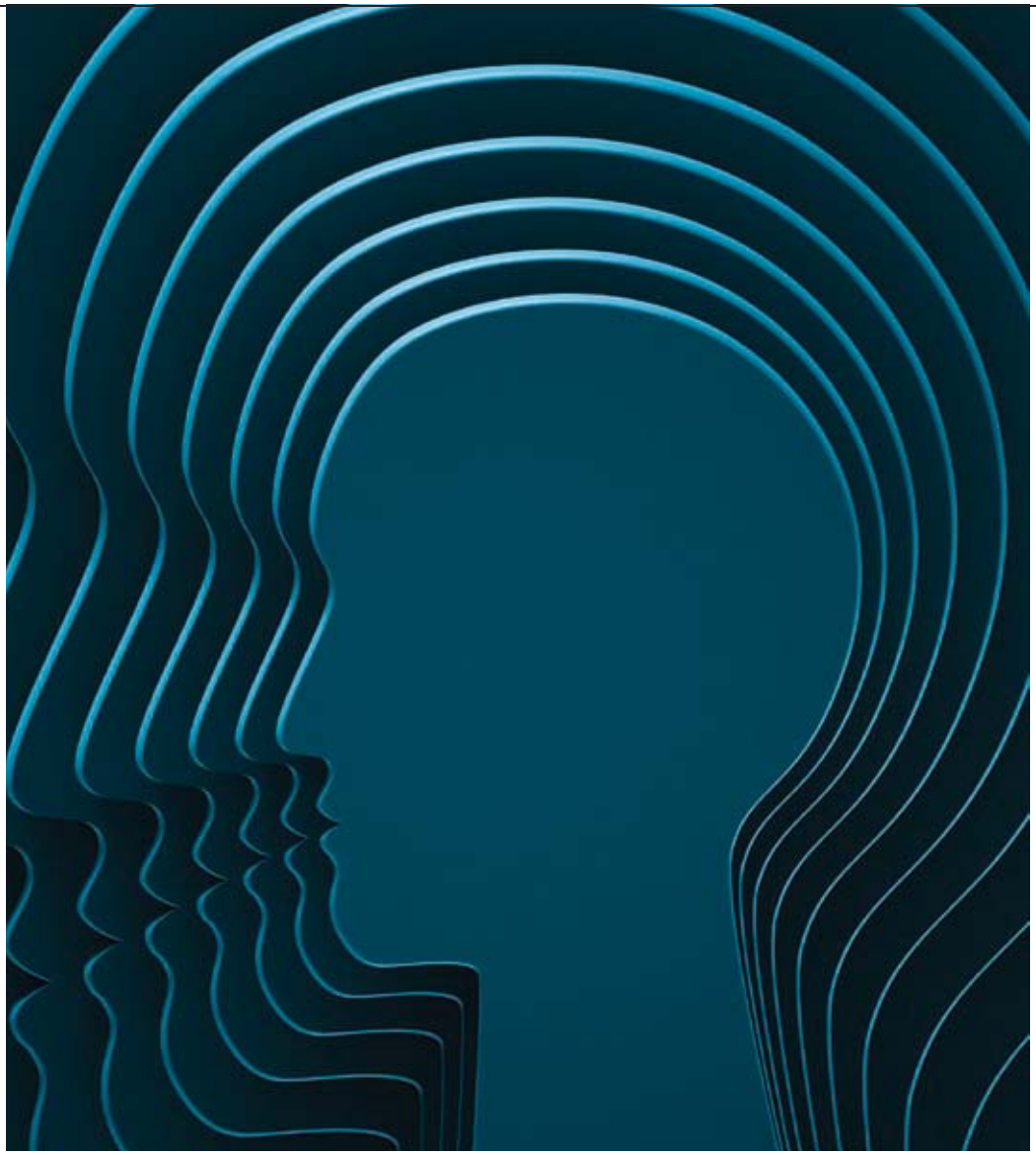
Head Lines



GETTY IMAGES

>> SENSES

Who Said That?

Specialized neurons sort out overlapping sounds

We live in a world full of echoes. Sounds reverberate, bouncing off walls, buildings, rocks and any other nearby surface. These sound waves pile on one another and hurtle down your ear canals from different angles, the echoes from one noise jumbling together with new sounds and their echoes. In spite of that barrage, the neurons in the auditory midbrain, an area that responds before the auditory cortex does, are able to sort out which were the original sounds and where they came from. How they do so has long puzzled scientists, but new research suggests the trick is simpler than expected.

In an April study, neuroscientists led by Sasha Devore at the Massachusetts Institute of Technology tested the widely held hypothesis that specialized cells in the brain actively suppress neuronal response to echoes. Using electrodes in a cat's midbrain, researchers measured cells' responses to a sound and its reverberations. They found that the cells that sense a sound's direction of origin responded more strongly to the first 50 milliseconds of sound waves than they did to the later waves—their activity simply tapered off after the onset of the sound. The tapering response, a much simpler mechanism than the earlier theory of suppression, allows the brain to easily tune in to original sounds and pinpoint who or what is making noise. *—Robert Goodier*

>> HORMONES

You Snooze, You Lose

Getting enough rest promotes weight loss

Lose weight while you sleep? It sounds too good to be true—but recent research indicates that there is a connection between how much you weigh and the amount of shut-eye you get per night.

Two hormones, ghrelin and leptin, help to control appetite. When you do not get enough rest, levels of ghrelin, which increases hunger, rise; levels of leptin, which promotes feelings of fullness, sink. A study in the May issue of *Psychoneuroendocrinology* found a significant disruption in nighttime ghrelin levels in chronic insomniacs. According to the study, this hormone imbalance leads insomniacs to experience an increase in appetite during the day, leading to weight gain over time.

In addition to creating an imbalance in ghrelin and leptin, sleep deprivation causes levels of the stress hormone cortisol to rise, which increases cravings for high-carb, high-calorie "comfort foods." Furthermore, the brain secretes growth hormone during the deep-sleep phase, helping the body convert fat to fuel. Without enough deep sleep, fat accumulates.

Sleep expert Michael Breus, clinical director of the sleep division at Southwest Spine & Sports in Scottsdale, Ariz., says that there is no magic number of hours people should sleep but that the average adult needs about five 90-minute sleep cycles per night, so 7.5 hours seems optimal as a minimum.

But simply getting under the covers is probably not a sufficient strategy to achieve long-term weight loss, Breus says. "What these findings suggest is that there's a new triad to achieving a healthy weight: diet, exercise and enough sleep." —*Christina Frank*

GETTY IMAGES (*snooze*); PAMELA COWART-RICKMAN *iStockphoto* (*robin*); MELANIE TAYLOR *iStockphoto* (*owl*); ROBERT DALE *Getty Images* (*man*)

>> SLEEP

Early Risers Crash Faster

Night owls belie slacker reputation by staying alert longer

Early birds may get the best worms—or at least the best garage sale deals—but they also tire out more quickly than night owls do. In a new study researchers Christina Schmidt and Philippe Peigneux, both at the University of Liège in Belgium, and their colleagues first asked 16 extreme early risers and 15 extreme night owls to spend a week following their natural sleep schedule. Then subjects spent two nights in a sleep lab, where they again followed their preferred sleep patterns and underwent cognitive testing twice daily while in a functional MRI scanner.

An hour and a half after waking, early birds and night owls were equally alert and showed no difference in attention-related brain activity. But after being awake for 10 and a half hours, night owls had grown more alert, performing better on a reaction-time task requiring sustained attention and showing increased activity in brain areas linked to attention. More important, these regions included the suprachiasmatic area, which is home to the body's circadian clock. This area sends signals to boost alertness as the pressure to sleep mounts. Unlike night owls, early risers didn't get this late-day lift. Peigneux says faster activation of sleep pressure appears to prevent early birds from fully benefiting from the circadian signal, as evening types do. —*Siri Carpenter*

Men Are Choosy, Too

In numerous studies of speed dating—a rapid-fire matchmaking tool that has men hop from table to table for quick encounters—women have proved choosier than the guys about whom they flag for a second date. Ladies must be picky because they invest more in their offspring, according to the oft-repeated evolutionary theory. But when researchers made the simple switch of having women do the table hopping while men stayed seated, the two sexes suddenly became equally choosy, suggesting social norms and physical cues play an underappreciated role in mate choice. Read more in the October issue of *Psychological Science.* —*JR Minkel*

>> COGNITION

Memory Maintenance

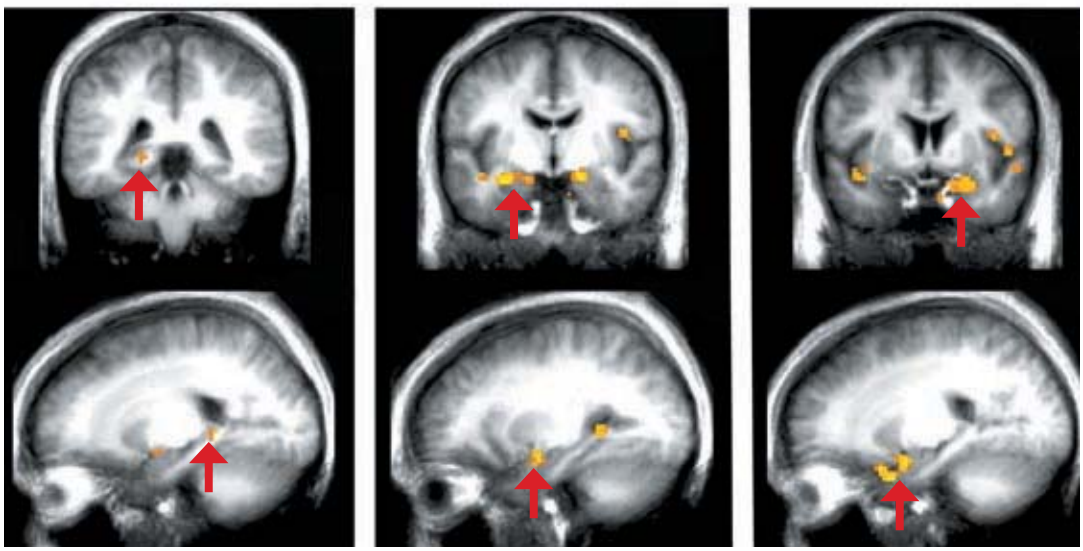
As recollections age, different brain areas take charge of the upkeep

The brain's ability to learn and form memories of day-to-day facts and events depends on the hippocampus, a structure deep within the brain. But is the hippocampus still maintaining the memory of, say, the commencement address at your college graduation 20 years ago? The latest evidence suggests that as memories age, the hippocampus's participation wanes.

In a 2006 study, neuroscientist Larry R. Squire of the University of California, San Diego, and the Veterans Affairs San Diego Healthcare System studied patients who had hippocampal damage. These individuals did not remember details of newsworthy events that occurred in the five to 10 years prior to their injuries, but they did recall older events.

Building on those results, Squire turned to healthy brains. His team questioned 15 people in their 50s and 60s about events in the news over the past 30 years while scanning the participants' brains with functional MRI. To single out brain activity related to the date of the event, the researchers separately evaluated activity tied to learning and remembering the test questions. They also accounted for the richness of participants' recollections of events, to make sure the degree to which someone was able to recall an event did not influence the data.

Squire's team reported in January that activity in the hippocampus steadily declined as subjects remembered events that were up to 12 years old. With more remote memories, the structure's activity leveled off. In contrast, areas in the frontal, temporal and parietal lobes displayed increasing activity for recalled events from those dozen years, then reached a plateau during older remembrances.



The arrows on each pair of MRI images point to an area where activity decreases as memories age: the hippocampus (*left*), the amygdala (*center*) and the temporopolar cortex (*right*).

COURTESY OF CHRISTINE N. SMITH, SOURCE: "MEDIAL TEMPORAL LOBE ACTIVITY DURING RETRIEVAL OF SEMANTIC MEMORY IS RELATED TO THE AGE OF THE MEMORY," BY C. N. SMITH AND LARRY R. SQUIRE, IN *JOURNAL OF NEUROSCIENCE*, VOL. 29, NO. 4; JANUARY 28, 2009 (*brains*); GETTY IMAGES (*suitcase*)

The biology behind how the brain makes and keeps memories is not fully understood, Squire notes, but it appears that, initially, a memory resides in the hippocampus and in areas the structure connects to in the neocortex, the outer part of the cerebral cortex. "A time comes when the cortical regions important to a memory are connected [to one another] heavily enough to form a stable representation," Squire says. "Then the hippocampus isn't needed to hold the whole thing together."

—Aimee Cunningham

>> CULTURE

Pack Your Bags for Creativity

Ernest Hemingway and Pablo Picasso were on to something: a recent study suggests that by living abroad artists may be fueling their creativity. Researchers from the French business school INSEAD and Northwestern University studied responses from subjects in five separate experiments, finding that those who had lived abroad—and had adapted to a nonnative culture—more consistently showed innovation and creativity in negotiations, in the use of ordinary items, and in drawings. More research is necessary to discern if an already creative person benefits more from living abroad than a noncreative one does or if the noted higher levels of creativity are permanent. *—Elizabeth King Humphrey*

>> PSYCHOLOGY

Say Cheese

Kids' smiles predict their future marriage success

Pictures of grinning kids may reveal more than childhood happiness: a study from DePauw University shows that how intensely people smile in childhood photographs, as indicated by crow's feet around the eyes, predicts their adult marriage success.

According to the research, people whose smiles were weakest in snapshots from childhood through young adulthood were most likely to report being divorced in middle and old age. Among the weakest smilers in college photographs, one in four ended up divorcing, compared with one in 20 of the widest smilers. The same pattern held among even those pictured at an average age of 10.

The paper builds on a 2001 study by psychologists at the University of California, Berkeley, that tracked the well-being and marital satisfaction of women from college through their early 50s. That work found that coeds whose smiles were brightest in their senior yearbook photographs were most likely to be married by their late 20s, least likely to remain single into middle age, and happiest in their marriage; they also scored highest on measures of overall well-being (including psychological and physical difficulties, relationships with others and general self-satisfaction).

The scientists speculate that one's tendency to grin—an example of what psychologists call "thin slices" of behavior that can belie personal traits—reflects his or her underlying emotional disposition. Positive emotionality influences how others respond to a person, perhaps making that individual more open and likely to seek out situations conducive to a lasting, happy marriage.

But there could be a more cynical explanation, according to Matthew Hertenstein, a psychologist at DePauw who led the new study. "Maybe people who look happier in photos show a social face to others," he says. "Those may be the same people who are likely to put up with partners because they don't want to appear unhappy."

—Jordan Lite

>> MUSIC

Dancing with the Starlings

Birds' rhythmic abilities offer clues to the origins of dance

Researchers have long assumed that humans were the only animals that could dance—even our close primate relatives cannot keep a steady beat or be taught to move to a rhythm. But new evidence shows that birds can dance, revealing that the mysterious ability could be a by-product of vocal learning.

Aniruddh Patel of the Neurosciences Institute, Adena Schachner of Harvard University and their colleagues studied several birds, among them a cockatoo that dances to the Backstreet Boys' "Everybody." When Patel sped up or slowed down the song, the bird adjusted its moves to match the tempo, eliminating the possibility that it was in sync with the music by chance. Intrigued, Schachner and her colleagues started searching YouTube for videos of other dancing animals. They found 15 bopping species (14 parrot and one elephant) that also share an additional trait: the capability to imitate sounds. That correlation suggests our musical ability grew out of the vocal learning system instead of being "a special-purpose ability," Patel says.

This cockatoo is one of very few animals known to have a sense of rhythm. Watch a video of it dancing at http://tinyurl.com/dgq4ko

The findings could help advance research on movement disorders, he adds. Hearing music helps Parkinson's patients to walk, for example. So far scientists do not understand the underlying mechanisms, but if bird brains share certain key circuits with humans, then scientists may find answers by studying them.

—Nicole Branan

MARC DEBNAM *Corbis (kids)*; COURTESY OF IRENA SCHULZ *Bird Lovers Only Rescue (cockatoo sequence)*

>> EMOTIONS

Smile! It Could Make You Happier

Making an emotional face—or suppressing one—influences your feelings

We smile because we are happy, and we frown because we are sad. But does the causal arrow point in the other direction, too? A spate of recent studies of botox recipients and others suggests that our emotions are reinforced—perhaps even driven—by their corresponding facial expressions.

Charles Darwin first posed the idea that emotional responses influence our feelings in 1872. "The free expression by outward signs of an emotion intensifies it," he wrote. The esteemed 19th-century psychologist William James went so far as to assert that if a person does not express an emotion, he has not felt it at all. Although few scientists would agree with such a statement today, there is evidence that emotions involve more than just the brain. The face, in particular, appears to play a big role.

This February psychologists at the University of Cardiff in Wales found that people whose ability to frown is compromised by cosmetic botox injections are happier, on average, than people who can frown. The researchers administered an anxiety and depression questionnaire to 25 females, half of whom had received frown-inhibiting botox injections. The botox recipients reported feeling happier and less anxious in general; more important, they did not report feeling any more attractive, which suggests that the emotional effects were not driven by a psychological boost that could come from the treatment's cosmetic nature.

"It would appear that the way we feel emotions isn't just restricted to our brain—there are parts of our bodies that help and reinforce the feelings we're having," says Michael Lewis, a co-author of the study. "It's like a feedback loop." In a related study from March, scientists at the Technical University of Munich in Germany scanned botox recipients with fMRI machines while asking them to mimic angry faces. They found that the botox subjects had much lower activity in the brain circuits involved in emotional processing and responses—in the amygdala, hypothalamus and parts of the brain stem—as compared with controls who had not received treatment.

Women whose botox injections make it difficult to frown are happier than women who are able to frown.

ADRIANNA WILLIAMS
Corbis

The concept works the opposite way, too—enhancing emotions rather than suppressing them. People who frown during an unpleasant procedure report feeling more pain than those who do not, according to a study published in May 2008 in the *Journal of Pain*. Researchers applied heat to the forearms of 29 participants, who were asked to either make unhappy, neutral or relaxed faces during the procedure. Those who exhibited negative expressions reported being in more pain than the other two groups. Lewis, who was not involved in that study, says he plans to study the effect that botox injections have on pain perception. "It's possible that people

may feel less pain if they're unable to express it," he says.

But we have all heard that it is bad to repress our feelings—so what happens if a person intentionally suppresses his or her negative emotions on an ongoing basis? Work by psychologist Judith Grob of the University of Groningen in the Netherlands suggests that this suppressed negativity may "leak" into other realms of a person's life. In a series of studies she performed for her Ph.D. thesis and has submitted for publication, she asked subjects to look at disgusting images while hiding their emotions or while holding pens in their mouths in such a way that prevented them from frowning. A third group could react as they pleased.

As expected, the subjects in both groups that did not express their emotions reported feeling less disgusted afterward than control subjects. Then she gave the subjects a series of cognitive tasks that included fill-in-the-blank exercises. She found that subjects who had repressed their emotions performed poorly on memory tasks and completed the word tasks to produce more negative words—they completed "gr_ss" as "gross" rather than "grass," for instance—as compared with controls. "People who tend to do this regularly might start to see the world in a more negative light," Grob says. "When the face doesn't aid in expressing the emotion, the emotion seeks other channels to express itself through."

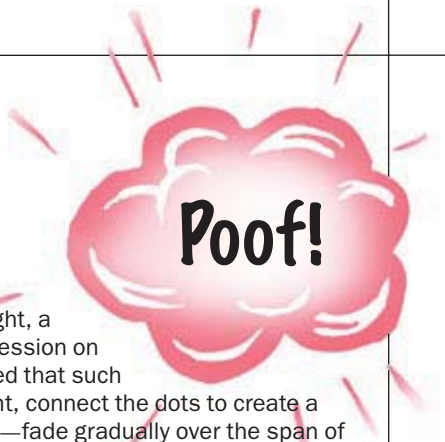
No one yet knows why our facial expressions influence our emotions as they seem to. The associations in our mind between how we feel and how we react may be so strong that our expressions simply end up reinforcing our emotions—there may be no evolutionary reason for the connection. Even so, our faces do seem to communicate our states of mind not only to others but also to ourselves. "I smile, so I must be happy," Grob says. *—Melinda Wenner*

CORBIS

>> MEMORY

Abruptly Forgotten

Certain memories die suddenly rather than fading away



When you go from bed to bathroom on a dark night, a quick flick of the lights will leave a lingering impression on your mind's eye. For decades evidence suggested that such visual working memories—which, even in daylight, connect the dots to create a complete scene as the eyes dart around rapidly—fade gradually over the span of several seconds. But a clever new study reported in the journal *Psychological Science* finds that such memories actually stay sharp until they are suddenly lost.

Cognitive psychologists Weiwei Zhang and Stephen J. Luck, both at the University of California, Davis, tested subjects' recall for the hues of colored squares flashed briefly on a screen up to 10 seconds earlier. Subjects marked their answer on a color wheel. If memories decay gradually, the guesses should have become increasingly imprecise as time wore on, evidenced by participants selecting yellow or red, for example, when the correct choice was orange. Instead subjects went straight from fairly accurate answers to random choices—no better than chance—indicating the memories were decaying all at once. According to Zhang and Luck's mathematical analysis, most subjects' memories went "poof" somewhere between four and 10 seconds after the stimulus.

Researchers say a sudden die-off is to be expected if working memories are stored in circuits that feed back on themselves. Luck says the system is like a laptop as compared with a flashlight. "The laptop is an active system that uses feedback circuits to limit how much power it draws," he says. So whereas a flashlight dims when it runs low on juice, "the computer runs perfectly normally while the battery drains," he says, "until suddenly the laptop shuts off." *—JR Minkel*

>> SOCIOLOGY

Confidence Wins over Smarts

Speaking up counts more than competence in becoming a leader

When a group of people works to complete a task, a leader usually emerges. New research shows such leaders are not necessarily more intelligent than the other group members, but rather they simply speak up more often. Researchers at the University of California, Berkeley, gave groups of college students 45 minutes to lay the groundwork for a business and then asked the students to rate one another on intelligence, judgment and other traits. The students believed that the people who spoke more often were the smartest in each group—even when, during another group exercise involving math problems, they offered more incorrect answers than did others who were less talkative. Those who did not say much were judged as averagely intelligent and not so creative. A later look at the participants' SAT scores revealed that, on average, the leaders had the same scores as the rest of the group. "The main reason dominant people took charge is they jumped in first and nobody questioned what they said," says psychologist Cameron Anderson, who led the study. "Dominant people seem really good at things because they speak with so much confidence."

—Robert Goodier

>> PAIN

Underwater Suffering?

A study suggests fish consciously experience discomfort

Many a seafood fan has parroted the popular idea that fish and crustaceans do not feel pain. New research, however, suggests that they may, revealing that their nervous system may be more complex than we thought—and our own awareness of pain may be much more evolutionarily ancient than suspected. [For more on pain, see the special section beginning on page 34.]

Joseph Garner of Purdue University and his colleagues in Norway report that the way goldfish respond to pain shows that these animals do experience pain consciously, rather than simply reacting with a reflex—such as when a person recoils after stepping on a tack (jerking away before he or she is aware of the sensation). In the study, the biologists found that goldfish injected with saline solution and exposed to a painful level of heat in a test tank "hovered" in one spot when placed back in their home tank. Garner labels that "fearful, avoidance behavior." Such behavior, he says, is cognitive—not reflexive. Other fish, after receiving a morphine injection that blocked the impact of pain, showed no such fearful behavior.

Although Garner's findings fit with previous work that tentatively suggests that fish feel pain, some experts remain unconvinced that the reaction was not an instinctive escape behavior. Still, the new study raises ethical concerns. "If we're going to use animals in experiments, and we're going to use animals as food, then it is really important to understand the consequences of our actions for those animals," Garner says.

—Harvey Black

>> HEALTH

Inflammation Brings on the Blues

Our immune system may mean well, but it might also cause depression

As if being stuck sick in bed wasn't bad enough, several studies conducted during the past few years have found that the immune response to illness can cause depression. Recently scientists have pinpointed an enzyme that could be the culprit, as it is linked to both chronic inflammation—such as that found in patients with coronary heart disease, type 2 diabetes and rheumatoid arthritis—and depressive symptoms in mice.

In the new study, immunophysiologist Keith Kelley and his colleagues at the University of Illinois exposed mice to a tuberculosis vaccine that produces a low-grade, chronic inflammation. After inoculation, production in the mice brains of an enzyme called IDO, which breaks down tryptophan, spiked. The animals exhibited normal symptoms of illness such as moving around and eating

less. Yet even after recovering from the physical illness induced by the vaccine, they showed signs of depression—for example, struggling less than control mice to escape from a bucket of water. Surprisingly, their listlessness was solved relatively simply. "If you block IDO, genetically or pharmaceutically, depression goes away" without interfering with the immune response, Kelley explains.

The research makes a solid case that the immune system communicates directly with the nervous system and affects important health-related behaviors such as depression. The findings could bring relief to patients afflicted with obesity, which leads to chronic inflammation, as well as to cancer patients treated with radiation and chemotherapy drugs that produce both inflammation and depression. "IDO is a new target for drug companies to aim for, to treat patients with both clinical depression and systemic inflammation," Kelley says.

—Corey Binns

IAN SANDERSON *Getty Images (fish)*; CORBIS (*woman*)

>> DRUGS

Marijuana Hurts Some, Helps Others

Cannabis can kill or rescue neurons—children are at risk, whereas adults may benefit

Clinton didn't inhale, Obama did—and maybe Reagan should have. New research suggests that THC, the chemical that gives marijuana its mind-bending properties, kills developing neurons, yet oddly, the same chemical saves neurons in adults with Alzheimer's disease.

"Marijuana is not the 'soft drug' people like to think it is," says neuropharmacologist Veronica Campbell of Trinity College in Dublin, whose latest study uncovered the harmful effects of THC on young neurons. When Campbell and her co-workers treated brain cells from newborn or adolescent rats with THC, the neurons died, but THC did not have such deadly effects on neurons taken from adult rats. In fact, work from other labs shows that THC benefits adult neurons. "We don't know why," Campbell says. Several possibilities are being investigated for this "Jekyll and Hyde" effect.

JOSH WESTRICH *Corbis*

Marijuana, like tobacco and opium, has powerful effects on the brain because certain compounds in the plant happen to have a chemical resemblance to naturally occurring substances in the body. Called endocannabinoids, these natural chemicals regulate important brain functions by controlling synapses in neural circuits that process thought and perception. According to several recent studies, these chemicals have many other functions in the brain and immune system, too—including regulating development and aiding survival of young neurons, as well as controlling the wiring of neurons into circuits for learning and memory. Smoking marijuana during the period of life when the brain is still developing obscures these critical chemical signals, Campbell suspects.

The slaughter of young neurons by THC could explain the developmental cognitive impairment seen in children born to women who smoked marijuana during pregnancy. In addition, some research on adolescent marijuana abusers shows brain damage in neural circuits that are still developing at that age.

In older brains, however, THC seems to have a protective effect. Campbell's findings indicate that the biochemistry of neurons changes as the cells mature. The role of endocannabinoids shifts to regulate different functions—most important, assisting in the survival of aged neurons. In patients with Alzheimer's disease, THC protects neurons from death in several ways. THC boosts depleted levels of the neurotransmitter acetylcholine, which, when diminished, contributes to the weakened mental function in Alzheimer's patients. THC also suppresses the toxic effects of the so-called a-beta protein that may kill neurons in Alzheimer's disease. It stimulates secretion of neuron growth by promoting substances such as brain-derived neurotrophic factor, and it dampens release of the excitatory neurotransmitter glutamate, which kills neurons by overstimulation. THC and other cannabinoids also have powerful anti-inflammatory and antioxidant actions that protect neurons from immune system attack.

Despite these benefits, THC and other compounds in marijuana also have many undesirable side effects on the brain. The trick for scientists will be to isolate the active ingredients in marijuana that are beneficial and develop drugs that can be applied in the proper dose for the specific age of the patient. Campbell finds that the beneficial effects of THC are seen in much lower concentrations of the chemical than are found in the plants people use to get high. "It's a matter of trying to balance that low concentration within a nice safety margin," she explains. Synthetic THC-like drugs are already available, as is a naturally derived drug called Sativex that contains THC and other cannabinoids, approved in Canada for treating pain from multiple sclerosis and cancer.

In contrast to these well-controlled drugs, the weed itself is a complex witches' brew of many brain-altering chemicals. The cannabis plant contains about 60 different cannabinoids, so the challenge lies in trying to tease out which are the important ones for protecting neurons, Campbell explains, echoing the views of other marijuana researchers. "Depending on how the plant is cultivated, the relative proportion of the different types of cannabinoids changes," she says. "The 'joints' that are available now are much stronger in terms of their THC content than those that would have been around when people were thinking of cannabis as being quite a soft drug."

—R. Douglas Fields

Forget Survival of the Fittest: It Is Kindness That Counts

A psychologist probes how altruism, evolution and neurobiology mean that we can succeed by not being cutthroat INTERVIEW BY DAVID DiSALVO

Why do people do good things? Is kindness hardwired into the brain, or does this tendency arise via experience? Dacher Keltner, director of the Social Interaction Laboratory at the University of California, Berkeley, investigates these questions from multiple angles and often generates results that are both surprising and challenging. In his recent book, Born to Be Good: The Science of a Meaningful Life *(W. W. Norton, 2009), Keltner weaves together scientific findings with personal narrative to uncover human emotion's innate power to connect people with one another, which he argues is the path to living the good life. Here Keltner discusses altruism, neurobiology and the practical applications of his findings with David DiSalvo.*

COURTESY DACHER KELTNER (*Keltner*); IMAGES.COM/CORBIS (*Illustration*)

SCIENTIFIC AMERICAN MIND: What, in a nutshell, does the term "born to be good" mean to you?

DACHER KELTNER: "Born to be good" means that our mammalian and hominid evolution has crafted a species—us—with remarkable tendencies toward kindness, play, generosity, reverence and self-sacrifice, which are vital to the classic tasks of evolution—survival, gene replication and smoothly functioning groups. These tendencies are felt in the wonderful realm of emotion—feelings such as compassion, gratitude, awe, embarrassment and mirth. Recent studies have revealed that our capacity for caring, play, reverence and modesty is built into our brains, bodies, genes and social practices.

MIND: One of the structures in our body that seems especially adapted to promote altruism is the vagus nerve, as your team at U.C. Berkeley has found. Tell us a bit about this research and its implications.

KELTNER: The vagus nerve is a bundle of nerves that originates in the top of the spinal cord. It activates different organs throughout the body (such as the heart, lungs, liver and digestive organs). When active, it is likely to produce that feeling of warm expansion in the chest—for example, when we are moved by someone's goodness or when we appreciate a beautiful piece of music. Neuroscientist Ste-

Our capacity for **caring, play, reverence and modesty** is built into our brains, bodies, genes and social practices.

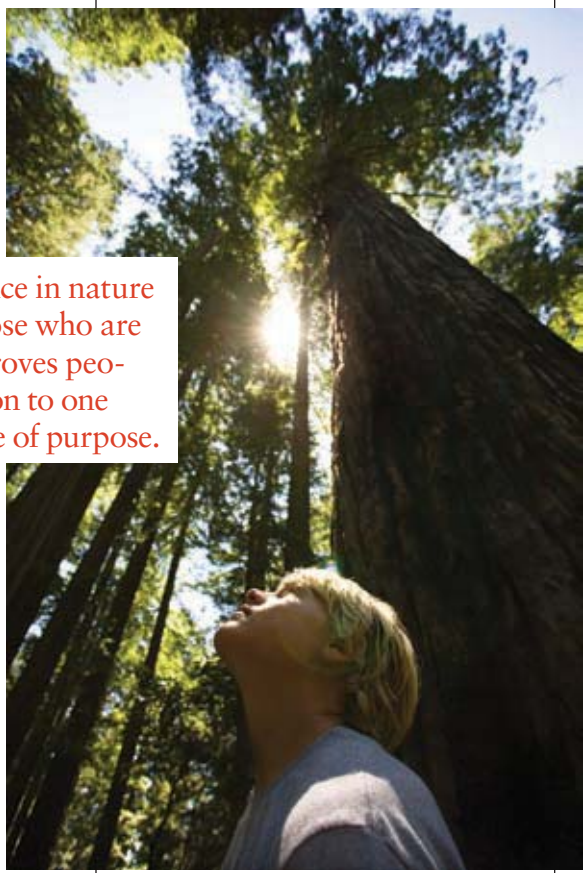
phen W. Porges of the University of Illinois at Chicago long ago argued that the vagus nerve is [the nerve of compassion] (of course, it serves many other functions as well). Several reasons justify this claim. The vagus nerve is thought to stimulate certain muscles in the vocal chamber, enabling communication. It reduces heart rate. Very new science suggests that it may be closely connected to receptor networks for oxytocin, a neurotransmitter involved in trust and maternal bonding.

Our research and that of other scientists suggest that activation of the vagus nerve is associated with feelings of caretaking and the ethical intuition that humans from different social groups (even adversarial ones) share a common humanity. People who have high vagus nerve activation in a resting state, we have found, are prone to feeling emotions that promote altruism—compassion, gratitude, love and happiness. Arizona State University psychologist Nancy Eisenberg has found that children with high-baseline vagus nerve activity are more cooperative and likely to give. This area of study is the beginning of a fascinating new argument about altruism: that a branch of our nervous system evolved to support such behavior.

MIND: Often when we learn about this type of intriguing academic work being done on emotions, morality and related areas, we are left asking, "Is there anything we can make actual use of here?" As you look down the road, what do you want the impact of your work to be out in the world?

KELTNER: In summarizing the new science of emotion in *Born to Be Good*, I was struck by how useful it is. Recent research is suggesting that our capacities for virtue and cooperation and our moral sense are old in evolutionary terms, and these capacities are found in the emotions I write about.

A new science of happiness is finding that these emotions can be readily cultivated in familiar ways, bringing out the good in others and in oneself. Here are some recent empirical examples:



- Experiences of reverence in nature or of being around those who are morally inspiring improves people's sense of connection to one another and their sense of purpose.
- Meditating on a compassionate approach to others shifts resting brain activation to the left hemisphere, a region associated with happiness, and boosts immune functions.
- Talking about what we are thankful for—in classrooms, at the dinner table or in a diary—boosts happiness, social well-being and health.
- Devoting resources to others, rather than indulging a materialist desire, brings about lasting well-being.

This kind of science gives me many hopes for the future. At the broadest level, I hope that our culture shifts from a consumption-based, materialist culture to one that privileges the social joys (play, caring, touch, mirth) that are our older (in the evolutionary sense) sources of the good life. In more specific terms, I see this new science informing practices in almost every realm of life. Here again are some well-founded examples: Medical doctors are now receiving training in the tools of compassion—empathetic listening, warm touch—that almost certainly improve basic health outcomes. Teachers now regularly teach the tools of empathy and respect. In prisons and juvenile detention centers, meditation is being taught. And executives are learning the wisdom of emotional intelligence—respect, building trust—and that there is more to a company's thriving than profit or the bottom line. **M**

DAVID DiSALVO is a science, technology and culture writer based in Florida.

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JUPITERIMAGES

When Does Consciousness Arise?

In the womb, at birth or during early childhood?

BY CHRISTOF KOCH

MOTHERS will want to crucify me for this seemingly cruel question, but it needs to be posed: How do we know that a newly born and healthy infant is conscious? There is no question that the baby is awake. Its eyes are wide open, it wriggles and grimaces, and, most important, it cries. But all that is not the same as being conscious, of experiencing pain, seeing red or smelling Mom's milk.

It is well recognized that infants have no awareness of their own state, emotions and motivations. Even older children who can speak have very limited insight into their own actions. Anybody who has raised a boy is familiar with the blank look on your teenager's face when you ask him why he did something particularly rash. A shrug and "I dunno—it seemed like a good idea at the time" is the most you'll hear.

Although a newborn lacks self-awareness, the baby processes complex visual stimuli and attends to sounds and sights in its world, preferentially looking at faces. The infant's visual acuity permits it to see only blobs, but the basic thalamo-cortical circuitry necessary to support simple visual and other conscious percepts is in place. And linguistic capacities in babies are shaped by the environment they grow up in. Exposure to maternal speech sounds in the muffled confines of the womb enables the fetus to pick up statistical regularities so that the newborn can distinguish its mother's voice and even her language from others. A more complex behavior is imitation: if Dad sticks out his tongue and waggles it, the infant mimics his gesture by combining visual information with proprioceptive feedback from its own movements. It is therefore likely that the baby has some basic level of unreflective, present-oriented consciousness.

When did that newborn become consciously aware?

FATMA IMAMOGLU (*Koch*); CORBIS

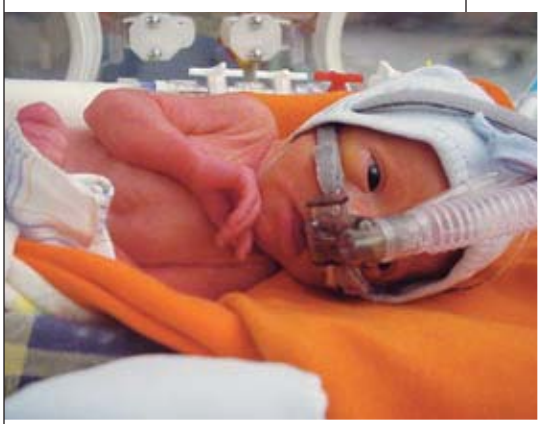
The Road to Awareness

But when does the magical journey of consciousness begin? Consciousness requires a sophisticated network of highly interconnected components, nerve cells. Its physical substrate, the thalamo-cortical complex that provides consciousness with its highly elaborate content, begins to be in place between the 24th and 28th week of gestation. Roughly two months later synchrony of the electroencephalographic (EEG) rhythm across both cortical hemispheres signals the onset of global neuronal integration. Thus, many of the circuit elements necessary for consciousness are in place by the third trimester. By this time, preterm infants can survive outside the womb under proper medical care. And as it is so much easier to observe and interact with a preterm baby than with a fetus of the same gestational age in the womb, the fetus is often considered to be like a preterm baby, like an unborn newborn.

Suspended in a **warm and dark cave**, connected to the placenta, the fetus is asleep.

But this notion disregards the unique uterine environment: suspended in a warm and dark cave, connected to the placenta that pumps blood, nutrients and hormones into its growing body and brain, the fetus is asleep.

Invasive experiments in rat and lamb pups and observational studies using ultrasound and electrical recordings in humans show that the third-trimester fetus is almost always in one of two sleep states. Called active and quiet sleep, these states can be distinguished using electroencephalography. Their different EEG signatures go hand in hand with distinct behaviors: breathing, swallowing, licking, and moving the eyes but no large-scale body movements in active sleep; no breathing, no eye movements and tonic muscle activity in quiet sleep. These stages correspond to rapid-eye-movement (REM) and slow-wave sleep common to all mammals. In late gestation the fetus is in one of these two sleep states 95 percent of the time, separated by brief transitions.



A two-week-old preterm infant born in the 25th gestational week. Although the newborn may already have some conscious experiences, a fetus of the same gestational age is kept actively sedated by the intrauterine environment.

USED WITH PERMISSION OF ANN-SOFI GUSTAFSSON AND HUGO LAGERCRANTZ

What is fascinating is the discovery that the fetus is actively sedated by the low oxygen pressure (equivalent to that at the top of Mount Everest), the warm and cushioned uterine environment and a range of neuroinhibitory and sleep-inducing substances produced by the placenta and the fetus itself: adenosine; two steroidal anesthetics, allopregnanolone and pregnanolone; one potent hormone, prostaglandin D_2; and others. The role of the placenta in maintaining sedation is revealed when the umbilical cord is closed off while keeping the fetus adequately supplied with oxygen. The lamb embryo now moves and breathes continuously. From all this evidence, neonatologists conclude that the fetus is asleep while its brain matures.

Dreamless Sleep?

One complication ensues. When people awaken during REM sleep, they often report vivid dreams with extensive narratives. Although consciousness during dreams is not the same as during wakefulness—most noticeably insight and self-reflection are absent—dreams are consciously experienced and felt. So does the fetus dream when in REM sleep? This is not known. But what would it dream of?

After birth, dream content is informed by recent and more remote memories. Longitudinal studies of dreaming in children by retired American psychologist David Foulkes suggest that dreaming is a gradual cognitive development that is tightly linked to the capacity to imagine things visually and to visuospatial skills. Thus, preschoolers' dreams are often static and plain, with no characters that move or act, hardly any feelings and no memories. What would dreaming be like for an organism that spends its time suspended in a sort of isolation tank, with no memories, and no way to imagine anything at all? I wager that the fetus experiences nothing in utero; that it feels the way we do when we are in a deep, dreamless sleep.

The dramatic events attending delivery by natural (vaginal) means cause the brain to abruptly wake up, however. The fetus is forced from its paradisic existence in the protected, aqueous and warm womb into a hostile, aerial and cold world that assaults its senses with utterly foreign sounds, smells and sights, a highly stressful event.

As Hugo Lagercrantz, a pediatrician at the Karolinska Institute in Stockholm, discovered two decades ago, a massive surge of norepinephrine—more powerful than during any skydive or exposed climb the fetus may undertake in its adult life—as well as the release from anesthesia and sedation that occurs when the fetus disconnects from the maternal placenta, arouses the baby so that it can deal with its new circumstances. It draws its first breath, wakes up and begins to experience life. M

CHRISTOF KOCH is Lois and Victor Troendle Professor of Cognitive and Behavioral Biology at the California Institute of Technology. He serves on *Scientific American Mind*'s board of advisers.

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Two Eyes, Two Views

Insights into the nuances of depth perception provided by our two eyes' slightly different views of the world

BY VILAYANUR S. RAMACHANDRAN AND DIANE ROGERS-RAMACHANDRAN

HUMANS enjoy stereoscopic vision (*a*). As we mentioned in our essay last issue, because our eyes are separated horizontally images we see in the two eyes are slightly different and the difference is proportional to the relative depth (*b*). The visual areas in the brain measure these differences, and we experience the result as stereo—what we all have enjoyed as children playing with View-Master toys.

Visual-image processing from the eye to the brain happens in stages. Rudimentary features such as the orientation of edges, direction of motion, color, and so on are extracted early on in areas called V1 and V2 before reaching the next stages in the visual-processing hierarchy for a progressively more refined analysis. This stage-by-stage description is a caricature; many pathways go "back" from stage to stage—allowing the brain to play a kind of 20-questions game to arrive at a solution after successive iterations.

Returning to the concept of stereo, we can ask: At what stage is the comparison of the two eyes' images made? If you are looking at a scene with hundreds of features, how do you know which feature in one eye matches with which feature in the other eye? How do you avoid false matches? Until the correct matching is achieved, you cannot measure differences. In stereopsis, this conundrum is called the correspondence problem.

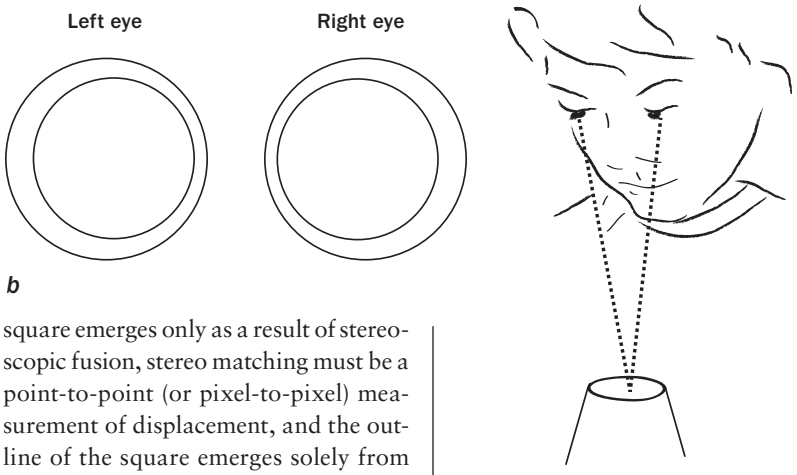
Questions about Boundaries

To address this issue, the great 19th-century German physicist, ophthalmologist and physiologist Hermann von Helmholtz asked: Is the comparison done very early, before object boundaries are recognized, or does the brain first separately extract contours in each eye before comparing them? He concluded, without a great deal of evidence, that form perception of outlines in each eye occurs prior to interocular comparison. "Monocular form perception precedes stereopsis," he said, arguing that the task of comparing the images in the two eyes is horrendously complex and happens very high up. The brain solves the correspondence problem by initially recognizing forms and then comparing the extended outlines of the forms. This strategy allows the brain to avoid (or minimize) false matches.

This idea was challenged nearly 100 years later by the late Hungarian scientist Béla Julesz, a non-self-effacing man of unparalleled genius, while working at Bell Labs. He employed a different stereogram (*c*), using computer-generated random-dot patterns rather than photographs or line drawings. In neither the left nor right eye image is there any recognizable contour or form—at all. Although these are made using a computer (as schematized in *d*), the principle can be understood by using a digital camera and random-dot images. Begin with a random-dot pattern about five square centimeters in size. Use a pair of scissors to cut out a one- by one-square-centimeter patch from another random-dot pattern (call it S, for square). Center this square atop the first pattern and take a photo to produce the left eye's image (L). If S is correctly positioned, it becomes virtually invisible because of camouflage from background dots. Now, slightly shift S horizontally to the right (making sure to position it so that no boundary of overlapping dots is seen from the small square). Take another picture to make the right eye's image, R.

Julesz presented just one image from his random-dot stereogram to each eye and was astonished to see a small square float out so vividly that he was almost tempted to grab it, even though no square is visible in either eye. The original experiment was done with digitally generated pixels rather than bits of paper, and the shift was also exactly digital. So it is not as if there is a square *hidden* in each eye's image; mathematically, it does not even *exist* in either eye alone. It is defined exclusively by the *difference*—the horizontal shift of S (shown by the column of Xs and Ys in *d*). Julesz concluded that von Helmholtz was wrong. Because the

RADIUS IMAGES/JUPITERIMAGES



b

square emerges only as a result of stereoscopic fusion, stereo matching must be a point-to-point (or pixel-to-pixel) measurement of displacement, and the outline of the square emerges solely from this comparison. Stereo precedes detection of form ("form" being used interchangeably with extended outlines and boundaries in this context).

Julesz's demo inspired a young medical student, Jack Pettigrew (then at the University of California, Berkeley), to look at the physiology of binocular nerve cells in the earliest stage of binocular processing. Until then, the problem of stereoscopic vision seemed intractable because, if von Helmholtz were right, researchers would have had to tackle the physiology of form perception first—about which no one had the foggiest idea how to proceed. Pettigrew found, however, that his hunch was right—these cells were extracting the horizontal shifts and signaling stereo (as we discussed in our previous column).

That is the simple story, but the picture got more complicated when a student (Ramachandran) from India found that in some circumstances form perception *preceded* stereo, showing the flexibility of the brain's visual centers. He created a stereogram that had a texture-defined square in each eye. He then shifted this entire square instead of shifting the dots that defined the textures (*e*).

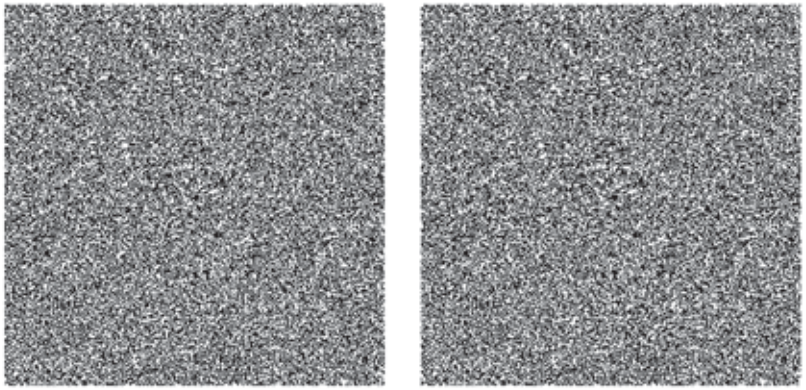
He had two random-dot patterns, one in each eye. But this time there is a square visible in each eye separately—unlike Julesz patterns. It is still made of random dots yet, because of a difference of texture, a square is visible separately in each eye. The dots that constitute the left eye's image (including S) are completely *different* in the two eyes; unlike Julesz's pictures, they are uncorrelated. This stereogram is the converse of Julesz's—a square is visible in each eye, but the dots that constitute it (and its background) is unrelated in the two eyes.

Ramachandran found that when he viewed this image through a stereoscope, the central square floated out. Because the dots defining the squares were uncorrelated in the two eyes, he and his colleagues concluded that, in this case, form perception occurred prior to stereo. The square was recognized separately in each eye before the shift across the eyes was measured. The Julesz rule could be violated. The brain often uses multiple tricks to achieve the same goal. In a noisy camouflaged environment, it makes sense to use both strategies.

The second display he invented makes the same point. It takes advantage of a curious visual effect dubbed illusory contours (*f*). Four "pacmen" are made of four black disks with pie-shaped wedges cut out from each. What you see, though, is not pacpeople facing each other; you see an opaque illusory white square occluding four black disks in the background. The brain says, in effect, "What is the likelihood that an evil scientist has precisely aligned these disks? More likely it is an opaque square, so that is what I will see." You hallucinate the edges, called image segmentation.

Now can these illusory edges provide an input for stereo? Begin with the left eye's picture in *f* and shift the illusory square to the left to create the right eye's image. (This shift entails taking bigger bites out of the pie.) When you view the images through a viewer—lo and behold—the illusory square floats

c



d

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0	0	1	1	0	1	1	0	1	0
0	1	0	A	A	B	B	1	0	1
1	1	1	B	A	B	A	0	0	1
0	0	1	A	A	B	A	0	1	0
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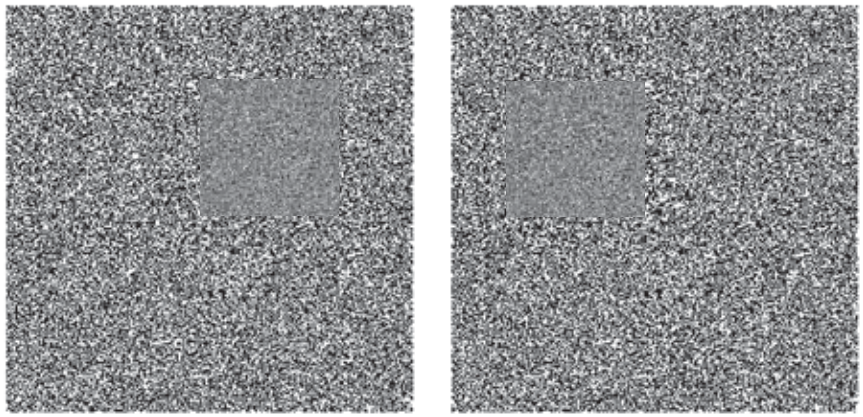

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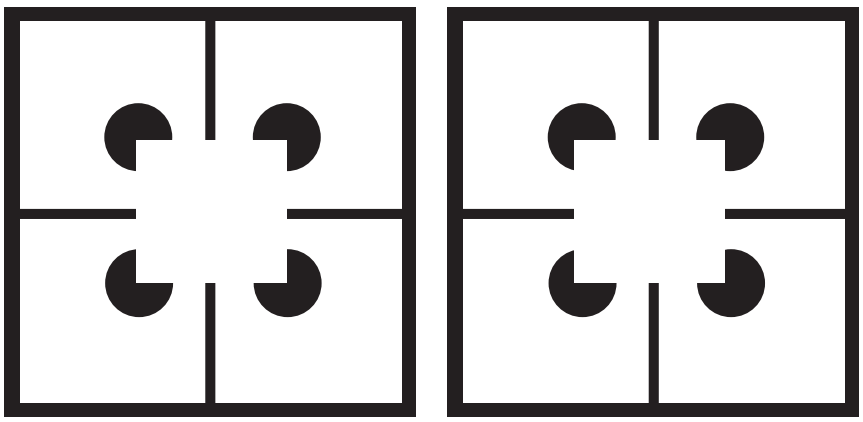

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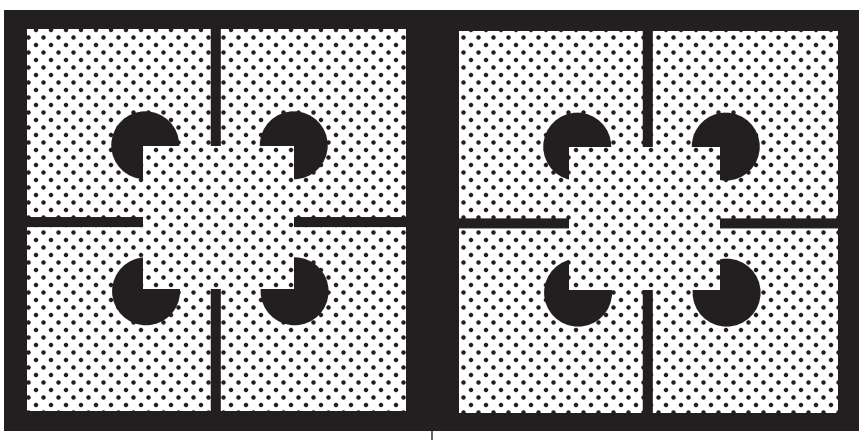


g

out! Again, form processing and image segmentation occur prior to stereo.

It gets better. Let us take a template of this stereogram and paste it on repeating wallpaper made of columns of dots (*g*). The dots are identical in the two eyes; they convey no disparity information. Yet amazingly, the dots inside the illusory square float out along with it—an illusion we call stereo capture; the dots are captured by the illusory square and dragged forward even though they themselves are not shifted.

This result suggests that Julesz's claim was not entirely correct: stereo involves more than comparing pixels across the two eyes. Even if you consider Pettigrew's disparity cells, they must be extracting tiny oriented clusters (not points) and "looking for" identical clusters to match. But the experiments of Ramachandran (and very similar results from psychologist Lloyd Kaufman of New York University) showed that the mechanism was even more sophisticated than that; it could segment the image based on *implied* occlusion and "hallucinate" illusory contours that can serve as tokens for stereoscopic matching. Once this information has been extracted and disparity measured, the brain constructs a 3-D illusory surface. The fact that the enclosed dots are dragged forward implies that the 3-D surface feeds back to be applied to the dots.

Thus, we may conclude that von Helmholtz, Julesz, Pettigrew and Ramachandran are all right; the visual processing of stereo is more complex than we thought. We have no inkling of the physiological mechanisms underlying these interactions. Cells signaling disparity are in V1 (as shown by Pettigrew), but cells that extract illusory contours (from implied occlusion) are extracted in area V2, the next stage up, as shown by Rudiger von der Heydt of Johns Hopkins University. These findings imply that messages from V2 must be fed back to V1 to modulate processing of smaller features. This idea has yet to be tested. M

VILAYANUR S. RAMACHANDRAN and DIANE ROGERS-RAMACHANDRAN collaborate on studies of visual perception at the Center for Brain and Cognition at the University of California, San Diego. They are on the board of advisers for *Scientific American Mind*.

(Further Reading)

- **Foundations of Cyclopean Perception.** Bela Julesz. University of Chicago Press, 1971. (MIT Press edition, 2006.)
- **The Role of Contours in Stereopsis.** V. S. Ramachandran, V. Madhusudhan Rao and T. R. Vidyasagar in *Nature,* Vol. 242, pages 412–414; April 6, 1973.
- **Capture of Stereopsis and Apparent Motion by Illusory Contours.** V. S. Ramachandran in *Perception and Psychophysics,* Vol. 39, No. 5, pages 361–373; May 1986.
- **Seeing in Depth:** Vol. 1: **Basic Mechanics** and Vol. 2: **Depth Perception**. Ian P. Howard and Brian J. Rogers. Oxford University Press, 2008.

(calendar)

September

7 When artists, anthropologists and neuroscientists gather at **The Brain Unravelled** in London through September 19, their creative efforts will range from paintings to performances to mixed-media works. In addition to the

exhibition, which includes a children's area, the event offers a daily program of film screenings, concerts, artist talks and lectures by renowned scientists. Informed by the latest research, the speakers will delve into the relation between brains and minds, plumbing the deepest reaches of human experience: our consciousness.
London
www.thebrainunravelled.com

9 Charles Darwin, in his 1871 book *The Descent of Man,* provoked his contemporaries by suggesting not only that our physical traits had evolved over time but also that our mental faculties had not always been as keen as they are today. At the conference **Evolution of Brain, Behaviour & Intelligence** in Cambridge, England, international scientists will discuss advances made since Darwin's time, drawing on results from species as diverse as unicellular organisms and Neandertals. Darwin biographer James Moore will deliver the keynote lecture.
Cambridge, England
https://registration.hinxton.wellcome.ac.uk/display_info.asp?id=130

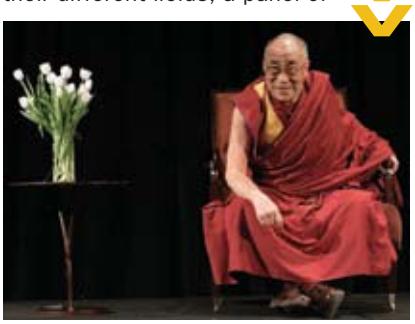
12 In Huntington's disease, genetic mutations cause a protein known as Huntingtin to become toxic to the brain, leading to movement disorders, problems swallowing and speaking, and eventually dementia and death. Hundreds of researchers and clinicians will convene in Vancouver at the 2009 **Congress on Huntington's Disease** to discuss advances in our understanding of the disorder as well as the latest results from experimental treatments.
Vancouver, B.C.
www.worldcongress-hd.net

24 Will neuroscience transform national security? Is a brain-dead person alive or dead? Could new findings in brain science undermine moral and criminal responsibility? These are a few of the controversial questions that experts will take on at **BRAIN Matters: New Directions in Neuroethics,** a cross-disciplinary conference hosted by the Novel Tech Ethics research team at Dalhousie University.
Halifax, Nova Scotia
www.noveltechethics.ca/site_brainmatters.php

October

1 As many as half of us may suffer from a mental disorder at some point in our life. Added to the burden of illness is the social stigma that people with mental health problems face. **The Scottish Mental Health Arts and Film Festival,** sweeping over the country until October 22, tries to raise positive awareness about these issues through a series of concerts, film screenings and theater performances. This will be the third year for the festival, which is the largest of its kind in the world.
Scotland
http://mentalhealthfestival.dreamhosters.com

8 Ancient wisdom and modern neuroscience will collide at **Mind and Life XIX,** a two-day conference presided over by the Dalai Lama himself. Exploring the emerging intersections among their different fields, a panel of educators, scientists and contemplatives will discuss ways to promote personal and societal health. The Mind and Life Institute's ultimate goal? To inspire a view of education that will "create compassionate, engaged, and ethical world citizens."
Washington, D.C.
www.educatingworldcitizens.org



17 Fraudulent mind reading, sleight-of-hand illusions and con artistry—not exactly what you would expect to find at the world's largest forum for brain scientists, **Neuroscience 2009.** But among its myriad presentations of cutting-edge research, the 39th annual meeting of the Society for Neuroscience features a different take on perception and memory as three renowned magicians present "Magic, the Brain and the Mind." Workshops, posters and lectures will put a more serious face on the science, exploring the nervous system from every possible angle.
Chicago
www.sfn.org/am2009

26 When we send criminals to prison, it is partly to punish them. But if a brain scan were to reveal faulty emotional circuitry at the root of their misdeeds, should that change the way we treat them in court? At **Law and Neuroscience: Our Growing Understanding of the Human Brain and Its Impact on Our Legal System,** international researchers will explore how neuroscience influences legal practice in Europe.
Acquafredda di Maratea, Italy
www.esf.org/index.php?id=5679

Compiled by Frederik Joelving. Send items to editors@SciAmMind.com

JOHN GIANNICCHI *Photo Researchers, Inc. (illustration with heads);* ROBERT SORBO *Reuters/Corbis (Dalai Lama);* CORBIS *(magic trick)*

The Social Cure

By Jolanda Jetten, Catherine Haslam, S. Alexander Haslam and Nyla R. Branscombe

Membership in lots of groups—at home, work, the gym—makes us healthier and more resilient. Here's how—and why

You have turned up for your annual medical checkup. The doctor has taken your blood pressure, inquired about your diet and exercise patterns, and asked whether you smoke. Then come some rather pointed questions about your social life: Do you have many friends? Do you socialize? Which groups do you belong to? How diverse are they? How important are these groups to you?

Even though these questions are unexpected, you go through the long list of your active memberships: your book club, volleyball team, hiking group, work colleagues, and so on. Your doctor congratulates you and says that you are doing exactly the right things. You even learn that because you belong to so many social groups you should not worry if you skip your gym visit every now and then.

This exam, of course, is not how doctor visits typically go. Checkups usually end after the medical tests and a cursory exchange of pleasantries. But they shouldn't end there.

Belonging to social groups and networks appears to be an important predictor of health—just as important as diet and exercise. This point is demonstrated by a

PHOTOILLUSTRATION BY AARON GOODMAN

In one study, the least sociable people were twice as likely to get colds as those who were the most sociable.

Joining a group is one of the best ways to arrest the cognitive decline associated with aging.

MICHAEL PRINCE *Corbis (man sitting alone)*; SCIENTIFIC AMERICAN MIND *(seniors at a museum)*

study of 655 stroke patients reported in 2005 by Bernadette Boden-Albala, professor of sociomedical sciences and neurology at Columbia University, and her colleagues. Patients who were socially isolated were nearly twice as likely to have another stroke within five years as were those with meaningful social relationships. In fact, being cut off from others appeared to put people at far greater risk of another stroke than traditional factors such as having coronary artery disease or being physically inactive (each of which increased the likelihood of a second stroke by about 30 percent).

Such effects are not restricted to those who have a significant health problem. In a 2008 study epidemiologists and health researchers Karen Ertel, Maria Glymour and Lisa Berkman of the Harvard School of Public Health tracked 16,638 elderly Americans over a period of six years. The findings, published in the *American Journal of Public Health,* revealed significantly less memory loss in those who were more socially integrated and active.

Using an even more prosaic health indicator, a 2003 study by Carnegie Mellon University psychologist Sheldon Cohen and his colleagues showed that a diverse social network made people less susceptible to the common cold. Their work, published in *Psychological Science,* indicated that the least sociable people in their sample were twice as likely to get colds as those who were the most sociable—even though the more sociable people were probably exposed to many more germs.

FAST FACTS

Community Minds

1» **Membership in a large number of groups was once thought to be detrimental because it complicated our lives and caused stress.**

2» **Now, however, research shows that being part of social networks enhances our resilience, enabling us to cope more effectively with difficult life changes such as the death of a loved one, job loss or a move.**

3» **Not only do our group memberships help us mentally, they also are associated with increased physical well-being.**

Such discoveries take us beyond the old debate about body-mind dualism, which explores the nature of the link between physical and mental health (soma and psyche). There is now compelling evidence that the health risk of social isolation is comparable to the risks of smoking, high blood pressure and obesity, even after controlling for other variables known to affect health.

Eggs in Many Baskets

A body of recent research shows that belonging to multiple social groups is particularly critical in shielding people from the health hazards of important life changes. Consider the marathon runner whose injury prevents her from ever running again. Anyone might be devastated by such an injury, but the consequences are greater for a person who defines herself exclusively in terms of being a runner. Likewise, think of the workaholic who never has time for his family or friends and therefore finds adjustment to retirement particularly difficult.

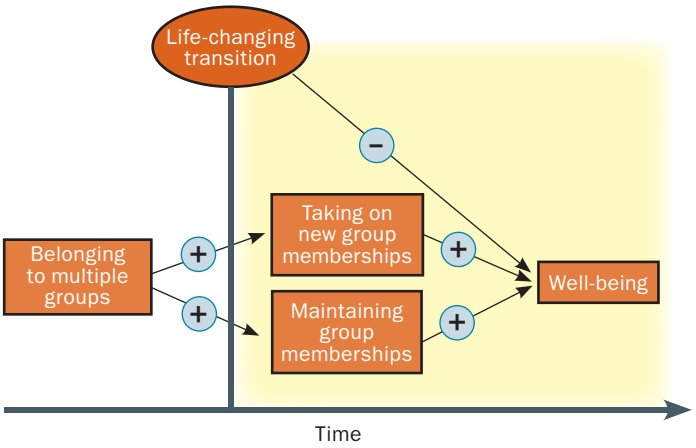
We hypothesize that it is best not to have all of your eggs (social identities) in one basket in case misfortune strikes. It is better, research suggests, to spread your metaphorical eggs around a number of baskets (that is, to have multiple social identities) so that the loss of one still leaves you with others.

Three of us (Haslam, Haslam and Jetten) recently examined this notion in a study we conducted with other clinical and social psychologists—Abigail Holmes, W. Huw Williams and Aarti Iyer—at the University of Exeter in England. In the study, published in 2008 in *Neuropsychological Rehabilitation*, we examined the changing circumstances of 53 people who had recently suffered a stroke. Life satisfaction after the stroke was much higher for those who had belonged to more social groups before their stroke. Further analysis suggested the reason for this finding was that stroke patients who had previously belonged to a lot of groups had a bigger social support network to fall back on. This was especially critical for those who had incurred the most cognitive losses (problems with directions, forgetting names, having trouble making up their mind). Patients who saw themselves as more damaged in this way tended to describe a lower quality of life, in part because these cognitive losses made it harder for them to maintain their social relationships—stripping them of the support group life had provided.

In another study that Jetten and S. A. Haslam recently published in the *British Journal of Social Psychology* with social psychologists Iyer, Dimitrios Tsivrikos and Tom Postmes, we monitored first-year university students over a period of four months, beginning two months before they enrolled in school and ending two months after. A key question for us was whether we could predict which individuals were most likely to embrace their new identities as university students. As in our stroke study, one of the best predictors of healthy adjustment was the number of groups that each student had belonged to before starting school. Those who

Protecting Well-Being during Change

We weather life transitions better if we have multiple social identities. For example, if people lose their job they are also likely to lose a network of colleagues that over the years has been important to them. This will tend to compromise their well-being. Yet they may still belong to the local tennis club or be a volunteer at the local church, and maintaining these identities will probably help them through the transition. *—J.J., C.H., S.A.H. and N.R.B.*



Adapted from "Maintaining Group Memberships: Social Identity Continuity Predicts Well-Being After Stroke," by Catherine Haslam et al., in Neuropsychological Rehabilitation, *Vol. 18; 2008, and "The More (and the More Compatible) the Merrier: Multiple Group Memberships and Identity Compatibility as Predictors of Adjustment after Life Transitions," by Aarti Iyer et al., in* British Journal of Social Psychology *(in press).*

(The Authors)

JOLANDA JETTEN is professor of social psychology at the University of Queensland in Australia and at the University of Exeter in England. She is currently editor of the *British Journal of Social Psychology*. CATHERINE HASLAM is associate professor of neuropsychology at the University of Exeter. S. ALEXANDER HASLAM is professor of social psychology at the University of Exeter and serves on the board of advisers for *Scientific American Mind*. NYLA R. BRANSCOMBE is professor of social psychology at the University of Kansas.

The authors' work on this paper was funded by the Economic and Social Research Council in the U.K., the Australian Research Council and the Canadian Institute for Advanced Research.

had belonged to more groups in the past had lower levels of depression, even after adjusting for other factors that could influence this transition—including uncertainty about college, the availability of social support, and academic obstacles.

Can Groups Also Bring Us Down?

So do groups always make us healthier? Can they also have a negative influence, perhaps when there is a lot of internal conflict in our group? What if our group is marginalized and stigmatized by society at large? Do we feel stronger when the groups with which we identify are strong but embattled when our groups are not respected or fail to achieve?

Group failure has been found to have one of two outcomes: sometimes people distance themselves from the group and report lower levels of group identification, but often their affiliation grows stronger and they feel greater group solidarity. And people are remarkably creative in explaining away group failure, as when they root for teams that always lose. One of us (Branscombe), along with psychologist Daniel L. Wann of Murray State University, looked at baseball and basketball fans in the U.S. and found that their degree of team identification bore no relation to the team's success or failure. For the die-hard fans—for whom the team was central to their sense of who they were—there was no question of doing anything other than sticking with the team through thick and thin.

What about membership in a group that experiences discrimination and devaluation? Again, people can take one of two routes: either distancing themselves from the group or emphasizing their commitment to it.

This point emerges clearly from a study that Branscombe conducted at the University of Kansas with social psychologists Michael T. Schmitt and Richard D. Harvey, published in 1999 in the *Journal of Personality and Social Psychology*. African-Americans who felt they had been the targets of racial discrimination reported lower levels of well-being—yet at the same time, the more they felt discriminated against, the more tightly they held on to their racial identity. What was particularly interesting was that those who identified more strongly as African-American in response to perceived racial discrimination experienced better psychological

Too Many Groups?

For a long time, researchers warned against belonging to too many groups, reasoning that the more groups we are in, the busier and more stressful our lives. But recent studies have suggested that what matters is not the *number* of social groups but the relations among them. For example, researchers have noted that in addition to work-family conflict, people can experience work-family facilitation. Psychologists Elianne F. van Steenbergen and Naomi Ellemers of the University of Leiden in the Netherlands found that women who were the most energetic and effective at work believed that they managed work life so well *precisely because they had an active family life.* The reverse pattern was also found—women who were more energetic at home said it was because working gave them an energy boost. Further, work-family facilitation was associated with improved physical health as indexed by people's cholesterol levels and body mass.

—J.J., C.H., S.A.H. and *N.R.B*

***SOURCE:** "Is Managing the Work-Family Interface Worthwhile? Employees' Work-Family Facilitation and Conflict Experiences Related to Objective Health and Performance Indicators," by Elianne F. Van Steenbergen and Naomi Ellemers, in* Journal of Organizational Behavior *(in press).*

GETTY IMAGES

Die-hard fans will continue to back losers when the team is central to their sense of who they are.

GETTY IMAGES

People are found to cope better with prejudice, and feel more able to resist it, if they embrace their group identity.

well-being than those who felt discriminated against yet identified less strongly with their racial group.

Similar findings also emerge from more recent studies by Branscombe and her colleagues of women, the elderly and minority cultural groups. Feeling discriminated against has the direct effect of compromising individuals' well-being. At the same time, people are found to cope better with prejudice, and to feel more able to resist it, if they embrace their group identity rather than denying it. Such results confirm that social groups can be the source of suffering, if they attract discrimination but in addition can be the means of dealing effectively with the slings and arrows of that very discrimination.

A similar conclusion was reached by Stephen D. Reicher, a social psychologist at the University of St. Andrews in Scotland, and S. A. Haslam on the basis of findings from their BBC Prison Study [see "The Psychology of Tyranny"; Scientific American Mind, October 2005]. In this research, male volunteers were randomly assigned to one of two groups, as "prisoners" or "guards" in a laboratory "prison." Over the course of eight days the prisoners were transformed from a group of dispirited individuals into a well-functioning, upbeat collective. The opposite process occurred among the guards, however. Their sense of shared identity decreased over time, associated with an increasing sense of powerlessness and depressed mood. Because the conditions of the "prison" made them socially isolated, the guards came to experience high levels of burnout [*see box on next page*].

Over time these changes in group members' social identification were reflected not only in stated levels of stress and depression but also in physiological indicators of stress—specifically, the participants' cortisol levels. Here again is evidence that social identities and membership in social groups [become internal to] the individual, leading to changes in basic autonomic functioning.

Real or Imagined Groups?

To answer the question of why identities have a positive effect on health, it helps to examine what happens to a person when social identity is impaired or no longer functioning as it should. This is how many neuropsychologists work: they attempt to understand a particular process by looking at what happens when it breaks down.

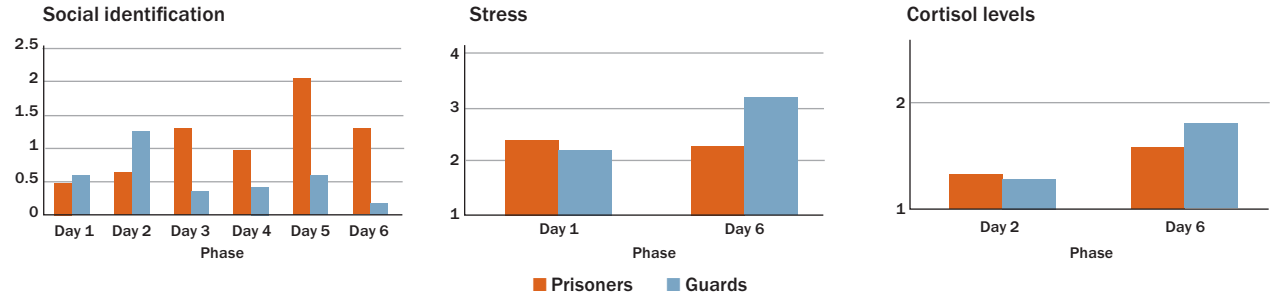
A recent study that Haslam, Haslam and Jetten conducted at the University of Exeter, together with

Group life and a sense of social identity have a profound influence on our general health and well-being.

clinical psychologists Cara Pugliese and James Tonks, examined this issue in a group of people with dementia. This research will soon be published in the *Journal of Clinical and Experimental Neuropsychology*. We started with the assumption that the more severe the dementia, the less people would be able to remember details of their past lives (that is, what they used to be and how they interacted with others), leading to a reduction in overall health. Indeed, our results showed that people with early signs of dementia experienced more health problems than those whose memories were largely intact. Surprisingly, though, we found no difference between the reported health of participants in the early stages of dementia and those with more advanced dementia. If anything, people in the latter group—who typically did not know what day of the week or even what year it was—tended to feel

Stressing the Group

The BBC Prison Study shows how changes in group members' shared social identity as prisoners or guards (*left*) are associated with corresponding changes in stress, as assessed through self-reporting (*center*) and physiological measures (*right*). As social identification increases among the prisoners (*red bars*), they are protected from an increase in stress. Similarly, among the guards (*blue bars*), a decline in social identity leads to greater stress. *—J.J., C.H., S.A.H. and N.R.B.*



In the BBC Prison Study, the guards became more stressed and depressed as their sense of shared social identity declined.

Among the prisoners, an increase in shared social identity brought improved mood and a sense of collective self-efficacy.

BRITISH BROADCASTING CORPORATION

SOURCES: "Stressing the Group: Social Identity and the Unfolding Dynamics of Responses to Stress," by S. Alexander Haslam and Stephen D. Reicher, in Journal of Applied Psychology, *Vol. 91, No. 5; September 2006, and "Rethinking the Psychology of Tyranny: The BBC Prison Study," by Stephen D. Reicher and S. Alexander Haslam, in* British Journal of Social Psychology, *Vol. 45, No. 1; March 2006; see also www.bbcprisonstudy.org*

How Social Is Social Networking?

With more than 220 million people worldwide using online networks such as Facebook and MySpace, the capacity to interact with people around the world has rapidly expanded. Such developments open up new ways to build social networks. Simply by going online, we can find out what our friends are up to, go through their photo albums and know what is on their minds—even when they are on the other side of the planet. Do such virtual social networks contribute to better health the way real networks do? Some speculate that Facebook is particularly valuable for those who are less mobile (such as older adults or the disabled) and therefore represents an excellent way to avoid social isolation.

There are also warnings, however, that in some cases, rather than reducing social isolation, tools such as Facebook could actually add to it. In a survey of 184 MySpace users, media researchers Rob Nyland, Raquel Marvez and Jason Beck of Brigham Young University found that the most frequent users reported being less involved in the communities around them than the least frequent users. This assessment suggests that virtual-world networking can become a substitute for real-world engagement. —*J.J., C.H., S.A.H.* and *N.R.B.*

healthier than those whose dementia was still relatively mild.

At first, this pattern was puzzling. But further analysis showed that the people with more advanced dementia tended to indicate they belonged to more groups than did those with mild dementia. In addition, groups from the past (their community group or bridge club) were in their minds in the present; unlike those with mild dementia, those with severe dementia did not remember that they were no longer active in these groups. It was this perception of group belonging that was responsible for their surprisingly higher levels of professed well-being.

This finding is consistent with the observations of neurologist Oliver Sacks of Columbia University Medical Center, who often writes about people whose lives have remained remarkably intact in the face of severe neurological impairment. In *The Man Who Mistook his Wife for a Hat* (Touchstone, 1998), Sacks concludes that when appraising patients' quality of life, it is not necessarily the severity of the disorder that matters so much as a person's ability to maintain a coherent sense of self.

A Group a Day …

Group life and a sense of social identity have a profound influence on our general health and well-being. This finding reflects something fundamental about human nature: we are *social animals* who live (and have evolved to live) in groups. For humans, membership in groups is an indispensable part of who we are and what we need to be to lead rich and fulfilling lives.

Recognizing the importance of social identity opens up new thinking not only in psychology but also in sociology, economics, medicine and neuroscience. Such work has practical ramifications, too, suggesting that groups can offer a social *cure*. "As a rough rule of thumb," wrote Harvard University political scientist Robert D. Putnam in his book *Bowling Alone* (Simon & Schuster, 2000), "if you belong to no groups but decide to join one, you cut your risk of dying over the next year in half."

In other words, participation in group life can be like an inoculation against threats to mental and physical health. This is much cheaper than the pharmaceutical pathway, with far fewer side effects. And as a means of keeping the doctor at bay, it is also likely to prove much more enjoyable. M

(Further Reading)

- **Perceiving Pervasive Discrimination among African Americans: Implications for Group Identification and Well-being.** Nyla R. Branscombe, Michael T. Schmitt and Richard D. Harvey in *Journal of Personality and Social Psychology,* Vol. 77, No. 1, pages 135–149; July 1999.
- **Bowling Alone: The Collapse and Revival of American Community.** Robert D. Putnam. Simon & Schuster, 2000.
- **Social Identity, Health and Well-being.** Edited by S. Alexander Haslam, Jolanda Jetten, Tom Postmes and Catherine Haslam. Special issue of *Applied Psychology: An International Review,* Vol. 58, pages 1–192; 2009.
- **The Social Cure: Identity, Health and Well-being.** Jolanda Jetten, Catherine Haslam and S. Alexander Haslam. Psychology Press (in press).

ROB KIM *Landov*

WHEN PAIN LINGERS

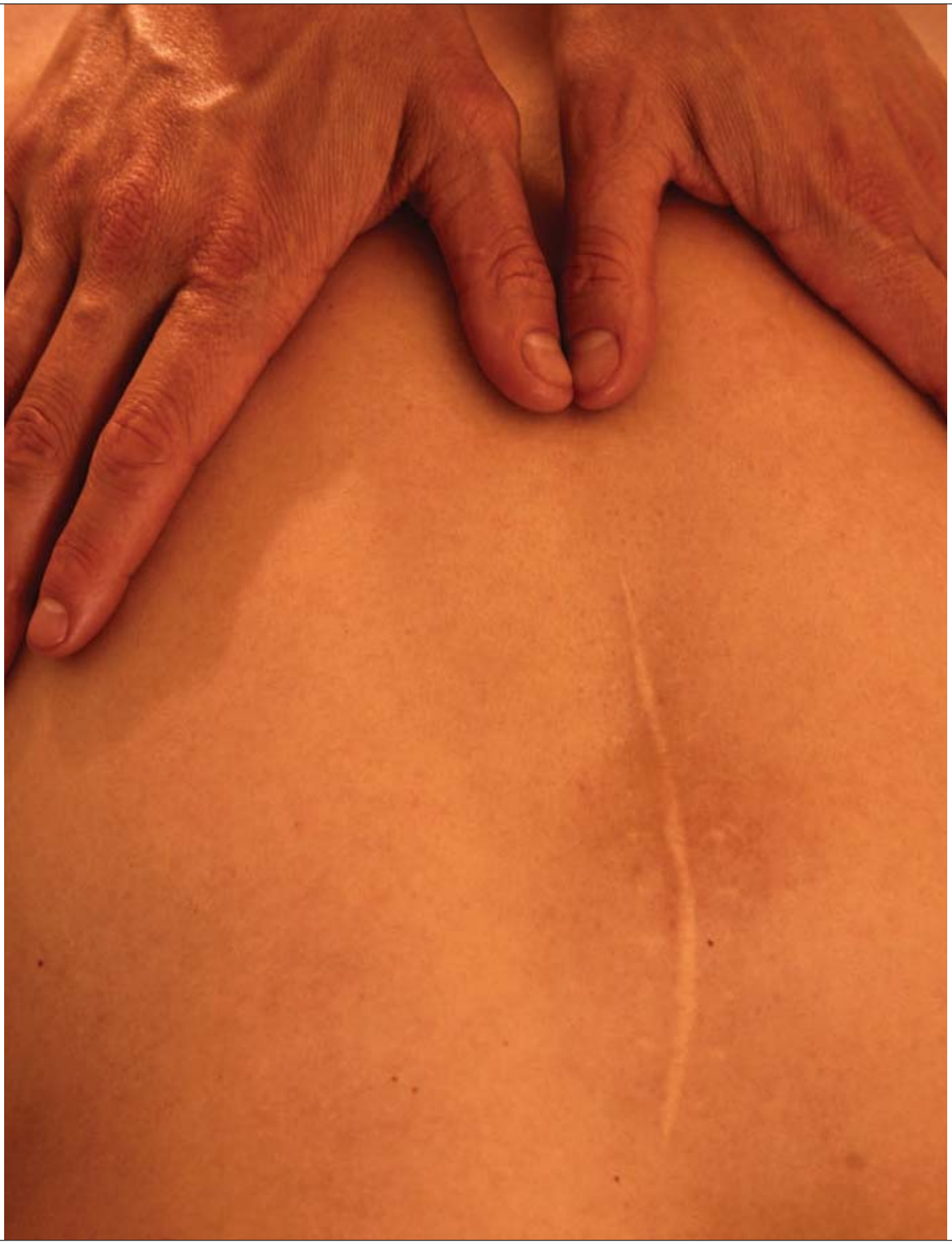
Researchers are revealing the biological basis of persistent, pathological pain—and providing clues to better treatments

BY FRANK PORRECA AND THEODORE PRICE

Imagine you are a doctor treating a patient who has been in nearly constant pain for four years, ever since the day he sprained his ankle stepping off a curb. Physical therapy only briefly dulled the agony. Painkillers were not much better, and the most effective drugs made your patient exhausted and constipated. He is now depressed, sleeping poorly and having difficulty concentrating. As you talk with him, you realize that his thinking also seems impaired. Your exam confirms that the original injury has healed. Only pain and its consequences remain—and your options for helping this man are running out.

This scenario plays out every day in doctors' offices around the world. Fifteen to 20 percent of adults worldwide suffer from persistent, or chronic, pain. Half the primary care patients who develop a chronic pain condition fail to recover within a year, according to surveys conducted by the World Health Organization. Common causes of such unrelenting discomfort include physical trauma, arthritis, cancer, and metabolic diseases such as diabetes that can damage nerves. In many cases, however, the pain's origins are mysterious.

Indeed, despite decades of intense research into the biology of pain and how pain is perceived, many mysteries still surround chronic pain and its treatment. No one knows for sure why some injuries, even minor ones, result in persistent pain or why it occurs in some people but not in others. Nevertheless, researchers are pinpointing telltale changes in the neurons that underlie persistent pain. In particular, they have documented abnormal excitability among neurons at every level of the body's pain network. For instance, in the spinal cord, some cells aberrantly amplify pain signals after undergoing a type of molecular "learning" that is similar to what happens in the brain during the formation of long-term memories.



KEITH BROFSKY *age Fotostock*

For some people, a windy walk is agonizing because a stiff breeze activates pain nerves.

Chronic pain is more emotionally fraught than acute pain—which comes on quickly but lasts a relatively short time. Changes in brain regions governing feelings and complex thoughts in chronic pain states may help explain some of the unwanted emotional and cognitive problems, from depression to attention deficits, that can sometimes emerge after years of suffering. Researchers have even uncovered signs that chronic pain might be a type of neurodegenerative disease, affecting parts of the brain that deal with attention, memory and decision making. A firmer understanding of these processes could lead to new treatments that would alleviate the relentless chronic pain experienced by millions of people worldwide.

Disease of Discomfort

We sense pain using specialized sensory neurons called nociceptors; these cells extend to most of the body, their fibers running alongside other sensory neurons in large bundles that make up peripheral nerves. Nociceptors normally respond selectively to strong stimuli, such as pressure, heat or cold. They then send their messages to neurons in the spinal cord, which, in turn, relay neuronal indications of potential or real tissue damage to the brain centers where pain perception occurs [*see box on opposite page*]. Activation of this pain pathway is critical for reflexive and coordinated protective responses to escape something that could damage the body, such as a stinging insect or a hot stove. Detecting circumstances in which we might experience harm is a vital protective function of our nervous system.

But the protective pain we experience as a result of daily living is quite different from that which leads patients to seek medical attention. Instead of becoming active only in the presence of strong and potentially damaging stimuli, the pain transmission pathway can become pathologically revved up in reaction to movement of joints, light touch or other actions that are normally innocuous—a phenomenon termed allodynia. In some sufferers, donning clothes, taking a shower or going for a walk on a breezy day is excruciating because the fabric, water or wind on their skin abnormally stimulates pain pathways.

In other cases, pain can occur spontaneously, without any obvious cause. Patients who have endured nerve damage as a result of diabetes, for example, may feel intense burning pain while doing nothing more than sitting quietly in a chair.

Unlike ordinary pain messages, spontaneous pain and pain produced by mild stimulation do not signal impending damage to tissues and do not provide a survival advantage. Pain produced under these conditions reflects pathological changes in pain pathways and represents a disease in and of itself.

Too Much Excitement

In the early 1980s researchers began to learn the sources of such pathological pain. Studies in rats by neuroscientist Clifford Woolf of University College London and Harvard University and his colleagues revealed, for example, that following an injury to a rat's paw, neuronal signals from nociceptors near the skin to neurons in the spinal cord became amplified, much like turning up the volume on an iPod. These altered neurons unleash exaggerated reactions to tissue-damaging input; in addition, they become more easily excited, responding to stimuli that are ordinarily too mild or weak to produce a reaction.

Hormones or inflammatory molecules that the body produces in response to injury may sensitize nociceptors, making them more impulsive, a change that could instigate the development of chronic pain and abnormal sensitivity to

GETTY IMAGES

FAST FACTS

Pain but No Gain

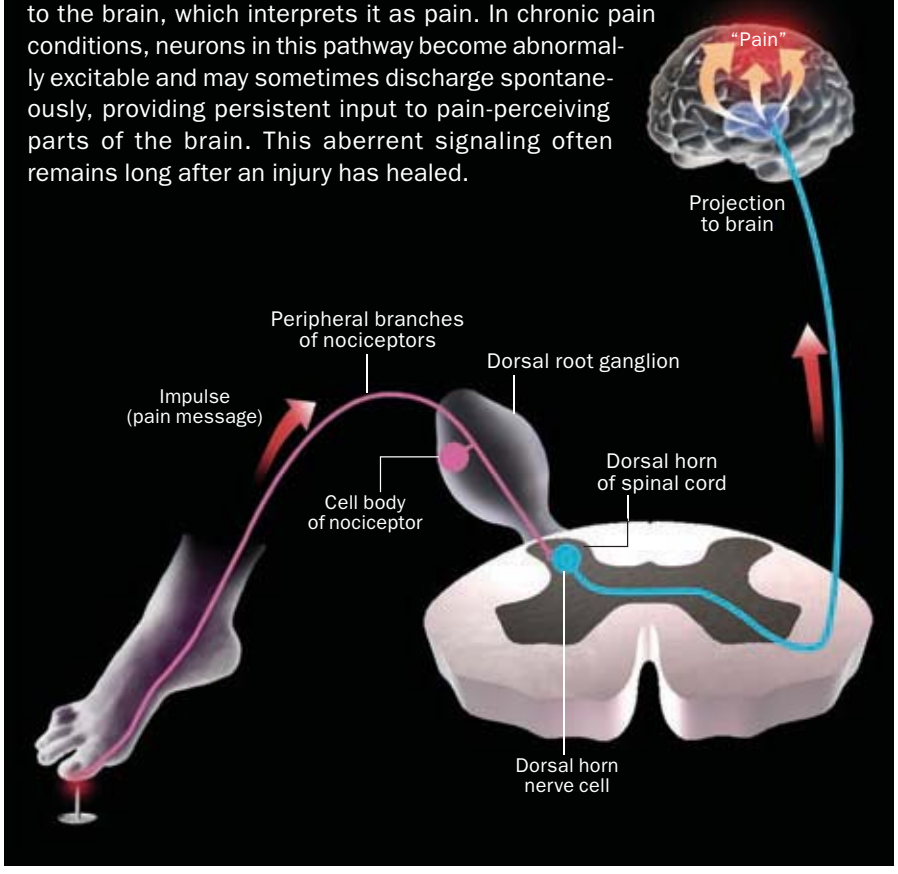
1» **Researchers are pinpointing telltale changes in neurons that underlie persistent pain. In particular, they have documented abnormal excitability among neurons at every level of the body's pain network.**

2» **Chronic pain is more emotionally fraught than short-lived pain. Changes in brain regions governing feelings and complex thoughts in chronic pain states may help explain some of the unwanted emotional and cognitive problems, from depression to attention deficits, that can sometimes emerge after years of suffering.**

3» **A firmer understanding of the biology of chronic pain could lead to new treatments that would alleviate the debilitating condition in millions of people worldwide.**

Perceiving Pain

In a healthy system for perceiving pain, a tissue injury causes pain-sensing nerve cells, or nociceptors (*pink*), to send a message to nerve cells in the dorsal horn of the spinal cord. These spinal cord cells pass the message to the brain, which interprets it as pain. In chronic pain conditions, neurons in this pathway become abnormally excitable and may sometimes discharge spontaneously, providing persistent input to pain-perceiving parts of the brain. This aberrant signaling often remains long after an injury has healed.



mild stimuli. (Such molecules also account for the aches a person may feel during normal movement a day after lifting weights, an activity that can lead to mild muscle damage.) Chronic pain conditions often begin when a peripheral nerve is injured, making that nerve—a bundle of fibers of which some are nociceptors—and neighboring ones more excitable. Hyperexcitability within the uninjured nerves that intermingle with the wounded nerve is probably paramount for the persistence of pain after the original injury is gone because many of the damaged nerves degenerate.

In addition to becoming more excitable, injured neurons may sometimes start signaling spontaneously. Injuries to peripheral nerves from trauma, diseases such as diabetes and cancer, drug treatments or excessive use of recreational drugs such as alcohol can spark such relentless electrical discharge, or ectopic activity, in the damaged nerves. These nerves then provide persistent input to the rest of the pain transmission pathway, a process that is believed to drive spontaneous pain. Often the recalcitrant signaling that underlies the pain remains long after an injury has healed.

In recent years researchers have revealed a molecular basis for this low-level ectopic activity. Voltage-gated sodium channels—proteins that conduct sodium ions into a cell in response to voltage changes—on the membranes of these neurons are essential for their ability to transmit electrical messages; their abundance and activity—how often they open and shut, for example—play an important role in how sensitive or excitable a neuron is. The latest data indicate that in chronic pain states these channels cluster where they count most, at the endings of the neurons near the skin and all along the nerve, most likely making the neurons more responsive to input.

For example, in a 2003 study one of us (Porreca) and his colleagues used fluorescent molecules to visualize a sodium channel called $Na_v1.8$ in the peripheral nerve cells of rats after a type of nerve injury that leads to chronic pain. We saw that the nerve membrane undergoes a "remodeling" so that the $Na_v1.8$ channels accumulate near the injury. This study suggests that injury prompts the nerve cells to ship lots of these proteins from their neuronal cell bodies near the spinal cord outward to the nerve terminal. This redistribution appears to be critical to the experience of neuropathic pain, because blocking the cells from producing this sodium channel made the rats' pain disappear, as evidenced by a return to their normal behavior. Neuroscientists have also discovered support for a similar transport of sodium channels in human tissues from studies on patients who have nerve injuries that produce persistent pain.

Other researchers have been homing in on the underpinnings of chronic pain in the dorsal horn of the spinal cord, where the peripheral pain fibers end. In 1999 neuroscientist Patrick W. Mantyh, then at the University of Minnesota, and his colleagues found that a subset of these dorsal horn neurons—just 1 to 3 percent of cells in this region—of the spi-

AMADEO BACHAR

After an injury, signals from pain-sensing cells in the body's periphery become amplified, much like turning up the volume on an iPod. The exaggerated input can lead to pathological pain.

A molecular mechanism that brain cells use to form certain types of memories may also underlie the ability of spinal cord neurons to sustain a state of chronic pain.

nal cord are major culprits in chronic pain. Using a Trojan horse strategy, they chemically coupled a toxin to a neurotransmitter, a neural signaling substance, so that when the neurotransmitter bound to its receptor on another cell, the receptor-transmitter complex served as a chemical "scalpel," deleting (killing) the recipient cell. Without these dorsal horn neurons, rats failed to show signs of chronic pain after local inflammation or nerve injury—symptoms that plagued rats that still had these neurons. The elimination of this neuronal subset did not affect ordinary pain perception, however, implicating these cells primarily in pathological discomfort.

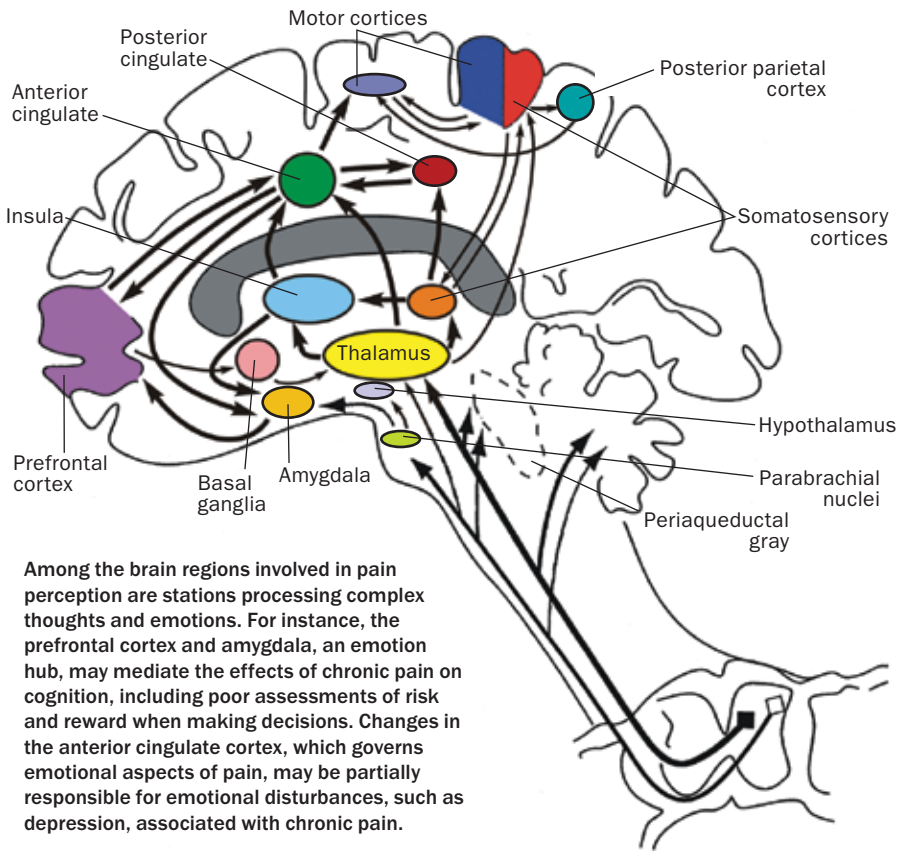
But what happens in these spinal cord neurons when pain becomes chronic? Recent data hint that they undergo a process called long-term potentiation (LTP), a long-lasting improvement in communication between two neurons that also underlies the formation of certain types of memories in the brain. Although LTP in the brain generally requires high-frequency input, 100 hertz or above, in a 2006 study neurophysiologist Jürgen Sandkühler of the Medical University of Vienna and his colleagues demonstrated that low-frequency stimulation from injured peripheral nerves in rats can lead to LTP in some dorsal horn neurons. In LTP, input from one neuron leads to a heightened response in the recipient cell, an effect that should enable spinal cord cells to amplify incoming pain signals. And just as LTP represents a molecular mechanism of memory storage in brain cells, it may underlie the ability of spinal cord neurons to sustain a state of chronic pain.

Nerve circuits that arise in the brain and lead down to the spinal cord can also profoundly influence the incoming pain signals and the resulting experience of pain. In this circuit, cells in the periaqueductal gray area of the midbrain receive input from the various regions of the brain's outer layer (the cortex) as well as from interior sections, such as the amygdala and the hypothalamus. This midbrain region then relays information to the rostral ventromedial medulla (RVM) in the brain stem, the lower part of the brain adjoining the spinal cord. Activation of this circuit mediates the powerful suppression of pain that occurs during trauma, intense stress or excitement [see illustration on page 44 of "The Psychology of Pain," by Howard L. Fields].

This same circuit, and in particular the RVM, also plays a major role when pain from an acute injury persists. Work from our laboratories has shown that when nerves are injured in rodents, a specific set of cells in the RVM sends out a signal that amplifies, rather than diminishing, incoming pain signals and sets the stage for chronic pain. In 2001, for example, a team led by Porreca used the toxin-based Trojan horse strategy to selectively snip out these RVM neurons in rats. Without these cells, the rats still developed pathological pain in their hind paw after a nerve injury, but that pain was short-lived, suggesting the RVM harbors a critical "switch" for the maintenance of chronic pain.

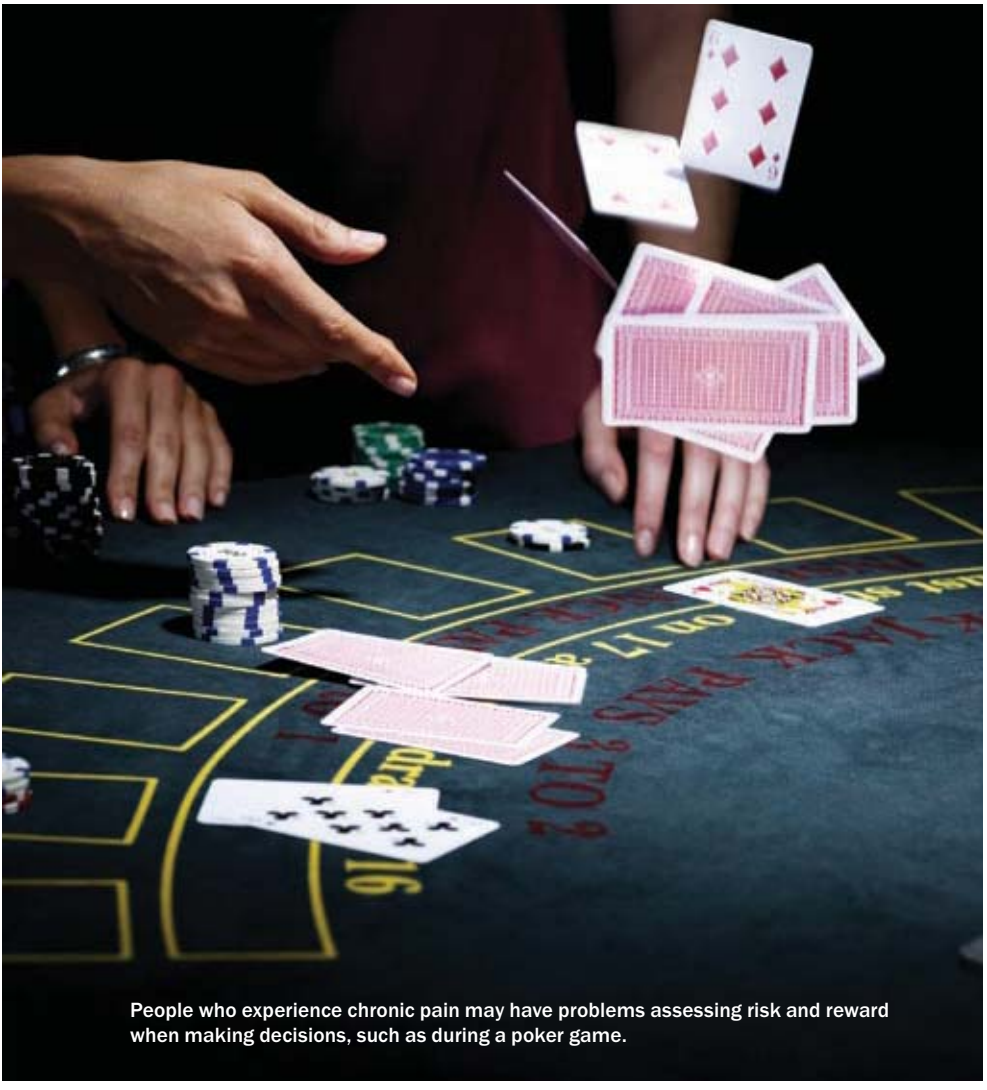
In an important 2008 study neuroscientist Irene Tracey of the University of Oxford and her colleagues found that neural activity in this brain stem region

Pain in the Brain



Among the brain regions involved in pain perception are stations processing complex thoughts and emotions. For instance, the prefrontal cortex and amygdala, an emotion hub, may mediate the effects of chronic pain on cognition, including poor assessments of risk and reward when making decisions. Changes in the anterior cingulate cortex, which governs emotional aspects of pain, may be partially responsible for emotional disturbances, such as depression, associated with chronic pain.

FROM "HUMAN BRAIN MECHANISMS OF PAIN PERCEPTION AND REGULATION IN HEALTH AND DISEASE," BY A. VANIA APKARIAN, M. CATHERINE BUSHNELL, ROLF-DETLEF TREEDE AND JON-KAR ZUBIETA IN *EUROPEAN JOURNAL OF PAIN*, VOL. 9; 2005. REPRINTED WITH PERMISSION FROM ELSEVIER



People who experience chronic pain may have problems assessing risk and reward when making decisions, such as during a poker game.

GETTY IMAGES

in human volunteers paralleled the duration of painful symptoms (induced by exposure to the hot pepper compound capsaicin) that were similar to those of chronic pain patients. Current evidence suggests that ectopic input from injured nerves may alter these RVM cells so that their messages to the spinal cord facilitate, instead of inhibiting, incoming pain signals.

Painful Feelings

In addition to operating the pain-control circuit, pain-processing regions of the brain interpret input from the spinal cord and from other brain regions to create an overall impression of the discomfort. This interpretation depends on the setting and on a person's past experience, attentiveness and mood, among other psychological factors [see "The Psychology of Pain," on page 42]. To that end, pain not only stimulates sensory areas of the brain but also powerfully activates brain areas involved in emotion, such as the anterior cingulate cortex (ACC), a region governing emotional aspects of pain, and the amygdala, which mediates fear and other feelings. These areas—which are part of a so-called pain axis in the brain—can become hyperactive in chronic pain conditions and may, in turn, play a significant role in enhanced responses to stimulation in these patients.

Various known triggers of chronic pain seem to alter the ACC in particular. Peripheral nerve injury and chronic inflammation precipitate neural restructuring in the ACC. In addition, psychological factors such as mood, expectation and hypnotic suggestion can modulate pain responses in the ACC, according to human imaging studies [see "The Truth and the Hype of Hypnosis," by Michael R. Nash and Grant Benham; Scientific American Mind, June 2005]. Thus, the ACC may integrate sensory input with emotional state and may partially underpin some of the "affective" disturbances associated with chronic pain, such as depression, sleep disorders and pain catastrophizing, a condition in which patients expect and fear that pain will be intense and unmanageable. (Neuroscientists have shown that pain catastrophizing specifically engages the ACC.) The involvement of the ACC and the pain axis in general might also help explain the common occurrence of pain in patients with conditions such as depression and post-traumatic stress disorder.

A hyperactive pain axis not only increases pain intensity but also augments the aversive qualities of the experience. Chronic pain may thus reflect a switch from a bottom-up condition in which painful sensory information dominates to a top-down state in which emotional and cognitive assessments control pain behavior.

Certain cognitive deficits may also result from the toll chronic pain takes on patients. In 2004 neuroscientist A. Vania Apkarian of Northwestern University's

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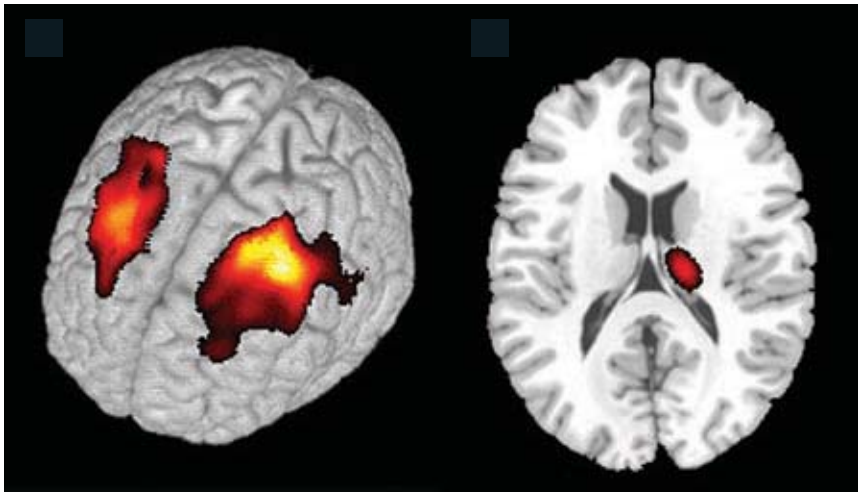
Feinberg School of Medicine and his colleagues demonstrated that individuals with chronic back pain or complex regional pain syndrome, a debilitating condition that can develop after trauma, showed a decreased ability to accurately assess risk and reward when making decisions. All the patients took part in the Iowa Gambling Task, a card game in which players choose between "bad" decks of cards that yield high immediate gain but substantial future losses and "good" decks that produce lower immediate gain but minimal losses later. Painhardo of the University of Porto in Portugal showed that arthritic rats display a similar impairment. Given a choice between a "high-risk" food-dispensing lever that yields three food pellets in three out of 10 visits and a "low-risk" lever promising one pellet eight times out of 10, arthritic rats over time developed a preference for the high-risk lever (risking going hungry in seven of 10 visits), whereas normal rats more consistently picked the low-risk lever (missing only two snacks in 10). In this study the researchers associated a change in the brain with

Recent findings hint that pain might actually be a neurodegenerative disease leading to remodeling of the prefrontal cortex and possibly other cognitive regions of the brain.



Chronic pain may lead to brain atrophy. In one study patients who suffered from long-lasting back pain had a lower density of neurons in the prefrontal cortex (*left, colored regions*) and in the right thalamus (*right, red oval*) compared with pain-free individuals.

free participants chose cards from the good decks—the most profitable strategy—more frequently than the pain patients did. The patients also tended to be fickle, frequently switching between decks, suggesting that the unpleasant emotions that accompany a state of persistent agony may interfere with judgments in other situations, such as weighing options in a gambling game.

In recent work presented at international pain meetings, neuroscientists Volker Neugebauer of the University of Texas Medical Branch and Vasco Gal- the inappropriate risk assessment: alterations in chemical signaling within neural circuits connecting the amygdala to the prefrontal cortex—a region governing higher cognitive functions, including attention, decision making and working memory—of the arthritic rats.

Previous work by Neugebauer and his colleagues suggests that chronic experimental pain in rats can lead to amplification of neural signals coming into the so-called nociceptive amygdala, a part of the amygdala governing pain. This augmented input then magnifies the messages—which are inhibitory in nature—that the nociceptive amygdala sends to the prefrontal cortex. The increased inhibition of the prefrontal cortex may impair an animal's (or human's) ability to accurately assess the risks of options when making important decisions.

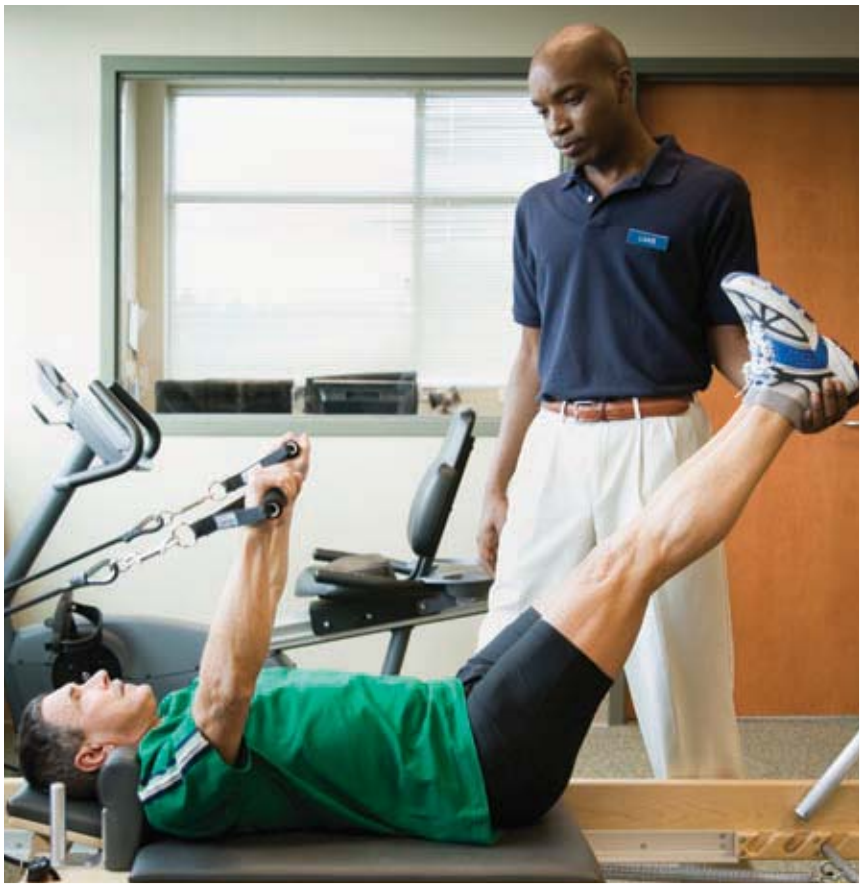
More obvious brain changes may underlie other types of cognitive decline, among them muddled thinking and difficulty concentrating, in chronic pain patients. In 2004 Apkarian and his colleagues reported a shrinking of the prefrontal cortex in patients with very long-lasting back pain. The decreased brain volume was proportional to the duration of the pain in these patients but roughly equivalent to that seen in 10 to 20 years of aging. Since then, other research teams have revealed preliminary evidence of possible atrophy in the brains of some patients afflicted with other persistent pain conditions. These results hint that pain might actually be a neurodegenerative disease leading to remodeling of the prefrontal cortex and possibly other cognitive regions of the brain.

No one knows for sure how chronic pain could lead to neurodegeneration, but the increased neuronal excitability that we now know characterizes chronic pain may provide a clue. Such excitability often leads to excessive release of the neurotransmitter glutamate, and glutamate is known to be toxic to neurons in large quantities. At this point, however, the glutamate explanation is purely speculative, and researchers are actively investigating various possible molecular causes of this neurodegeneration.

Calming Nerves

The recent insights into the mystery of why pain becomes chronic may point to new therapies. Medical researchers are attempting to block amplification of neuronal signals at every stage of the body's pain network. A few current and emerg-

FROM "CHRONIC BACK PAIN IS ASSOCIATED WITH DECREASED PREFRONTAL AND THALAMIC GRAY MATTER DENSITY," BY A. VANIA APKARIAN ET AL., IN *JOURNAL OF NEUROSCIENCE*, VOL. 24, NO. 46; NOVEMEBER 17, 2004



Exercise and intellectual challenges such as puzzle solving might help chronic pain patients combat the cognitive decline that can occasionally accompany their condition.

ing medicines are geared toward countering abnormal activation of nociceptors. Some of these therapeutics act as "sponges" to absorb inflammatory proteins or nerve growth factors that are thought to boost the excitability of these pain-transmitting neurons. Other compounds that target neuronal hyperexcitability include sodium channel blockers and inhibitors of enzymes such as nitric oxide synthase that yield active neurotransmitters.

In the future, new analgesics might target the small subset of cells in the dorsal horn of the spinal cord that Mantyh, now at the University of Arizona, and his team tied to chronic pain or analogous cells in the RVM. A better understanding of the role of the ACC in chronic pain conditions might lead to novel therapeutic strategies that ameliorate pain, along with its psychological consequences. Ideally, these antiamplification therapies will not only ease patients' suffering but also prevent structural brain changes and possibly neurodegeneration that accompany extreme forms of chronic pain. That is, the best treatments would not just reduce symptoms but also reverse the disease process.

Drug treatments might make up just a part of the eventual strategy for ending intractable pain. Advanced diagnostic techniques might help determine the underlying cause of persistent pain. Some researchers are trying to identify "biomarkers," or molecular signs, of chronic pain that they could find in a blood or tissue sample, enabling early detection—and treatment—of abnormal changes in the nervous system that signal chronic pain. This technique could also point to the therapies most likely to work in an individual.

For patients who have a long-standing problem, doctors may want to prescribe behavioral techniques to address any emotional and cognitive fallout from the pain. Patients might be advised, for example, to supplement their medication with mind-preserving strategies, including intellectual challenges such as puzzle solving and physical exercise. Such a multipronged attack on relentless pain and its consequences should ultimately offer greater hope for the afflicted. M

(Further Reading)

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- **Chronic Pain Patients Are Impaired on an Emotional Decision-Making Task.** A. Vania Apkarian et al. in *Pain,* Vol. 108, Nos. 1–2, pages 129–136; March 2004.
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- **Identifying Brain Activity Specifically Related to the Maintenance and Perceptual Consequence of Central Sensitization in Humans.** Michael C. Lee, Laura Zambreanu, David K. Menon and Irene Tracey in *Journal of Neuroscience,* Vol. 28, No. 45, pages 11642–11649; November 5, 2008.
- **Morphological and Functional Reorganization of Rat Medial Prefrontal Cortex in Neuropathic Pain.** Alexia E. Metz et al. in *Proceedings of the National Academy of Sciences USA,* Vol. 106, No. 7, pages 2423–2428; February 17, 2009.

GETTY IMAGES (*exercising*); AGE FOTOSTOCK (*puzzle solving*)

THE PSYCHOLOGY

Our expectations, mood and perspective on pain powerfully influence how much something actually hurts—and the decisions we make every day

BY HOWARD L. FIELDS

OF PAIN

GETTY IMAGES

Several years ago an elderly man came into the emergency room at Cook County Hospital in Chicago with a large, painful abscess (boil) on the back of his neck. When I told him he needed a minor procedure to lance the boil and drain it, he became ashen, asking, "Doc, is this going to hurt?" I told him that if at any time the treatment hurt too much, he could tell me to stop—and I would. I opened the boil with a very sharp scalpel. He did not make a sound for some time. "When are you going to start?" he finally asked. "It's done," I said. "How did you do that?" he replied. "I didn't feel anything."

Most people think of pain as resulting from physical injury or disease, but psychological factors play a huge role in pain perception. In the case of my elderly patient, my reassurance that the treatment would not significantly worsen his pain—because he could stop me if it did—produced an analgesic effect. In addition, reducing the man's fear enabled him to look forward to pain relief instead, and that positive expectation also eased his pain.

The importance of mind-set to pain perception should come as no surprise. Pain is a warning sign of injury, but for such a sign to be useful, pain must influence human behavior in a way that increases survival. Thus, pain must be intimately tied to brain functions that govern behavior and decision making, including expectation, attention and learning. By way of these links, a painful blister on your foot can motivate you to stop walking or to protect the area with moleskin. It may also teach you to shop for more comfortable shoes or wear thicker socks in the future.

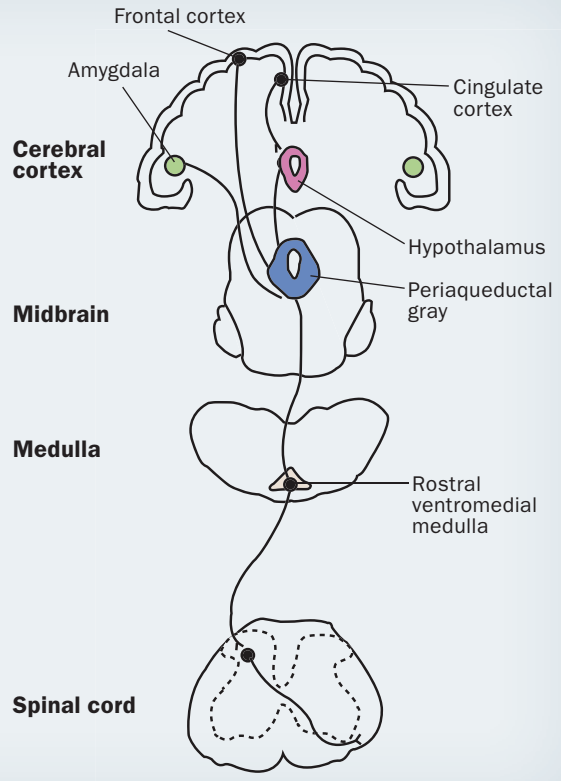
The interaction between the pain message and the brain centers that mediate motivation and learning accounts for the powerful effect of a person's state of mind on the severity of pain he or she experiences with any injury. It explains the placebo effect: the expectation that a sugar pill will relieve pain reduces the extent of the agony even though the pill has no pharmacological effect. Conversely, if you are convinced that an injection, say, will be very painful, you are likely to unwittingly amplify the sting. Mood also interacts with agony. Depressed people, for example, may feel more pain as a result of their sour state of mind. In fact, worsening of a long-standing

pain problem, such as headache, often is the first sign of depression or at least the complaint that first brings a depressed patient to the attention of a physician.

Recent investigations are unraveling the mystery of how and when factors such as expectation of reward or punishment, fear, stress and mood alter perceived pain intensity and affect our daily decisions. Some of these psychological factors also influence the risk of developing a chronic pain condition. The research not only reveals just how far pain reaches into our psyches but also may lead to better ways of controlling pain and hastening recovery from painful injuries.

Mind Control

A circuit in the brain and spinal cord acts as a volume control for pain, adjusting its perception depending on circumstances. This pathway contains two classes of neurons: off cells, which are activated by endorphins and morphine and inhibit pain transmission, and on cells, which facilitate pain signals and are stimulated by noxious stimuli and certain psychological factors.



Mind over Matter

In the classic view of pain perception, a stimulus to the body excites pain-sensitive sensory neurons in the body's periphery; these neurons then transmit information in the form of electrical signals that eventually activate parts of the brain that enable us to perceive pain [see "When Pain Lingers," by Frank Porreca and Theodore Price, on page 34]. But for decades doctors have noted that a person's mental state can also dramatically affect pain perception.

For example, Harvard University anesthesiologist Henry K. Beecher noted in an article published in 1956 that soldiers who had been wounded in battle complained of much less pain than did patients with similar injuries in a civilian hospital. Beecher reasoned that in the context of having survived a battle and heading for home, an injury has a different meaning than it does for people hurt in the course of ordinary life. In the war scenario, a wound has honorable connotations, and such a positive spin on pain can lessen the sensation, Beecher speculated. Doctors have also long known about the analgesic powers of traumatic stress and of dummy pills that patients believe to be painkillers.

How could cognitive and emotional influences affect how much agony we feel? Over the past few decades researchers have uncovered a circuit in the brain and spinal cord that functions as a kind of volume control for pain, adjusting the amount a person perceives depending on the circumstances. In the early 1970s scientists at the University of California, Los Angeles, discovered that excitation of a small area in the midbrain of the rat produced profound pain relief. When they sent electricity through small wires implanted into that region of the brain, the rodent would no longer respond to intense, tissue-damaging stimuli that otherwise would make it squeak and flee. Later in the decade scientists showed that patients with severe chronic pain obtain significant, though temporary, relief from electrical stimulation of the same midbrain site, the periaqueductal gray.

FAST FACTS

Mentality of Misery

1» **Most people think of pain as resulting from physical injury or disease, but psychological factors play a huge role in pain perception. Pain is intimately tied to brain functions that govern behavior and decision making, including expectation, attention and learning.**

2» **Recent investigations are unraveling how factors such as expectation of reward or punishment, fear, stress and mood alter perceived pain intensity and affect our choices.**

3» **Scientists are not only revealing just how far pain reaches into our psyches but are also using their findings to devise ways of better controlling pain and hastening recovery from painful injuries.**

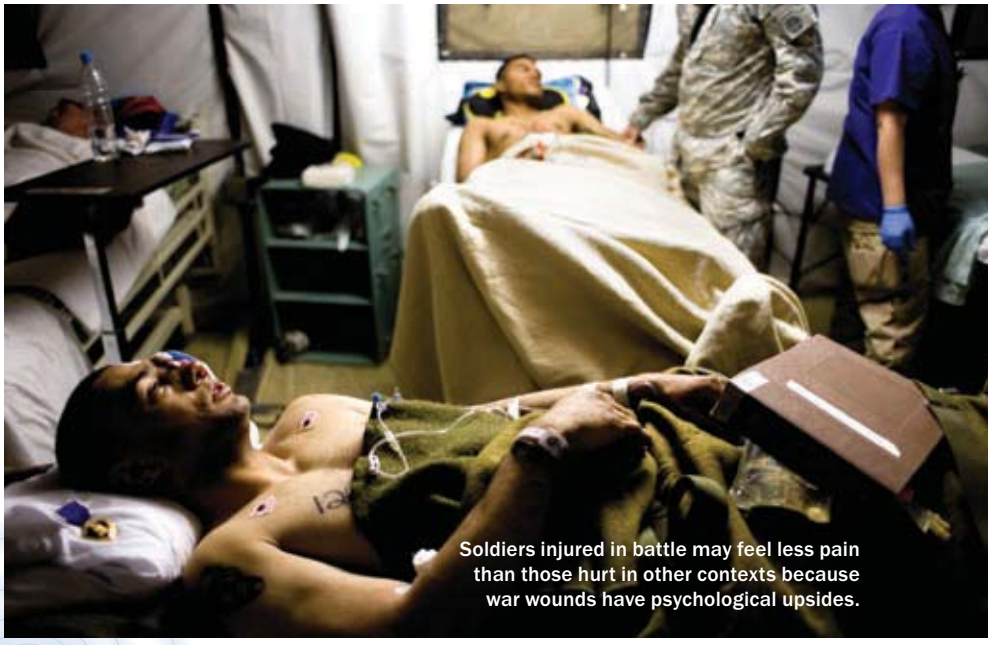
FROM "PAIN MODULATION: EXPECTATION, OPIOID ANALGESIA AND VIRTUAL PAIN," BY HOWARD L. FIELDS, IN *PROGRESS IN BRAIN RESEARCH*, VOL. 122; 2000. EDITED BY E. A. MAYER AND E. B. SAPER, WITH PERMISSION FROM ELSEVIER

Since then, researchers have mapped other parts of the body's pain-control circuit [*see box on opposite page*]. It stretches from the brain's cerebral cortex in the frontal lobes through underlying brain structures, including the periaqueductal gray, to the spinal cord, where pain-sensitive nerve fibers connect to neurons that transmit pain signals from the rest of the body. Neurons in this pathway synthesize peptides known as endorphins that have pharmacological properties identical to the powerful opioid morphine. Endorphins, the body's natural painkillers, and opioids (which also include opium and heroin) act at the same receptors, called mu opioid receptors, along this pain modulatory pathway to produce their analgesic effects.

Great Expectations

Neuroscientists are finding that cognitive influences on pain operate through this modulatory pathway. The circuit is the conduit for a variety of expectation effects, including the prospect of pain relief from a placebo pill. In 2004, for example, neuroscientist Tor D. Wager, now at Columbia University, and his colleagues found that a placebo produced increased activity in this pain-control circuit. Endorphins seem to be important in transmitting the pain-suppressing signal: my colleagues and I found that blocking mu opioid receptors with the drug naloxone erases the placebo effect in patients experiencing pain from a recent surgery. [For more on placebos, see "Cure in the Mind," by Maj-Britt Niemi; Scientific American Mind, February/March 2009.]

Recent data from my laboratory implicate the same circuit in other forms of expectation while underscoring their power over pain. In a study published in 2006 my research team showed volunteers color cues generated on a computer monitor just before exposing them to a painful stimulus through a metal probe taped to their hand. The words "low temperature" against a blue background were followed by mildly painful heat, and the words "high temperature" against a red background by more intense heat. Afterward subjects were placed in a magnetic resonance imaging scanner and randomly shown the red-high and blue-low cues beginning just before the mild or intense painful stimuli were applied.



EROS HOAGLAND *Redux Pictures*

Soldiers injured in battle may feel less pain than those hurt in other contexts because war wounds have psychological upsides.

The pain we experience is a synthesis of what happens in our body and what we expect, which depends on what we are told or have otherwise learned.

We found that the blue-low temperature cue, which had previously preceded milder discomfort, reduced the reported pain to the intense stimulus. In contrast, the red-high temperature cue, which had been paired with greater pain, amplified the discomfort of the mild stimulus. When the red-high cue preceded the intense stimulus, the pain magnitude was greatest. The brain sites known to be part of the pain transmission system in the thalamus and cortex were fully activated only when both stimulus intensity and high pain cues were given together. Thus, the pain we experience is a synthesis of what happens in our body and what we expect, which depends on what we are told or have otherwise learned.

We isolated the brain regions involved in the expectation effect by subtracting activity in the brain areas that lit up when the stimulus was intense and a person anticipated more pain from those excited by the same painful peripheral stimulus given when a person expected less pain. The net result was activation in cortical and brain stem regions that we now know are involved in the control of pain.

In addition to predictions about the pain itself, the expectation of a reward—say, from food or drugs—can profoundly affect pain intensity. In a classic 1984 ex-

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Looking forward to eating a delicious dessert can reduce pain.

periment pharmacologists J. Dum and Albert Herz of the Max Planck Institute for Psychiatry in Munich fed rats every day while the rodents were standing on a metal plate, which was at room temperature. Some of the rats ate regular rat chow, whereas the others feasted on chocolate-covered biscuits. After two weeks, the researchers placed the rats on the plate, which they then gradually heated to a painful temperature. The rats that had previously consumed their regular chow responded to the pain after four seconds; the rats that expected to receive chocolate endured the heat for twice as long. When the rats received a drug that prevents endorphins from relieving pain, however, the animals would no longer wait twice as long for their chocolate treat. Thus, the anticipation of the food reward had served as an analgesic, effectively raising the rats' tolerance for pain.

Food, sex and other natural enticements—and even the mere anticipation of such pleasures—activate the brain's reward circuitry in both rodents and humans. In doing so, they can also produce pain relief. The effects of opioid drugs further suggest that reward and pain relief have a partially shared neural basis. After all, the most powerful of these drugs, such as morphine and oxycodone

Food, sex and other natural enticements—and even the mere anticipation of such pleasures—activate the brain's reward circuitry. In doing so, they can also produce pain relief.

(OxyContin, a prescription painkiller that has been widely abused), can relieve severe pain but also unleash a "high"—leading to their addictive potential.

Painful Choices

Pain and reward interact at mu opioid receptors. Mice engineered to lack a functioning mu receptor experience neither pain relief nor reward from morphine. In addition, rats given naloxone (which blocks opioid receptors) no longer experience pain suppression when they are expecting a food reward such as chocolate. Thus, when a person anticipates a reward such as a delicious dinner, the body releases endorphins, activating the mu receptors along the descending pain-control pathway and controlling pain signals as they enter the central nervous system.

A brain region called the nucleus accumbens plays a critical role in both signaling reward and controlling pain. Inactivating this region, which contains mu receptors, prevents animals from experiencing pleasure from either recreational drugs or natural rewards such as food and sex. What is more, injecting rewarding substances into this region can suppress pain responses.

The ability of an imminent prize to suppress pain can influence decision making in situations in which reward seeking and escape from pain are in conflict. An athlete, for example, may face a choice between giving in to physical discomfort and enduring it in hopes of winning a race or a game. A person with a painful blister on his foot might have to choose between resting the injury and going out for pizza and a movie. Such decisions depend on a cost-benefit analysis inside the brain. How painful is the injury, and how much do you expect to enjoy the victory, movie or pizza? These expectations influence your decisions, in part through the pain-control circuit.

If you are a highly motivated athlete

LISE GAGNE *iStockphoto (woman)*; ELKE DENNIS *iStockphoto (cheesecake)*

or you expect the pizza or movie to be extremely good, your expectation will—through the release of endorphins and their stimulation of mu receptors—not only enhance the predicted enjoyment of the victory, food or film but also suppress pain. The overall effect biases you toward tolerating the pain to reach your goal or reward. In addition, you will actually *feel* less pain as you compete or head to town.

Similarly, rats that anticipate chocolate subconsciously "decide" to bear the pain of a hot plate to get the chocolate, both because they expect it to taste delicious and because that expectation alone reduces their pain. Such a resolution of pain-reward conflicts may have survival value. Animals often must endure pain to fight off a competitor for food or for a desirable mate.

The analgesic properties of anticipated rewards are consistent with the placebo effect. If relief of pain is rewarding, then a placebo pill is a sign of a forthcoming reward, leading to pain suppression. Thus, the expectation of relief becomes a self-fulfilling prophesy. Conversely, predicting pain has the opposite effect, amplifying activity in the pain transmission pathway and leading to greater pain perception.

Victims of traffic-related whiplash injuries who expected to return to work recovered faster than those who were less optimistic.

Positive expectations for healing from painful injuries can lead to faster actual recovery from those wounds. In 2009 epidemiologist J. David Cassidy of the University of Toronto and his colleagues reported that among 2,335 Saskatchewan residents who endured traffic-related whiplash injuries, which are a major source of neck pain, those who expected to get well enough to return to their regular job reported recovering 42 percent faster than did those who were less positive. Previous studies have also shown that expectations for recuperating are consistently associated with going back to work among patients who have lower back pain, suggesting that a person's outlook on the future can strongly influence how much pain impinges on his or her life.

Skirting Danger

In addition to expectations of recovery or reward, a sense of danger can squelch pain. Researchers, including psychologists Fred. J. Helmstetter of the University of Wisconsin–Milwaukee and Michael S. Fanselow of U.C.L.A., have

A boxer who badly wants to win may subconsciously "decide" to endure pain to reach his goal. While the athlete is envisioning victory, his anticipation of that reward will relieve his pain.

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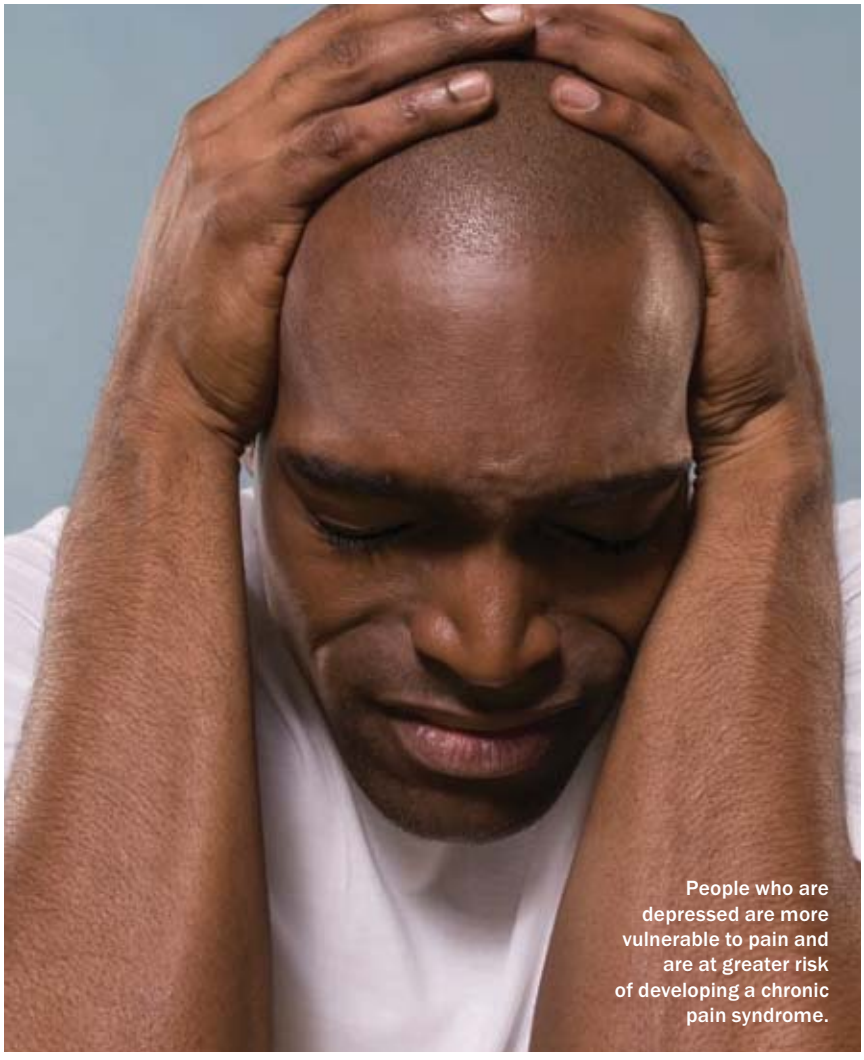
shown that rats do not respond to painful stimuli in the presence of a predator or when the rats are in an environment that provokes fear because, say, they had previously experienced a painful stimulus in it. Naloxone blocks this analgesic effect of fear in rats, indicating that the presence of imminent danger suppresses the experience of pain through release of an endogenous opioid.

People will often feel no pain during or immediately after severe trauma—say, a traffic accident or incident on a battlefield or during an athletic contest. Situations that produce acute tissue injury may signify an ongoing hazard and thus unleash fear or acute stress in humans and animals. The resulting suppression of pain may enable a person or animal to get to safety before being hobbled by agony.

Although acute stress can suppress pain, if stress persists and becomes chronic, pain usually intensifies. A bad mood may also increase pain. People who suffer from depression, for instance, may be more vulnerable to or less tolerant of pain. A 2007 study of 131,500 Canadians showed that among chronic pain patients, 11.3 percent had major depressive disorder as compared with just 5.3 percent of individuals who did not experience chronic pain. Being in pain may be depressing, and depression itself is also thought to affect pain perception. Neurochemical changes associated with depression—such as the depletion of the neurotransmitters serotonin and norepinephrine—may reduce normal inhibition or increase facilitation within the descending pain pathway.



People who are depressed are more vulnerable to pain and are at greater risk of developing a chronic pain syndrome.

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Catastrophizing, or interpreting pain as unbearable and likely to worsen, tends to intensify pain. People who catastrophize feel greater discomfort after surgery.

In addition, catastrophizing, or interpreting pain as unbearable and likely to worsen, tends to increase the experience of pain. Patients who score high on catastrophizing on a standard questionnaire tend to experience more severe pain after surgery and show more sensitivity to experimentally induced pain than do those who score low on the questionnaire. Catastrophizing may worsen pain by making a person concentrate on it and attach additional emotion to it. In a study published in 2004 rheumatologist Daniel J. Clauw of the University of Michigan at Ann Arbor and his colleagues tested 29 fibromyalgia patients for their tendency to catastrophize and then measured their brain responses to blunt pressure on a thumbnail. They linked pain catastrophizing to increased activity in brain areas related to the anticipation of pain, attention to pain and emotional aspects of pain perception.

Psychological distress of various forms raises a person's risk of developing a pain syndrome. In a study published in 2007 neurobiologist William Maixner of the University of North Carolina at Chapel Hill and his colleagues tracked 244 initially pain-free women for up to three years to see who developed temporomandibular joint disorder, a condition characterized by persistent jaw pain, to determine the traits that foretell its development. They linked being depressed and feeling stressed, for example, with a twofold to threefold rise in the chance of getting the disorder. In earlier work, scientists at the University of Washington tied somatization—a ten-

The **Empathy** Effect

Among the more intriguing psychological effects on pain perception is empathy, a sense of knowing and even sharing the experience of another person. In 2006 a team led by neuroscientist Jeffrey S. Mogil of McGill University showed that mice respond more readily to pain when they see cage mates, but not strangers (of the rodent variety), in pain. In 2008 Mogil, along with McGill pain researchers M. Catherine Bushnell and Marco L. Loggia, reported that empathy similarly heightens pain perception in humans.

The researchers exposed volunteers to a painful heat stimulus before and after showing them a video designed to evoke either empathy or distaste for an actor, depending on the version the viewer watched. When the participants felt the heat a second time, they watched the same actor being exposed to painful and nonpainful stimuli. The viewers who felt empathetic toward the actor rated their own pain as more intense and unpleasant than did those who felt negatively toward the actor (no matter whether or not they perceived the actor to be in pain). In fact, the more a person said he or she identified with the actor, the more pain the individual reported having, supporting the idea that empathy itself alters pain perception.

The scientists theorize that activation of the brain areas associated with vicarious emotional distress from the high empathy video may have boosted the stimulation of the neuronal pathways that govern pain, because physical pain and distress activate similar brain regions. They also speculate that the empathy effect may be greater in more established relationships; it may, for example, help explain why the spouses of chronic pain patients so often say they are also in pain.

—Ingrid Wickelgren, staff editor

dency to report numerous symptoms in excess of that expected from a physical injury—with more than a doubling of the incidence of the disorder and less improvement after five years.

Parting with Pain

Research into the psychology of pain may lead to new ways of helping people overcome or cope with pain caused by injury, medical treatment or disease, whether minor or significant. Already, increased knowledge of the brain circuits that mediate the interaction of reward and pain relief is beginning to provide clues for strategies to dissociate the addictive potential of drugs from their pain-relieving power. The findings may lead to effective painkillers that are significantly less addictive than opiates.

In addition, understanding the powerful effects of mood, expectation and other psychological factors on pain is important for helping friends, patients or loved ones deal with their pain. Telling people in pain about individuals who have done well can often ease their distress and discomfort, whereas informing them of others who have had serious illnesses with similar symptoms will very likely worsen their suffering.

Doctors should be on the lookout for mood-related factors such as depression or chronic stress that might be abetting a patient's pain. They also need to carefully query patients about, or otherwise assess, their expectations regarding their discomfort. If a patient is overly pessimistic, a physician can reassure him or her by providing more accurate information, as I did with the man I treated for the abscess. Ultimately, the new understanding of the effects of mind-set on pain promises to revolutionize our approach to pain treatment. M

(Further Reading)

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I DO NOT FEEL YOUR PAIN

Researchers are unraveling why some people are more sensitive to pain than others. Their efforts could lead to more accurate diagnoses, better pain prevention and safer, more powerful painkillers

BY INGRID WICKELGREN

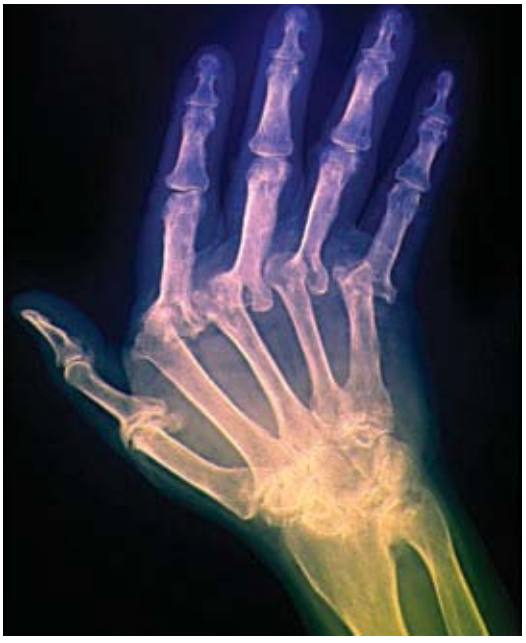
One day as a child Billy Smith (not his real name), a resident of Newfoundland, could not take off his shoe. No amount of twisting or tugging would loosen its grip on his foot. The reason for his struggle eventually surfaced: a nail had pierced the sole and entered Smith's flesh, tightly binding the two. Removing the nail freed the foot, but solving that problem only underscored a bigger one: Smith had not noticed.

Smith is among a tiny cluster of people, fewer than 30 in the world, who harbor a genetic quirk that renders them incapable of perceiving pain. "These humans are completely healthy, of normal intelligence, but don't know what pain is," says clinical geneticist C. Geoffrey Woods, who studied a group of such patients from northern Pakistan. They can sense touch, heat, vibration and their body's position in space. Yet for them, root canals are painless, as are falls, fires and whacks on the head with a baseball bat. One woman with so-called congenital indifference to pain (CIP) delivered a baby without discomfort.

"The children have lots of bruises, cuts and scalds from exploring like kids do, but with no pain to restrict their activities," Woods says. One Pakistani boy entertained others by sticking knives in his arms and leaping out of trees. Before Woods could see the child, he died jumping off a roof. The kids who survive are often deformed and disabled by self-mutilation or broken bones that they failed to notice or refused to rest. When Smith was three, he fractured a bone in his foot but kept walking on it as if nothing had happened.

Although such cases are exceptional, doctors and scientists have known for decades, if not centuries, that human beings at large differ greatly in how sensitive they are to pain. Much of the variation is apparently random. But gender matters. Women tend to hurt more than men do. Ethnicity can also interface with ache; some ethnic groups are more tolerant of discomfort than others are.

In the past few years, as technological advances have eased the deciphering of the human genome, researchers have begun unearthing the genetic roots of these differences. They are also pinpointing social, cultural and psychological factors that play parts in pain sensitivity. The multitude of influences on pain refutes the conventional conception of this sensation as an index of tissue damage. Thus, assessing patients' vulnerability to anguish may be essential to accurately judging the severity of their condition. It is also critical to deciding how to treat their pain. Revealing the molecular causes of individual variation in pain perception is already helping to



Pain does not always parallel injury. The amount of pain a person has may not correspond to the degree of damage displayed on an x-ray.

unravel the biology of agony and providing targets for novel pain medications.

Spectrum of Suffering

Physicians have long noticed wide disparities in the pain tolerance of the people they treat. Among patients with the same condition, pain ratings typically range from "no pain" to "the worst pain imaginable." And although some disorders are more painful than others, the variation in distress among individuals with the same physical malady is far greater than the difference in the discomfort people feel, on average, from one condition to the next. "Two soldiers may be shot in the same nerve," says Stephen G. Waxman, a neurologist at Yale University and the Veterans Administration Connecticut Health Care Center. "One has sensory loss but is otherwise okay; the other has intractable burning pain."

Objective indicators of physical harm often correspond poorly to perceived pain. In one study the amount of inflammation in rheumatoid arthritis patients did not parallel the degree of suffering they reported. In people with osteoarthritis, the tissue damage shown on an x-ray often bears little relationship to the amount of discomfort a patient feels. Even when a scientist carefully controls the intensity of a painful procedure—say, a cold bath or compression of a limb—people significantly differ in how much they say it stings. (On the other hand, an individual's evaluations of agony are surprisingly consistent. If you ask someone to hold an object that becomes increasingly hot to tell you when the pain starts, that moment will be the same—within 0.2 degree Celsius—every time you repeat the procedure, even a few years later.)

What a person says about pain does jibe with changes in the brain if not the body. In a 2003 investigation neurobiologist Robert C. Coghill of the Wake Forest University School of Medicine and his colleagues asked 17 adults to evaluate the pain they felt from a hot metal device touching their lower leg. At the same time, the researchers scanned the volunteers' brains using functional magnetic resonance imaging. Pain-related regions of the brain were more active in the individuals who judged the twinge as more intense than they were in less sensitive subjects, Coghill and his colleagues found.

Verbal pain ratings also predict a person's vulnerability to chronic pain. In 2007 neurobiologist William Maixner of the University of North Carolina at Chapel Hill and his colleagues tested healthy female volunteers for pain sensitivity and psychological functioning. The researchers then tracked them for three years to determine who would acquire temporomandibular joint disorder (TMJD), which causes persistent discomfort in the joints on either side of the ear where the upper and lower jaw meet. Sixteen of 243 women came down with classic TMJD, and the disorder was three times more likely if a woman was very sensitive to pain than if she was relatively insensitive, Maixner says. His

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FAST FACTS

Diversity in Discomfort

1» Human beings at large differ in how sensitive they are to pain. Much of the variation is apparently random. But gender matters. Women tend to hurt more than men do. Ethnicity can also interface with ache; some ethnic groups are more tolerant of discomfort than others are.

2» In the past few years researchers have begun unraveling the genetic roots of these differences. They are also pinpointing social, cultural and psychological components that play parts in pain sensitivity.

3» Assessing patients' vulnerability to anguish may be essential to accurately judging the severity of their condition. It is also critical to deciding how to treat individuals' pain. Revealing the molecular causes of individual variation in pain perception is already providing potential targets for novel pain medications.

team has also associated elevated sensitivity to painful stimuli with other persistent pain syndromes such as fibromyalgia.

Gender Bias

For a decade or longer, researchers have known that women are at greater risk than men for a number of chronic pain conditions, including rheumatoid arthritis, lupus and fibromyalgia. Women are also more sensitive to noxious stimuli: in laboratory experiments the average woman exhibits a lower pain threshold (the point at which she first feels pain) and less pain tolerance (the degree or duration of pain she can stand) than the average man.

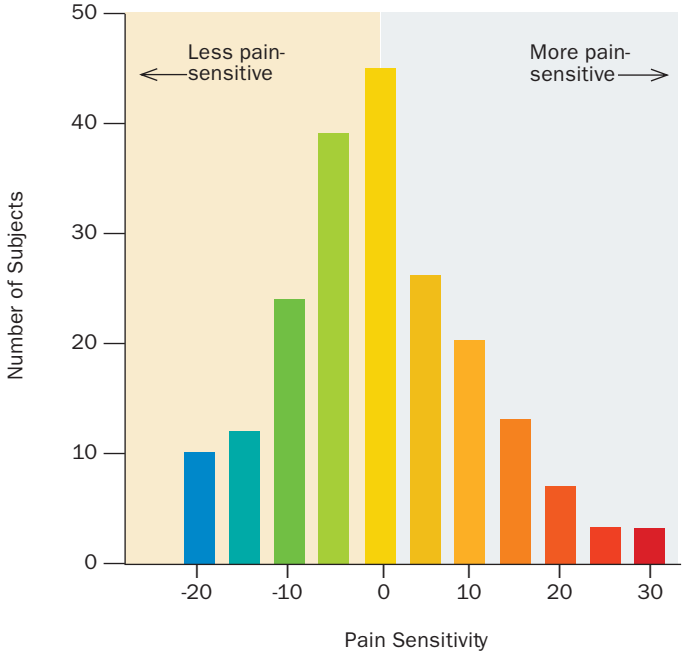
Sex hormones may contribute to this gender difference. Estrogen, for example, can often increase pain, in part by acting at receptors that sit on pain nerves. During her menstrual cycle, a woman perceives more pain after ovulation when progesterone—and to a lesser extent, estrogen—levels are high, consistent with the idea that female hormones intensify pain. In addition, hormone replacement therapy increases pain sensitivity in women, whereas drugs that stymie estrogen's actions provide long-term pain relief in certain situations. (In other circumstances, such as pregnancy, high levels of female hormones are accompanied by diminished pain perception; scientists do not fully understand why.)

Very pain-sensitive women were three times more likely to develop a common persistent pain syndrome than women who were relatively indifferent to pain.

Male and female brains seem to register discomfort differently. In 1999 Coghill's team reported that women perceived the same painful stimulus as more intense than men did and showed more activity in brain regions involved in processing pain. This excess excitement may stem in part from a weaker network for blocking pain. In 2002 psychiatrist Jon-Kar Zubieta, now at the University of Maryland, and his colleagues gave 14 men and 14 women an excruciating injection of saline into their cheeks while scanning their brains, focusing on parts of a "descending" pain-thwarting pathway in which endorphins, the body's natural painkillers, bind to mu opioid receptors to squelch the pain signal after acute injury [see "The Psychology of Pain," by Howard L. Fields, on page 42]. In the males this pain-curbing network was flooded with more endorphins and activity at mu opioid receptors than it was in females—a sign of a more powerful pain-control system.

Other evidence points to weaker pain inhibition in women. Intense or long-lasting pain applied to one part of the body, say, an arm, can suppress pain at another site, such as a tooth. The initial pain is thought to invoke the body's descending pain suppression system. In 2003 neuroscientist Donald D. Price of the University of Florida College of Dentistry and his colleagues showed that this phenomenon was less pronounced in women: in men, dunking one hand into a painfully hot bath diminished the discomfort of a scorching object touching the other hand, but the women felt no such relief.

Emotional and social factors may also contribute to women's enhanced pain sensitivity. For instance, women tend to engage in



Pain perception varies among people. Researchers gave 202 healthy women 16 pain sensitivity tests, subjecting them to heat, pressure and constriction, and reported a range of overall sensitivity scores (*above*).

SOURCE: "GENETIC BASIS FOR INDIVIDUAL VARIATIONS IN PAIN PERCEPTION AND THE DEVELOPMENT OF A CHRONIC PAIN CONDITION," BY LUDA DIATCHENKO ET AL., IN *HUMAN MOLECULAR GENETICS*, VOL. 4, NO. 1; 2005

Pain is not necessarily a sign of weakness. Women's tendency toward discomfort might enable females to better detect threats and thereby protect their offspring.

Women are more sensitive to painful stimuli than men are. Female hormones, weaker pain inhibition in women, and emotional as well as social factors may explain this gender difference.

pain-related catastrophizing—that is, expecting that pain will be awful and unbearable—more than men do. On the other hand, men are typically less willing than women to admit to being in pain because men want to appear tough and strong.

But pain is not necessarily a sign of weakness. In fact, women's tendency toward discomfort might be adaptive. Women are generally more attuned to bodily sensations than men are and have a greater capacity to sense all environmental stimuli, such as light, noise and odor, which may improve their ability to detect threats. Some scientists argue that evolutionary pressures may have promoted such a trait in women to enable them to better protect their offspring.

Not only are women more prone to pain, so are certain ethnicities. African-Americans display greater sensitivity to painful stimuli in the laboratory and report more negative emotional responses to pain than Caucasians do.

Cultural, social and psychological factors probably contribute to this disparity. In a study published in 2007 clinical psychologist Roger B. Fillingim, also at the University of Florida College of Dentistry, and his colleagues demonstrated that a person's ethnic identity—that is, the degree to which a person relates to a minority group's ancestry, language, physiology and culture—strongly affects his or her pain sensitivity. The researchers tested 63 African-Americans, 61 Hispanics and 82 non-Hispanic whites for their susceptibility to pain from a hot object touching their arm, very cold water surrounding a hand, and constriction of blood flow to an arm. Each person also filled out a questionnaire called the Multigroup Ethnic Identity Measure (MEIM).

The researchers found that the range of temperatures and the time that a person was willing to endure pain were lower for members of the two minority groups than they were for whites. And for the African-Americans and Hispanics, but not the whites, the stronger a participant's ethnic identity as judged by the MEIM, the greater his or her sensitivity to any of the types of pain. "Within a minority group the greater your ethnic identity, the greater your pain sensitivity," Fillingim concludes. Cultural factors related to ethnic identity such as religion, education or social expressiveness might bestow specific meanings on pain or suggest coping strategies, he posits. Such shared beliefs and practices may not only influence people's outward expressions of pain; they may also sculpt the biological infrastructure that underlies the experience of pain.

Some of that physiology apparently differs between African-Americans and whites. In 2008 Fillingim and his colleagues tested the natural pain suppression elicited by a strong or prolonged sensation of pain in 29 African-Americans and 28 whites. They induced ischemic pain, depriving an arm muscle of oxygen, by squeezing the arm with a tourniquet; during that procedure, they electrically shocked each person's ankle. The researchers found that ischemic pain produced greater reductions in

SHELBY ROSS *Getty Images (top left)*; GETTY IMAGES *(top right and bottom)*

electrical pain ratings in whites than it did in African-Americans, who may have a weaker inhibitory pathway. "This suggests that African-Americans are less effective at controlling pain than whites," Fillingim says.

Spectacular Mutations

Of course, individuals within a gender or ethnic group also vary in their sensitivity. Genes account for 22 to 60 percent of the variance, according to studies comparing the correspondence in this trait between fraternal twins, who share about half of their genes, with that between identical twins, who have virtually the same DNA.

In rare cases, such as those with a congenital indifference to pain, a single gene has a huge effect. Smith and others like him have a mutation in a gene for a tiny molecular gate, or channel, that sits on the endings of nerves that sense pain. The channel ordinarily serves as an amplifier of neural signals and appears to be necessary for all types of pain perception. In patients with the mutation, the channel does not work, knocking out pain perception. "This spectacular observation seals the case, at least in the extreme, that genetics can have profound effects on sensitivity to pain," says Stanford University anesthesiologist David Clark.

Other mutations in the same channel protein make its gate flip open more readily and stay open too long, turning up the amplifier instead of knocking it out. This molecular mishap results in the flip side of Smith's perilous indifference to pain: an existence infused with agony. Patients experience mild warmth as searing or scalding heat. They liken slipping on socks to pouring hot lava on their feet, Waxman says. One teenager's pain gets so severe that he requires anesthesia in an intensive care unit [see "The Pain Gate," by David Dobbs; Scientific American Mind, April/May 2007].

Subtler genetic tuning of this channel could underlie more ordinary variation in pain sensitivity. Woods has unpublished data fingering a relatively uncommon change in a single base pair that makes the channel more responsive and its bearers feel a moderate amount of additional pain, about the level that could be countered by codeine.

Inherited Ache

Common variants of genes for other proteins, including enzymes, appear to underlie a hardiness to hurt, or the opposite. The enzyme catecholamine-O-methyltransferase (COMT) breaks down the stress hormones adrenaline and noradrenaline (also known as epinephrine and norepinephrine) as well as dopamine, a brain chemical involved in reward and mood. If this enzyme is scarce or not working properly, stress hormone and dopamine levels rise, and that chemical bounty apparently intensifies pain. Fibromyalgia patients and people with facial pain have higher levels of these chemicals. People who are disposed to pain such as females or chronic pain patients also often have relatively sluggish COMT.

Lethargic COMT can result from an alteration in the gene for the enzyme, leading to a threefold to fourfold reduction in its function. In a study published in 2003 Zubieta and his colleagues found that people who had at least one genetic blueprint for the less active enzyme were more sensitive than those with only active COMT to pain from intramuscular injections of saline, requiring less saline to reach the same level of agony.

STEWART TILGER *Getty Images*

"At the end of the day, there will be scores to hundreds of genes related to explaining individual differences in pain," predicts one behavioral geneticist.

Responses of patients to opioid painkillers vary widely. Some of this variation may stem from differences in the gene for the receptor in the body at which these drugs act.

In recent years Maixner, geneticist Luda Diatchenko, also at the University of North Carolina, and their colleagues linked two other versions of the same gene, along with the one Zubieta studied, with distinct levels of pain sensitivity—low, average and high—as well as with vulnerability to chronic pain. (Zubieta evaluated the "average" version, for the enzyme with lower activity.) The researchers analyzed the gene in 202 healthy women, whom they also tested for sensitivity to 16 types of painful stimuli and followed for three years to determine which ones developed TMJD. Compared with the other versions of the gene, the variant conferring low pain sensitivity gives rise to vastly greater quantities of COMT and lowers a woman's risk for TMJD more than twofold.

These COMT alternatives account for 11 percent of the variability in human pain perception, the largest contributor to pain sensitivity people have found so far, Diatchenko says. COMT type is a better predictor of the risk of developing a chronic pain condition than cholesterol level is for cardiovascular disease risk, Maixner adds.

The link between COMT and pain turns out to involve intermediaries called beta-adrenergic receptors that sit on pain-sensitive nerve endings. Adrenaline stimulates these receptors, whose activation (by drugs) can result in an agonizing arthritislike syndrome. Variation in the genes for these receptors, too, can shape pain perception. Maixner's group has nabbed one version of the gene for the beta-adrenergic 2 receptor, which is especially responsive to epinephrine and thereby sensitizes a person to pain.

Diversity in pain sensitivity may also arise from different forms of the mu opioid receptor, which also influence responses to opioid drugs. Opioids such as morphine and the body's endogenous painkillers exert their pain-suppressing effects by acting on this receptor. Responses of patients to opioid painkillers vary widely. The lowest effective dose may be five to 10 times higher for some patients than for others, and in 25 percent of patients morphine is ineffective or causes intolerable side effects.

In 2009 Diatchenko and her colleagues looked at the mu opioid receptor gene in 196 females who were also scored for their sensitivity to a battery of painful stimuli, including those that were hot, piercing and squeezing. After analyzing the gene at 25 places at which a chemical unit tends to vary between individuals—so-called single nucleotide polymorphisms (SNPs)—the researchers found one site associated with pain sensitivity. The rarer version of this SNP, carried by 6 percent of the population, seemed to make a person pain-prone and relatively unresponsive to opioid medication; its more common counterpart, on the other hand, conferred high pain tolerance and a good morphine response.

Other genetic differences may also impinge on a person's response to opiates. Certain human enzymes metabolize medications, and the results of their actions may be required to make the drugs effective and nontoxic. For example, a liver enzyme known as CYP2D6 converts codeine into morphine, the substance that relieves pain. In 7 to 10 percent of Caucasians, however, codeine does not work, because these individuals' CYP2D6 enzyme cannot accomplish the conversion. On the other hand, 1 to 7 percent of whites have multiple copies of the same gene. These individuals break down codeine extremely quickly, making even low doses of the drug potentially toxic. In one 62-year-old man with this gene duplication, a small dose of codeine nearly killed him, according to a report from Geneva University Hospital in Switzerland.

The genes nabbed so far probably represent just a tiny fraction of the body's Lilliputian conspirators in creating or modulating pain. "At the end of the day, there will be scores to hundreds of genes related to explaining individual differences in pain," predicts behavioral geneticist Jeffrey S. Mogil of McGill University.

Tailoring Treatments

Careful assessment of a patient's pain sensitivity could be invaluable for preventing and treating pain. Pain-sensitive patients are, for example, likely to experience a lot of discomfort after surgery and thus may require a higher-than-average dose of a painkiller. "Even in people who had identical surgeries, there can easily be a severalfold difference in the amount of pain reliever a person will need during recovery," Clark says.

An awareness of such differences may also help doctors better assess the severity of a person's illness. Low pain sensitivity might, for example, mask the true seriousness of a patient's condition. In contrast, an unusually strong reaction to a painful event might exaggerate the degree of physical injury it caused.

AJ PHOTOS/HOP AMERICAIN/PHOTO RESEARCHERS, INC.

Evaluating the pain tolerance of healthy patients may help doctors identify who is most vulnerable to developing persistent pain syndromes and thus who might want to forgo elective surgeries or take preventive analgesics after accidents or trauma. Genetic tests may further clarify a patient's risk. "Combining a couple of these genes together could give us good predictive value for who is likely to develop several persistent pain syndromes," Maixner says.

Testing people for variations in the mu opioid receptor or metabolic enzymes might further reveal who will respond well to opioids and at what dose and who might benefit from alternative therapies. Responses to future generations of analgesics might also depend on a patient's genetic makeup. "It's critical to understand the impact of genetics on the treatment of a patient," Clark says.

Unearthing genes involved in pain perception, or lack thereof, can also pave the way toward new therapies. Pharmaceutical and biotech scientists, including those at Xenon Pharmaceuticals in British Columbia, are trying to discover and build molecules that silence the sodium channel that is out of order in congenital insensitivity to pain. "It looks very hopeful that people will have a new generation of painkillers" that target this molecule, Woods says.

Blocking beta-adrenergic receptors may help treat pain conditions stemming from either low COMT activity or high adrenaline levels, or both. In 2007 Maixner's team found that inhibiting beta-adrenergic receptors in rats that had poor COMT function prevented the animals from showing signs of heightened pain sensitivity. In a study published in 2009 Maixner, along with neuroscientist Kathleen C. Light of the University of Utah and colleagues, found that propranolol, which treats high blood pressure by blocking beta-adrenergic receptors, decreased pain in 10 fibromyalgia and 10 TMJD patients as compared with a dummy medication.

Even without genetic tests, doctors may one day base their prognosis and treatments on a person's gender, ethnicity and individual psychology. Some genetic differences seem to be more common among certain genders or races, in accordance with group differences in pain sensitivity. Unpublished work by Fillingim and his colleagues, for example, indicates that a form of the mu opioid receptor associated with stronger natural pain control is far less frequent in African-Americans than it is in whites.

The science of pain peculiarities also helps all of us to gain a better appreciation of pain in those around us. We cannot assume that another person's pain is inconsequential even if the injury looks unimpressive or would not be painful to us. Indeed, the pain perceived, almost by definition, exaggerates or minimizes the damage inflicted, given that pain stems from biological quirks particular to the sensation itself, along with cultural, social and psychological influences.

Another person may be in a lot of pain even if her wound appears minor.

Of course, extreme cases of pain indifference put the survival value of our aches in stark relief. Despite the unpleasantness of pain and the commercial quest for ever more powerful analgesics, humanity cannot afford to wipe out pain the way it might strive to end cancer or heart disease. "We might joke that we wish we felt no pain, but that would be terrible—and is terrible for those who can't experience pain," Clark says. Aside from their physical injuries, people like Smith must endure a dollop of emotional isolation resulting from their inability to experience a virtually universal sensation. They keep quiet about this void. When they fall, they pretend that it hurts, because they want to be normal. M

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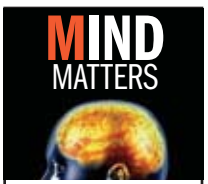
Why Don't Babies Talk Like Adults?

Kids go from goo-goo to garrulous one step at a time

BY JOSHUA HARTSHORNE



This article was adapted from Mind Matters, www.ScientificAmerican.com/MindMatters, a column edited by Gareth Cook, a Pulitzer Prize–winning journalist at the *Boston Globe*, and Jonah Lehrer, the science writer behind the blog The Frontal Cortex, http://scienceblogs.com/cortex

The setting: a nursery. A baby speaks directly to the camera: "Look at this. I'm a free man. I go anywhere I want now." He describes his stock-buying activities, but then his phone interrupts. "Relentless! Hang on a second." He answers his phone. "Hey, girl, can I hit you back?"

This E*Trade commercial is only the latest proof of what comedians and movie directors have known for years: few things are as funny as a baby who talks like an adult because, as everyone knows, babies can't do that. This comedic law obscures an important question: Why don't young children express themselves articulately?

By attempting to answer that question, researchers are uncovering clues about brain development and the mysterious process of learning a language. Recent work supports the seemingly counterintuitive idea that the way children learn to talk—in baby steps—remains the same no matter what age they are when they start to learn a language. In other words, a baby's degree of mental development has very little to do with the fact that he or she does not speak in complete sentences.

FAST FACTS

Baby Steps

1» As young children learn to talk, they progress through stages of imperfect grammar, such as speaking in one-word sentences or dropping articles and word endings ("Mommy get bowl").

2» Scientists have long questioned whether these stages exist because a toddler's brains cannot handle complex grammar or whether they are necessary stepping-stones in language development at any age.

3» By studying international adoptees of varying ages, researchers found evidence that the stages of language usage are essential and not dependent on mental development.

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Having a **more mature brain** did not help the adoptees avoid the toddler-talk stage.

International adoptees offer researchers a chance to study language learning in kids with varying degrees of mental development.

Many people assume children learn to talk by copying what they hear. In other words, babies listen to the words adults use, and the situations in which they use them, and imitate accordingly. Behaviorism, the scientific approach that dominated the American study of cognition for the first half of the 20th century, made exactly this argument.

This "copycat" theory cannot explain why toddlers are not as fluent as adults, however. After all, when was the last time you heard literate adults express themselves in one-word sentences ("bottle," "doggie") or in short phrases such as "Mommy open box"? Of course, it is easy to show that a copycat theory of language acquisition cannot explain these strange patterns in child speech. Actually explaining one-word sentences is much harder. Over the past half a century scientists have settled on two reasonable possibilities.

First, the "mental development hypothesis" states that one-year-olds speak in baby talk because their immature brains cannot handle adult speech. Children do not learn to walk until their body is ready; likewise, they do not speak multiword sentences or use word endings and function words ("Mommy opened the boxes") before their brain is capable of doing so.

Beyond the Copycat Stage

The second theory, the "stages of language hypothesis," states that the incremental-step progress in child speech is a necessary process in language development. A basketball player cannot perfect his or her jump shot before learning to both jump and shoot, and children, similarly, learn to add and then to multiply—never in the reverse order.

In language learning there is also evidence of such necessary movements toward fluency. For instance, in a 1997 review article cognitive scientists Elizabeth Bates of the University of California, San Diego, and Judith C. Goodman of the University of Missouri–Columbia found that studies of young children consistently show that kids do not usually begin speaking in two-word sentences until after they have learned a certain number of words. Until they have crossed that linguistic threshold, the word-combination process does not kick in.

The difference between these theories boils down to this: under the mental development hypothesis, patterns in language learning should depend on a child's level of cognitive development when he or she starts learning a language. Under the stages of language hypothesis, however, learning patterns should not depend on mental development. This prediction is difficult to test experimentally because most children learn language at around the same age—and thus at roughly similar stages of cognitive development.

In 2007 researchers at Harvard University found an ingenious way around this problem. More than 20,000 internationally adopted children enter the U.S. every year. Many of them are no longer exposed to their birth language after arrival, and they must learn English more or less in the same way in-

(The Author)

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The 1989 movie *Look Who's Talking* capitalized on the fact that most of us find the idea of a talking baby hilarious—because we all intuitively know that kids learn language step by step.

fants do—that is, by listening and by trial and error. International adoptees do not take classes or use a dictionary when they are learning their new homeland's tongue, and most of them do not have a well-developed first language. All these factors make them an ideal population in which to test these competing hypotheses about how language is learned.

The Adoption Effect

Harvard neuroscientists Jesse Snedeker, Joy Geren and Carissa L. Shafto studied the language development of 27 children adopted from China between the ages of two and five years. These children began learning English at an older age than U.S. natives and therefore had more mature brains to bring to bear on the task. Even so, just as with American-born infants, their first English sentences consisted of single words and were largely bereft of function words, word endings and verbs. The youngsters then went through the same hallmark language stages as typical American-born children, albeit at a faster clip. The adoptees and native children started combining words in sentences when their vocabulary reached the same size, further suggesting that what matters is not how old you are or how mature your brain is but the number of words you know.

This finding—that having a more mature brain did not help the adoptees avoid the toddler-talk stage—suggests that babies speak in baby talk not because they have a baby brain but because they only just got started learning and need time to accrue sufficient vocabulary to be able to expand their conversations. Before long, the one-word stage will give way to the two-word stage, and so on. Learning how to chat like an adult is a gradual process.

But this potential answer also raises an even older and more difficult question. Adult immigrants who learn a second language rarely achieve the same proficiency in that language as does the average child raised as a native speaker. Researchers have long suspected there is a "critical period" for language development after which it is unlikely to proceed with full success to fluency, yet we are still far from understanding this critical period. Nobody knows exactly when in a child's life it occurs or why it ends—and some experts question its existence entirely.

Paradoxically, although Snedeker, Geren and Shafto may have explained why there are no talking babies—a prospect so absurd it makes us laugh if we see it in commercials or movies—we still need to explain how babies become eloquent adults. M

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A New Vision for Teaching Science

Recent studies from neuroscience and psychology suggest ways to improve science education in the U.S.

By J. Randy McGinnis and Deborah Roberts-Harris

We face a real crisis in science education in America. Representative Bart Gordon of Tennessee, chair of the House Committee on Science and Technology, has warned that countries such as China and India will trample the U.S. economy in the near future without major improvements in teaching. Indeed, our schools are falling behind. In the 2006 Program for International Student Assessment (PISA)—a respected measure of achievement around the globe—the average science score of U.S. 15-year-olds dropped below that of teens in 28 out of 57 participating countries. (In math, U.S. students fared even worse, lagging behind their peers in 34 nations.)

Despite decades of reform, America has made only modest gains in the science classroom, particularly in high schools. Two recent reports from the National Research Council (NRC), however, offer novel strategies. Entitled *Taking Science to School* and *Ready, Set, Science!,* they call for changes in the way science is taught beginning in elementary school. Unlike previous recommendations, the new suggestions reflect recent findings from neuroscience and psychology about how young children think and how they acquire knowledge.

Whereas past reform has aimed primarily at placing the U.S. first among other nations, the latest reports offer a well-defined goal of science proficiency: students must be able to know, use and interpret scientific explanations of the natural world; they must be able to generate and evaluate scientific evidence and explanations; they must be able to understand the nature and development of scientific knowledge; and they must be able to participate meaningfully in scientific activities and discourse.

These four interrelated targets weave a "science as practice" approach, widely endorsed by education researchers. K-8 instruction needs to present science as a dynamic process. Currently most schools package science into two parts: the step-by-step scientific method and a collection of unproblematic facts. As a result, most children hold an absolute view of what they see as "the truth" and believe most knowledge results from direct observations. As these pupils grow, many never realize that science is an exercise in building and revising theories. But all students—not just those who intend to pursue a scientific career—should learn how scientific knowledge is constructed. Basic scientific literacy will be mandatory for anyone hoping to fully participate in our future society as a responsible adult.

Rethinking How Children Learn

A long-standing question in education concerns what students can learn at various ages. Until recently, educators and psychologists assumed that age alone determined this learning capacity. Abstract thinking, they believed, took considerable time to develop, and so with younger classes, teachers often focused on memorization over understanding. This limited view of children's cognitive abilities grew from a 1958 study, *The Growth of Logical Thinking from Childhood to Adolescence.* In it, Jean Piaget, the father of child psychology, and his colleague Bärbel Inhelder asserted that no form of instruction could hasten the onset—typically at age 12—of logical thinking.

CHIP SIMONS Getty Images

Educators and psychologists have long underestimated children's cognitive abilities. New research shows that even very young kids are capable of logical thinking and scientific reasoning under certain circumstances.

LINDA LEWIS Getty Images (*girl with ladybug*); GETTY IMAGES (*boy looking at test tube*)

More contemporary research, though, shows that children do possess the capacity for scientific reasoning long before age 12. In a 2004 study of third and fourth graders, David Klahr of Carnegie Mellon University and Milena K. Nigam of the University of Pittsburgh demonstrated that, given the appropriate instruction, young children can grasp abstract concepts such as controlled experimental design. Even earlier, from infancy to preschool, children observe and interact with the world around them: they start to understand how objects move and how creatures live; they realize that different people hold different ideas, and so forth. In certain situations, they can differentiate cause and effect, design experiments, and make use of models and symbols.

After children enter elementary school, their skills advance rapidly. Educators previously credited this leap solely to cognitive development. With greater maturity, children possess longer attention spans, greater self-discipline and faster processing speeds. But maturity is not the only force driving learning. Encouragingly, researchers have found that progress is largely contingent on a child's prior learning experiences. The quality of these educational experiences is the key, not the child's age or developmental stage or how early or late he or she starts school.

These findings affirm what learning theorists have long recognized: students master an idea more readily when they have some foundation of knowledge to build on. Researchers are now actively pursuing so-called learning progressions, the conceptual paths students take as they move from a simplistic to a sophisticated view of some subject. Seminal studies by Vanderbilt University professors of science education Richard Lehrer and Leona Schauble, for example, have examined how students develop an understanding of topics such as density, growth and motion using model-based reasoning. Working

FAST FACTS

The Science of Teaching

1» Two recent reports from the National Research Council call for significant changes in the way science is taught in elementary school. Unlike previous recommendations, the new suggestions reflect recent findings about how young children think and how they acquire knowledge.

2» Research shows that children learn best when they regularly revisit topics, moving from basic to sophisticated views. In keeping with this knowledge, education experts advocate curricula in which students deepen their understanding of a topic—and hone their abilities to practice science—across many grades.

3» The most effective teaching expands both the knowledge and the skills needed to engage with science authentically—that is, in a manner akin to how scientists work. To practice science in the classroom calls for problem- and project-based lessons, as well as considerable social interaction. As is the case among scientists, argumentation and discourse help students to refine one another's ideas and to articulate their own.

with children across several elementary school grades, they have observed a steady advancement in the pupils' ability to create models from straightforward depictions to more symbolic and mathematically valuable representations.

Given a solid foundation, teachers can easily build out extensions. When a child already has some idea about animals, for example, it is not difficult to introduce, say, the platypus. Other conceptual additions come only as children gain knowledge in other areas and in their ability to use mathematical representations, symbols and models. Some new ideas are so counterintuitive that students need to shift their entire way of thinking. Pupils must also develop a sense of metacognition and notice when their understanding varies from evidence generated in the classroom or from scientific theory. But the pattern remains the same: the most successful route to mastery in any subject follows a spiral path, in which students regularly revisit and refine their conceptual underpinnings.

More Effective Teaching

Based on these insights, the NRC reports advocate a science curriculum that revisits topics at increasing levels of sophistication. Students deepen their understanding of a topic—and hone their science abilities—across many grades. The authors criticize current standards on several fronts: the curricula lay out too many discrete pieces of knowledge, with no hierarchy or meaningful sequencing; they separate skills from content; and they overemphasize methods. Studies such as the PISA that compare U.S. curricula with those of other countries underscore the point: countries that teach fewer topics overall produce higher scores. To that end, the new vision proposes that U.S. science educators focus on core topics, such as atomic-molecular theory, evolutionary theory, cell theory, and force and motion.

In *Atlas of Science Literacy*, the American Association for the Advancement of Science has attempted to map out what facts should be linked to such core concepts in science, grade by grade, so that students eventually assemble a complete, detailed view. Curriculum writers are using these maps to develop lesson plans that advance students through carefully designed learning experiences, not simply the passing of time. The most effective learning experiences gradually expand both the knowledge and the skills needed to engage with science authentically—that is, in a manner akin to how scientists do science in the real world.

To actually do science in the classroom like a scientist calls for a wide variety of learning experiences, including problem- and project-based lessons and considerable social interaction. As is the case among scientists, argumentation and discourse help students to challenge and sharpen one another's ideas and to articulate and examine their own. Pupils must learn to speak and write using the specialized language of science, and they must be versed in the use of models and other mathematical tools. Kathleen Metz of the University of California, Berkeley, has demonstrated that even first graders can work in pairs to conceptualize and implement a study of their own design. Interviews showed that nearly all the children could express the aim, method and results of their study. Half of them could also analyze their design and devise ways to improve it.

Because children lack experience, though, they need carefully considered help from teachers to harvest the fruits of their independent investigations.

	Science score
Finland	563
Hong Kong-China	542
Canada	534
Chinese Taipei	532
Estonia	531
Japan	531
New Zealand	530
Australia	527
Netherlands	525
Liechtenstein	522
Korea	522
Slovenia	519
Germany	516
United Kingdom	515
Czech Republic	513
Switzerland	512
Macao-China	511
Austria	511
Belgium	510
Ireland	508
Hungary	504
Sweden	503
Poland	498
Denmark	496
France	495
Croatia	493
Iceland	491
Latvia	490
United States	489
Slovak Republic	488
Spain	488
LIthuania	488
Norway	487
Luxembourg	486
Russian Federation	479
Italy	475
Portugal	474
Greece	473
Israel	454
Chile	438
Serbia	436
Bulgaria	434
Uruguay	428
Turkey	424
Jordan	422
Thailand	421
Romania	418

In the 2006 Program for International Student Assessment (PISA), the average science score of U.S. 15-year-olds trailed that of teens in 28 out of the 57 participating nations (not all are listed here).

(The Authors)

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For most learners, only limited learning results from totally free explorations or, at the other extreme, cookbook-type guidance. Teachers must continuously grasp those critical times to provide instructional scaffolding. This scaffolding—which can take many forms, including oral feedback, supplementary handouts and software tools—enables students to reach what would not be possible through cognitive development alone.

It is essential for teachers to connect what pupils already know and guide them toward becoming science-informed citizens. Teachers should recognize each student's prior knowledge, respect the diverse backgrounds from which they come and, even more important, employ that information to make their class's scientific practices richer and more meaningful. To have the best chance at assisting children where and when they need it, the reports advise teachers to implant assessment into the learning process. By constantly probing their pupils' understandings, teachers are being afforded the best opportunities to connect with them.

Despite its promise, the new vision presents significant challenges. The cost of providing in-service teachers' professional development and redesigning classroom or laboratory space—as well as the ex-

Scenes from the Classroom

Deborah Roberts-Harris, a public school teacher in Phoenix, Ariz., designed a lesson plan incorporating many of the current recommendations for reforming science education to study ecosystems with her fifth-grade class. Here are some highlights.

SEAN JUSTICE Getty Images

"Science as Practice" Approach

Using two-liter plastic bottles, the children constructed terrariums, planting grass and alfalfa atop layers of gravel, sand and potting soil. Then they determined key aspects of the investigation, exercising and developing problem-solving skills. For instance, the students debated how much water to add, turning to one another and seed packets for guidance. They had some sense of the scientific process and decided that everyone should measure their additions. They agreed on one cup, which was more than the soil could easily absorb, but Roberts-Harris used it as an opportunity to discuss drainage, as well as to conduct further experiments about groundwater. Later, the children added crickets to their terrariums. They made daily observations and filled a white board with questions. Like a working lab group, the kids consulted one another, brought in library books, shared articles and looked for answers on the computer.

Spiral Curricula

Before designing her lesson plan, Roberts-Harris reviewed her state's and county's K-12 science curricula to see what her class already knew, so the investigation of ecosystems could build on concepts studied in earlier grades. For instance, the students had a rich notion of the sun as our planet's primary energy source. Because their classroom had only skylights for natural light, they decided to move the terrariums to the library, where there were large, sun-filled windows. The investigation into ecosystems also gave Roberts-Harris an opening to seed key ideas about habitats and pollution, which the students would examine in greater detail in middle and high school. For example, the children witnessed that terrariums having filter paper over their openings were thriving habitats of grass, alfalfa and crickets, whereas sealed bottles could not support life. They also built aquariums and learned how critical temperature, chemical balance and light are in a fish's environment.

Encouraging Discourse

Roberts-Harris frequently encouraged class discussions, responding to the youngsters' questions by asking, "What do you think? What do we already know that might help us answer that question? How can we find out?" When the children placed their terrariums on top of their aquariums, for instance, someone asked, "How will the fish get any air, since the terrarium fits snugly on top of it?"

"Fish don't need air like we do," one child said. "When they are in a river, they can't get any air."

"Yes, they do get air," another replied. "Where do you think the bubbles on top of the water come from? And my aquarium at home has a pump to put air into the water."

"Do fish breathe like we do?" Roberts-Harris interjected, focusing the conversation.

Another child answered, "Fish have gills, not lungs, and they can breathe underwater and they die in the air."

"How do gills work?" Roberts-Harris pressed on. "What do the fish need to survive?"

The dialogue continued, and after consulting the computer, the children were satisfied to discover that fish absorb from water some of the same gases we take in from the air.

—J.R.M. and D.R.-H.

Teachers play a critical role in supporting children as they investigate scientific topics. Given the correct instructional scaffolding, students can grasp more sophisticated concepts than would be possible on their own.

pense of additional materials—may prohibit disadvantaged regions from adopting the proposed changes. Also, although no one disputes the value of developing spiral curricula that focus on fewer core topics that are revisited over time, selecting those topics could prove contentious. It will also take serious coordination to implement such a curriculum: Will teachers have enough opportunity to interact with peers across schools and grades to ensure a seamless progression through any one topic? Will adequate class time be available for science instruction, given the strictures of the No Child Left Behind legislation? Are there enough science teachers available? In the proposed curriculum, science teachers play a central role—one that demands extensive planning and improvisatory skills, as well as vast knowledge, not only about science but also about the cultures and environments in which their students live.

Finally, the proposed curricula can succeed only if students are motivated to learn. If they lack the inherent interest or the support needed to sustain their interest, they will not reap the benefits of the newly designed experiences. According to feedback from teachers in the field, this issue may prove most difficult to surmount. Even though scientific thought processes are natural to most children, the NRC documents report that students often have negative attitudes toward science, based on poor academic experiences or inaccurate views of how it works. In some cases, pupils simply do not want to expend their mental energy on a subject they see as irrelevant to their life. But that notion could not be farther from the truth. As we face such dire threats as flu pandemic and global climate change, our very future hinges on how well the next generation learns science. M

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Environment and Weight

Researchers point to external causes of—and fixes for—the obesity pandemic

BY HAL ARKOWITZ AND SCOTT O. LILIENFELD

OBESITY is a "global epidemic," according to the World Health Organization. Two thirds of American adults and one third of school-age children are either overweight or obese (defined as extremely overweight). These proportions have been rising steeply, report the latest surveys. From 1960 to 2002 the population of overweight and obese adults increased by roughly 50 percent, and the corresponding increase for children was 300 percent. Compounding the problem, obesity rates in other countries are rapidly approaching those in the U.S.

What is causing this pandemic, and what can we do about it? Researchers have provided some tentative answers that fly in the face of commonly held beliefs. They suggest that the increase in obesity may be a result of environmental changes that tempt us into unhealthy habits and tend to overwhelm our psychological defenses against consuming too much and succumbing to fattening fare. In fact, environmental cues can exacerbate any innate tendency to use food as a balm for jittery nerves or sadness. Thus, many health experts advocate legislation—for instance, a tax on junk food—that promotes healthy eating. Others are trying to help individuals change their immediate eating milieu in ways that discourage overeating.

Obesity Myths

Many people, including health care professionals, believe that obesity can be attributed simply to a lack of self-control or willpower. It is true that obese people are often unable to adequately control their eating. But lack of self-control is merely a description, not an explanation. What remains to be explained is *why* they cannot exercise self-control.

Nevertheless, doctors routinely tell their obese patients to restrict what they eat. Diet books, articles in health magazines and on Web sites, and commercial weight-loss programs also encourage people to eat less and exercise more. Unfortunately, approaches based on self-control do not seem to work very well. As sales of weight-loss books have climbed recently—from 3.6 million copies in 2005 to 4.8 million in 2007—so has obesity. Further, two thirds of those who slim down in weight-loss programs regain their weight within a year, and almost all have put back the pounds within five years.

Other explanations of the increase in obesity are based on genes and psychological factors. It is true that many people are predisposed to gain weight because of their genetic makeup. But genetic factors cannot account for the sharp *increase* in the prevalence of obesity in society. The genes within a population relevant to weight do not change appreciably in 50 years. Some psychological factors may also play a role in obesity, including impulsivity, anxiety and a tendency among some people to eat during negative emotional states. But here, too, there is no reason to believe that these characteristics have become more prevalent in recent decades. Therefore, genetic and psychological factors cannot account for the rise in obesity.

Toxic Environment

Results of a large number of studies support the conclusion that environmen-

> The increase in obesity may be a result of environmental changes that tempt us into **unhealthy habits**.

COURTESY OF HAL ARKOWITZ (*top*); COURTESY OF SCOTT O. LILIENFELD (*bottom*); FRED R. CONRAD *New York Times*/*Redux Pictures* (*drive-through*)

tal cues exert a powerful influence on our eating behaviors. And unlike biological factors, our nutritional environment *has* changed radically in the past 50 years. In various publications, Yale University psychologist Kelly D. Brownell has used the term "toxic environment" to refer to this new dietary atmosphere, which is characterized by pervasive exposure to food that is energy-dense, heavily marketed, cheap and widely accessible, accompanied by a lack of physical activity.

A 1995 report by the Institute of Medicine set the stage for future work when it concluded that the root of the obesity problem "must lie in the powerful social and cultural forces that promote an energy-rich diet and a sedentary lifestyle." More recent studies have borne out this statement. These forces, Brownell postulates, include the explosion of fast food outlets, increasingly large restaurant portion sizes, "all you can eat" buffets, the proliferation of mini markets that sell high-calorie snacks and drinks, contracts between schools and fast food and soft drink companies to sell their products in school cafeterias, and widespread powerful food advertising.

Given the importance of the environment on obesity, many researchers, including Brownell, argue that we need new laws and social policies to combat obesity. Brownell's controversial proposals suggest, for example, regulating food advertising aimed at children, prohibiting fast foods and soft drinks in schools, and subsidizing healthy foods.

Taxation is another potentially effective means of reducing consumption of harmful products, as the tobacco tax has demonstrated. Brownell and Thomas Frieden, who now heads the Centers for Disease Control and Prevention, have argued for a tax on one of the biggest contributors to obesity: sugar-sweetened beverages. Recently the U.S. Senate Finance Committee recommended such a tax to help combat obesity. Although major soft drink corporations vehemently oppose such a tax, the proposal is now on the national agenda.

Cornell University researcher Brian Wansink and his colleagues have found that cues in our personal eating environment also exert pressure on our tendencies to overeat [*see box above*]. Based on these findings, they have suggested various ways of altering our environment to influence us to eat less. They advise, for instance, reducing portion sizes, keeping tempting food out of sight, never eating directly out of a package, and asking waiters to remove the chips or bread from the table.

Analyzing the power of environmental influences on obesity can lead to many practical suggestions for lessening their detrimental effects and encouraging lifelong healthy eating. And because obesity is a serious problem that has managed to spread to many corners of the globe, we must explore every possible avenue to reduce its prevalence. **M**

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Send suggestions for column topics to editors@SciAmMind.com

Conspicuous Consumption

Studies show that our surroundings greatly influence how much and what we eat. In his book *Mindless Eating: Why We Eat More Than We Think* (Bantam Dell, 2007), Brian Wansink, professor of consumer behavior and nutritional science at Cornell University, describes the environmental stimuli that numerous investigations have tied to overeating. Here are some of them:

- The larger the amount of food on a plate, the more we eat.
- The bigger the food container, the more we eat.
- When the food we prepare comes in large packages, we prepare and eat more than if the food comes in smaller packages.
- We eat more when the food is visible and conveniently located.
- We eat more when the food has an appealing name (such as Succulent Italian Seafood Filet) than when the same food has an ordinary name (such as Seafood Filet).
- Schoolchildren who live close to fast food outlets have a 5 percent higher obesity rate than do students who attend schools farther away from such stores.
- People who move from less modernized countries to more modernized ones show increased rates of obesity as compared with individuals who stay in their less modernized country.

—H.A. and S.O.L.

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Don't Know Much Biology

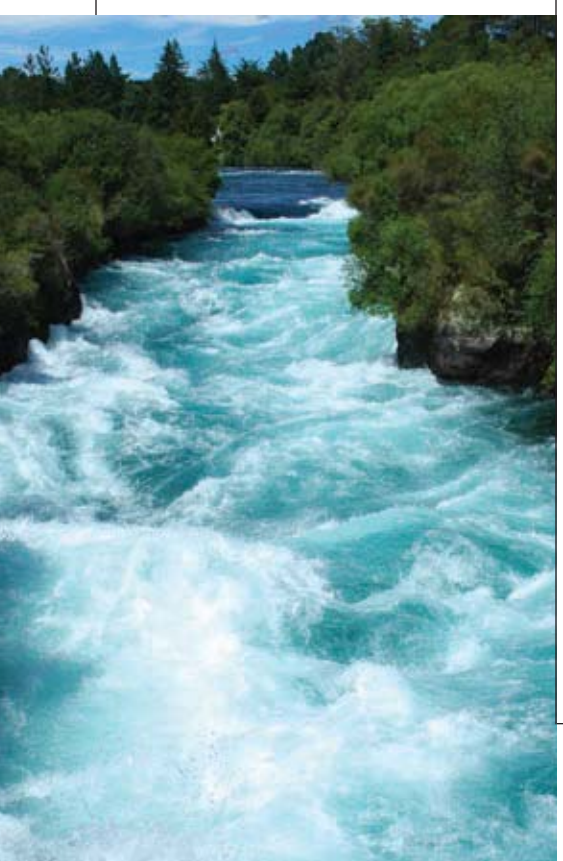
Learning to categorize the living world is surprisingly difficult for the human mind

BY WRAY HERBERT

THINK about what it takes to learn biology. Not textbook biology, the kind you learn in high school with microscopes and dissecting kits. Rather the kind you learn on your own, as a young child encountering the vast and diverse world of living things. How does the human mind link together organisms as varied as hippos and lichen and mosquitoes and rhododendrons? And how do we assemble this diversity into meaningful categories? In short, how do we think about life?

Psychologists are very interested in how the mature mind sorts the living world and where we put ourselves in relation to other life-forms. That is the stuff of philosophy and religion and morality. But how we recognize life—and arrange the living world in our mind—is not as obvious as one would think.

Take something as simple as motion, for example. Many living things move, but so do rivers and clouds and rocket ships. And some living things, such as coral and trees, do not appear to move at all. So it is not just the fact of motion that defines life, but the why and how things move. How does the movement of a bicycle differ from that of a horse? That is a fairly nuanced analysis for an immature mind, and indeed young children find this idea confusing. Kids make a lot of mistakes about what is animated and what is not. Only over time do we outgrow our simple, childish ideas and replace them with a sophisticated view of the natural world.

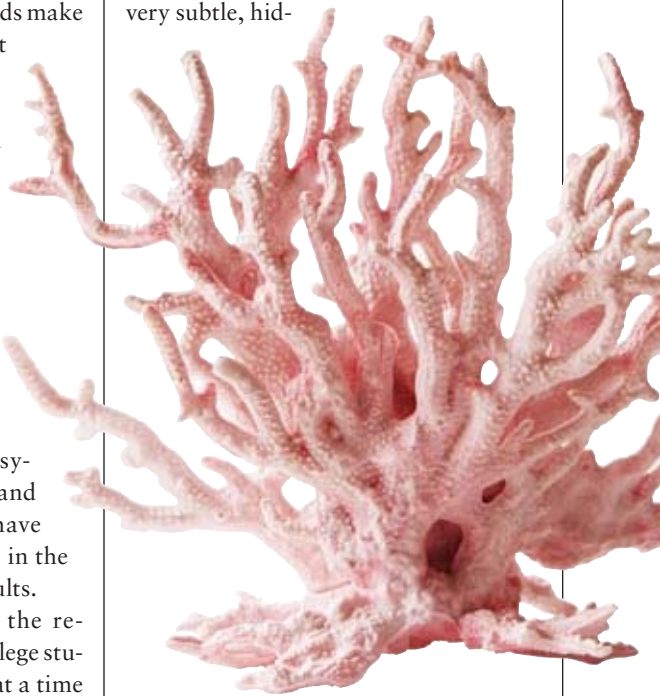


Confused by Motion

Or do we? Do we really discard all our naive thinking as we experience the world and learn about its complexity? University of Pennsylvania psychologists Robert F. Goldberg and Sharon L. Thompson-Schill have been exploring these questions in the laboratory, with intriguing results.

In one recent experiment the researchers showed a group of college students a long list of words, one at a time and very rapidly. Some of the words were the names of plants, others, animals, and still others, nonliving things. The nonliving items were further divided into nonmoving man-made objects such as brooms, nonmoving natural features such as boulders, moving artifacts such as trucks and, finally, moving natural phenomena such as rivers. The idea was to see how quickly and accurately the volunteers used movement and "naturalness" to classify something as living or nonliving. Mistakes and hesitation would be taken as evidence that the primitive ideas of childhood still retain some power.

The scientists were particularly interested in how we think about plants—where our mind tends to put them in the grand scheme of things. Plants are an interesting anomaly because—at least to young children—they do not "do" anything; instead we do things to them, such as climb, water and prune them. If plants move at all, their movement is very subtle, hid-



MATT MENDELSON (*Herbert*); GLOBALP /*Stockphoto* (*bulldog*); BJORN RASMUSSEN /*Stockphoto* (*river*); DOLE08 /*Stockphoto* (*coral*)

den to the casual observer. Not surprisingly, kids often misclassify plants as nonliving.

But how do college students think about plants? Well, it appears that they, too, make mistakes, even with all that formal education. The volunteers in the study were much more hesitant in classifying plants, suggesting that they had to slow down to deliberately overrule

their naive taxonomy. They also made more outright errors than they did when classifying animals. In addition, the students were slower to size up moving things in general as well as nonliving natural things—suggesting that movement and naturalness were the features that stymied them.

Stumbling over Plants

To be fair, these student volunteers were not biology majors. And we all know that kids can slip into college without much in the way of rigorous scientific training. But here is the really interesting part. The psychologists subsequently ran basically the same experiment but recruited biology professors—people who make their living teaching university students about the natural world. Indeed, the volunteers in this second study had been teaching college-level biology for a quarter of a century on average—and at two highly prestigious schools, Yale University and Johns Hopkins University.

And guess what? As reported in the April issue of the journal *Psychological Science*, the professors did better than the undergraduates but not as brilliantly as one might expect of the scientific elite. Even these experts were significantly worse at classifying plants than they were at categorizing animals. That is, even a lifetime of advanced scientific training did not trump the tendency to view plants as artifacts. What's more, the biologists were not much better than undergrads at classifying nonliving things such as clouds and rivers. Goldberg, Thompson-Schill and their colleagues are following up with neuroimaging work to see if they can identify the roots of such naive thinking in the developing brain.

Children may be natural-born taxonomists, but they are not all that good at it. That is because they have a deep-wired urge to see the world as designed and simple—and to be at the center of it all. Apparently that impulse never entirely goes away. M

> Even a lifetime of advanced scientific training **did not trump** the tendency to view plants as artifacts.

For more insights into the quirks of human nature, visit the "We're Only Human. . . " blog and podcasts at www.psychologicalscience.org/onlyhuman

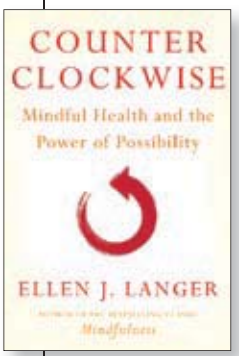
WRAY HERBERT is director of public affairs for the Association for Psychological Science.

(Further Reading)

◆ **Developmental "Roots" in Mature Biological Knowledge.** Robert F. Goldberg and Sharon L. Thompson-Schill in *Psychological Science,* Vol. 20, No. 4, pages 480–487; April 2009.

books

BEYOND THE PLACEBO EFFECT



Counterclockwise: Mindful Health and the Power of Possibility

by Ellen J. Langer. Random House, 2009 ($25)

When she was in her 20s, Harvard University psychologist Ellen J. Langer fainted occasionally, and doctors said she might have epilepsy. She decided to take the matter into her own hands, mentally "catching" herself sooner and sooner when she felt faint, until the fainting disappeared. That empowering experience set the tone for her remarkable 30-year career, much of which she has spent figuring out how to help people take almost miraculous control over their lives.

Her 1989 book *Mindfulness,* summarizing a decade of ingenious experiments, became an instant classic. Now, in her new book, *Counterclockwise,* with more of those experiments under her belt, she presents a more thoughtful and thorough look at the power of mindful thinking: "the simple process of actively drawing distinctions."

Langer says by changing the way we observe and label our experience—specifically, by becoming more aware of the *variability* we often mindlessly ignore—we can improve our health and quite possibly prolong our lives. In a recent study that makes the point, Langer and a Harvard colleague, psychologist Alia Crum, told cleaning personnel in Boston hotels that the considerable exercise they got every day in their job satisfied government guidelines for living an active lifestyle. Their activity levels did not change, but their perspective did, and they soon lost more weight and body fat than control subjects did.

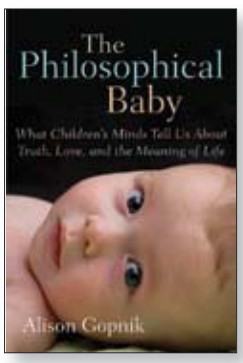
Langer attributes outcomes such as this one to the placebo effect: when people are persuaded to think mindfully about what they are doing, they adopt more positive and empowering beliefs about themselves, and they feel and perform better.

The book's title refers to a study conducted in 1979 in which two small groups of elderly men were housed for a week in quarters simulating the world of 1959. Members of one group were told to imagine themselves living in that time and that "you will feel as well as you did in 1959." The other group was told merely to *talk* about that year and that "you *may* feel as well as you did."

Signs of aging decreased in both groups, with greater gains for the experimental subjects—an effect, perhaps, of the difference in the instructions they received. Subjects in both groups also gained weight (a desirable outcome), with the experimental subjects gaining more. This change makes the study results difficult to interpret, however, because weight gain alone makes elderly people appear more youthful.

That said, *Counterclockwise* succeeds in presenting powerful ideas about largely untapped human abilities, grounded in a body of fiendishly intriguing research. —*Robert Epstein*

INFANT INTELLIGENCE



The Philosophical Baby: What Children's Minds Tell Us about Truth, Love, and the Meaning of Life

by Alison Gopnik. Farrar, Straus and Giroux, 2009 ($25)

Most parents want to believe their children are brilliant. But how much do babies really understand about the world around them? In her provocative new book *The Philosophical Baby,* Alison Gopnik, a developmental psychologist at the University of California, Berkeley, asserts that babies and young children are in some ways "actually smarter, more imaginative, more caring, and even more conscious than adults are."

These claims are bold, but Gopnik backs them up with dozens of empirical studies, many conducted in her own lab. At the heart of her argument is that children have evolved to be the "R&D department of the human species." While adults are kept busy seeking food and avoiding danger, children are free to let their minds wander in the "useful uselessness of immaturity." They can ask questions their parents would not conceive of, occasionally stumbling on solutions no adult could have taught them.

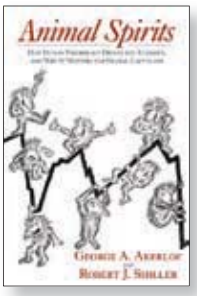
In this way, Gopnik claims, babies behave like little scientists. Toddlers build theories about the people and things around them not just by observation and imitation but also by running "experiments" on their surroundings—experiments their parents might not always appreciate, as they may be messy or disruptive. Comparing young children with researchers is a sug-

GETTY IMAGES (*man in store*)

Neuro-Economic **Boom**

Does sex really persuade us to buy a product? Why do economies slip into depressions? And how much do we let our emotions influence our decision making? A spate of new books tries to answer these and other questions about how we make our choices, why they are sometimes so far off the mark and what their consequences are.

Animal Spirits—How Human Psychology Drives the Economy, and Why It Matters for Global Capitalism (Princeton University Press, 2009) examines the relation between economic fluctuations and psychological forces. Economists George Akerlof and Robert Shiller explore how "animal spirits"—the term coined by economist John Maynard Keynes to describe levels of consumer confidence—lie at the core of such questions as why there is unemployment and why minorities are often particularly poor.



gestive analogy, although one that doesn't seem to capture the scope of childish curiosity.

So what is it like to be a baby? Gopnik ventures a guess. If adult attention is like a spotlight that can be directed at will, baby consciousness is more like a lantern beaming in all directions. She cites the work of psychologist John Hagen of the University of Michigan at Ann Arbor, who found that younger children were better than older children at recalling playing cards they had been instructed to ignore. Adults who want a taste of this kind of open-ended awareness, Gopnik suggests, should try travel or meditation.

It may not seem intuitive that a three-year-old playing with her imaginary friend is "exercising some of the most sophisticated and philosophically profound capacities of human nature," as Gopnik proposes. But even when Gopnik ventures to the limits of what can be inferred from behavioral research—taking on the human predilections for love, imagination and awe—she remains both credible and accessible. In the end one doesn't need to know much about cognitive science to grasp the essence of her argument: if we could only get inside our children's heads, we would learn something deep about ourselves. —*Jascha Hoffman*

DREAMWORKS/THE KOBAL COLLECTION

dvds

MADNESS AND MUSIC

The Soloist

Paramount Pictures.
DVD available Fall 2009

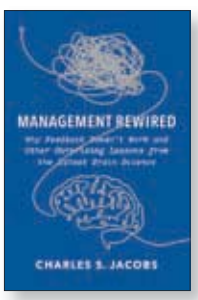
In 2005 *LA Times* columnist Steve Lopez befriended a homeless schizophrenic cellist named Nathaniel Ayers. Lopez often wrote about Ayers for the *Times,* introducing many readers to the reality of schizophrenia and the desperate plight of the Los Angeles slum known as Skid Row. *The Soloist,* based on Lopez's book of the same name, translates Lopez's powerful depiction of mental illness and urban desolation into a moving film.

Lopez (Robert Downey, Jr.) first meets Ayers (Jamie Foxx) in a park where the musician plays Beethoven on a two-stringed violin. Foxx portrays a breathless and rambling schizophrenic. He pounces on the natural pauses in speech, filling them with seemingly random words and riffing on tangents. Adding to Foxx's compelling performance, the film's soundtrack offers the audience a frightening taste of what schizophrenia might feel like. Viewers are tormented by the voices Ayers hears. The most disturbing is that of a woman who speaks in neutral tones about worthlessness and violence.

Foxx's performance rates well with an expert on schizophrenia, Tyrone Cannon, a psychologist at the University of California, Los Angeles, who happens to have met Lopez. "I thought his portrayal of thought disorder was convincing," Cannon says. "His difficulty relating socially was well done."

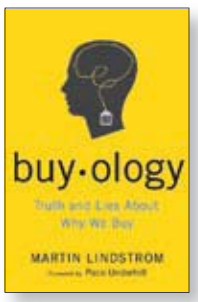
One of the film's recurring themes is friendship as a treatment for schizophrenia, and the sentiment is not off base, Cannon confirms. "Even as a neuroscientist," he says, "I wouldn't minimize the importance of friendship."

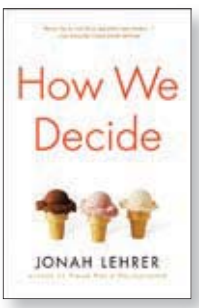
—*Robert Goodier*



In ***Management Rewired—Why Feedback Doesn't Work and Other Surprising Lessons from the Latest Brain Science*** (Portfolio, 2009), business consultant Charles S. Jacobs reveals that, contrary to popular belief, decisions in the business world are never purely based on facts and logic. Looking at new research, the book also shows that rewards and punishments are not as effective as many managers think and that relying on pay raises and bonuses to improve performance is likely to backfire.

In ***Buyology—Truth and Lies about Why We Buy*** (Broadway, 2008), best-selling author Martin Lindstrom reveals the findings of the world's largest neuromarketing study that examined the brains of 2,000 consumers. The book debunks myths about shoppers' behavior by showing, for example, that dire health warnings on cigarette packs actually make us want to smoke more.





But how does our brain make those calls? In ***How We Decide*** (Houghton Mifflin, 2009), *Scientific American Mind* contributing editor Jonah Lehrer provides some answers to that question. Drawing from a wealth of recent neuroscience studies, Lehrer reveals what exactly is going on in our heads when we ponder whether to order chocolate, strawberry or vanilla. Along the way he provides us with tools to make smarter decisions and shows us why it is important to have both gut instincts and rational thoughts participate in our decision-making process.

But in the end, why we buy what we buy comes down to our evolutionary history, explains psychologist Geoffrey Miller in ***Spent—Sex, Evolution, and Consumer Behavior*** (Viking, 2009). He argues that although we are not consciously aware of it, our product choices are rooted in our desire to advertise our personality and attract mates and friends. —*Nicole Branan*

askthe**Brains**

Compared with other animals, human babies take much longer to learn to walk. Does this have something to do with our big brains?

—Mahmoud Dhaouadi, via e-mail

John Bock, an anthropologist at California State University, Fullerton, provides a reply:

A HORSE can walk within an hour after birth. A newborn baboon baby can cling to its mother's hair while she jumps through the trees. Even among our closest evolutionary relatives—chimpanzees and bonobos—babies are far more agile than their human counterparts. That's because humans are born with brains that are largely immature, leaving babies with little control over their movements. This uniquely human attribute is the result of a lengthy evolutionary battle between big brains and narrow pelvises.

One of the first traits that differentiated humans from our ancestors was upright gait. There are several hypotheses about the emergence of this trait, but it seems to have offered a way to move more efficiently in open environments such as the savanna. Although our earliest human ancestors were very apelike in terms of their brains, their upright gait had changed their pelvis to look much like our modern one. This reshaped pelvis came with a narrower birth canal, making childbirth more difficult.

Meanwhile the new roaming grounds afforded advantages in acquiring resources and negotiating social relationships to those with flexible, problem-solving behavior. Over time, natural selection increased brain size in these early humans. But at some point, the selection for bigger and bigger brains collided head-on, so to speak, with the narrow pelvis. If babies' heads got any bigger, they would get stuck in the birth canal and kill both mother and child. Although natural selection worked to maximize what could be done—for instance, babies' heads compress as they twist their way around the bones in the pelvis—there simply is not enough room for a big, mature brain to pass through.

As it turned out, the evolutionary answer was to let the brain keep growing outside the womb before it matures. So in contrast to other mammals, humans have a good bit of development to do after birth. The result is a relatively undeveloped infant who needs lots of care and can do much less for itself than other newborn primates.

Physiologically, why is the sound of fingernails on a blackboard so unnerving? Is this effect particular to human beings, or are other creatures similarly affected?

—Rowan Snyder, via e-mail

Neuroscientist **Josh McDermott** of New York University explains:

PROBABLY A COUPLE of factors combine to make such sounds unpleasant. The first, perhaps unsurprisingly, is the presence of high frequencies. The range between two and four kilohertz—approximately that covered by the highest octave of a standard piano—seems to contribute the most to the nastiness of the sound. It is unclear why people tend to find these frequencies unpleasant, but we know that noise-induced hearing loss most commonly occurs in roughly this region, so it is conceivable that the aversive reaction partly reflects the ear's vulnerability.

The spectrum of screeching sounds is also much noisier than that of an instrument; that is, there is a strong random component to the sound. The noisiness probably results from the fingernails repeatedly catching on part of the chalkboard surface before sliding forward. This catching and sliding also causes rapid fluctuations in intensity, giving the sound a "rough" character.

Roughness is known to be unpleasant—car manufacturers, who aim to produce minimally unpleasant engine noise, for instance, find that smooth sounds with minimal variation in intensity are preferred by listeners over those that are rough. It's a bit harder to say why sound roughness is considered unpleasant—as far as we know it is not harmful to the ears.

Some scientists have proposed that screeching sounds are acoustically similar to screams, a sound to which we might plausibly have evolved an aversion. If this hypothesis were true, one might expect to find similar reactions in nonhuman primates, which also produce screams. Thus far only one species of monkey has been tested, and it did not display the same aversive response to screeches that humans have. It may therefore be more appropriate to simply regard screeching sounds as a "perfect storm"—combining two properties that we know to be unpleasant, resulting in a single sound that is awful to listen to. M

At some point, the selection for bigger and bigger brains collided head-on with the narrow pelvis. There simply is not enough room for a big, mature brain to pass through.

Have a question? Send it to editors@SciAmMind.com

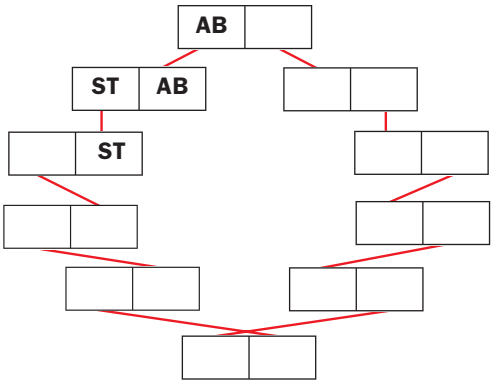
SARA E. JOHNSON (*Bock*); TALI SHAROT (*McDermott*)

Head Games

Match wits with the Mensa puzzlers

1 WORD WHEEL

Fill in each two-letter blank to create a four-letter word horizontally across. Red lines connect identical two-letter pairs. There may be more than one correct answer.



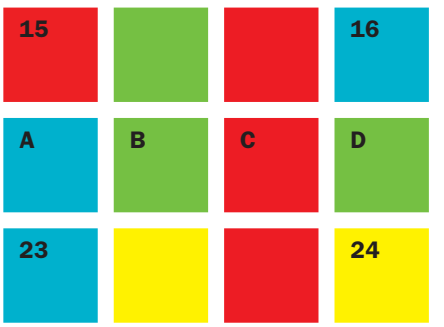
2 DECRYPTION

What is the next number in this list?

238,120
919
2,085
1,452,420
1,421,132,518
914
208,919
_____?

3 THREE-BY-FOUR

Fill in the grid with each of the numbers 1 through 12, using three types of clues:



COLOR

If a square is colored RED, it contains the number 1, 2, 6 or 10.
If a square is colored BLUE, it contains the number 4, 5 or 9.
If a square is colored GREEN, it contains the number 3, 7 or 8.
If a square is colored YELLOW, it contains the number 11 or 12.

LETTER

The letter A means the square is surrounded by only odd-numbered squares.
The letter B means the square is surrounded by more odd than even squares.
The letter C means the square is surrounded by more even than odd squares.
The letter D means the square is surrounded by only even-numbered squares.

SUM

A small number in a square's upper left corner represents the sum of the surrounding squares.

Example
The upper left square in a group of four may be clued with RED for color, C for letter or 9 for sum.

1	2
4	3

4 RHYME TIME

Find three rhyming words, each preceded by "a," that describe the clue words. For example:

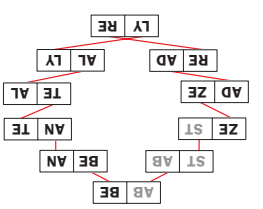
HORN **CONCORD** **MASKING**

The answer is: a cape, a grape and a tape. What are the three rhyming words that describe each of the following trios?

a) CORN	**ONION**	**WING**
b) FOOD	**CONFIRMATION**	**E-BAY**
c) IRISH	**SUPER**	**COORS**
d) PLYMOUTH	**GRANDFATHER**	**STICKER**

Answers

1. Our solution:



2. 1,291,920. The numbers stand for letters: 1 = A, 2 = B, 3 = C, etc.

W	H	A	T		20	9	19		E	X	T		N	U	M	B	E	R
23	8	1	20		8	I	S		5	24	20		14	21	13	2	5	18

I	N		T	H	I	S		L	I	S	T	?						
9	14		20	8	9	19		12	9	19	20							

3.

5	11	10	12
9	7	6	8
1	3	2	4

4. a) A chip, a dip and a tip
b) A fight, a rite and a site
c) A stew, a glue and a brew
d) A rock, a clock and a shock

SCIENCE COMICS PRESENTS:

HEADACHES

BY DWAYNE GODWIN AND JORGE CHAM

YOUR BRAIN HAS NO PAIN RECEPTORS, SO WHERE DO HEADACHES COME FROM?

ANCIENT CULTURES BELIEVED HEADACHES WERE CAUSED BY HEADACHE DEMONS, AND SOME WOULD EVEN DRILL HOLES INTO THE SKULL TO RELEASE THEM.

ONE ANCIENT EGYPTIAN REMEDY PRESCRIBED CLAY CROCODILE HATS:

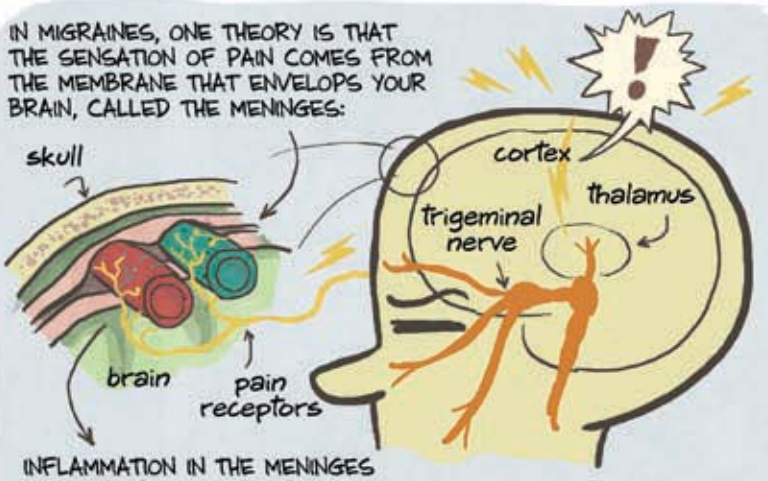
HEADACHES ARE STILL MYSTERIOUS, BUT THERE ARE SEVERAL KNOWN TYPES:

CLUSTER (sharp pain behind the eye)

TENSION (like a vise squeezing your head)

MIGRAINE (intense, may affect vision)

IN MIGRAINES, ONE THEORY IS THAT THE SENSATION OF PAIN COMES FROM THE MEMBRANE THAT ENVELOPS YOUR BRAIN, CALLED THE MENINGES:



INFLAMMATION IN THE MENINGES CAUSED BY SWOLLEN BLOOD VESSELS TRIGGERS THE TRIGEMINAL NERVE, WHICH RELAYS THE PAIN SIGNAL TO THE BRAIN CORTEX.

THE CAUSE OF THIS SWELLING IS STILL UNKNOWN, BUT IT'S OFTEN PRECEDED BY WAVES OF DEPRESSED NEURAL ACTIVITY.

CLUSTER HEADACHES HAVE BEEN LINKED IN STUDIES TO THE BODY'S INTERNAL CLOCK IN THE HYPOTHALAMUS...

PEOPLE WHO SUFFER FROM THEM CAN OFTEN SET THEIR CLOCKS TO WHEN THE HEADACHES OCCUR.

WHAT WE DO KNOW FOR SURE IS THAT MOST COMMON HEADACHES ARE TRIGGERED BY STRESS.

SO REMEMBER TO RELAX. YOU NEED ANOTHER HEADACHE LIKE YOU NEED A HOLE IN THE HEAD!