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1. **Please read the following article.**
2. **As you read, put a box around any word you do not know the definition of.**
3. **As you read, selectively highlight main ideas and write appropriately short 'quick lookup' notes in the box.**
4. **Then write a précis statement.**
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"When Our Eyes Deceive Us" by Dahlia Lithwick, Newsweek, 23 March 2009

Describe the last person who served you coffee. What if I helped refresh your memory? Showed you some photos of local baristas? Pulled together a helpful lineup? Cheered exuberantly when you picked the "right" one? Now imagine that instead of identifying the person who made your venti latte last week, we had just worked together to nail a robber or a rapist. Imagine how good we would feel. Now imagine what would happen if we were wrong.

Last month, a Texas judge cleared Timothy Cole of the aggravated-sexual-assault conviction that sent him to prison in 1986. Although his victim positively identified him three times—twice in police lineups and again at trial – Cole was ultimately exonerated by DNA testing. The real rapist, Jerry Wayne Johnson, had been confessing to the crime since 1995. Unfortunately for Cole, he died in prison in 1999, long before his name was cleared.

Our eyes deceive us. Social scientists have insisted for decades that our eyewitness-identification process is unreliable at best and can be the cause of grievous injustice. A study published in the journal of the American Psychology-Law Society, reveals just how often those injustices occur: of the more than 230 people in the United States who were wrongfully convicted and later exonerated by DNA evidence, approximately 77 percent involved cases of mistaken eyewitness identification, more than any other single factor.

Wells has been studying mistaken identifications for decades, and his objection to the eyewitness-identification system is not that people make mistakes. In an interview, he explains that eyewitness evidence is important, but should be treated – like blood, fingerprints and fiber evidence – as trace evidence, subject to contamination, deterioration and corruption. Our justice system allows juries to hear eyewitness-identification evidence shaped by suggestive police procedures. In a 1977 case, Manson v. Braithwaite, the Supreme Court held that such evidence could be used if deemed "reliable." Today we know you can have a good long look, be certain you have the right guy and also be wrong. But Manson is still considered good law.

Jennifer Thompson was 22 the night she was raped in 1984. Throughout the ordeal, she studied her attacker, determined to memorize every detail of his face and voice so that, if she survived, she could help the police catch him. Thompson soon identified Ronald Cotton in a photo lineup. When she – after some hesitation – again picked Cotton out of a physical lineup a few days later, a detective told her she'd picked the same person in the photo lineup.

But in this case Thompson got it wrong, although Cotton served 10 years before DNA evidence exonerated him and decisively implicated another man, Bobby Poole. The curious part of the story is that despite Thompson's determination to memorize every detail, when she first saw Poole in court she was certain she had never seen him before. Indeed, according to Wells and Quinlivan, "even after DNA had exonerated Cotton and Thompson herself had accepted the fact that Poole was her attacker, she had no memory of Poole's face and, when thinking back to the attack, she says, 'I still see Ronald Cotton'."

In their paper, Wells and Quinlivan suggest a host of tricks the mind can play, ranging from incorporating innocent "feedback" from police investigators to increasing certainty in one's shaky memories that become reinforced over time. Add to that Thompson's determination to regain control over her life, and her need to believe that the justice system was just, and it would have been doubly hard for her to look at a police lineup that, as it happened, did not include an image of the real rapist, and walk away. To hear Thompson and other victims tell it, being part of a system that identified and ultimately convicted the wrong man became another form of victimization, and for that reason alone the system needs to be reformed.

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"Gender Marketing 101"

You cannot market to men the same way you market to women. It's not a simple transformation of changing colors, fonts or packaging. Men and women are different biologically, psychologically and socially.

Of course, when it comes to attractiveness, both sexes want to garner attention, but each for different reasons. For men, looking good is looking strong, confident, authoritative, adventurous – a standout. Men concentrate on looks to the extent that it signals something about what they do, have done or can do. Regardless of how much a woman wants to attract in the contest of beauty and brains, their focus is on hope and details, and they concentrate on how appearance reflects their inner being.

Consider three fundamental gender differences and their impact on marketing:

Time – Men tend to hone in, more quickly than women, on what they're looking for. Men are not browsers; they shop for what they need "now." Women can shop for something and put it away for "later."

Causality – Women often think, "it depends." You rarely hear a man voice this sentiment. These different ways of defining what leads to what also impacts what goes with what. Men dislike ensembles (or groups of things). Men tend to buy individual items. In contrast, many women like to think about how they can put together "outfits" and are creative in selecting, say, a variation on a scarf or a belt that will change the nature of one basic dressing.

Space – Men structure and relate to space as compartmented and sequential. To men, space is not relational, as it is for women. For example, when a woman gives directions, she will say, "Go three blocks south (as she points or orients in the direction indicated), then bear right, and when you see the clock tower, watch for your street on the right." Men say, "Go three blocks to Pullman Street and turn left on to Main, the turn left to Brighton Street."

These kinds of underlying, fundamental gender differences can have critical implications not only for what makes an item compelling, but also for store design and product layout. For example, many women like the challenge, the somewhat disorganized variety and the catch-as-catch-can nature of places such as TJ Maxx or Marshalls. Men, even men who shop in such places because of price, are not there out of joy or desire.

For the male it's every man for himself. Men prize individuality and self-reliance. They conceive of other people as "my competition." Daily life for them is a contest with winners and losers. This is in contrast to women, who often view other people as a source of strength. Note, too, that men never shop together. Women often shop with a friend and make a "day" of it. A man focuses on himself – the "me" while a woman is focused on the "we."

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"Gender Marketing 102"

For years, marketing executives have sought to refine their target markets through advertising angles. Beer commercials showing scantily clad women are geared toward a male audience, and chocolate commercials for women. Fundamental gender differences may affect how we shop, even if men and women resent being stereotyped and demand more equality in marketing.

Brand vs. Price – Women care more about comparison shopping, bargain hunting and getting a lower price at. Men were more willing to shop at traditional department stores to find well-known brand names (trust stems from familiarity), regardless of the price.

Emotion vs. Function – Women are looking for an emotional, tactile (hands on) experience when they shop. Men aren't looking for words like "touch" or "smile" in ads, but women find this approach thoughtful. To target women, advertisers have to think about how women use the product differently. Women are looking for decisions that are "right for their families" or that "help them bring out the best version of themselves." Women want goods that make their friends happier, get the job done faster and provide a little bit of encouragement along the way.

Practicality vs. Luxury – Men and women homebuyers respond to different "must-haves" and "deal-breakers" while shopping. The survey showed that a higher percentage of women reported storage space and a large yard as a high priority, compared with men. Also, 60% of women (compared with 49% of men) said they wouldn't consider a home with small bedrooms. Men looked for perks like a great view (44% vs. 33% of women) and a luxurious bathroom (28% vs. 23% of women) and guest bedrooms (70% vs. 63% of women). In general, men are more turned off by photos of outdated furniture or unkempt landscaping.

Metro vs. Hetero – "Men are portrayed in a buffoonish, sophomoric way or as sensitive, feminized men; the bulk are somewhere in the middle, and those are men who haven't been spoken to." One survey found 79% of men do not recognize themselves in advertisements today. Men can be sold traditional "women's products" like beauty products and vacuum cleaners if advertisers promote the functionality, engineering and brand-name image, using men who aren't overly burly or overly feminine.

Detail vs. Simplicity – There are some fundamental variations in males and females. For instance, female brains have stronger connections between the right and left hemispheres, meaning that their language and memory is very much in touch with their emotions. Women also have a larger hippocampus region, which translates to better recall of detailed information. Men have superior spatial ability and problem-solving processes. When marketing to women, they use loads of detail and emotional cues. For men, they keep the message simple but show important design elements.

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"Mountain Biking with the Blind" by L.Rosenblum, Psychology Today, 21 July 2009

I expect some stares as we walk into the bike shop, and we get them. My companions are both blind, leading with white canes, and one is rolling in his ailing mountain bike. I'm also not surprised when the salesman approaches me to ask what we need. But then one of my companions, Daniel Kish, answers that he's looking for a new tube "24 inches, latex, with a valve". The salesman quickly realizes that despite appearances, Daniel is the experienced rider.

In fact, Daniel has been leading his group of blind mountain bikers and hikers—Team Bat—for over 15 years. Today's group is small. I'm along to document the experience for my book.

We leave the safety of Brian's driveway and turn onto the residential street leading to the mountain trail. That's when the tongue-clicking begins. Daniel, Brian, and Megan are making loud, sharp clicking sounds with their tongues so that they can hear what I can see. Using this form of human echolocation allows them to detect sounds reflected from parked cars, trash cans, and other silent obstacles along the street. Daniel and Brian are experienced echolocators and can actually hear the location of the curbs, driveways, and even hedges on the side of the street.

Like bats, dolphins, and whales, humans can echolocate. The skill involves implicitly listening to how objects reflect sounds in distinct ways. It helps if the emitted sound is produced by the listener him or herself, but this isn't necessary. There's evidence, in fact, that blind individuals can perceive the location of a wall based only on how it reflects the normal, quiet ventilation sounds in a room. Sighted individuals can also echolocate. In experiments conducted in my own lab, we're able to get blindfolded college freshman to successfully echolocate the position of a small wall within 10 minutes. And with practice, both blind and sighted listeners can determine the rough shape, size, and even texture of objects based on how they reflect sound.

To get a sense of how echolocation works, try this. Hold your hand up about one foot in front of your face with your palm facing your mouth. Put your front teeth together, open your lips, and make a continuous shhhhhh sound. As you make this sound, slowly bring your hand toward your mouth. You will hear the shhhh sound change. What you're hearing is the sound reflecting from your hand colliding with the sound leaving your mouth. This interference turns out to be one of the most important types of sound dimensions we use to echolocate objects at close distances.

But this demonstration is exaggerated. The interference patterns used for echolocation are usually too subtle to be consciously heard. This highlights one of the most amazing aspects of echolocation: It's rarely experienced as sound. Try using your shhhh sounds to walk slowly toward a wall with your eyes closed. As you come close to the wall, you'll experience its presence as more of a feeling than a change in sound. It may feel as if there are air pressure changes on your face, an experience also reported by the blind. Echolocation is truly one of your implicit perceptual skills: It allows you to detect aspects of your environment without even knowing which sensory system you're using. And it could very well be that you're constantly using the skill to recognize properties of the rooms you occupy.

A recent study confirms that the types of tongue clicks used by Daniel Kish and his colleagues are acoustically ideal. In fact, Daniel and Brian have reportedly had good success in training other Team Bat members to use clicks to guide biking and hiking.

Back on the bike trail, I hear clicks approaching from behind and Brian zooms past me. I then realize that when it comes to our riding, our most important difference is that he's in much better shape. But I'm comforted in knowing that we both have auditory systems that allow us to hear reflections from the silent world.

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"Selective Mutism"

Selective Mutism is a type of social disorder that primarily affects children. It was originally called Elective Mutism about two decades ago, because doctors mistakenly believed that children electively chose not to talk. Even if this was done by the child to gain attention or just out of curiosity, Elective Mutism was eventually discovered to be an uncontrollable phenomenon.

While the symptoms of Selective Mutism can vary, there are some distinctive characteristics of this disorder. The most prominent symptoms are seen in the differences of communication between social situations. Usually, a child has the ability to speak clearly and normally at home with family. On the contrary, children with this disorder tend to have a difficult time in certain social situations. The child experiences a failure to verbally communicate, particularly in situations where the child feels uncomfortable. Furthermore, the child is typically shy or afraid of others, especially strangers.

The symptoms of this disorder usually appear at a very early age, typically around the time the child starts preschool. It is typically recommended to contact a doctor only if the child's function in school or other social settings becomes altered. It may also be beneficial to talk directly to the child's teacher or counselor.

Furthermore, if the child is predisposed to another form of social phobia based on hereditary factors, it is more likely that they could develop Selective Mutism. Many people believe that inheriting social anxiety traits is the only way to develop this disorder. Likewise, if the child is already diagnosed with a social phobia, there is a significantly greater risk of developing Selective Mutism.

Currently, no cure-all treatments are available for Selective Mutism. A great number of strategies can be employed by counselors, physicians and family members to help children deal with this disorder. Selective Mutism can be combated with prescription medication, as well as counseling and other therapy techniques. A combined approach is probably the most effective. Prescription medications, such as those used for other social disorders, like Prozac, fluoxetine and other antidepressants, have been proven effective for the treatment of Selective Mutism.

Selective Mutism is more common than many people would think, as recent studies have suggested that one out of every 1,000 children around the world is affected by this disorder.

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"Pink Brain, Blue Brain – the Claim of Sex Differences Fall Apart"

Among certain parents, it is an article of faith not only that they should treat their sons and daughters alike. If Jack gets Lincoln Logs and Tetris, and joins the soccer team and the math club, so does Jill. Lise Eliot, a neuroscientist, doesn't think these parents are lying, exactly. But she would like to bring some studies to their attention.

In one, scientists dressed newborns in gender-neutral clothes and misled adults about their sex. The adults described the "boys" (actually girls) as angry or distressed more often than did adults who thought they were observing girls, and described the "girls" (actually boys) as happy and socially engaged more than adults who knew the babies were boys. Dozens of such disguised-gender experiments have shown that adults perceive baby boys and girls differently, seeing identical behavior through a gender-tinted lens. In another study, mothers estimated how steep a slope their 11-month-olds could crawl down. Moms of boys got it right to within one degree; moms of girls underestimated what their daughters could do by nine degrees, even though there are no differences in the motor skills of infant boys and girls. But that prejudice may cause parents to unconsciously limit their daughter's physical activity. How we perceive children - sociable or remote, physically bold or reticent - shapes how we treat them and therefore what experiences we give them. Since life leaves footprints on the very structure and function of the brain, these various experiences produce sex differences in adult behavior and brains - the result not of innate and inborn nature but of nurture.

For her new book Eliot immersed herself in hundreds of scientific papers (her bibliography runs 46 pages). Marching through the claims, she explains that assertions of innate sex differences in the brain are either "blatantly false," "cherry-picked from single studies," or "extrapolated from rodent research" without being confirmed in people. For instance, the idea that the band of fibers connecting the right and left brain is larger in women, supposedly supporting their more "holistic" thinking, is based on a single 1982 study of only 14 brains. Fifty other studies, taken together, found no such sex difference - not in adults, not in newborns. Other baseless claims: that women are hard-wired to read faces and tone of voice, to defuse conflict, and to form deep friendships; and that "girls' brains are wired for communication and boys' for aggression." Eliot's inescapable conclusion: there is "little solid evidence of sex differences in children's brains."

Yet there are differences in adults' brains, and here Eliot is at her most original and persuasive: explaining how they arise from tiny sex differences in infancy. For instance, baby boys are more irritable than girls. That makes parents likely to interact less with their "nonsocial" sons, which could cause the sexes' developmental pathways to diverge. By 4 months of age, boys and girls differ in how much eye contact they make, and differences in sociability, emotional expressivity, and verbal ability - all of which depend on interactions with parents - grow throughout childhood. The message that sons are wired to be nonverbal and emotionally distant thus becomes a self-fulfilling prophecy. The sexes "start out a little bit different" in fussiness, says Eliot, and parents "react differently to them," producing the differences seen in adults.

Those differences also arise from gender conformity. You often see the claim that toy preferences - trucks or dolls - appear so early, they must be innate. But as Eliot points out, 6- and 12-month-olds of both sexes prefer dolls to trucks, according to a host of studies. Children settle into sex-based play preferences only around age 1, which is when they grasp which sex they are, identify strongly with it, and conform to how they see other, usually older, boys or girls behaving.

The belief in blue brains and pink brains has real-world consequences. It encourages parents to treat children in ways that make the claims come true, denying boys and girls their full potential.

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"When A Brain Forgets Where Memory Is"

Every so often, seemingly normal people suddenly walk out of their lives and disappear, with no recollection of who they are, where they are from or what their previous life was like. It is the stuff of fiction, but it happens in real life too.

Last year a Westchester County lawyer – a 57-year-old husband and father of two, Boy Scout leader and churchgoer – left the garage near his office and disappeared. Six months later he was found living under a new name in a homeless shelter in Chicago, not knowing who he was or where he came from. Library searches and contact with the Chicago police did not help the man. His true identity was uncovered through an anonymous tip to "America's Most Wanted." But when he was contacted by his family, he had no idea who they were.

Known technically as dissociative fugue or dissociative amnesia, people with this problem suddenly and unexpectedly take leave of their usual physical surroundings and embark on a journey that can last as little as a few hours or as long as several months. During the fugue state, individuals completely lose their identity, later assuming a new one. They don't know their real names or anything about their former lives, and they do not recognize friends or family. They may not even remember how they got to where they are. While loss of memory can occur for many reasons, dissociative fugue has no direct physical or medical cause. Rather, it is precipitated by a severe stress or emotionally traumatic event that is so painful the mind seems to shut down and erase everything, like a failed computer hard drive. But unlike a computer whose unsaved information is lost forever, most if not all patients suffering from dissociative fugue eventually recover their memories, typically just as suddenly as the memories disappeared.

While in the fugue state, people are unaware that their identity and memory have been lost, said David Schacter, professor of psychology at Harvard. They wander off, often traveling far from home. It is only when they are forced to reveal some piece of biographical information that they realize they do not know who they are, which may lead to a desperate search to uncover their identity.

In a telling case, a man who felt increasingly trapped in his father-in-law's business one day failed to show up at the store in Boston and later found himself in New York. Not until he had to provide his name for a hotel did he discover he did not know who he was. After many failed attempts to uncover his identity, his past revealed itself while he was being quizzed by a doctor at Bellevue Hospital, he recalled. "All of a sudden, I knew, I remembered. I jumped up and shouted. I yelled, 'I know – I can remember! I remember my wife's name. We live in Boston. I can even tell you the address."

For some, an underlying neurological problem is the usual cause of amnesia. When amnesia has a physical basis, memory loss is usually not complete, but rather covers a part of someone's life. The more recent memories are often lost, while memories of more distant events are preserved.

When examining a patient with memory loss that has no obvious physical cause, the first step is to look for a cause like a head injury, stroke, or temporal lobe epilepsy. In such cases, in addition to incomplete memory loss, there is usually a loss of individual facts

Key themes and comments about the article:

like biographical information. However, when memory loss includes generic knowledge about whole classes of things, like how many wings birds have, the underlying cause is more likely to be psychogenic. When amnesia has an organic cause, people's memories of who they are usually are not disrupted, Dr. Schacter said. Nor are memories usually lost of events that occurred before the physical problem. But such people may be unable to form new memories.

And sometimes cases of fugue have a precipitating psychological cause along with some form of underlying brain damage that is revealed, say, through a MRI or PET scan.

Another challenge clinicians face in diagnosing amnesia, Dr. Schacter said, is to differentiate between genuine cases of lost memory and the concocted amnesia of malingerers – people who are fleeing financial problems, for example, who have committed a crime or who simply wish to gain notoriety. Neuropsychological tests performed by experts can often pinpoint the malingerers who may do so poorly on certain tests that they are obviously faking their answers.

Other checks for malingering include assessing whether patients are making a genuine effort to answer questions correctly and whether there are apparent motives for pretending they do not know who they are.

In the case of the Westchester lawyer, who had lost all memory of his former life, his wife provided background information suggesting that his amnesia may have resulted from prolonged post-traumatic stress. He was a Vietnam War veteran who happened to have walked between the twin towers of the World Trade Center on Sept. 11, 2001, minutes before the first plane hit. He subsequently experienced a return of painful memories of his war experience and required treatment for depression.

"You never lose your memory. It's always there. It just falls out of the file cabinet."

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"Taking on Tourettes"

Marg was just 3 when her parents noticed the first signs of what turned out to be Tourette syndrome. The blond toddler began sniffing her fingers repeatedly, and over the next six years, her uncontrolled tics came to include clicking, whirring and scrunching her nose. Her condition was manageable (she attends school with other kids) until last year, when, at the age of 9, she began to suffer about 60 episodes a day of repeated head jerks that left her sore and spent by nighttime. So when MacKrell's parents learned about an old but little-used therapy called habit-reversal training (HRT), they decided to try it. Last November, Marg started learning new ways to pre-empt her most severe tics. When she felt a head jerk coming on, she was taught to drop her head and stare at the second hand on her watch for a minute. "Soon [the head jerking] was down by 90 percent," says Marg's mother, Diane MacKrell. "I couldn't believe it."

An estimated 200,000 Americans have the most severe form of Tourette syndrome, a neurological disorder sometimes marked by outbursts of jerking or swearing. In addition, as many as 1 in 100 exhibits milder symptoms of the disorder. TS is usually treated with drugs, including antipsychotics and antidepressants. While they can be effective in controlling symptoms, they are often accompanied by bothersome side effects, including lethargy and weight gain. Now, with the help of a $5.4 million grant researchers at six universities are taking a fresh look at HRT, which was originally developed in 1973 as a treatment for problems like chronic nail biting. The results so far have been encouraging. HRT has "the potential to bring significant relief to TS sufferers without the serious side effects characterizing most TS medications," says Dr. John Piacentin.

In theory, HRT substitutes a competing action—looking at a watch, for example—for a disabling or socially embarrassing tic. Patients undergoing HRT learn to recognize the so-called premonitory urges that precede their tics. They are then taught how to counteract the approaching tic with an opposing response. Before a tic, Marg says, she feels "energy in my body that feels like it needs to get out. It's like a balloon." Rick Shocket, 9, also undergoing HRT therapy at Duke, has had a tic that involves squatting after every step. To divert it, he's been taught to lock his knees and count to 60 when he feels it coming on. After just three therapy sessions, he has managed to get a handle on the squatting and many of his other tics, which include neck jerking and eye movements. HRT is "another tool in my tool belt," he says.

Yet the treatment remains controversial. Many doctors believe tics can't be suppressed, or that suppressing them only aggravates them, though more than two dozen studies suggest that isn't so. "We don't see a particularly important role for the behavioral therapy," says Dr. Roger Kurlan, who usually uses medication when his TS patients need treatment. "I'm not sure it has any effect on the underlying condition." Critics also complain that depicting tics as "habits" that can be reversed ignores the underlying biology of Tourette's. (Imaging studies suggest that tics involve a disruption of normal brain processes.) "We in the Tourette community have fought hard to get insurance companies to understand that this is a genetic neurological disorder," says Leonard Misner, 39, who has Tourette's and opposes HRT. Dr. Alan Peterson, says TS, like other disorders, can be behavioral and biological. "There are all kinds of medications and genetic work being done to help people with type 2 diabetes. But the largest study done proved lifestyle modification can cure it."

David, 34, welcomes the new approach. He tried HRT after years of severe tics, including swearing and punching himself. He's managed to control nearly all the tics involving his face and head. A troubling one remains, though: his tendency to thump his wife when they embrace. "I just don't hug her much," he says. Perhaps with continued HRT, they'll finally be able to snuggle in peace.

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"Ethics of Scan and Tell"

As part of my job reporting on neuroscience, I found myself in an unusual situation 10 years ago. During an interview, I offhandedly told a researcher to contact me if he ever needed a volunteer for a study. Months later the neurologist actually called, and I enrolled in a project on Parkinson's disease.

I was soon lying in a positron-emission tomography machine. Scientists injected a radioactive dye into my left arm, which felt warm and tingly as it coursed toward my brain. It settled in the regions that produce dopamine, a chemical that becomes depleted in Parkinson's. The researchers hoped that brain scans of middle-aged people could reveal the earliest signs of dopamine loss.

My dopamine levels turned out to be very high, "the highest we've seen in a normal volunteer," the neurologist told me. But he and his colleagues had also found something unsuspected. They wanted me to undergo magnetic resonance imaging (MRI) to highlight one particular area. Alarmed, I agreed, and a few days later they took the MRI scan. There, in a dark corner of my cerebellum, was a large ghostly-white mass. It wasn't pretty. The researcher, who was not a physician, shrugged uncomfortably. The radiologist said nothing. Many hours later the neurologist called and told me the shadow indicated a cyst and not to worry. I had probably had it since I was born.

No one volunteers for a study expecting that something sinister may appear. But now, after more than a decade of brain-imaging research, scientists have run across the likes of me often, and they have finally pulled together to discuss the issue of such "incidental findings"--IF, for short. The findings range from tumors and blood clots to cysts and other structural abnormalities. Investigators simply don't know what to do when they happen on these anomalies in what are supposed to be "normal" test subjects.

This past January dozens of scientists, lawyers, ethicists and policymakers convened at the National Institutes of Health to debate the issue. It seems that incidental findings show up in 20 percent of subjects in research studies--a huge number--and there are still no official procedures for handling such discoveries. Judy Illes, a senior research scholar at the Stanford Center for Biomedical Ethics who organized the meeting, notes that researchers typically are not medical doctors and shouldn't be put in the position of practicing medicine. Yet they become good at sizing up scans. What should they do when they spot something? When should they scan and tell?

The answer is not easy, Illes and other experts note. Scans are like Rorschach tests: in the best hands, scans can still be interpreted differently. No one even agrees what a normal brain should look like.

"It's a judgment call," says David Eidelberg, director of neuroscience at the North Shore Long Island Jewish Health System in Manhasset, N.Y. "There are lots of variants of normal. Do you tell a person that they have a cyst in their brain that will never alter the course of their life? I'm not sure."

This position, in essence, is that there is no reason to alarm a test subject unnecessarily. But others say volunteers should be told about any kind of unusual indications, whether it might worry them or not. It's their brain, and they should be informed about it. The sticking point is that scientists have no uniform way of handling incidental findings. Illes

Key themes and comments about the article:

and her colleagues want to adopt a basic framework to follow. "The idea is to come up with solutions to protect our research volunteers, our patients and our institutions," Illes says.

B. J. Casey, a psychologist at Weill Medical College of Cornell University who took part in the NIH meeting, recounted the first time that her team stared at an odd white mass on the MRI scan of a normal volunteer. "We all realized it was something that shouldn't be there," Casey says. "But we aren't doctors, we aren't neuroradiologists, we're researchers."

Casey did call in a neuroradiologist, who concluded that the mass was a brain tumor. "We saved this person's life," she states. Nevertheless, the situation and others like it are uncomfortable. "We don't want to enter into a patient-doctor interaction in a research study," Casey says. "We have to separate research from clinical practice." Then, she adds, the problem becomes, "How do you even know something is important enough to tell a person? Anything abnormal should be confirmed" first. Casey now runs pediatric scanning studies and shudders at the thought of "telling parents something is wrong with their child when it isn't."

Who tells a parent or an adult subject about an incidental finding is an issue, too. Scientists could simply say nothing; there is no requirement in a study scenario. Others might call in a doctor, and if the physician agrees something is suspicious he or she could refer the volunteer to a specialist. Alternatively, a researcher could advise a subject to contact his or her own doctor for follow-up. Or the scientist could call the doctor directly and have him or her address the patient.

Most studies do not include a physician, and participants at the meeting disagreed over whether they should factor a doctor into the cost of a study. "Researchers may be able to detect obvious abnormalities, but they don't have the basic knowledge to make diagnoses," says Ruth Macklin, a professor of bioethics at the Albert Einstein College of Medicine. Therefore, she maintains, the cost of a doctor or radiologist should be included.

Another vexing issue is that scans used for research are typically not as sophisticated as those in a clinical setting. As a result, the images might be harder to read and interpret by untrained eyes. Buying more expensive machines would further drive up study costs.

By meeting's end the group at least agreed that the consent forms volunteers sign should lay out the possibility that a normal brain might not always look "normal." The form could ask patients whether they want to know about what seem to be minor findings. And the document should specify that markers of potentially major abnormalities, such as a blood clot, aneurysm or tumor, would trigger immediate attention, whatever that might be. The group agreed to begin drafting guidelines for IFs, including the recommendation to inform subjects and when to refer them to a physician.

Given my own experience, I'm heartened that scientists are paying more attention to this problem. After all, I represent the normal volunteer who was abnormal. I'm glad to know that my dopamine levels are so high that I will probably never get Parkinson's disease. And I've got a picture of my brain, the cyst hogging a good chunk of my cerebellum--the area that controls movement. If I do have any complications from that, it's nothing more than a sore toe on my dancing partner's foot.

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"A Tough Pill To Swallow"

This year 2.25 million Americans will get married—and a million will divorce. Could birth control be to blame for some of these breakups? Recent research suggests that the contraceptive pill—which prevents women from ovulating by fooling their body into believing it is pregnant—could affect which types of men women desire. Going on or off the pill during a relationship, therefore, may tempt a woman away from her man.

It's all about scent. Hidden in a man's smell are clues about his major histocompatibility complex (MHC) genes, which play an important role in immune system surveillance. Studies suggest that females prefer the scent of males whose MHC genes differ from their own, a preference that has probably evolved because it helps offspring survive: couples with different MHC genes are less likely to be related to each other than couples with similar genes are, and their children are born with more varied MHC profiles and thus more robust immune systems.

A study published in August in the Proceedings of the Royal Society B, however, suggests that women on the pill undergo a shift in preference toward men who share similar MHC genes. The female subjects were more likely to rate these genetically similar men's scents (via a T-shirt the men had worn for two nights) as pleasant and desirable after they went on the pill as compared with before. Although no one knows why the pill affects attraction, some scientists believe that pregnancy—or in this case, the hormonal changes that mimic pregnancy—draws women toward nurturing relatives.

Women who start or stop taking the pill, then, may be in for some relationship problems. A study published last year in Psychological Science found that women paired with MHC-similar men are less sexually satisfied and more likely to cheat on their partners than women paired with MHC-dissimilar men. So a woman on the pill, for example, might be more likely to start dating a MHC-similar man, but he could ultimately leave her less sexually satisfied. Then if she goes off the pill during the relationship, the accompanying hormonal changes will draw her even more strongly toward more MHC-dissimilar men. These immune genes may have a "powerful effect in terms of how well relationships are cemented," says University of Liverpool psychologist Craig Roberts, co-author of the August paper.

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"The Roots of Schizophrenia"

Brains today are expensive—metabolically speaking, that is. Pound for pound, the human brain demands a huge amount of energy to support its recently evolved language and social skills. Now a study offers some of the first strong evidence that the rapid development of our metabolically costly brain may have led to an unfortunate by-product: when energy problems arise, the result may be schizophrenia.

No one knows exactly what causes schizophrenia, a debilitating disorder characterized by psychosis and severe cognitive impairments. One theory, which suggests it is a consequence of our brain's high metabolism, has been around for years—but until now scientists had not developed a way to test it.

In the new study—a rare combination of evolutionary genetics and medicine—researchers in China, Germany and the U.K. compared gene expression (when and where in the body certain genes are active) and concentrations of metabolites (small molecules crucial for metabolic processes) in the postmortem brains of people without schizophrenia with those in the brains of chimpanzees, rhesus macaques and human schizophrenics. They determined that the genes and metabolites that are altered in schizophrenia appear to have changed rapidly in recent human evolution. More important, they are related to energy metabolism.

Because these changes may have happened recently (on an evolutionary scale), we may not yet have developed ways to cope with energy problems that arise, according to study co-author Philipp Khaitovich, an evolutionary biologist at the joint Max Planck/Chinese Academy of Sciences Institute for Computational Biology in Shanghai. Khaitovich suggests that the brain could be operating at the limit of its energy-regulating abilities, so it might be easy for something to go wrong, as in the case of schizophrenia.

This study may begin to explain why schizophrenia exists but not necessarily why some people are more predisposed to it than others, says Matthew Keller, an evolutionary behavioral geneticist at the University of Colorado at Boulder, who was not involved with the study.

Khaitovich agrees that the work is just a glimpse into the mechanisms responsible for our uniquely human abilities, but the findings do put metabolism in the spotlight for future research. Once we understand what makes our brains special, we can begin to understand what goes wrong in schizophrenia, he says.

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"When Your Mind Disowns A Limb"

Look down. There isn't a doubt in your mind that the body you are looking at is yours. But what if you could be fooled into thinking that one of your hands belonged to someone else? Scientists recently incited this false perception through an illusion—and they found that when people felt dissociated from a limb, their brain devoted less processing power toward that limb and even interfered with its temperature regulation. These findings, building on a smattering of other studies in disembodiment, suggest that the conscious mind's control over basic body function is much stronger than scientists once thought.

The science of out-of-body experiences seems to have begun with an impromptu party trick. In 1998 a psychiatrist asked a fellow guest to hide one of his hands behind an opaque screen, then placed a rubber hand next to the screen where the person could see it. When he simultaneously tickled the man's hidden hand and the rubber hand with brushes, the person felt as if the rubber hand was his own.

But how does it work? Seeing the touch delivered on the rubber hand "captures the sense of touch experienced by the subject's real hand," explains another psychologist. "The rubber hand illusion shows that the integration of different senses is powerful enough to fool the brain into treating a fake hand as a real one."

Last year scientists used a similar tactic to fake an entire out-of-body experience: they gave subjects goggles that played live feeds from two video cameras located eye distance apart two meters behind them. The experimenter stood beside the subject and used two rods to touch the person's actual chest and the "illusory chest," the space that would correspond to the chest of someone whose eyes were located at the two video cameras. After two minutes, subjects began to feel as if they were sitting two meters behind their bodies.

The stronger a person experiences these types of illusions, the stronger the activity in his or her brain's premotor and parietal cortices, which integrate sensory and movement information. The brain's fear circuits are also affected: although subjects know they are experiencing an illusion, they become protective of their new body part. When experimenters threaten the fake hand, brain areas corresponding to threat responses and withdrawal urges become more active.

In the latest study, scientists used the illusion to figure out what happens to the hand that becomes "disowned." Immediately after the brain begins thinking of the rubber hand as its own, the temperature of the disowned hand drops (while the rest of the body's temperature remains the same). When an experimenter touches the disowned hand, the subject's brain responds more slowly than it does when his or her other real hand is touched. These results suggest that when the brain forgets about a limb, the body responds accordingly.

Disease-associated problems in temperature regulation have always been attributed to central nervous system defects, never to thought alone—but these new results offer the tantalizing suggestion that conscious control may be possible, too. These types of illusion-based experiments could help scientists understand what is required for amputees to accept prosthetic limbs as "real" body parts, and they could lead to treatments for people who disown their limbs, such as stroke patients who stop recognizing their paralyzed body parts

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"Of Tics and Compulsions"

On the surface, Tourette's syndrome and obsessive-compulsive disorder (OCD) seem to have little in common. Tourette's is characterized by repetitive involuntary facial or vocal tics, whereas OCD sufferers have all-consuming thoughts and overwhelming urges to perform certain actions. But 50 to 70 percent of people with Tourette's also have OCD, and recent studies suggest that the same genetic roots may underlie both conditions. Now a new study may help scientists further understand how the disorders overlap and differ by revealing several key differences in the brain activity of Tourette's patients with and without OCD.

Andrew Feigin and his colleagues at North Shore LIJ Health System in Manhasset, N.Y., scanned the brains of 12 unmedicated Tourette's patients—some of whom also had OCD—and 12 healthy subjects using positron-emission tomography, which reveals patterns of brain activity. Compared with healthy controls, those who had Tourette's exhibited more activity in the premotor cortex and cerebellum, regions that handle motor control, and less activity in the striatum and orbitofrontal cortex, areas involved in decision making and learning. These findings support the idea that the symptoms of the disorder may arise from the brain's inability to suppress abnormal actions using decision-making skills.

When the researchers compared the Tourette's patients who had OCD with those who did not, they found that the patients who had both disorders exhibited greater activity in the primary motor cortex and precuneus, an area that plays a role in self-awareness. Previous research has suggested that in patients who suffer from both disorders, OCD might show up more in the form of compulsions than obsessions, and these findings support that idea: the increased activity of the precuneus may reflect individuals' efforts and ability to resist obsessive thought, and the motor cortex may be more active because OCD is manifesting itself more physically than mentally.

Although the neural networks that seem to be affected in Tourette's and OCD are distinct, they nonetheless involve brain regions with similar functions—in particular, motor activity and decision making. The discovery isn't all that surprising, Feigin says, given that tics are actually quite similar to compulsions—both, after all, involve extra involuntary movements

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"Why Autism Strikes More Boys Than Girls"

Autism, a developmental disorder that causes deficits in social behavior and communication, affects four times as many boys as girls. Because of this extreme gender imbalance, some scientists posit that sex hormones may contribute to the disease. Now researchers have identified for the first time a gene that may help explain the gender discrepancy and underlie some common autism symptoms.

In 2010 biologist Valerie Hu of the George Washington University Medical Center and her colleagues found that brains of people with autism have low levels of a protein produced by a gene called retinoic acid–related orphan receptor-alpha (RORA). Now they report in a study published in PLoS ONE on February 16 that this gene interacts with certain types of estrogen and testosterone found in the brain.

Hu and her team examined neural cells in their lab. They found that RORA controls the production of an enzyme called aromatase, which converts testosterone to estrogen. But in their tests, the presence of testosterone made RORA less active, leading to a decline in aromatase and a buildup of even more testosterone. Estrogen had the opposite effect. In a typical brain the balance of sex hormones regulates RORA activity and keeps hormone levels steady, but any imbalance can be exacerbated by this loop.

Next, the researchers confirmed that brain tissue from donors who had autism indeed contains low amounts of the RORA protein and aromatase. The authors suggest that a deficiency in these molecules causes the chemical loop to spiral out of control, resulting in an accumulation of testosterone that may cause autism. In most females, higher levels of estrogen could be protecting them from the disorder.

In addition to the gender bias, RORA might be implicated in the abnormal routines that characterize autism. For instance, mice that lack this gene fixate on objects and show limited exploratory behavior, similar to individuals with autism. "I don't think any single gene is going to explain all of the pathology associated with autism, but RORA does explain quite a few of them," Hu says.

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"Predicting Attraction Through Language"

What distinguishes a fling that ends in tears from long-term love? Past research suggests that the most successful couples share common interests, values and personality traits. Now new research published in Psychological Science proposes that the simplest words lovebirds use to speak to each other also make a difference—both in determining how attracted they are and how likely they are to stay together.

James Pennebaker and his colleagues at the University of Texas at Austin recorded 40 men and 40 women as they participated in a speed-dating exercise in which they talked to 12 strangers of the opposite sex for four minutes apiece. Later, the subjects rated each date based on how much they seemed to have in common and whether they wanted to see the person again. Pennebaker analyzed the participants' conversations based on their use of pronouns and articles, such as "him," "the," "and," "as" and "be." These function words are used in most contexts and are processed rapidly and unconsciously.

The pairs who used similar types of function words with similar frequencies, he found, were more likely to want to see each other again, regardless of how much they felt they had in common. In a follow-up study, Pennebaker compared the language used by 86 couples in committed relationships via writing samples from instant messages. He found that the more their function words matched, the more likely they were to be together three months later, irrespective of how happy they said they were in their relationships at the time.

The big question is whether individuals feel more aligned to others who already talk the way they do or whether they adapt their language to match that of individuals they really like. Pennebaker admits that both are possible, but he believes the latter is the driving force: language, he says, predicts relationship success because it reflects how well couples listen to each other. What is Pennebaker's advice for living happily ever after with a loved one, then? "Pay closer attention to the other person," he says.

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"Hyper One Day, Calm The Next"

Attention deficit hyperactivity disorder (ADHD) is a chronic condition, and if left untreated, it can set a child up for a lifetime of difficulties in learning and forming relationships. At least that is the assumption that has guided the popular approach to treating ADHD for decades. But new research suggests that ADHD might be much less persistent than previously thought.

A team led by Prudence Fisher and J. Blake Turner, both at Columbia University and the New York State Psychiatric Institute, reviewed the records of nearly 1,500 children from four studies that had used a standard diagnostic interview to screen for ADHD. They found that a majority of children who qualified for an initial diagnosis had lost their diagnosis by two years later.

ADHD has three subtypes: hyperactive, inattentive and both combined. More than half the children with the hyperactive and inattentive subtypes of the disorder had reverted to no ADHD at a two-year follow-up interview. Although the combined subtype was more persistent, between 18 and 35 percent of children in that group had also lost their diagnosis by the follow-up. Kids with many symptoms and significant impairment were just as likely to lose their diagnoses as children with milder forms of the disorder. Nor were the losses attributed to successful treatment.

To Turner, the findings suggest that the current definition of ADHD would benefit from greater specificity. If a disorder is, by definition, long-lasting, "then we are over-diagnosing ADHD," Turner says. He and Fisher are advisers to the ongoing revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the handbook of guidelines for diagnosing psychiatric disorders. Turner recommends a cautious approach to labeling and medicating kids whose behaviors, though irritating to many adults, are likely to be transient.

Joel Nigg, a professor of psychiatry at the Oregon Health and Science University who was not involved with the study, says that the finding reflects our evolving understanding of ADHD. Fifty years ago experts believed that most children "grew out of it." In the 1970s and 1980s new studies appeared to show that ADHD is lifelong. The truth might lie somewhere in between. "The corrective here," Nigg says, "may be that it's chronic sometimes, a fluctuating condition in other cases, and it may be that some kids get better."

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"The Link Between Alzheimer's and Mitochondria"

Five million Americans suffer from Alzheimer's disease, but scientists still have more questions about it than answers. Arguments abound over whether the hallmark protein clusters that accumulate in the brain are a cause or an effect of the illness, and current treatments do not address the main problem that causes impaired thinking: broken synapses, the junctions that allow neurons to communicate with one another. Researchers are now zeroing in on a promising missing link: mitochondria, the cell components responsible for energy regulation. In October researchers at Columbia University reported that young mice predisposed to acquiring Alzheimer's accumulate protein clusters in synaptic mitochondria and that these clusters directly impair synapse function.

The link between mitochondria and Alzheimer's is not exactly new. In the 1990s studies suggested that in the diseased brains of people and mice, mitochondria do not produce and distribute energy normally. And as early as 1994 researchers at the University of Kentucky showed that amyloid-beta protein fragments, the type found in Alzheimer's, interfere with mitochondrial function. But no one knew how, exactly, mitochondria were linked to synaptic problems, if at all.

To find out, Shirley ShiDu Yan and her colleagues at the Columbia University Medical Center genetically engineered mice to overproduce a compound that leads to the formation of amyloid-beta clusters. When the mice were at various ages, the researchers isolated mitochondria from their synapses and from other brain regions. When the mice were just four months old—well before they showed symptoms of the disease—their synaptic mitochondria had accumulated approximately five times more amyloid protein than nonsynaptic mitochondria had.

The affected mitochondria could no longer provide the synapses with enough energy, which ultimately prevented the synapses from functioning—providing the first direct link between cellular injury caused by amyloid protein and the characteristic breakdown of neuronal communication that occurs in Alzheimer's patients.

The findings could provide new treatment possibilities. In earlier research Yan reported that cyclosporin D, a drug used to treat patients with organ transplants and autoimmune diseases, prevents amyloid-beta proteins from injuring mitochondria. Although the drug has serious side effects, Yan hopes that she can develop a similar but safer compound that prevents synaptic problems early on. Scientists need to "stop the disease early, before neurons have already died," she says.

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"<u>Shades of Grief: When Does Mourning Become A Mental Illness?</u>"

Sooner or later most of us suffer deep grief over the death of someone we love. The experience often causes people to question their sanity—as when they momentarily think they have caught sight of their loved one on a crowded street. Many mourners ponder, even if only abstractedly, their reason for living. But when are these disturbing thoughts and emotions normal—that is to say, they become less consuming and intense with the passage of time—and when do they cross the line to pathology, requiring ongoing treatment with powerful antidepressants or psychotherapy, or both?

Two proposed changes in the "bible" of psychiatric disorders—the Diagnostic and Statistical Manual of Mental Disorders (DSM)—aim to answer that question when the book's fifth edition comes out in 2013. One change expected to appear in the DSM-5 reflects a growing consensus in the mental health field; the other has provoked great controversy.

In the less controversial change, the manual would add a new category: Complicated Grief Disorder, also known as traumatic or prolonged grief. The new diagnosis refers to a situation in which many of grief's common symptoms—such as powerful pining for the deceased, great difficulty moving on, a sense that life is meaningless, and bitterness or anger about the loss—last longer than six months. The controversial change focuses on the other end of the time spectrum: it allows medical treatment for depression in the first few weeks after a death. Currently the DSM specifically bars a bereaved person from being diagnosed with full-blown depression until at least two months have elapsed from the start of mourning.

Those changes matter to patients and mental health professionals because the manual's definitions of mental illness determine how people are treated and, in many cases, whether the therapy is paid for by insurance. The logic behind the proposed revisions, therefore, merits a further look.

The concept of pathological mourning has been around since Sigmund Freud, but it began receiving formal attention more recently. In several studies of widows with severe, long-lasting grief in the 1980s and 1990s, researchers noticed that antidepressant medications relieved such depressive feelings as sadness and worthlessness but did nothing for other aspects of grief, such as pining and intrusive thoughts about the deceased. The finding suggested that complicated grief and depression arise from different circuits in the brain, but the work was not far enough along to make it into the current, fourth edition of the DSM, published in 1994. In the 886-page book, bereavement is relegated to just one paragraph and is described as a symptom that "may be a focus of clinical attention." Complicated grief is not mentioned.

Over the next few years other studies revealed that persistent, consuming grief may, in and of itself, increase the risk of other illnesses, such as heart problems, high blood pressure and cancer. Holly G. Prigerson, one of the pioneers of grief research, organized a meeting of loss experts in Pittsburgh in 1997 to hash out preliminary criteria for what she and her colleagues saw as an emerging condition, which they termed traumatic grief. Their view of its defining features: an intense daily yearning and preoccupation with the deceased. In essence, it is the inability to adjust to life without that person, notes Mardi J. Horowitz, professor of psychiatry at the University of California, San Francisco, and

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another early researcher of the condition. Prigerson, then an assistant professor at the Western Psychiatric Institute and Clinic in Pittsburgh, hoped the meeting would begin the process of finding enough evidence to support changing the DSM. “We knew that grief predicted a lot of bad outcomes—over and above depression and anxiety—and thought it was worthy of clinical attention in its own right,” says Prigerson, now a professor of psychiatry at Harvard Medical School.

A spate of studies since then—not only of widows but of parents who had lost a child, tsunami survivors and others—has further confirmed and refined that initial description. In 2008 researchers got their first hint of what complicated grief disorder looks like at the neurological level. Mary-Frances O’Connor of U.C.L.A. scanned the brains of women who had lost their mother or a sister to cancer within the past five years. She compared the results of women who had displayed typical grief with those suffering from prolonged, unabated mourning. When, while inside the scanner, the study participants looked at images of the deceased or words associated with the death, both groups showed a burst of activity in neurological circuits known to be involved in pain. The women with prolonged grief, however, also showed a unique neural signature: increased activity in a nub of tissue called the nucleus accumbens. This area, part of the brain’s reward center, also lights up on imaging scans when addicts look at photographs of drug paraphernalia and when mothers see pictures of their newborn infant. That does not mean that the women were addicted to their feelings of grief but rather that they still felt actively attached to the deceased. Meanwhile clinical studies have shown that a combination of cognitive therapy approaches used to treat major depression and post-traumatic stress may help some people with complicated grief work through it.

As these and other studies began to pile up, a few researchers turned to complex statistical analysis to validate more precisely the exact combination of features that define the condition. In 2009, more than 10 years after the Pittsburgh panel, Prigerson published data collected from nearly 300 grievers she had followed for more than two years. By analyzing which of some two dozen psychological symptoms tend to cluster together in these participants, she devised the criteria for complicated grief: the mandatory presence of daily yearning plus five out of nine other symptoms for longer than six months after a death [see box at right]. This is exactly the type of rigorous, quantitative study that is needed before a condition makes it into the DSM. “People who meet the criteria for complicated grief do not necessarily meet criteria for either depression or post-traumatic stress disorder,” says Katherine Shear, a professor of psychiatry at Columbia University. “If you didn’t have this disorder [in the DSM], then those people would not get treatment at all.”

The case for diagnosing people as depressed and treating them accordingly when they are still newly bereaved is more contentious. Although some symptoms of grief and depression overlap (sadness, insomnia), the two conditions are thought to be distinct. Grief is tied to a particular event, for example, whereas the origins of a bout of clinical depression are often more obscure. Antidepressants do not ease the longing for the deceased that grievers feel. So in most cases, treating grieving people for depression is ineffective.

A few studies, however, have suggested that mourning may trigger depression in the same way that other major stresses—such as being raped or losing one’s job—can bring about the condition. If so, some people who grieve may also be clinically depressed. It seems unfair, advocates of changing the DSM argue, to make mourners wait so long for medical help when anyone else can be treated for depression after just two weeks of consistent depression. “On the basis of scientific evidence, they’re just like anybody else with depression,” says Kenneth S. Kendler, a member of the DSM-5 Mood Disorder Work Group, which reviews all proposed changes to the manual related to anxiety, depression and bipolar disorder (a condition characterized by extreme mood swings). It is for this reason that the group recently suggested deleting the clause that specifies a two-month wait before mourners can receive a diagnosis of, and therefore treatment for, depression.

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Critics of the move counter that it will lead to unwarranted diagnoses and overtreatment. “It’s a disastrous and foolish idea,” says Allen Frances, who chaired the task force that produced the fourth edition of the DSM. He worries about how the DSM-5 may be used by sales representatives from pharmaceutical companies to urge doctors to write more prescriptions. Indeed, Frances believes that changes in the edition that he oversaw inadvertently sparked an unwarranted explosion of diagnoses for bipolar disorder in children. Prigerson, for her part, predicts a general backlash against the idea that mourners might ever need psychiatric treatment. “There will be vitriolic debates when the public fully appreciates the fact that the DSM is pathologizing the death of a loved one within two weeks,” she says.

In many ways, parsing the differences between normal grief, complicated grief and depression reflects the fundamental dilemma of psychiatry: mental disorders are diagnosed using subjective criteria and are usually an extension of a normal state. So any definition of where normal ends and abnormal begins will be the object of strongly held opinions. As Frances says, “There is no bright line—it is always going to be a matter of judgment.”

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"More Vitamin D Could Prevent Some Psychosis"

Could some cases of schizophrenia boil down to something as simple as vitamin D deficiency? The idea was first put forth more than a decade ago by schizophrenia researcher John McGrath of the University of Queensland in Australia. The circumstantial evidence fit: people born in winter or spring or at high latitudes are at slightly increased risk of developing schizophrenia, and vitamin D deficiency is also more common in winter months and at high latitudes because of lack of sunlight. It may be that a deficit of vitamin D leaves expecting mothers more vulnerable to illnesses such as influenza, which could in turn sensitize the maturing brain to stress-related damage later in life. [For more on how prenatal infections can lead to mental illness, see "Infected with Insanity," by Melinda Wenner; Scientific American Mind, April/May 2008.]

Now McGrath and his colleagues have put the hypothesis to the test. They analyzed blood samples taken from 424 Danish newborns who went on to develop schizophrenia as well as an equal number of babies who never acquired the disease. In each sample, they measured the amount of the chemical 25OHD, which the body converts into vitamin D. The researchers found that infants who had low levels of 25OHD in their blood—and therefore mothers who were deficient in vitamin D while they were pregnant—were at a higher risk of developing schizophrenia when they grew up.

The result, published in the September issue of Archives of General Psychiatry, could be especially interesting for communities of black immigrants living in northern countries. Researchers have found a striking increase in schizophrenia risk for the children of dark-skinned migrants living at high latitudes—a finding neatly explained if vitamin D plays a role, because dark skin blocks ultraviolet B radiation, the component of sunlight necessary for the body to synthesize vitamin D.

There are some loose ends to tie up, however, before recommending vitamin D supplements for at-risk mothers. The group found that infants with high levels of 25OHD were also at increased schizophrenia risk. McGrath speculates that these infants might have been relatively incapable of generating vitamin D, leading to a buildup of the precursor in their blood—but more research is necessary to say for sure. All told, 44 percent of the schizophrenia cases in the study were attributable to either low or high vitamin D levels. "Even if vitamin D supplements can prevent only a small fraction of schizophrenia," McGrath says, "it will be a fantastic outcome."

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"Things That Go Bump in The Night"

Some people wake up at the drop of a pin; others snooze through their alarms every morning. Whether you can sleep through noise has a lot to do with the brain waves you produce while you sleep, according to a new study published in Current Biology. And good news for insomniacs: it might one day be possible to manipulate these waves to ensure a good night's rest.

Previous research has shown that when people sleep, the thalamus—a brain structure that connects the high-level thought areas with the sights and sounds of the outside world—produces brief, high-frequency brain waves called spindles. Scientists speculated that these spindles shut out environmental sounds during sleep. To find out, Jeffrey Ellenbogen, chief of the division of sleep medicine at Harvard University's Massachusetts General Hospital, and his colleagues asked 12 healthy people to spend three nights in his sleep lab. The first night the researchers measured spindle activity while the subjects slept individually in quiet rooms. The second and third nights the researchers relentlessly bombarded each snoozing participant with recordings of common noises such as toilets flushing, phones ringing and people talking, starting each noise at a low volume and repeating it more and more loudly until the subject was aroused from sleep. Then they repeated the process as soon as the person fell asleep again.

The researchers discovered that "the more spindles one has, the more likely they are able to stay asleep when they are confronted with sounds," Ellenbogen explains. Compared with subjects who produced few spindles on the first night, those who produced many had to be bombarded with louder sounds to wake up. Because spindle production dwindles with age, the findings could explain why older people frequently complain of poor sleep.

In future research, Ellenbogen plans to explore why some people produce more spindles than others. Eventually he hopes to find drugs or devices that will boost natural spindle production and induce better sleep. "The capacity of our brain to block out at least some sound is truly amazing, given that our ears are wide open all night long," he says

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"Less Sleep Linked to Blues in Teens"

Despite kids' protests, enforcing early bedtimes may be good for their mental health. Teens who are allowed to go to bed later are more likely to suffer from depression—probably for the simple reason that they are not getting enough sleep, a recent study suggests.

Columbia University scientists found that depression was 24 percent more common in teens whose parents let them go to bed at midnight or later than in kids whose moms and dads required them to hit the pillow by 10 p.m. The night owls were also 20 percent more likely to have suicidal thoughts.

Teens with bedtimes of midnight or later got an average of seven and a half hours of sleep, whereas those with a lights-out of 10 p.m. or earlier got an average of eight hours and 10 minutes. Although the association between later bedtimes and depression was greater before controlling for parents' marital status and poverty level, it remained statistically significant after taking those things into account—as well as teens' perceptions of how much their parents cared about them. The researchers looked at parent-enforced bedtimes—as opposed to simply logging hours slept—to rule out the possibility that depression was causing some kids to sleep less, rather than the other way around.

Earlier work supports the idea that too little sleep may lead to depression. Research at the University of London showed that children who suffer from insomnia are at increased risk of developing depression in their tweens and teens. And a University of Pittsburgh study of youth at risk for hereditary depression found that the one biological predictor of resilience—in other words, not getting depressed—was adequate sleep. Although too little sleep is unlikely to be solely responsible for a teen's low mood, in those with a genetic or environmental predisposition sleep loss may raise risk and satisfying rest may be protective.

Recent studies at Walter Reed Army Medical Center and the University of California, Berkeley, are starting to tease out why. During brain scans, sleep-deprived but otherwise healthy people showed increased activity in the amygdala (the brain's emotional center) and decreased activity in the prefrontal cortex (an area that puts our experiences in context, and by extension, makes us rational)—the same changes seen in people who are depressed. In one army study, subjects started to show symptoms of depression, and the Berkeley subjects became more distressed than rested participants when confronted with upsetting images.

All these neurobiological effects may hit teens especially hard, says psychologist William D. "Scott" Killgore of Harvard Medical School–affiliated McLean Hospital, a co-author of the army research. As teens cope with increasingly complicated daily life, they need more sleep than younger kids or adults, Killgore explains, and so "not getting enough sleep is especially problematic."

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"Ambidexterity and ADHD"

One of the first things that anatomy students learn is that the brain is divided down the center. In most people, one half, or hemisphere, plays a dominant role. Handedness has long been a crude measure of hemispheric dominance, because each side of the brain controls the opposite side of the body. Right-handers, for instance, are likely to have dominant left hemispheres. Today researchers are realizing that studying ambidextrous children (who have no dominant hand) could yield insights into the consequences of an unusually symmetrical brain.

A team of European researchers recently assessed nearly 8,000 Finnish children and showed that mixed-handed children are at increased risk for linguistic, scholastic and attention-related difficulties. At age eight, mixed-handed kids were about twice as likely to have language and academic difficulties as their peers. By the time the children were 16, they also were twice as likely to have symptoms of ADHD—and their symptoms were more severe than those of right- or left-handed students.

Ambidexterity is not causing these problems. Rather "handedness is really a very crude measure of how the brain is working," says Alina Rodriguez, a clinical psychologist at King's College London and the study's lead author. In typical brains, language is rooted in the left hemisphere, and networks that control attention are anchored in the right—but brains without a dominant hemisphere may be working and communicating differently.

Consistent with this theory, a 2008 study by scientists at the University of California, Los Angeles, found anomalies in cross-hemisphere communication in children with ADHD. On tasks that should be the domain of the left hemisphere—such as linguistic processing—children with ADHD seemed to be getting too much input from their right hemispheres. Rodriguez is quick to point out, however, that mixed handedness does not, by itself, indicate a malfunctioning brain and is "just one risk factor among many others."

So why do some kids have overly symmetrical brains? The answer may lie in epigenetics—the mechanism by which environmental influences affect gene expression. In 2008 Rodriguez found that women who experienced stressful life events or depression during pregnancy were more likely to give birth to children who became mixed handed, adding evidence to the idea that the experiences of a mom-to-be affect her fetus's brain development. [For more about prenatal influences on mental health, see "Infected with Insanity," by Melinda Wenner; Scientific American Mind, April/May 2008.] That means that handedness, Rodriguez says, "can be used with other markers to predict who's going to have problems with behavior" and give parents, teachers and doctors the opportunity to intervene at the first sign of trouble.

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"The Origins of Suicidal Brains"

Suicide rates in the U.S. have increased for the first time in a decade, according to a report published in October by the Johns Hopkins Bloomberg School of Public Health. But what leads a person to commit suicide? Three new studies suggest that the neurological changes in a brain of a suicide victim differ markedly from those in other brains and that these changes develop over the course of a lifetime.

The most common pathway to suicide is through depression, which afflicts two thirds of all people who kill themselves. In October researchers in Canada found that the depressed who commit suicide have an abnormal distribution of receptors for the chemical GABA, one of the most abundant neurotransmitters in the brain. GABA's role is to inhibit neuron activity. "If you think about the gas pedal and brakes on a car, GABA is the brakes," explains co-author Michael Poulter, a neuroscientist at the Robarts Research Institute at the University of Western Ontario.

Poulter and his colleagues found that one of the thousands of types of receptors for GABA is underrepresented in the frontopolar cortex of people with major depressive disorder who have committed suicide as compared with nondepressed people who died of other causes. The frontopolar cortex is involved in higher-order thinking, such as decision making. The scientists do not yet know how this abnormality leads to the type of major depression that makes someone suicidal, but "anything that disturbs that system would be predicted to have some sort of important outcome," Poulter says.

Interestingly, this GABA receptor problem is not the result of abnormal or mutated genes. Rather the change is epigenetic, meaning some environmental influence affected how often the relevant genes were expressed—that is, made into proteins. In the frontopolar cortex of suicide brains, the gene for the GABA-A receptor often had a molecule called a methyl group attached to it, the team found. When a methyl group is attached to a gene, it keeps that gene hidden from cells' protein-building machinery—in this case, preventing the cells from manufacturing GABA-A receptors.

The addition of this methyl tag, called methylation, occurs more extensively in rodents that are handled by humans than in rodents that are not. Less is known about what causes methylation in the human brain, but another recent study suggests it could be related to abuse during childhood. In May researchers at McGill University reported that the gene responsible for creating cells' protein-building machinery is more frequently methylated in the hippocampus—the brain region responsible for short-term memory and spatial navigation—of depressed suicide victims who suffered child abuse than in the brains of nonsuicide victims who were not abused.

Again, the researchers do not yet know how problems with protein-building machinery lead to depression and suicide. But "it makes sense that if you have some limited capacity for protein synthesis, you gradually are depriving yourself of building critical synapses," or connections between neurons, which could be important for staying happy, says co-author Moshe Szyf, a pharmacologist at McGill. "Our hypothesis is that there are social events early in life that kind of epigenetically program the brain," he says. He and his colleagues are now comparing the brains of suicide victims who were abused with those of suicide victims who were not abused to see if their methylation patterns differ.

Even in the womb, epigenetic influences can change the developing brain in ways that increase the risk of eventual suicide. In February 2008 a study revealed that baby boys who are born either short or with low birth weight are more likely to commit violent suicide as adults than longer and heavier babies are, irrespective of their height and weight as adults. Similarly, baby boys born prematurely are four times more likely to attempt violent suicide than those born at full term.

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The experiences of prisoners held in solitary confinement—the despair, the disorientation, the hallucinations—are well documented, but laboratory observations of isolated human subjects and the profound effects of extreme confinement are exceedingly rare, in part because such experiments might have trouble getting past institutional review boards these days. But that wasn't the case during the '50s, when Donald O. Hebb, a professor of psychology at Montreal's McGill University, set out to study how sensory isolation affects human cognition.

Hebb had previously examined the effects of visual deprivation in rats as a doctoral candidate at Harvard University. In 1951, he secured a $10,000 grant from the Canadian Defence Research Board to expand his research to human subjects. The results were dramatic. Depriving a man of sensory input, he soon discovered, will break him in a matter of days.

Hebb's experiments went well beyond the level of isolation prisoners typically experience in solitary. He offered male graduate students $20 a day — excellent pay for the time — to stay in small chambers containing little more than a bed. "It would be a bit more than a meter wide and a couple of meters long, probably enough for a table or something," recalls Peter Milner, one of Hebb's former graduate students who is now an emeritus psychology professor at McGill.

At the time, Milner was working on another project for Hebb, but he saw the sensory deprivation rooms firsthand. "They were given food by human beings, and also when they needed to use the washrooms and things they would be escorted there by other human beings. So they weren't completely alone," Milner says. He recalls watching as the subjects were led down the hall to the bathroom clad in frosted-over goggles. "They wore goggles and earphones and [there was] some sort of noise, just white noise, from a loudspeaker," he says.

Prone in their isolation rooms, the volunteers also wore gloves and cardboard tubes over their arms to limit their sense of touch. A U-shaped pillow covered their ears and the hum of an air conditioner further obscured outside noise. "According to his theory, the brain would deteriorate if it didn't have a continuous stream of sensory input," Milner told me. "It was really just a test of this theory, which in any case didn't really hold together much, although these sensory deprivation experiments tended to support it."

Hebb had reportedly hoped to observe his subjects for six weeks. As it turned out, the majority lasted no more than a few days in isolation — and none more than a week. "Most of the subjects had planned to think about their work: Some intended to review their studies, some to plan term papers, and one thought he would organize a lecture he had to deliver," wrote Woodburn Heron, one of Hebb's collaborators, in "The Pathology of Boredom," a 1957 *Scientific American* article describing the experiments. "Nearly all of them reported that the most striking thing about the experience was that they were unable to think clearly about anything for any length of time and that their thought processes seemed to be affected in other ways."

A series of cognitive tests showed that the volunteers' mental faculties were, in fact, temporarily impaired. While in isolation, for instance, the subjects were played tapes arguing that supernatural phenomena, including ghosts and poltergeists, were real; when interviewed later, they proved amenable to such beliefs. They performed poorly on grade-school tasks involving simple arithmetic, word associations, and pattern recognition. They also experienced extreme restlessness, childish emotional responses, and vivid hallucinations. "The subjects had little control over the content" of their visions, Heron wrote. "One man could see nothing but dogs, another nothing but eyeglasses of various types, and so on."

Nor were their hallucinations merely visual: One volunteer repeatedly heard a music box playing; another heard a full choir accompanying his vision of the sun rising over a church. "One had a feeling of being hit in the arm by pellets fired from a miniature rocket ship he saw; another reaching out to touch a doorknob in his vision felt an electric shock," Heron wrote. Inspired by Hebb's work, D. Ewen Cameron, head of McGill's psychiatry department during the 1950s, began employing sensory deprivation as part of a technique called "psychic driving," his unsuccessful attempt to "reprogram" the minds of mentally ill patients, some of whom later sued Cameron, according to Milner. In 1956, Cameron wrote in the *American Journal of Psychiatry* that he would hypnotize his schizophrenic patients "under stimulant drugs and after prolonged psychological isolation."

Cameron's experiments were torture, Milner told me, because unlike Hebb's volunteers, Cameron's subjects were entirely under his control. "They were sick people," he says. "They came to him because they had a mental illness, and his job was to cure them. If they had been day patients they would have not bothered to come back. But because they were hospitalized there wasn't much the patient could do. Hebb thought it was not only stupid, but rather wicked. And he was right."

Hebb's work wasn't driven entirely by academic curiosity. There was a concern during the 1950s that the Soviets were using sensory deprivation to brainwash Canadian POWs in Korea, and the McGill researchers viewed their own work — some of which the Canadian government forbid Hebb from publishing — as an attempt to understand sensory deprivation so that some sort of defense might be devised against it. Yet this type of knowledge was famously put to use as part of the Bush-era program of "enhanced interrogation" (a.k.a. torture) of US detainees. As *The New Yorker*'s Jane Mayer has reported, psychologists versed in techniques of "Survival, Evasion, Resistance, and Escape" — a military program wherein soldiers were exposed to extreme conditions, including isolation, that they might encounter as POWs — were enlisted to advise interrogators at

Guantanamo Bay. According to Mayer's sources, they essentially "tried to reverse-engineer" SERE techniques to extract information from enemy combatants.

In any case, there's a big difference between *voluntary* isolation, however extreme, and the situation in which thousands of American prisoners find themselves today—stuck in tiny cells for an indefinite length of time with minimal human contact and no clear process by which they might earn their way out. "The really scary thing," noted Sara Shourd, one of three Americans taken captive by Iranian forces in 2009, in a recent interview with *Mother Jones*' James Ridgeway, "is that the US government and many governments were very critical of Iran for holding me in solitary for 13 and a half months, but when I got out I was shocked to find that the US had more people in solitary confinement than any other country — and in this country it is used routinely as an administrative practice, not as a very last resort."