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The unhealthy truth about income inequality

As this graph shows, health in developed nations rises with income equality. In “Why Poverty Is Bad for All of Us,” epidemiologist Richard Wilkinson explains why income distribution matters.
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On the Cover
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For more than 40 years, Jhpiego’s mission has been to prevent the needless deaths of women and children. We take the science of Johns Hopkins and develop low-cost solutions for today’s global health challenges.

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Welcome to the inaugural issue of Johns Hopkins Health Review, a new magazine that aims to lead an informed and intelligent conversation about health—the health of your body, your brain, your family, your planet.

Johns Hopkins is a leader in health and understands that a smart, sophisticated discussion on that topic is about a lot more than eating an apple a day and getting some exercise. It involves biomedical engineering, economics, politics, the food system, neuroscience, computational science, global interconnectedness, climate change, population dynamics, and ethical quandaries that didn’t exist 20 years ago.

This magazine is not going to tell you how to lose 10 pounds in 10 days, or get six-pack abs, or make a green smoothie. Rather, it will offer everything from practical ideas about nutrition, exercise, sleep habits, and relationships to long reads about breakthrough scientific discoveries involving our bodies and our minds, our community, and our environment—all evidence-based and grounded in Johns Hopkins research.

In our inaugural issue we explore everything from the mechanics of the common itch [“Why Do We Itch?,” pg. 26], to what happens to our bodies and brains when we are chronically tired [“Cheating Sleep,” pg. 70], to how our understanding of cancer cells changes when scientists work in three dimensions [“Beyond the Petri Dish,” pg. 44]. We also look at poverty’s impact on the health of the poor—and everyone else [“Why Poverty Is Bad for All of Us,” pg. 58]. And that’s just a few of the stories you’ll find in these pages.

Please enjoy the magazine, our compliments. If you’d like to let us know what you think or sign a friend up for a free subscription, visit us at johnshopkinshealthreview.com.
New links between skin conditions and internal health have come to light in recent years. Mary Sheu explains the latest research and gives advice on the best ways to protect your skin.
**What Can Our Skin Tell Us About Our Health?**

**MARIANNE AMOSS**

Your skin can give you clues about your overall health, showing signs of hypothyroidism, diabetes, cardiovascular disease, and even too many carbs.

Not feeling well? You may see it in your skin. “There are well-established connections between internal problems and skin issues,” says dermatologist Mary Sheu, medical director of the Johns Hopkins Dermatology and Cosmetic Center at Green Spring Station. Some are quite familiar: For example, an excess of bilirubin, a waste product in our bloodstreams that is normally filtered out by the liver, causes jaundice and its characteristic yellowing of the skin. A common (and easily resolved) affliction in infants, jaundice also affects adults and can indicate conditions both benign and serious, from gallstones to pancreatitis.

Other connections are more unexpected. One sign of hypothyroidism—an underactive thyroid gland—is hair loss along the outer third of the eyebrows. This condition has been dubbed “Queen Anne’s sign” because a portrait of Anne of Denmark, who married James I of England in 1589, shows her with shortened eyebrows. (It was never confirmed that the queen actually suffered from a thyroid condition, but the nickname for the ailment stuck.)

New links between skin conditions and internal health have come to light in recent years. Sheu points to research that has found a connection between psoriasis and increased risk of cardiovascular disease. Psoriasis—a chronic skin condition that causes red patches of scaly skin that can be itchy or painful—has already been linked to increased risk of diabetes. In both cases, some researchers believe the inflammation that accompanies psoriasis could be the culprit, as it can affect major arteries and cause insulin resistance. “Now I routinely recommend to my patients that, if they have psoriasis, they discuss cardiovascular...
risk factors with their primary physician and get screened for diabetes and heart disease, and try to be in the best cardiovascular health that they can be to prevent problems later on," says Sheu.

When it comes to taking care of skin itself, Sheu has one answer: sunscreen. It’s advice we hear constantly, but we’d do well to heed it, as a 2010 study has shown that correct sunscreen use may reduce the risk of melanoma, the deadliest kind of skin cancer. In the study, researchers followed 1,621 adults in a subtropical region of Australia for five years. About half of them applied sunscreen only when they thought it was needed, while the other half applied sunscreen daily. Ten years after the trial ended, the discretionary sunscreen group reported 22 melanomas, while the daily sunscreen group had only 11. “UVA—which is present year-round, even if there’s cloud cover—might play a larger role in skin cancer development, especially melanoma, than we had previously thought,” Sheu explains.

To combat that, she recommends daily application of a broad-spectrum sunscreen, which protects against both UVA and UVB rays, with an SPF of 30 or higher. “If [patients] do nothing else for their skin, the one thing they can do to improve their health, to prevent skin cancer, and to improve appearance of their skin—both short- and long-term—is to wear sunscreen every day,” Sheu says. “Even if it’s overcast, I recommend that they apply it just like they would brush their teeth.”

Sun exposure doesn’t just cause skin cancer; it also causes skin to age. And so can our diet: Some researchers now say that those high-glycemic foods shunned by adherents of the “glycemic index diet”—white bread, russet potatoes, pretzels—can also age our skin. According to one of the many theories about aging, the consumption of these high-glycemic foods is related to higher accumulation of AGEs, or advanced glycation end products, which are toxins that build up in the body over time and can cause the structural changes we see in aging skin. “These are things that we can definitely have some control over,” says Sheu. “There’s not a lot of control over our genetics, but our diet is certainly something that we can all do something about.”

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MORE THAN SKIN DEEP

1. **Jaundice**
   A yellowing of the skin caused by an excess of bilirubin, jaundice can be a sign of gallstones, pancreatitis, and other conditions.

2. **Hair loss along the outer third of your eyebrows**
   Called “Queen Anne’s sign,” the loss of eyebrows can indicate hypothyroidism.

3. **Psoriasis**
   Itchy or painful patches of scaly skin could mean an increased risk of diabetes or cardiovascular disease.

4. **Premature aging**
   Too much sun exposure causes our skin to age. So might high-glycemic foods. So put on your sunscreen and watch those carbs!
If you’re doing some reorganizing, don’t forget your kitchen pantry, says Gerard Mullin. Your pantry should be a go-to resource for the building blocks of healthy family meals. But for many, it’s a dusty jumble of junk food and old, unmarked who-knows-what. Fall is as good a time as any to pull everything out, toss the garbage, and give the insides a good cleaning. Mullin suggests taking the “Five R” approach: Reevaluate the space (size, shelving, etc.). Rid yourself of junk. Replace bad foods with better choices. Reorganize so everything is easy to reach. Include remedies for ailments in the form of various health-boosting foods. Here are 10 items every healthy pantry should have on hand. /


10 Must-Haves for Your Kitchen Pantry

**SPECIALIST**  Gerard Mullin, Gastroenterologist

---

1 / **Beans**
All types have antioxidants, are anti-inflammatory, high in fiber, and promote colon health.

2 / **Brown Rice**
Rich in antioxidants, vitamins, and fiber.

3 / **Cinnamon**
High in antioxidants, stabilizes blood sugar levels, and may aid weight loss.

4 / **Dark Chocolate**
Reduces hypertension and boosts intestinal immunity.

5 / **Flax Seeds**
May protect against breast and colon cancers; source of fiber and omega-3s.

6 / **Ginger**
Anti-inflammatory, helps with nausea, and may aid weight loss.

7 / **Nuts**
Contain vitamins and minerals that promote heart health and lower cholesterol.

8 / **Olive Oil**
Reduces risks for coronary heart disease, prevents bone loss, and lowers cholesterol.

9 / **Tea**
Promotes cardiovascular health, protects against colorectal and pancreatic cancer, and reduces risk of stroke.

10 / **Turmeric**
Fights inflammation, has anti-cancer qualities, and may protect against Alzheimer’s and digestive disorders.
Vanya Jones
Injury researcher
Johns Hopkins Bloomberg School of Public Health

I WISH THERE WERE...
a way to help older drivers know when it's time to turn over their keys—before they cause some catastrophic crash, or get hurt themselves, or put their family in the difficult position of having to make the decision for them.

Vanya Jones develops programs aimed at assessing older adults' cognitive and physical abilities so that, when the time comes, they can make a reasoned—and comfortable—decision to stop driving. It's complex because not driving might be safer, but it can put a person at risk of isolation. “We just don't know enough to help physicians talk with their patients, for the DMV to make policy,” says Jones, who is developing a driving retirement workbook. “Hopefully, in the next 10 to 12 years, we'll be able to come up with strategies that are solid and grounded in research to help people make an educated decision.”
Survey

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   Freeze-Dried Snacks

WORLD/
24. Urban Gardens
   Supply and Demand
25. Toxic Time Bombs?
   Rewarding Altruism
Diet Soda’s Downside

[NUTRITION]

If your idea of “going on a diet” is switching from regular soda to diet soda, you might want to reconsider. According to new research, overweight adults who drink diet soda are likely compensating (calorically speaking) by eating more food.

“Although overweight and obese adults who drink diet soda eat a comparable amount of total calories as heavier adults who drink sugary beverages, they consume significantly more calories from solid food at both meals and snacks,” says Sara Bleich, an associate professor in Johns Hopkins’ Department of Health Policy and Management and lead author of a paper published by the American Journal of Public Health. If people hope to lose weight by drinking diet soda, they will also need to eat less, she says.

“Overweight and obese adults looking to lose or maintain their weight—who have already made the switch from sugary to diet beverages—may need to look carefully at other components of their solid-food diet, particularly sweet snacks, to potentially identify areas for modification,” says Bleich.

Statins Revisited

[MEDICINE]

Do statin medications cause cognition problems? In February 2012, the FDA ordered changes on their labels, warning about memory problems with short-term use. As a result, some people have been reluctant to take the drugs even though they help prevent heart disease.

Researchers from Johns Hopkins’ Ciccarone Center for the Prevention of Heart Disease conducted a review of the literature, analyzing the impact of short-term statin use on cognitive function and, in studies where patients took statins for more than a year, whether there was a correlation with a later diagnosis of Alzheimer’s disease or vascular dementia.

They found no threat to short-term memory. In fact, when statins were taken for more than a year, the risk of dementia was reduced by 29 percent. “Because of their effect on arteries to reduce or stabilize plaque and prevent strokes,” says senior study author Seth Martin, “it makes sense that statins could be protective in the brain against vascular dementia.”
Asking About Abuse

[Women's Health]

Don't be surprised during your next checkup if your doctor asks whether your spouse hits you. Along with routine questions about diet, exercise, smoking, and alcohol consumption, many physicians are asking female patients about domestic violence.

Whether this encourages women to seek help is up for debate. Success may hinge on how a caregiver asks the questions, according to Nancy Glass, a Johns Hopkins nurse clinician who studies gender inequality, poverty, and violence against women.

A recent study published in BMJ found that domestic violence screening works—at least most of the time. Doctors who ask their patients about violence do indeed find more victims, but the victims don’t always seek assistance.

Yet having these delicate conversations with patients is important, if you ask Glass, associate director of the Johns Hopkins Center for Global Health. She recently told NPR that there are many reasons women won’t report abuse, including fear of further harm, so it’s the duty of the health care community to keep respectfully and privately asking careful questions.

Oh, Well, Resveratrol

[Food]

There’s something in red wine, dark chocolate, and berries that reduces inflammation, but it isn’t the resveratrol.

The antioxidant is definitely in there, but it turns out that an as-yet-unidentified element produces the heart-healthy benefits once attributed to resveratrol, according to a new study led by Richard D. Semba, a professor of ophthalmology at the Wilmer Eye Institute at Johns Hopkins.

So keep the Chianti flowing (in moderation, of course), but don’t say cheers to resveratrol.

Treating Sprains and Strains

[Exercise]

If a new or increased exercise routine has left you wincing in pain from a sprain or a strain, don’t despair.

“Most minor sprains [injured ligaments] and strains [overstretched or torn muscles or tendons] are easy to treat at home,” says Sameer Dixit, a sports medicine physician at Johns Hopkins.

For self-care, he suggests the PRICE technique:

Protect the injured area.
Rest the affected limb.
Ice or apply a cold pack to the swelling.
Compress the area using a bandage.
Elevate the injured limb.

“If you don’t see noticeable improvement within a week, see your doctor,” Dixit says. “If you want a quicker return to activity, rehabilitation and physical therapy can help get you there.”
Skip Those Vitamins

[NUTRITION]

Save your money—and possibly your good health—by skipping the vitamin supplements.

Study after study has shown that taking vitamins won’t stave off chronic diseases or untimely death. So from a medicinal perspective, there is no reason for well-fed people to take them, according to researchers at Johns Hopkins and several other institutions.

It’s not just that the experts have deemed vitamins useless: They’ve also determined that taking bonus beta-carotene, vitamin E, and vitamin A may contribute to a heightened risk of death.

For many lifetime vitamin poppers, this news is hard to swallow. After all, mom doled out that chalky, vaguely fruity multivitamin tablet every morning as part of a nutritious breakfast. It’s time to cast sentimentality aside because the facts show that vitamins are not a magic bullet, even though they might be shaped like one.

Hepatitis C

[DISEASE]

Think hepatitis C should be off your health radar? About 3.2 million people in the United States are infected. The vast majority just don’t know it yet.

Hepatitis C is a viral infection that can lie dormant for decades. “Think of the virus as finding a nice warm spot in your body, and liking it. From the virus’s perspective, why mess that up by hurting your home?” says David Thomas, a Johns Hopkins infectious diseases expert.

Those infected are typically asymptomatic. Over time, however, a chronic infection can scar and slowly destroy the liver, leading to cirrhosis or liver cancer. Heavy alcohol use can worsen conditions.

Historically, the virus is transmitted through intravenous drug use, risky sexual behavior, and dirty tattoo needles. But Thomas warns that others might still have the disease, specifically baby boomers who now account for three-fourths of newly reported infections. As a result, the CDC recommends that everyone born from 1945 to 1965, or who received a blood transfusion or organ transplant before 1992, be screened for the virus.

“It’s a simple blood test, a finger stick. In the past people have said, I’m not a drug user, why should I be tested? Why take any chance when we have the capacity to cure them totally?” A regimen of FDA-approved pills taken for eight to 12 weeks can effectively kill the virus in more than 90 percent of patients.
Friends With Neurotransmitter Benefits

Yo, bro, you really activate my brain’s social reward circuit.

Who you keep in your inner circle of friends might be prompted and reinforced by a synaptic release of oxytocin that then triggers a sprinkle of serotonin, a neurotransmitter linked to feelings of happiness, according to Gul Dolen, an assistant professor in the Johns Hopkins Brain Science Institute. Dolen has studied oxytocin’s role in the social behaviors of mice. It’s been suggested that oxytocin, aka the love drug, is central to posse-forming behaviors that have evolved in species as diverse as humans, singing squirrels, honeybees, and pair-bonding penguins.

Don’t Worry, Be Healthy

People with cheerful temperaments are significantly less likely to suffer a heart attack or sudden cardiac death. A study published last year in the American Journal of Cardiology found that a general sense of well-being reduces the chances of a heart attack.

“If you are by nature a cheerful person and look on the bright side of things, you are more likely to be protected from cardiac events,” says study leader Lisa R. Yanek, an assistant professor of general internal medicine at Johns Hopkins.

Yanek cautions that a cheerful personality is likely something we’re born with, not something we can easily change. Some have suggested that people lucky enough to be born with such a trait are also more likely to take better care of themselves and have more energy to do so. Yanek says her research shows that people with higher levels of well-being do still have many risk factors for coronary disease, but on the whole, they had fewer serious heart events.

“A happier temperament has an actual effect on disease, and you may be healthier as a result,” she says.
Jackpot Fantasies

[CONSUMER BEHAVIOR]

Hyeong-Min Kim, an assistant professor of marketing at the Johns Hopkins Carey Business School, has found that the mere act of buying a lottery ticket triggers materialistic thinking and can make someone throw prudence out the window.

In a series of experiments involving research subjects, some of whom were given lottery tickets and some of whom weren’t, he found that those with a ticket in hand ate more M&M’s than those without one. “Self-control failure means choosing an immediate reward over a larger, longer-term reward,” Kim says. “When people have a chance at a large windfall, say $1 million in lottery winnings, they tend to choose immediate gratification.”

Kim’s study was published last year in the Journal of Consumer Research.

Hurt Less, Sleep More

[PSYCHOLOGY]

People who are in chronic pain often experience trouble sleeping, which means they’re deprived of the healing power of a restful night. It’s a vicious cycle. Luis Buenaver, director of the Behavioral Sleep Medicine Program at Johns Hopkins, believes he has found a way to help manage this situation with cognitive behavioral interventions.

Many people who have chronic pain, Buenaver explains, make the situation worse by dwelling on the problem, magnifying it, and convincing themselves there’s nothing they can do to feel better. This thought pattern, known as catastrophizing, is also linked to insomnia.

“These folks are definitely worrying a lot about their pain, and that’s impacting their sleep, which is making them more pain sensitive,” he says. His research of 214 people suffering from chronic face and jaw pain, published online in the journal Pain, suggests that simple techniques, such as teaching people to identify their negative thoughts and replace them with more balanced thinking, can improve sleep and, by eliciting positive emotions and relaxation, ease pain. “This is a skill people can learn,” he says.
Caregiving’s Just Rewards

Taking on the responsibility of caring for a loved one who is disabled or chronically ill can be stressful and difficult. But it also may be life-prolonging for the caregiver, according to a new study led by David Roth, director of the Center on Aging and Health at Johns Hopkins. Roth analyzed data from a long-range national study of stroke risk factors, and he found that caregivers lived nine months longer over a six-year period, on average, than a matched sample of noncaregivers.

One reason for added longevity may be that, within a family, the healthiest person signs on for the role of caregiver. Another may be that caring for others, and all that entails, is in itself beneficial to the helper.

The challenge now is providing a more balanced view of caregiving and recognizing that caring for a disabled family member is usually not bad for your health. “Providing care to a family member with a chronic illness or disability can be stressful, but this stress is not associated with an increased risk of death in most cases,” Roth says. “Caregiving is also a positive and healthy activity in many families, and it appears to be associated with modest survival benefits for the caregivers.”

Time Out for Young Athletes

Overuse injuries used to happen primarily to professional athletes. Now, they are sidelining a sizable number of preteen athletes. Sports specialization and excessive play are to blame, say the experts.

“Every sport stresses different joints,” says Teri McCambridge, a sports medicine pediatrician and assistant professor of pediatrics at the Johns Hopkins University School of Medicine. “If, each season, you change which joints are taking the most impact, the other joints get a rest.”

Young athletes who play one sport year-round miss the opportunity to rest stressed joints during an off-season.

Too much play within a single season also increases risk of injury. “Some kids are going from a school team lacrosse practice to their club team practice for another two hours. There’s no way they’re going to be able to handle that,” McCambridge says, adding that tournaments also can be troublesome. “Playing five or six games in one day is going to injure your child eventually.”

In a recent statement, the American Academy of Pediatrics quantified the dangers of excessive competitive play. The pediatric authority asserted that children playing organized sports more hours per week than their age—for instance, 12-year-olds who play competitive basketball 12-plus hours weekly—are more likely to incur injuries.

The takeaway message? Maintaining a balanced approach to sports when kids are young may keep them in the games when they’re older.
Keep Calm and Pass It On

[PSYCHOLOGY]

If you have an anxiety disorder, you might be handing it down to your kids without realizing it. Golda Ginsburg, a child psychologist at Johns Hopkins, explains why—and what you can do to prevent it.

How are parents creating anxiety in their children? Research has shown that there is a trickle-down effect: Children of parents who have anxiety disorders are up to seven times more likely to develop anxiety themselves. There is good evidence to show that this is due to a combination of nature and nurture. In some families, there is a biological component. Other parents may be inadvertently causing anxiety in their kids through their own behaviors.

What kinds of parental behaviors are responsible? Parents who frequently highlight all the dangers and threats in the world can make their children fearful. They can model anxiety by saying things like, “Don’t touch that doorknob because it’s full of germs and you’ll get sick.” Parents who are overprotective can subtly communicate to their children that they don’t have good coping skills, and this can cause anxiety.

Can parents do anything to change this pattern? We’re learning that they can. In a pilot study at Johns Hopkins of parents who have anxiety and their children, we provided interventions to some children, and none of those children developed anxiety. Among the children who didn’t receive intervention, 30 percent developed an anxiety disorder. We just completed a larger trial at Johns Hopkins, and the findings are similar.

Are there resources that can help? Interventions focus on both parents and children. First, they help identify the signs of anxiety, such as stomachaches and avoidance of certain situations. Then, they open the door to developing coping skills. Parents might work on not modeling anxiety and not being overprotective. Kids can learn to change their thoughts, such as realizing nothing terrible will happen if they give a wrong answer when they’re called on in class. Early intervention is key to preventing a full-blown disorder. Professional guidance is a good idea if anxiety interferes with daily activities.
Safe at Home

[AGING]

With simple, inexpensive home modifications, some older people can “age in place”—that is, stay in their homes and out of costly nursing homes or assisted living facilities—longer, says Johns Hopkins occupational therapist Ally Evelyn-Gustave. She’s involved with the CAPABLE (Community Aging in Place—Advancing Better Living for Elders) study, which has found that expenses totaling $1,300 per individual can make a big difference.

The key, she says, is tailoring the modifications to the individual and his or her goals, based on an assessment by an occupational therapist. “We look at their everyday activities, how they care for their home, and how they move around in and outside their home,” she says.

Purchases might include rug grips that hold floor coverings in place, antislip strips for bathtubs, second railings on staircases, and chain extenders for ceiling lights and fans. Evelyn-Gustave also recommends strategies such as scooting to the edge of a chair and rocking three times to get up, and navigating stairs by leading with the stronger leg going up and the weaker leg going down.

Teen Obesity Surgery

[WEIGHT MANAGEMENT]

Growing numbers of severely overweight children and teens are turning to weight-loss surgery to reduce their risk for cardiovascular disease and diabetes, and to help address issues of self-esteem and depression.

But kids might not be ready for such measures, says Shawna Mudd, an assistant professor in the Johns Hopkins University School of Nursing. What’s more, she says, guidelines regarding adolescent weight-loss surgery can be inconsistent. Mudd and a colleague conducted a review of literature and found that guidelines vary significantly, particularly when it comes to age, body mass index, and co-occurring health problems.

She did find one area in which experts agree: Weight-loss surgeries could put younger teens and children at risk for significant malabsorption that could affect growth and development. Younger patients may also lack the emotional maturity needed for success because weight-reduction surgery is just one part of a larger process. Weight-loss surgery should be considered only when an adolescent has achieved close to full physical and emotional maturity.
**ADHD Treatment and Your Child’s Weight**

**[MEDICINE]**

About 9 percent of U.S. kids have attention-deficit hyperactivity disorder, and many of them are given stimulants like Adderall and Ritalin to treat it. In fact, ADHD medication is the second most prescribed treatment among children. A study published in the journal *Pediatrics* found that those drugs have an effect on kids’ body mass index. According to the study, children who take stimulants for ADHD experience a slower BMI growth in early childhood, followed by a rapid rebound in late adolescence, typically after they’ve stopped taking the medication. Brian Schwartz, a professor in the Johns Hopkins Bloomberg School of Public Health and the study’s lead author, cautions that we need to pay attention to the role these drugs may play in the development of childhood obesity. “Given the dramatic rise in ADHD diagnosis and stimulant treatment for it in recent decades,” he says, “this is an interesting avenue of research regarding the childhood obesity epidemic, because the rises in each of these roughly parallel one another.”

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**Before You Pack Those Freeze-Dried Snacks ...**

**[NUTRITION]**

How healthy are those freeze-dried fruit and vegetable snacks in the grocery store? Turns out, they’re a mixed bag. Jed W. Fahey, a nutritional biochemist with the Johns Hopkins University schools of Medicine and Public Health, explains. “They’re really just dehydrated fruits and vegetables,” he explains, “and thus, good for you.”

**Downsides?** Some contain added sugar. And overindulging in these fiber-heavy snacks could cause intestinal discomfort, Fahey cautions. But consumers needn’t be dissuaded. “I’d sure rather see kids munching on dehydrated fruits and vegetables than a lot of other products,” he says. The key to picking healthy freeze-dried snacks is reading the label for added ingredients, like sugar. Also, all that fiber means you should consume these snacks in moderation.
Plant a Safer Urban Garden

[URBAN LIVING]

Community gardens offer city dwellers myriad benefits—bringing neighbors together to work on a common project, creating green spaces in an otherwise asphalt-and-concrete environment, even teaching kids where food really comes from.

But hold on a minute, say researchers from the Johns Hopkins Center for a Livable Future. Located so close to industrial sites and heavily trafficked roads, urban gardens can contain heavy metals, petroleum products, and asbestos. People who work or play in that soil, or eat the food grown in it, can be at risk of exposure to those kinds of contaminants.

A new study that appeared online in *PLOS ONE* found that urban gardeners generally understand that they should know how an urban garden site was used in the past, but they might lack the information and expertise to sufficiently determine the history. They also might not know how best to minimize their risk of exposure through things like soil testing.


Supply and Demand

[MEDICINE]

Shortages of lifesaving medicine are the new norm. At any moment, some 300 drugs or more could be in low supply. In a consensus statement published in the journal *Pediatrics*, a group of prominent health care experts has proposed concrete steps for preventing and managing future shortages.

“Some hospitals and health centers are simply better equipped than others to deal with the shortages and have more resources to bring to bear,” says Yoram Unguru, co-author of the document and a faculty member at the Johns Hopkins Berman Institute of Bioethics. This “Blueprint for Action” recommends such measures as the sharing of scarce drugs between health care institutions and developing a central online drug supply database.

The reasons for the shortages are complex but boil down to simple economies of scale. Once a drug can be produced generically, manufacturers might halt or limit production since it’s less lucrative. Some gray-market companies then gobble up the remaining drugs and mark up the cost as much as 650 percent. Most hospitals refuse to buy from these folks, but not all do, which creates an imbalance.

“Our primary aim is to create a kind of ethical agreement and set of practices,” Unguru says, “so that all hospitals can play in the same sandbox.”
Toxic Time Bombs?

[ENVIRONMENT]

In a quest to create more durable plastics, manufacturers have started using something called carbon nanotubes, or CNTs, in products like bicycles, tennis racquets, sailboats, and packaging materials.

Some of these CNT-containing polymers are biodegradable, so they’re “greener” than petroleum-based plastics. Good, right? Maybe not, says Ed Bouwer, a professor of environmental engineering at Johns Hopkins. CNTs are toxic, and they could be released during wear of a product’s surface or during biodegradation. Their ultimate fate and impact will be dependent upon interactions with microorganisms present in landfills, soils, or surface waters.

“CNTs are like fibers. They have thin diameters and long lengths,” says Bouwer. “Their toxicity is analogous to asbestos fibers. They are sharp needles and can cause physical irritation to organisms. One health concern is inhalation and exposure to our lung tissue.”

Bouwer and other researchers want to further study the health risks and determine acceptable levels of CNTs in these products, and how best to dispose of them.

Rewarding Altruism

[SOCIETY]

Conventional thinking on compensating blood donors comes down to this: Giving should be its own reward. For four decades, the World Health Organization has frowned upon offering economic incentives for blood donation.

A recent study published in Science, however, argues that something more than a pat on the back can indeed motivate donors in certain instances and locations. Mario Macis, an assistant professor at the Johns Hopkins Carey Business School, says his team of researchers examined existing studies and conducted field research in Europe, Argentina, and the United States. They found that out of the 19 incentives tested in the field, 18 of these increased donations. T-shirts and supermarket coupons led to 16 percent more donations at American Red Cross drives, and a one-day paid leave was associated with 40 percent more annual donations in Italy. In Switzerland, the offer of a $5 lottery ticket increased donations by 5 percent, and a $10 gift card in the United States increased donations by 50 percent. Only one reward, a free cholesterol test, had no appreciable impact.
why do we itch?

Is itching the same as pain, or entirely different? Are you itching right now? Before scientists can ease itching, they need to figure out what, exactly, it is.

GREG RIENZI
It starts when it wants.

It’s on my right leg now—middle front calf to be exact.

Think of something else. Baseball.
No good. It wins.

It always wins.

It is an itch. (And excuse me while I scratch it.) All humanity can relate to the sensation. We itch every day, sometimes plenty. But why, and just how does the feeling start and stop?

Long overshadowed by its sensation sibling, pain, itching is now getting its due. Within just the last decade, the literature on possible causes of itching and how to find relief has increased exponentially. Temple University dermatologist Gil Yosipovitch, dubbed “The Godfather of Itch,” has vocally led a crusade to classify itching as a disease, not just a nuisance.

In 2001, Yosipovitch organized the first worldwide summit on itching, held in Singapore. Four years later he founded the International Society for the Study of Itch, a multidisciplinary association of clinicians and researchers dedicated to improving understanding and treatment of what science calls pruritus.

“The field is growing, no question,” says Yosipovitch, author of Living with Itch: A Patient’s Guide (Johns Hopkins University Press, 2013). “For me, this is the most exciting time in the 20 years I’ve been in the field. Not that long ago, I could count on one hand the number of professionals interested in itching. Today, there are more than 200 researchers focused on this issue.”

These scientists want to identify the mechanisms of itching (and scratching) and how our brain interprets itch signals and differentiates them from pain, in essence distinguishing the sensation of a brush of a fuzzy sweater from a hand held too close to the fire. A team of Johns Hopkins and Yale molecular biologists and neuroscientists believe they have identified an itch-mediating neuron activated in mice. In a 2013 study published in the journal Nature Neuroscience, the researchers sought to end the Why itch at all? Many call it an evolutionary warning sign to stay clear of itch-inducing toxic plants and insects that might spread diseases like malaria or dengue fever.
decades-long debate on whether humans and animals possess separate pathways for pain and itch, or if those sensations travel along a similar route.

Xinzhong Dong, a professor of neuroscience at Johns Hopkins and senior author of the study, says the findings were significant but fall short of being the final say on the argument. “We now have some compelling evidence that itch-specific neurons exist in the mouse, but how that translates to primates is not clear,” he says. “The sensory neurons that mediate pain and itch in humans are very much alike, so it’s hard to know which one mediates itch or pain, or if they are the same group. We still don’t fully understand the itch, although we appreciate the mystery more and more.”

More than 350 years ago, German physician Samuel Hafenreffer first defined the itch as an “unpleasant sensation” that makes people want to scratch. We itch on our skin and in our throat, but mercifully not internally. Imagine an itchy spleen? The common itch sensation, scientists believe, results from the excitation of nerve endings in our skin. This feeling forms in the cortex of the brain and is encoded in the central nervous system. Some of what we know relies on the groundbreaking work of Glenn J. Giesler, a neuroscientist from the University of Minnesota who recorded spinal cord neuron itch circuits in monkeys and rats. Giesler’s work, published in 2009, tells us that primary sensory neurons fire in the presence of an itchy substance. Those neurons then relay the signal to the spinal cord, thalamus, and then the cortex.

And then we want to scratch.

But why itch at all? Many call it an evolutionary warning sign to stay clear of itch-inducing toxic plants and insects that might spread diseases like malaria or dengue fever. “People generally believe itching provides a protective mechanism,” Dong says. “You feel bad, you scratch it.”

Xinzhong Dong is a professor of neuroscience at Johns Hopkins and author of a study that tried to determine whether itch and pain are the same. “We still don’t fully understand the itch,” he says.
Maybe it removes something from the skin that shouldn't be there. But what if an insect bite or poison ivy is already on the skin? Maybe that's nature telling you to try to avoid the substance, or area, in the future."

All itches, Dong says, are not created equal. They're broken down into two forms: acute and chronic. Acute itch is typically produced by mechanical means like a gentle touch, pressure, or contact with an itchy substance such as wool. Heat and friction can also produce itch, like wearing tight pants or walking into a room with the central heating kicked up high. The colder months yield what is known as “winter itch,” as low humidity levels draw the moisture from our skin, leaving it dry and flaky.

Perhaps the most common type is spontaneous itch, the one you might be feeling reading this story. It's itching without an obvious stimulus—and it's contagious, like a yawn. Dong theorizes that unlike pain nerve fibers, where the body needs a high threshold to fire, our itchy receptors balance on a hair trigger so that even the thought of itching can set them off. “Maybe the itchy fiber is always near the threshold,” he says. “Your guess is as good as mine.”

It's chronic itch that makes someone truly miserable.

In his clinical practice, Yosipovitch has witnessed the debilitating effects of chronic itch, which lasts six weeks or longer and can cause sufferers to scratch uncontrollably and without relief. Some people have trouble sleeping, scientists believe, because their itching intensifies with the evening rise in skin temperature (think indoor heating and being under sheets); studies have also suggested the nerves that secrete itch-causing chemicals are more active during nighttime. Chronic itch sufferers have to live with the embarrassment of constant scratching in public, and may be forced to cover up eroded areas on the skin due to excessive scratching. “Without question, chronic itching can negatively impact quality of life,” Yosipovitch says. “I've seen cases where the urge to scratch is so powerful you can't turn it off. It can be misery.”

The affliction impacts millions, and it's estimated that nearly 20 percent of children and 5 percent of adults have some form of chronic itch, which has multiple causes. A host of diseases—kidney, liver, HIV, leukemia, lymphoma, and others—carry what is called neurogenic
itching as a symptom. Unlike itch activated by primary sensory fibers on the skin, neurogenic itch involves neurons in the spinal cord or brain that somehow get triggered. The result can leave you scratching all over.

Severe burns and nerve damage, as with shingles, can also cause chronic itch. There’s also psychogenic itch, or phantom itching, which happens when you’re not as engaged with your surroundings or otherwise distracted by an activity like talking or walking, or it can be linked to psychiatric conditions such as obsessive-compulsive disorder.

“So itching is very complex, and we are only just starting to learn the very basic mechanism of the itch right now,” Dong says. “You can imagine there are many stimuli, chemical and mechanical, that can induce itch, including itch mediators inside the body like histamine.”

Over-the-counter antihistamines relieve some types of itching and are effective for treating mild allergies and conditions where histamines are the major culprit, such as insect bites and hives. These drugs block the itch receptors and can inhibit scratching—a good thing. For all its pleasures, scratching can make matters worse by damaging the upper layers of the skin, leaving it exposed to other irritants. In the case of poison ivy, scratching spreads the toxin to other parts of you. But the urge to scratch is strong. Studies have found that scratching your ankle can feel as good as sex.

Nearly two-thirds of chronic itch, and some acute forms, however, cannot be treated with antihistamines because, as the name suggests, these drugs can only target histamine receptors. Currently, the medical profession has no good remedies for people who have such severe itching. To help find a cure, Dong and his colleagues wanted to understand how primary sensory neurons—the nerve endings that first detect itchy compounds—work in mice.

“We’re trying to block the sense of itch. If we’re successful, we will probably develop a nonsteroid cream because FDA approval is easier than for a pill or injection.”
capsaicin, the chemical that makes chili peppers hot and painful. They rubbed capsaicin on the mice’s skin and, instead of the mouse equivalent of “ouch,” the animal scratched, demonstrating that these cells transmitted only itching. In another experiment, researchers killed these same nerve cells with a toxin and then exposed the mice to itchy chemicals. The mice scratched much less—compared with control animals in which the itch-sensing neurons weren’t killed—but still received pain signals. The findings gave researchers a new target for clinical treatment. Here were cells that, if we could turn them off, would reduce itch but wouldn’t inhibit the practical ability to sense pain.

The work on itch-mediating nerves found in mice now needs to be translated to primates. That’s the type of work currently being done in the labs of Yale neuroscientist Robert LaMotte and Johns Hopkins neuroscientist Matthias Ringkamp. LaMotte is looking at inflammation on the skin by experimenting with contact allergens, similar to the chemical in poison ivy. His task is to look at how the properties of cutaneous sensory neurons react to this stimulation and then contribute to itch. “We basically want to know how the itch is presented in humans,” LaMotte says. “What is being activated, and what is being turned off when we scratch.”

In Ringkamp’s lab, researchers are investigating electrical activity in cutaneous nerves, which are the ones responsible for providing sensory stimulation to the skin. Bigger picture: Ringkamp wants to find out more about how the central nervous system encodes itch versus pain in humans.

The association of itch and pain comes together neatly in the form of scratching. A prevailing theory is that the act of digging fingernails into skin activates the pain pathways and inhibits the itching response. The pain overrides the itch, so you’re in effect trading sensations. One piece of research suggested that scratching “quiets” nerve cells associated with itch, releasing inhibitor interneurons that stop the nerve cells from firing. “Of course, in some cases, the act of scratching simply removes the irritant,” Dong says.

Perhaps the most common type is spontaneous itch, the one you might feel reading this story. It’s itching without an obvious stimulus—and it’s contagious, like a yawn.
Dong’s hypothesis is that this scratching pain signal activates a bigger population of neurons that drowns out itch. But it doesn’t work the other way around. “We can’t scratch pain away. Only if an itch-inducing signal goes in can we inhibit. Why? That is hard to understand.”

Dong says that once we better understand the basic mechanisms of itch in humans, we can start working on clinical trials. The answers the medical community finds could provide much-needed comfort for those suffering from acute and chronic itch that cannot be treated by antihistamines or topical creams. They have identified some receptors in mice and humans, the genes expressed in the nerve pathways to detect those itchy signals, and currently have a drug development program in the lab to develop blockers for those receptors.

“We’re trying to block the sense of the itch. But it’s a hard job,” he says. “If we’re successful, we will probably develop a nonsteroid cream because FDA approval is easier than for a pill or injection.”

Dong says there has been a general lack of interest from the pharmaceutical companies to develop anti-itch drugs. One reason, he says, is that drug companies think the market is not particularly big. Another is that clinical trials will be difficult because the placebo effect is strong for topical treatments. “When people see something applied to the skin, they feel better, even though there is nothing in it. That can really mask the effect of these drugs.” Finally, Dong says that research into pain tends to get more industry attention. “If you present two projects and one is treating pain and one is treating itch, the drug company will pick the pain every time,” he says.

Dong and his colleagues are currently focusing their research on the spinal cord in mice to see how the itch signal is received from the skin. Having already identified the primary neurons to detect itch information, they now want to identify the secondary neurons involved in this signal chain. It’s a game of connect the dots. “You need a series, or chain, of neurons connected with each other through their nerves in order to have sensory information like itch be relayed to the brain,” Dong says. “A single neuron cannot send the information to the brain. There are at least three to four neurons connected in the series, called a circuit.”

Science is now a few years closer to unlocking all of itching’s mysteries. “Yet in some ways,” Dong says, “we are still at the very beginning of this type of research using mice genetics and molecular approach to studying the mechanism. Previously, most of what we knew was observations. Now, we’re getting down to the details.”

Just don’t think about the itchy details too much. You’ll regret it. /
created a virtual juggling scenario in which participants used a real-world paddle connected to a computer to bounce a virtual ball. They repeatedly bounced the ball up to an area between two lines that were shown on the computer monitor. In some trials, participants used only their vision. In others, they received a brief impulse on their paddle whenever the digital ball hit the onscreen paddle, similar to what they would have felt if a real ball had hit their paddle. Receiving touch, or haptic, feedback proved helpful to participants’ performance—but not exactly how researchers thought it would. “We expected, based on previous literature, "Juggling provides an interesting window into many of the same questions that you try to answer when you study forms of locomotion, such as walking or running.”

Noah Cowan is an associate professor of mechanical engineering who supervised a study on how the brain uses vision and touch to control rhythmic motor coordination because it’s a simpler system to study.”

For their study, which appeared in the *Journal of Neurophysiology*, the researchers and animals move their limbs in a repetitive way, as when they’re running. Their work may eventually help treat people with neurological diseases and could lead to prosthetic limbs and robots that move more efficiently.

Noah Cowan, an associate professor of mechanical engineering (and an amateur juggler himself), supervised the Johns Hopkins research team as it sought to learn more about how the brain uses vision and touch to control rhythmic motor coordination. “Juggling provides an interesting window into many of the same questions that you try to answer when you study forms of locomotion, such as walking or running,” Cowan says. “In our study, we had participants stand still and use their hands in a rhythmic way. It’s very much like watching them move their feet as they run. But we used juggling as a model for rhythmic motor coordination because it’s a simpler system to study.”

Science of Juggling

Chances are that when you think of juggling, you picture circus clowns and street performers, not scientists. But a team of Johns Hopkins engineers is studying juggling to understand how humans...
that people would recover more quickly from their errors with haptic feedback. They didn’t recover faster. Rather, the participants made fewer errors in the first place,” Cowan explains.

“The human nervous system gets feedback all the time from our sense of vision. But the important thing about the sense of touch while juggling is that we get a precise timing cue that complements the continuous visual feedback. This timing cue is very important for us to get the rhythm of the juggling task.”

More significantly, however, the researchers concluded that the brain’s ability to integrate both visual and haptic information improves the overall performance of activities that require rhythmic, repetitive movements.

Applications could go well beyond fine-tuning one’s juggling performance. A surgeon might suture more accurately with some sort of timed haptic feedback input, suggests Cowan. A neurological patient with damage to the cerebellum, which influences motor control, might also benefit. “The question is: How could we augment or enhance sensory feedback, and could it help patients overcome motor deficits more quickly, or learn to cope with them?” // ELIZABETH HEUBECK
The Future Flu

In 2009, the first flu pandemic of the 21st century hit hard. A new strain of influenza called H1N1 affected almost every country and appeared in a population not normally so susceptible: relatively young, otherwise healthy people. Unlike seasonal flu, most people had little or no immunity to it, and, at least initially, there was no vaccine.

It was a frightening time, and it pointed to a real problem: Our approach to viruses is reactive, not proactive. Viruses mutate unexpectedly and sometimes very quickly. Vaccines and treatments that respond to these mutations can take months, or even years, to develop. Predicting how a virus might mutate and affect humans, and devising a treatment for it ahead of time, has not been possible.

Now, though, with a three-year, $20 million investment from Defense Advanced Research Projects Agency, an interdisciplinary team—including Johns Hopkins’ Department of Emergency Medicine and School of Public Health, Harvard University, the University of Michigan, the University of Pittsburgh, and the Donald Danforth Plant Science Center—has taken the first steps toward getting out in front of disease. It’s why the program, which is being managed at the
Johns Hopkins University Applied Physics Laboratory with Harvard as the prime contractor, is called Prophecy.

The team has devised a new way to facilitate and monitor viral evolution. Here’s how it works: Using revolutionary microfluidics technology, the researchers isolate individual viruses in droplets of water. These drops of water are submerged in oil, like tiny test tubes, then each virus is subjected to stress—an antiviral drug, for example—to gauge its reaction. “We can screen [the water droplets] at a rate of a billion a day. If there are rare viruses that have a mutation that has found a way around our drug, we could probably find that in a day,” says Andrew Feldman, DARPA Prophecy project manager and principal investigator in APL’s Research and Exploratory Development Department. “It’s really cool.”

What’s special about the microfluidics approach is that it allows each virus to come into its own, so to speak. In typical lab cultures, viruses compete with each other, and the stronger viruses naturally overpower the weaker ones. Because the microfluidics approach isolates individual viruses, some weaker viruses survive. Why is that helpful? Given the right conditions, a weaker virus could develop into the next uber-virus. And that’s something researchers would like to happen in the lab—before it happens in nature. “We’re going to see all of what would be the favorable mutations to get around our drug before they start occurring in people,” says Feldman.

With all this new data on virus behavior so quickly available, researchers may one day be able to effectively predict which viruses could become future threats. The team is starting to study the human flu virus now and plans to tackle dengue fever before DARPA funding ends this year. The research is in its infancy, but the potential applications of the microfluids technology are many—from screening for drug-resistant bacteria and malaria, to stronger defense against threats of emerging viruses, to better flu vaccines that could protect us from another pandemic. // MARIANNE AMOSS

Richard Huganir is a professor and director of the Solomon H. Snyder Department of Neuroscience at Johns Hopkins and an investigator with the Howard Hughes Medical Institute.

Forgetting Fear

Memories can be one of life’s greatest joys, but for some people who have gone through devastating trauma, a single memory can blight an otherwise happy life and keep them from moving forward. If only there were a way to erase those fearful memories or simply “unlearn” to be afraid. Though that’s the goal of countless programs aimed at treating post-traumatic stress disorder, this condition is often intrac-table despite years of therapy, with patients relapsing after triggers as simple as the sound of a car backfiring.

However, new research led by Johns Hopkins neuroscientist
What is it that embeds fear into our brains? Johns Hopkins neuroscience researchers are examining how to combat the chemical reaction that triggers fear memories. It may help to treat conditions like PTSD in the future.

Richard Huganir shows that forgetting fear is indeed possible and has already been achieved many times—at least, in mice. Huganir, who directs the Solomon H. Snyder Department of Neuroscience, discovered the ability to unlearn fear while studying its flip side, how mice encode and retain scary memories. His lab has long focused on learning and memory in general—a passion for Huganir since at least high school, where some of his earliest experiments were bent on learning more about memory making in goldfish. More recently, his lab has taken on the task of better understanding the basis of fearful memories, such as the deeply ingrained recollections many Americans have of 9/11.

Huganir and his postdoctoral fellow Roger Clem created fearful memories in their mice by playing an audible tone, then delivering a brief, mild electric shock to the animals’ feet. Within a day, mice froze in fear after hearing the tone to prepare for the shock, regardless of whether it came or not. Searching for the mechanism behind how this fear became wired into the rodents’ brains, Huganir and Clem looked at connections between nerve cells in a part of the brain called the amygdala, often referred to as the brain’s “fear center.”

Through tinkering with their mouse models, the researchers eventually realized that the unusual receptors played an important role in fortifying memories.
As in other types of memories, brain cells cement what they’ve recently picked up by changing the strength of their connections to other brain cells at junctions called synapses. Huganir and Clem saw evidence of this strengthening by the presence of familiar receptors for brain chemicals newly present on cell surfaces in the amygdala. But they also saw something completely unexpected: About 24 to 48 hours after mice learned to fear the tone, the researchers detected another, unexpected receptor in amygdala synapses, a rare type called calcium-permeable AMPA receptors. In an even more surprising twist, these new receptors were unstable and short-lived, disappearing within a week of their arrival.

Through tinkering with their mouse model, the researchers eventually realized that the unusual receptors played an important role in fortifying fearful memories. Once the calcium-permeable AMPA receptors completed their usual weeklong stay, the rodents’ fearful memories of the tone seemed fully ingrained. But if the researchers knocked them off prematurely—a feat achievable by activating a different receptor called mGluR1—the animals forgot to be afraid.

Replicating this scenario in human brains would only work for PTSD if patients were treated quickly. “We’d have to treat PTSD like we do for stroke patients, within hours or days after trauma,” Huganir explains. Luckily, some of his newest research suggests that the window for unlearning fear could be extended, perhaps indefinitely. He and Clem have learned that when their fearful mice heard the same tone again weeks after their initial conditioning, the same unusual receptor type reappeared, offering another chance to knock them off and erase fear.

Huganir and his colleagues are currently working with Johns Hopkins’ Brain Science Institute to find drugs that could facilitate this process in people, perhaps eventually to enhance the behavioral therapy already used for PTSD to prevent relapse. // CHRISTEN BROWNLEE

Fat to the Rescue

Of all the primary malignant brain tumors that affect adults, glioblastomas are the most common—and the deadliest. Currently, standard treatments for glioblastoma are chemotherapy, radiation, and surgery, but even a combination of all three rarely leads to more than 18 months of survival after diagnosis. Glioblastoma tumor cells are particularly mobile, migrating across the entire brain and establishing new tumors. This traveling capability is
thought to be a key reason for the low cure rate.

But a tissue type often considered a bodily pariah could hold the key to new and more effective glioblastoma treatments. In laboratory studies, Johns Hopkins researchers, led by neurosurgeon Alfredo Quiñones-Hinojosa, have found that stem cells from a patient’s own fat may have the potential to deliver new treatments directly into the brain after the surgical removal of glioblastoma tumors.

The investigators say mesenchymal stem cells—multipotent stem cells that can be isolated from a variety of tissues, including bone marrow, muscle, and adipose tissue—have an unexplained ability to seek out damaged cells, such as those involved in cancer. This quality may provide clinicians with a new tool for accessing difficult-to-reach parts of the brain where cancer cells can hide and proliferate anew. Furthermore, harvesting mesenchymal stem cells from fat is less invasive and less expensive than getting them from other sources.

“The biggest challenge in brain cancer is the migration of cancer cells,” says Quiñones-Hinojosa. “Even when we remove the tumor, some of the cells have already slipped away and are causing damage somewhere else. Building on our findings, we may be able to arm a patient’s own healthy cells with the treatment needed to chase down those cancer cells and destroy them.” //CHRISTEN BROWNLEE

Stem cells from a patient’s own fat may have the potential to deliver new treatments directly into the brain after the surgical removal of deadly glioblastoma tumors. These stem cells have the ability to seek out damaged cells that may remain postop from the cancer.
Sociologist Stefanie DeLuca is an associate professor at Johns Hopkins. Her research focuses on the way social context affects outcomes for disadvantaged young people.

Smart Move

For more than a decade, Stefanie DeLuca, a sociologist, has studied the impact of housing mobility programs on social inequality: Can families living in impoverished communities improve their situations if they receive assistance to move to better neighborhoods?

Almost since the start of the federal government’s Housing Choice Voucher Program (formerly known as Section 8) in the 1970s, the assumption has been that people would jump at the opportunity to move to a neighborhood with better schools, less crime, and safer streets. Over the past 40 years, though, voucher programs have seen poor people simply move from one bad neighborhood to another.

In 2005, DeLuca, an associate professor at Johns Hopkins, and her research team began following more than 2,000 families relocated through the Baltimore Housing Mobility Program (BHMP), a private contractor-managed, federal court-awarded remedy intervention. BHMP requires families to move to communities where the poverty rate is below 10 percent, the population is less than 30 percent African-American, and less than 5 percent of the population is already participating in housing assistance. BHMP requires families to stay in the new neighborhood for at least two years.

As DeLuca documents in a paper titled “Living Here Has Changed My Whole Perspective” — How Escaping Inner-City Poverty Shaped Neighborhood and Housing Choice,” co-authored with Jennifer Darrah, a lecturer at the University of Hawaii at Manoa, one to eight years later, more than two-thirds of BHMP families were still living in their new neighborhoods. “Once they had a chance to live in high-performing school districts with low crime rates, there were some pretty profound changes in how these parents thought about neighborhoods and schools and what was best for their kids,” says DeLuca, who conducted the research with Darrah.

DeLuca believes BHMP’s success can teach us how to improve voucher programs in the future. But she also thinks the study tells us something important about the way people make choices about their lives.

“There’s this implicit assumption that if you give people choice, that reduces inequality. And then we make assumptions about the choices poor people make, that they make bad ones.”

Almost since the start of the Housing Choice Voucher Program, the assumption has been that people would jump at the opportunity to move.
Families relocated through the privately managed Baltimore Housing Mobility Program have the benefit of neighborhood tours, credit counseling, and walk-throughs of available apartments.

ones”—like choosing to stay in a bad neighborhood—“as if they exist independent of social structure.”

Her study suggests that offering a different set of experiences, including a better social structure, enables people to make better choices instead of just following old patterns. “Whether it would help all outcomes—health, mental health, employment—is still a mixed bag of answers,” she says, adding that this paper is neither the first nor the last set of results coming out of her study. “I think we know, given the status quo [housing] policies, if we gave them some of the features of the Baltimore Housing Mobility Program, it would help families increase their opportunities to live in neighborhoods that are better for them and their kids.” // BRET MCCABE

DeLuca’s study suggests that offering a different set of experiences, including a better social structure, enables people to make better choices.
Our immune system may play a big role in preventing the spread of cancer. Could special immune-boosting cells be sent to the lymph nodes? The first test was whether injected cells would disperse through the body. If so, researchers could then work on adding agents to trigger antibody production.

**Research**

Jeff Bulte’s lab injected cells that carry a fluorine tracer into the tails of mice. The dye would glow under an MRI.

**Result**

Glowing red markers in the mice’s lymph nodes.
Beyond the Petri Dish

For decades, biologists have studied cancer cells in petri dishes. But to a cell, the dish is a two-dimensional environment. Put cancer cells in a three-dimensional environment and everything changes—how they are shaped, how they move, how they behave. That may have critical implications for cancer research.

DALE KEIGER
Galen of Pergamos was a physician with a practice in second-century Rome. Among his clientele was the Roman Emperor Marcus Aurelius. Galen was also a prolific scribbler, so we know a lot about his ideas regarding disease, including his theory of cancer. Cancer, he wrote, was caused by “black bile” that flowed through the body; when it became trapped somewhere, it formed a malignant tumor.

He was wrong about black bile, though it is one hell of a good metaphor. But he was strikingly close to the mark with the flow theory. There are cancers, such as glioblastomas in the brain, in which the primary tumor can be deadly. But for most cancers, the original malignancy does not pose mortal peril. In more than 90 percent of cancers, what kills is metastasis. Cancer cells have a terrifying ability to move through the body and form new tumors in the bones, in the lymph nodes, in the lungs, in the liver and other internal organs. If a physician finds your tumor before the cancer has spread, you may survive. If the tumor has metastasized, cancer will probably kill you. Medicine still cannot do much to counter the flow of black bile.

What if that inability derives, in part, from the fact that a substantial portion of cancer cell biology and cancer drug testing has been hindered by reliance on a ubiquitous piece of lab equipment?

German bacteriologist and military physician Julius Richard Petri invented the
petri dish in 1887. Unless it was invented two years before that by Emanuel Klein, or by a pair of microbiologists, André Victor Cornil and Victor Babeş. Unless it was invented a year before that—we’re back to 1884 now—by English researcher Percy Faraday Frankland. Whatever its provenance, the two-piece flat, cylindrical glassware (frequently polystyreneware now) has been used by scientists for decades to culture and study cells of all kinds, including cancer cells.

Denis Wirtz is a professor of chemical and biomolecular engineering in Johns Hopkins’ Whiting School of Engineering and director of the Wirtz Lab in the Physical Sciences-Oncology Center. He has dedicated the past few years to developing methods of studying cancer cells in three-dimensional environments. In a petri dish, cells are cultured on a substrate so thin as to be two-dimensional. Wirtz believes that this 2-D microworld-in-a-dish so distorts the cells and cell behavior as to cast doubt on a significant portion of critical cancer biology. He and his research team have been creating 3-D matrices that more resemble human tissue, growing cancer cells in them and observing how those cells move about. The difference has been so dramatic to Wirtz that when he talks about it, he becomes an evangelist for cell biology in three dimensions. To figure out metastasis, he says, scientists must work in 3-D. And it would be a good idea to test hundreds of drugs that were deemed failures after testing them on cells in a dish and test them again in three dimensions. Wirtz is convinced pharmaceutical companies may have missed drugs that will work because of their reliance on Herr Petri’s invention.

In journal articles, cancer researchers refer to “the metastatic cascade.” It is a remarkable process. At the disease’s point of origin, cancer cells proliferate and clump, creating the primary tumor and forming blood vessels to nourish themselves in a process called vascularization. Before long, malignant cells begin to detach from the original tumor and move through the surrounding tissue until they run into a blood vessel. Able to deform themselves, they squeeze between the endothelial cells that form the blood vessel’s wall and enter the bloodstream—this is known as intravasation. As the heart pumps the blood, it pumps the cancer cells as well to other parts of the body. The cells tumble and bump into the blood vessels’ walls, and when that happens some of them adhere and push between the endothelial cells once again and exit the bloodstream (extravasation). Now they are inside a lung, or the liver, or some other organ. Malignancies constantly shed cells by the millions, and almost all of them die before they can do more harm. But enough survive the rough ride through the blood to lodge somewhere new and begin the explosive proliferation characteristic of cancer.
Crucial to understanding this process is figuring out cell motility—how cells move. Until about a dozen years ago, researchers commonly studied motility by the straightforward method of growing cancer cells in a petri dish and observing how they moved about. When cultured on a substrate in a dish, the cells typically flatten and move in a slow, seemingly random fashion by pulling themselves along by their leading edges. They also form strong attachments, called focal adhesions, to the floor of the dish. These adhesions consist of proteins that aggregate on the bottom of the cell. Until a few years ago, that was the picture scientists had of how cancer cells move.

In 2010, Stephanie Fraley, then a doctoral student in Wirtz’s lab, was studying motility, but not in a petri dish. She had become curious about the possibly distorting effects of the planar environment of the dish. What would happen if she placed cancer cells in something that more resembled human tissue? From a fibrosarcoma cell line called HT-1080, she took cells and inserted them in a gel prepared from collagen I, the protein that forms most connective tissue in the human body. The gel, formed in a cylindrical well, was only a few millimeters thick, but that was enough to constitute a three-dimensional environment for something as tiny as a cancer cell. Then Fraley watched what happened.

What she saw was startling. For one thing, the cells were no longer flat. They were more spherical, with long protrusions at each end that had not been observed in a dish. The proteins that correlate with the metastatic potential of cancer and, in a dish, were located mostly on the bottom of the cell, now were diffuse throughout the cell. Focal adhesions barely existed. The cells did not crawl along in the laborious, erratic fashion observed in two dimensions but moved rapidly through the 3-D environment by first extending protrusions fore and aft and anchoring them in the collagen matrix, then contracting like springs and releasing one protrusion to snap the cell in the opposite direction.

Fraley’s work suggested that much of how cells behaved in a petri dish was an artifact of the 2-D environment. The proteins that correlate with the metastatic potential of cancer and, in a dish, were located mostly on the bottom of the cell, now were diffuse throughout the cell. Focal adhesions barely existed. The cells did not crawl along in the laborious, erratic fashion observed in two dimensions but moved rapidly through the 3-D environment by first extending protrusions fore and aft and anchoring them in the collagen matrix, then contracting like springs and releasing one protrusion to snap the cell in the opposite direction.

“There was no turning back. You forget that in research sometimes the reason you are doing things a certain way is for convenience, not because it faithfully reproduces what we already know from studying cancer in vivo.”
environment behave so differently from cells grown in a dish, then much of what scientists thought they knew about motility, which is central to metastasis, had to be reconsidered.

For decades, some cell biologists had been thinking about experiments using various extracellular matrices, or ECMs, because cancer cells move through the human body's connective tissue, which is an ECM. As far back as 1980, a paper by three University of California Medical Center researchers in the journal *Cell* noted that tumor cells “are more likely to resemble their in vivo counterparts when maintained on extracellular matrix than on plastic.” A National Institutes of Health investigator, Kenneth M. Yamada, published an influential paper 21 years later in *Science* titled “Taking Cell-Matrix Adhesions to the Third Dimension.” It became the most-cited paper to date by Yamada, whom Google Scholar ranks second among all cell biologists in research citations. Wirtz, too, had been pondering the implications of working in three dimensions. Fraley’s results convinced him that 3-D was going to be transformative for cancer research. “Stephanie’s paper was the trick,” he says. “There was no turning back. You forget that in research sometimes the reason you are doing things [a certain way] is for convenience, not because it faithfully reproduces what we already know from studying cancer in vivo.” Scientists know how to work in a dish. Vital sources of research dollars, like the National Institutes of Health, fund studies of cells in a dish. Pharmaceutical companies have developed sophisticated automated processes that screen cancer drugs by testing them on malignant cells — in a dish.

The petri dish has even influenced the fundamental direction of much cancer research, in Wirtz’s view. Because the two-dimensional environment of the dish lends itself much more to studying cancer cells’ explosive proliferation than motility, that’s what scientists have studied. Wirtz argues that the result has been a diversion from the vital study of how cancer cells migrate. Roger Kamm, another oft-cited researcher and a professor of biological and mechanical research tools work well only in a dish.
engineering at the Massachusetts Institute of Technology, says, “I agree, and would go a little further. It’s not only migration that matters, but all steps in metastasis: epithelial-mesenchymal transition [vital to enabling cancer cell invasion], intravasation, extravasation. And all of these involve studies that cannot be done by standard cell culture techniques. The bottom line is that we now have excellent methods for controlling primary tumors but have precious little knowledge about how to prevent cancer cells from spreading to remote sites.”

“Ninety-five percent of funding from the National Cancer Institute is about tumor shrinkage,” Wirtz says. “Why? Because we can see it! Because then everyone is happy! Pharmaceutical companies are happy; they’re selling stuff. The scientists are happy; they’re publishing, they’re getting funding. The patients are happy, for a while, until it doesn’t work.”

Wirtz adds, “We’ve obsessed about proliferation. The first statement in any textbook about cancer is that cancer is a disease of high proliferation. I say this is completely wrong! Completely wrong! We are discovering that often the very cells that successfully metastasize are those that proliferate the least. We’re not trained to think about metastasis because it’s harder. We have blinders to the point where we don’t even think about blinders.”

Wirtz is slender, boyish in appearance, bespectacled. When he gets excited, his accented English becomes emphatic and he uses his hands a lot. He has formidable instant command of detail when talking about his work but says he is entirely reliant on his assistant, Tracy Smith, to keep him on schedule and tell him where he needs to go. His office and lab are in Croft Hall on Johns Hopkins’ Homewood campus in Baltimore, and one day as he walked to a meeting for which he was already late, he had to ask for directions to nearby Gilman Hall. Teased about this, he held up his hands and said, “I’ve only been here 18 years! How am I to know?”

He works in cancer cell biology but is not a biologist. “I’ve yet to take my first course in biology,” he says. His background is in physics, which he studied as an undergraduate at the Free University of Brussels. He says he picked that course of study because he figured physics afforded the best opportunity for graduate study in California, where he wanted to live for a while. “I figured I’d do that for a couple
of years and then go back to Belgium and be a nuclear physicist, or something like that. I had no intent to stay in the U.S. or apply physics to biology. None of it.” He did indeed make it to California when he went to Stanford for a doctorate in classical physics. His PhD adviser, Gerald Fuller, steered him toward study of the long molecules known as polymers. Looking for polymers to investigate, he veered toward biology. “DNA, to me, was a polymer,” he says. “The laminate that makes up the nucleus was a bunch of polymers. I thought, ‘Wow, there are polymers everywhere! I’m going to have a fantastic time!’”

He studied physics at Stanford within the chemical engineering department, because the university’s physics department was more oriented toward classical physics. When he found a job at Johns Hopkins, it was in chemical and biomolecular engineering. This provided a path into cancer biophysics and connections to Johns Hopkins oncologists, pathologists, and cancer cell biologists who, he says, put him onto the right questions to ask. “There’s a beautiful back-and-forth between biology and physics,” Wirtz says, though 10 years ago he could not have predicted that because physicists had been reluctant to dive into the complexity of cells. “They think there’s beauty in simplicity and bringing phenomena down to fundamental interactions and universal behavior.” Cells vary in such complex ways, universal behavior is hard to discern. To a biologist, there’s beauty in complexity; physicists took a different view, Wirtz says. “We hate the alphabet soup of proteins. We hate this diversity of cells. I don’t; I love it, but most engineers and physicists tend to be kind of pushed away from it. It’s too bad because physicists and engineers are the best trained to handle complexity and extract what really matters.”

Wirtz looks at cancer metastasis as a mechanistic process involving forces that engineers and physicists are well-equipped to study. For example, the extracellular matrix that cancer cells inhabit subjects them to confinement forces, especially as the cells proliferate and become more densely packed in the tumor. When cancer cells force their way through the walls of blood vessels to enter the bloodstream, that subjects the cells to more compression forces, as does the reverse process of the cells pushing out of the blood vessels into other organs or tissues. During their migration through the circulatory and lymph systems, the cells are subject to shear forces. All of that can be studied as physics and engineering. “Ask biologists what are the units of force, and they haven’t a clue,” Wirtz says. “They know what a force is, of course, but they don’t know how to measure it.” Enter the physicists. Wirtz recalls attending meetings of the

“We’re not trained to think about metastasis because it’s harder. We have blinders to the point where we don’t even think about blinders.”
American Society for Cell Biology 10 or 15 years ago and finding two or three physicists like him. Now when he attends, he finds several hundred, he says, and more and more biologists who are learning physics.

The Wirtz Lab currently has a dozen doctoral students and seven postdocs; among them are electrical engineers, biophysicists, biologists, and a physician. He has turned the focus of the lab entirely toward the study of cancer cells in 3-D, with what he describes as dramatic results. For example, when a tumor grows in a human body, the tissue around it tends to stiffen. Often this stiffening is what the fingers detect when someone first finds a lump that turns out to be a malignancy. “Cells in 2-D dishes tend to migrate toward stiffer surfaces,” Wirtz observes. “The cells have a sense of touch called mechanosensing, and cells on flat surfaces love to go from soft, pliant surfaces to a stiff surface. That’s called durotaxis.” But that poses a paradox: If cancer cells prefer stiffer surfaces, and tumors stiffen the tissue around them, why do cancer cells migrate to softer tissue in the body to form new tumors? Why don’t they stay in the stiff collagen capsule that is home? When Wirtz observed migration in three dimensions, cancer cells did the opposite of what they do in a dish: “Cells will tend to go to the softer part of a 3-D gel from the stiffer part. So what we had learned in 2-D does not translate in the 3-D case.”

Scientists have long described the movement patterns of cancer cells as following a “persistent random walk model”—a sort of slow wandering without direction—because that is what they observed in petri dishes. A paper published in March by Wirtz and three co-authors in *Proceedings of the National Academy of Sciences Early Edition* announced that when they studied fibrosarcoma cells in a 3-D matrix, the cells followed direct, almost straight-line trajectories. In a press release about the study, Wirtz said, “This gives them a more efficient way to reach blood vessels—and a more effective way to spread cancer.”
Much of the Wirtz Lab’s research in the last few years has been figuring out how to do the research. “Life is hard in 3-D,” he says. “Every measurement that we take for granted in 2-D, like how to measure the protein content of a cell, or protein activity, or protein localization, the shape of cells, cell motility—all of those become so much harder, if not impossible.” Conventional electron microscopy becomes impossible in three dimensions. To achieve high magnification and high resolution, you have to work at very short distances of under one millimeter. Comparable three-dimensional microscopy will require a lens that does not yet exist. With current technology, researchers can’t even locate the nucleus or mitochondria in 3-D. “It’s just a moving blob of light, basically,” Wirtz says. “We don’t have the subcellular resolution that we have come to take for granted in 2-D.”

He is especially interested in how malignant cells create and exploit pathways out of the original tumor site and through the body to secondary sites. The tissue matrix in which a tumor begins to form may feel soft and pliant to the touch, but at the cellular level it’s dense, and this density inhibits the proliferation and motility of cancer cells. But those cells somehow effect structural changes in the collagen. Ordinarily, within collagen are fibers that are evenly distributed with no favored orientation; they are every which way. Cancer cells aggregate the fibers and orient them into thick strands that Wirtz calls “freeways from the tumor to the blood vessels.” To travel these freeways, the malignant cells must still get through the dense collagen matrix.

So they secrete enzymes that have no effect on the fibers but digest the surrounding collagen, clearing the way. It’s as if the cancer cells were hiking through a jungle, and to follow the path at their feet, they hack through the dense jungle growth. All of this needs to be studied in three dimensions, Wirtz says, because none of it happens when cells are grown in a petri dish.

When the topic moves to drug screening in 2-D environments, he says, “Before drugs are tested in clinical trials, the way pharmaceutical companies identify new compounds is to develop thousands, tens of thousands, hundreds of thousands of compounds, and then subject cancer cells to them in dishes to see how proliferation is affected. But that’s presuming the 2-D case is somewhat relevant to cell proliferation in 3-D.”

This brings him to the example of paclitaxel, a chemotherapy drug marketed as Taxol. Tested on cells in dishes, it was found to have little effect on motility but some ability to inhibit cell division when applied in high doses. So it has been used to treat ovarian, breast, and lung cancers for 50 years. Test paclitaxel on cells in a 3-D matrix, Wirtz says, and you get much different results. In 3-D, the drug has

He is especially interested in how malignant cells create and exploit pathways out of the original tumor site.
little effect on tumor growth but does inhibit migration. These results, Wirtz argues, suggest that the drug should be rethought. Stop using high dosages in an often futile attempt to stop growth of the primary tumor, and start using more targeted dosages to inhibit metastasis while the primary tumor is attacked by better weapons.

Wirtz says, “When you do a conventional drug screen, you end up with maybe 10 candidate drugs that are really the big killers of cancer cells in 2-D. All right. Then you move on into years of development not only to produce this in large quantities but do clinical trials in mice, blah blah blah blah.” Next are hugely expensive human trials. “Then eventually most of the drugs fail. So there is all that lost development cost, based on that very first promising 2-D screen.” A 2-D screen that, perhaps, could not produce meaningful results because the dish imposes so many changes on cell morphology and behavior. Conversely, a bad outcome in a 2-D experiment could lead to rejection of a drug that actually might be found to halt metastasis when tested on cancer in three dimensions. Wirtz believes many drugs abandoned after failed petri dish tests should be revived and tested again in 3-D matrices. There might already be something out there that can stop a prostate or breast tumor from exploding throughout the body.

Wirtz is moving full speed ahead on his conviction that three-dimensional matrices are essential to cancer cell biology. He is not alone in this conviction. Donald E. Ingber of the Wyss Institute at Harvard wrote in the journal Trends in Cell Biology that 3-D microenvironments still require validation but “could have profound effects on drug discovery and environmental toxicology testing.” (Ingber has been working on a marriage of microbiology and microtechnology to produce more sophisticated matrices he calls “organs-on-chips.”) Kenneth Yamada continues to work on and extol the value of 3-D matrices. At Johns Hopkins, a number of researchers are engaged with Wirtz on various research collaborations.

Winston Timp, an assistant professor of biomedical engineering in the Whiting School, says, “It is vastly underestimated how important this is, especially when it comes to motility. A 3-D microenvironment lets us bridge this gap between information we have from 2-D and the animal or human model. The more we can do in an in vitro environment faster and more effectively, the more we’ll be able to get to better drugs to help human
health, better screening that identifies diseases, and more information for plumbing the unknown in biology.”

Various researchers point out how much more complicated the science becomes when you add the third dimension. “The beauty of the petri dish is that it is a reductionist approach,” Timp says. “It’s easy to do and you get results which make sense. When you move stuff to three dimensions, everything becomes a lot more complicated.” Technology and protocols relied on for many years no longer work well. The number of variables that need to be controlled for in 3-D scales up fast. And while some commercially available 3-D matrices now in use greatly improve on the dish, they remain a long way from replicating the remarkable complexity of actual human tissue (though some research groups are getting closer, Timp says). Stephanie Fraley, now a postdoctoral research fellow in the School of Medicine, further cautions that collagen gels and other experimental matrices may create artifacts of their own that distort observations and research results. And a model will always just be a model. As John Isaacs, a professor of oncology and cancer biology in the School of Medicine, says, “I have always lived by the belief that ‘a model is a lie which can tell you a lot about the truth.’”

Timp, for one, isn’t ready to toss out every petri dish in the lab. “It makes sense to do the easiest experiment you can that will give you the information you want,” he says. “So sometimes there’s value in doing things in 2-D first. That said, validation in a 3-D environment is still a vital confirmation that the 2-D experiment is real.” Then he laughs and adds, “You have to avoid the tendency to do the shiniest experiment. Because 3-D is cool, there’s no question about it. It’s incredibly cool.”

Scientists commonly face a fundamental dilemma that Wirtz cites in talking about cancer cell biology: There is what can be measured, and then there is what should be measured. Andrew J. Ewald, an assistant professor of cell biology and oncology in the School of Medicine, says, “I think Dr. Wirtz is exactly right when he says that scientists do what they can get funding to do, and that enough biology is turning out differently in 3-D tissues than in 2-D petri dishes [to indicate] we have to revisit a large fraction of our existing conclusions. The challenge for funding is that this often means proposing to retest existing models and hypotheses, something study sections [that review grant applications] are loath to do.”

Wirtz says, “I am not in the business of wanting to prove people wrong. We are not in the business of dismantling knowledge. But here we are, years into it, discovering again and again how many things we took for granted.”
Do Brain Fitness Games Really Work?

BY GREG RIENZI

Consumers have been downloading smartphone and tablet apps such as Luminosity and Fit Brains that claim to boost cognitive ability and otherwise ward off mental decline. But do they actually improve memory, alertness, and focus?

“In essence, playing these games makes you better at, well, the game itself,” says David Linden, a professor of neuroscience at the Johns Hopkins University School of Medicine. “The effects are very small and don’t last very long.” Linden equates the effect to feverishly preparing for the SAT exam (as his son is doing). You get better at SAT-form tests, for a limited time.

Luminosity and Fit Brains, similar in concept with both free and monthly subscription versions, run the user through a gamut of quick brainteasers that test attention and memory. You match pairs, recognize patterns, and recall images and shapes. The more correct answers in a given time span, the higher your score. These types of games typically exercise short-term memory, which tends to decline as we age, Linden says.

Some studies suggest these games can have a small, generalized effect on cognitive function in the elderly. But the data is suspect, Linden says. “Many of the studies have limited data or involved those with strong ties to the financial interests of the game itself. At this point, I’m willing to believe that you can create a brain game that would have some reasonable benefit. But, to date, the evidence is weak.”

What about the assertion that these games help a 70-year-old regain the memory function of a 50-year-old? Sounds impressive, Linden says, but in reality there isn’t that much difference. “It’s really a small effect, if any at all.”

How, then, to keep your brain fit? Linden has two words: physical exercise. Walking 30 minutes a day has a greater effect than playing any brain game, he says. “I’m not talking killer exercises or running marathons. Even moderate, low-intensity exercise is five times more beneficial to cognitive tasks than playing brain games on your iPad,” he says.

Research has shown that exercise can increase the volume of the brain and elevate patterns of activity. The effects can be particularly beneficial for people who are middle-aged, making them better at real-world tasks like finding a car in the mall parking lot or recalling a phone number.

As for brain games, Linden has some final words on their benefit. “I work in a department of neuroscience with a few dozen men and women in their middle age, and guess what? Zero of us are doing these brain games,” he says. “If my colleagues thought [these games] would benefit our mental ability, we’d all be at our desks playing them right now. I mean, c’mon, we’re computer and brain geeks. This would be right up our alley.”
“At this point, I’m willing to believe that you can create a brain game that would have some reasonable benefit,” says David Linden. “But, to date, the evidence is weak.”
why poverty is bad for all of us

Income inequality is harmful to your health—whether you’re rich, poor, or part of the ever-shrinking population living between those two extremes.

LAWRENCE LANAHAN
few miles up the road, among the Victorian homes with wide wraparound porches, it’s 83 years.

Look closely at those life expectancy patterns and you’ll see them track with race and class. In America, the numbers will tell you: Whiteness and wealth mean better outcomes on many indicators of health.

But being well-off doesn’t necessarily make you well. Income inequality may be driving Americans further and further apart economically, but their lives continue to intersect through policy and in our communities. The consequences of growing poverty and inequality create tension that can be felt physically by the rich and poor alike. With every effort made to reinforce the social and geographical distance between more well-off Americans and the problems they see in poorer communities, that tension grows. And it’s making us all less well.

The afterschool art program was done for the day, and the children filed into the van. I went along for the ride as we took them back to their homes in West Baltimore, past blocks of boarded-up row houses.

As we pulled to the curb to drop off one little girl, I saw a man in dirty clothes stumbling down the sidewalk. The girl stepped out of the van right in front of him. He continued his awkward ambling just steps behind her. The longer the man followed the girl, the more I tensed up. She walked in a door. He walked in behind her.

He lives with her, I realized. The door closed.

I lived less than two miles away, but the distance felt much greater. Life expectancy in the little girl’s neighborhood is 68 years. It’s 75 years in the neighborhood of well-kept brownstones I went home to later that day. Just a

“I’m glad I’m living in the land of the free

Where the rich just get richer

And the poor you don’t ever have to see.”

RANDY NEWMAN, “THE WORLD ISN’T FAIR”
There’s no question that poverty is bad for poor people, particularly where their health is concerned. There is a growing body of research into the so-called “social determinants of health”—income, access to health care, food security, public school conditions, racial discrimination, and any other social condition, policy, or distribution of economic resources that can affect a person’s health. They’re a priority for the World Health Organization, which recently created a global plan of action to address them, and the Centers for Disease Control and Prevention, which has several initiatives, including the Racial and Ethnic Health Disparities Action Institute. One report cited by the CDC claims that social determinants actually have a greater effect on health than do genes, medical care, and health behavior combined. Poor people are more likely to have low birth weight at the beginning of life, the end of life is likely to come earlier, and they have more asthma, heart disease, and diabetes in between.

Researchers, however, have been investigating not just the effect of poverty on the health of individual poor people, but what happens to the health of entire societies when the rich get richer and the poor get poorer. British epidemiologist Richard Wilkinson has spent three decades studying the effects of income inequality on population health. As income inequality increases in rich nations, he argues, so do other health and social problems, among them infant mortality, low life expectancy, incarceration rates, and social mistrust. And that’s not just among the poor—that’s everybody. “America is one of the most unequal countries in the developed world,” he says. “And it does worst in terms of almost all the health and social problems.”

In The Spirit Level: Why Greater Equality Makes Societies Stronger, published in 2009, Wilkinson and co-author Kate Pickett compare Swedish health to that of England and Wales, the former having a more equal distribution of income than the latter two. He found that lower-class Swedes had lower death rates than those in the upper classes of England and Wales. He also found infant mortality to be lower in Sweden than in England and Wales at all levels.

One theory about the consequences of inequality on population health posits that if you’re at the bottom of the economic ladder with no hope of climbing up, anxiety and resentment can create a lifetime of debilitating stress and lead to coping mechanisms like smoking or drug abuse. Wilkinson cites a 2004 study that found the release of cortisol,
a hormone associated with stress, elevated for tasks related to “social evaluative threat”—the feeling of being rejected or judged by others.

Wilkinson sees this effect not just on the poor, though—he says it’s on all rungs of the economic ladder. “In a more unequal society,” he says, “where some people seem so important and other people seem almost worthless, I think we judge each other more by social status. I think we all get more worried about how we’re seen and judged, and there are lots of signs of that. Money becomes even more important, so people work longer hours and get into debt more and save less, because money becomes more important as a way of showing what you’re worth.”

“America is one of the most unequal countries in the developed world. And it does worst in terms of almost all the health and social problems.”

Earlier this year, a Baltimore resident and blogger named Tracey Halvorsen, who lives in one of the city’s recently gentrified neighborhoods by Patterson Park, wrote a post called “Baltimore City, You’re Breaking My Heart.” In response to several brutal crimes in her neighborhood—including a mugger knocking a man’s teeth out with a brick and burglars stabbing a woman to death in her park-front home—Halvorsen wrote that she is now scared to visit the park even during the day. She wanted to stay in the city, she said, and she made a long list of things she loves about living there. But the blog’s subhead made clear what was at stake: “This is why people leave.” The post went viral in Baltimore.

Halvorsen advocated for police to start arresting people in her neighborhood for minor offenses like littering, arguing that the offenders would eventually take their problems elsewhere. Some Baltimoreans criticized her “us versus them” mentality—including me. I pointed out that police had already tried “zero tolerance” quality-of-life policing, which they dropped after a lawsuit pointed out that in one year, they had made 100,000 arrests in a city of less than
650,000 residents. An “us versus them” mentality, I thought, undermined the fact that even people in Baltimore’s poorest neighborhoods care about quality of life and home values.

Yet an “us versus them” mentality prevails, not just in Baltimore, and the problem goes beyond urban environments. Across the country, middle-class neighborhoods—where Americans of a wide range of economic backgrounds go to the same schools, attend the same churches, and socialize in the same civic and fraternal organizations—are disappearing. According to US2010, a peer-reviewed research project on how America is changing in the 21st century, in the 1970s, two-thirds of American families lived in middle-income neighborhoods. Now less than half do. It’s hard to argue that a healthy community is one in which the most “comfortable” residents are actually so uncomfortable that they are retreating to exclusive neighborhoods or, like Halvorsen, are at least hatching escape plans.

Those with the greatest means are increasingly retreating behind locked gates. The number of American housing units in gated communities rose by 4 million between 2001 and 2009. “It used to be that only in the poorest of countries people lived in locked and gated communities,” says Robert Blum, director of the Johns Hopkins Urban Health Institute. “But increasingly, because of the recent economic disparity and the consequent sense of vulnerability, we have a proliferation of locked and gated communities to try to give those of us of higher income the illusion—or delusion—of safety.”

Social epidemiologist Ichiro Kawachi, chair of the Department of Social and Behavioral Sciences at the Harvard School of Public Health, has found that ruptures in the social fabric—memberships in social organizations, a sense that neighbors look out for one another and can be trusted—are correlated with mortality, and that mistrust is higher where income inequality is greater. Amid this social disorganization, his theory goes, mistrust fills the growing chasm between the “haves” and the “have-nots.” As middle-class neighborhoods disappear, so does the common ground upon which social bonds can grow between those on all rungs of the economic ladder.

The idea that social bonds make us healthier—and that a lack of them makes us less healthy—is not necessarily new. A 1999 study published in Sociological
Forum by sociologist David Williams and Chiquita Collins, currently associate dean for diversity and cultural competence at the Johns Hopkins University School of Medicine, found that in cities where blacks and whites “had little contact,” both groups’ mortality rates were higher. Citing a collection of 10 studies, a 2001 Russell Sage Foundation review that looked at the way social capital (the benefits of strong social relationships within a community), poverty, and community health all worked together showed that an equitable distribution of resources “translates into a lower burden of mortality for all members of the community.” The review also noted that “the positive contribution to health made by social integration and social support are said to rival in strength the detrimental contributions of several well-established biomedical risk factors” like smoking, obesity, and elevated blood pressure.

In the 1990s, the U.S. Department of Housing and Urban Development used a lottery to offer poor families in several cities housing vouchers to move out to mixed-income areas. A 2012 study of the Moving to Opportunity program found that even though such moves didn’t improve a family’s economic well-being, the program had a positive impact on people’s physical and mental health, including obesity, diabetes, and “self-reports of subjective well-being.”

Thomas LaVeist will tell you that poverty has real economic costs. A Johns Hopkins health policy professor and director of the university’s Center for Health Disparities Solutions, LaVeist co-authored a 2009 report for the Joint Center for Political and Economic Studies called The Economic Burden of Health Inequalities in the United States. He and two other researchers calculated that American health inequalities had a direct medical cost of over $230 billion between 2003 and 2006, plus $1.24 trillion in indirect costs from lower productivity and premature death.

The result, LaVeist says, is a workforce that is less healthy than it should be and an economy that is less productive than it should be, leaving us with what he calls “opportunity costs” affecting everyone. “Resources that are used to try to address inequalities are not available to use for other things,” LaVeist says. “If you’ve got people showing up in the emergency departments with illnesses
that shouldn’t necessarily need emergency care, that affects the ability to bring through the system people who really do need emergency care.”

Arguments for addressing those inequalities have, for the most part, been focused on social justice, LaVeist says. “It’s the right thing to do; it’s a shame; we shouldn’t do that.’ What we wanted to do was to create another strain of argument for why we should devote resources and to move beyond simply tugging at people’s heartstrings to say, ‘No, the cold, hard reality is that it is taking resources out of the economy, and it’s expensive to maintain disparities, and everyone’s affected by them.’”

But the economy is not the only thing at stake. In his book One Nation, Underprivileged: Why American Poverty Affects Us All, Mark Rank acknowledges the inefficiency of money “spent on the back end of the problem rather than on the front end.” But he also believes growing poverty and inequality are incompatible with fundamental American values. “The words ‘liberty and justice for all’ take on a hollow meaning when a significant percentage of the population is economically and politically disenfranchised,” he writes. “This undermines every citizen, for it suggests that the American ideals in which we profess to believe apply to some more than others. This contradicts the very core of the American promise, diminishing us all.”

At the end of the Economic Burden of Health Inequalities report, LaVeist and the other authors acknowledge that their analysis shows “social justice can be cost-effective.” But, they write, “sometimes the tremendous human suffering of health inequalities can be obscured by analysis such as was conducted for this report. “It is not our intent that the utilitarian argument replace moral deliberation or the application of social justice,” they continue. “We should address health disparities because such inequities are inconsistent with the values of our society. Addressing them is the right thing to do.”
MedTech

LOW-TECH TO HIGH-TECH SURVEY
Just some of the ways Johns Hopkins researchers are using technology to advance health and health science.

1 / Beyond Kisses
Could mistletoe be the latest weapon in the war on cancer? The Johns Hopkins Kimmel Cancer Center is studying mistletoe extract, which may boost the immune system and diminish chemotherapy side effects. (ALTERNATIVE MEDICINE)

Tongue Depressor
Doctors have been brandishing these humble wooden paddles for centuries, though 18th- and 19th-century versions were also made of bone, ivory, nickel, and silver.

2 / Brain Scan Bank
Johns Hopkins engineers and radiologists are building a searchable image bank of MRI scans from children. The cloud-based digital archive currently holds 5,000 images of brains sorted into 22 brain disease categories such as chromosomal abnormalities, congenital malformations, vascular diseases, and psychiatric disorders. Researchers described the image bank as Google for pediatric brain scans. (PEDIATRIC NEUROLOGY)

3 / Tweeting the Flu
When people get the flu, they often say so on Twitter. Researchers from Johns Hopkins and George Washington universities have been able to analyze Twitter data to track the spread of flu in New York City, which could be a model to help health care workers in other locales prepare. (SOCIAL MEDIA)

Medical Resident
SATISH MISRA
The Johns Hopkins Hospital
Satish Misra, a third-year medical resident at the Johns Hopkins Hospital, was one of the creators of iMedicalApps.com, a one-stop website for physician-led reviews of medical apps, separating cool tools from quackery. The site reviews an app’s data, discloses any conflicts of interest, and assesses its overall advantages versus potential harm.
Atlas Knows
Atlas, a wrist-mounted fitness tracker due out later this year, tracks exercises in three dimensions. It analyzes movement and can tell the difference between pushups, curls, and squats.  
(FITNESS)

Pocket Diet Coach
If you have a smartphone, you have a diet coach. The Johns Hopkins Bloomberg School of Public Health’s pilot program TRIMM—Tailored Rapid Interactive Mobile Messaging—sends personalized weight-loss messages to the phones of registered users, typically three or four times a day. In a six-month trial, TRIMM participants lost more weight than those who received an initial assessment and dietary advice, but not the daily dietary coaching.  
(WEIGHT MANAGEMENT)

Selfie-Medicating
Johns Hopkins technology is behind a new app called miDOT that reminds patients to take their pills on time and enables them to shoot video of themselves in the process so they can share the footage, along with notes about symptoms, with their doctors. The app is in the pilot phase.  
(PATIENT CARE)

Light Hearted
Defibrillators can jolt troubled hearts back to regular beating with strong bursts of electricity. Lives can be saved, but the process can damage tissue—and be painful. Biomedical engineers from Johns Hopkins and Stony Brook universities are exploring the possibility of using blasts of light instead. The process uses special light-responsive proteins that can create electricity in response to light in a way that is much more targeted than the broadly delivered zap from a pair of paddles.  
(HEART DISEASE)
Why Your Food Is Still Not Safe

If we want our food to be safer, our government policies need to be smarter.

Each year, 48 million Americans suffer from illnesses caused by dangerous microbial pathogens lurking in the food they eat. For most people, food poisoning leads to temporary stomachaches or diarrhea. But the effects can be much more serious. According to the Centers for Disease Control and Prevention, more than 125,000 Americans are hospitalized and 3,000 die each year from pathogens in our food. Foodborne illness is estimated to cost more than $75 billion a year for health care and lost time on the job for people who get sick.

Most of us believe that the United States fixed these problems more than a century ago after Upton Sinclair's famous book The Jungle revealed the ghastly facts about unsafe methods of commercial food processing for a mass-market economy. But in fact, the rules and regulations we assume will protect us are inadequate. Duplication and gaps in government responsibilities leave Americans highly vulnerable to a variety of risks from industrial food production.

In 1906, Congress took important steps toward protecting consumers by passing both the Pure Food and Drug Act and the Meat Inspection Act. The two laws

Adam Sheingate is an associate professor in the Department of Political Science at the Johns Hopkins University. This article is adapted from the original published by the Scholars Strategy Network (scholarsstrategynetwork.org/sites/default/files/ssn_key_findings_sheingate_on_food_safety.pdf).
divided authority for food safety between the U.S. Department of Agriculture and the Food and Drug Administration. Since then, authority and oversight have fractured even further.

Today, responsibility for ensuring U.S. food safety is scattered across at least 12 federal departments and agencies. Responsibilities are divided in ways that make little sense, and resources often do not match responsibilities. For example, five different agencies share authority over frozen pizza, with responsibilities divided according to the type of food topping. Cheese pizza facilities are inspected by the FDA, while companies that make pepperoni pizza are assigned to the Food Safety Inspection Service in the Department of Agriculture.

Another example: Federal rules require on-site inspectors to be stationed at all meat processing plants, and the FSIS employs more than 7,000 inspectors to carry out this task. Meanwhile, other food processing facilities do not require on-site inspections, so fewer than 3,000 inspectors monitor 65,000 domestic plants and oversee food imports. More than half of all the facilities in the United States have gone five or more years without a single inspection.

In 2010, Congress passed the Food Safety Modernization Act to begin to address long-standing problems. The FDA now has the authority to order mandatory recalls of tainted food (previously the recalls were voluntary). It can also conduct more frequent inspections and exercise greater control over imported foods.

But serious risks remain. For example, animals consume 80 percent of antibiotics in the United States, mostly in low doses intended to increase the quantity and speed of meat production. Despite mounting scientific evidence that routine use of antibiotics results in dangerous, drug-resistant strains of bacteria, the government has been slow to act. In 2012, the FDA finally acknowledged that giving antibiotics to healthy animals poses a threat to human health. Rather than regulate antibiotic use, the agency created “voluntary” guidelines.

Fixing food safety requires new efforts from government and citizen advocates alike. Instead of using multiple agencies, a smarter alternative would concentrate functions in those parts of the government that can do the job best. For instance, the FSIS could take charge of all inspections, freeing the FDA to focus its energies on food pathogens.

At the same time, food safety advocates must inform and arouse citizens, changing the way we talk and think about the issue. Today, industry and government often try to shift responsibility to everyday consumers—for example, by claiming that people can protect themselves by keeping clean kitchens, or by suggesting that foodborne illness is an unavoidable feature of the world we live in. But increasingly, people get sick because they are exposed to unsafe products. Food advocates need to get this message out and make the case for strong public regulations to reward companies that provide the safest food and allow adequately empowered public officials to root out harmful industry practices well before people get sick or die.

America knows enough to make our food safe. We just need to remove political obstacles and overcome governmental inefficiencies to get the job done.
Cheating Sleep

Lights out and technology on? Finishing up that last report for work, Facebooking with our friends, binge-watching *Game of Thrones*—there are so many options to occupy and entertain us late into the night. But our new nocturnal habits aren’t just making us a nation of sleepy people; they’re hurting our health in more ways than you may realize.

SARAH RICHARDS
but most people need between 7.5 and 8.5 hours of sleep a night.

What causes us to cheat on sleep? Perhaps the better question nowadays is, what doesn’t? Squeaking in work once the kids are in bed. Answering those last personal emails. Maybe there’s that final run of laundry spinning. And after all of it, when we’re truly exhausted, we collapse on the sofa to fiddle with our smartphones or catch up on the latest Netflix series. Soon, it’s way too late—already?—and the alarm clock is poised to squawk in just a few hours, beckoning us back awake and back to work.

It’s not just that our habits are creating a vast army of cranky individuals; we’re also putting ourselves at risk for a host of health issues. Lack of sleep can affect our cognitive abilities, mood, and ability to deal with pain and metabolize food, among other things.

“There are so many other distractions, we really see sleep as sort of this luxury item,” says neurologist Charlene Gamaldo, medical director of the Johns Hopkins Center for Sleep at Howard County General Hospital and an associate professor of neurology at the Johns Hopkins School of Medicine. Even Gamaldo admits to cheating sleep: She gets about six to seven hours per night. “There are 24 hours in a day; that’s never going to change,” she says. “But what’s being robbed is our health—our sleep.”

Teresa Hesley is a sleep cheater. A working mother with two young children, often the only occasion Hesley gets to spend one-on-one time with her husband is at night after the kids have gone to bed.

“That’s basically our time together,” says Hesley, a 33-year-old statistician who works for the U.S. Bureau of Labor Statistics. “A lot of times we’ll just watch TV, or we’ll talk for a while. Just relaxing and not going next-to-next-to-next. We’ll end up pushing that.”

Her 10 p.m. bedtime sounds OK. The problem is that, on most days, her alarm clock goes off at 4:20 in the morning so she can catch the 5:12 train from Arbutus, Maryland, to Washington, D.C., where she works. Her packed week includes a graduate course in statistics and two nights of recreational volleyball. “It kind of has a cumulative effect through the week,” Hesley says. “Monday, I’m a little sluggish, just because it’s Monday. By Wednesday, I’m like, Oh man . . .”

Hesley isn’t alone—we’re a nation of sleep cheaters. According to the Centers for Disease Control and Prevention, roughly 41 million working adults say they get six or fewer hours of sleep each day. Everyone is different,
Although sleep medicine is a growing field of study, there’s a lot we are still trying to understand about sleep—including why we even do it in the first place. Researchers do know, however, that not getting enough shut-eye affects our health.

“Sleep really impacts pretty much every physiologic function that you have, both your brain and how your body and organs perform,” says sleep researcher Michael Smith, a psychologist who directs the Johns Hopkins Center for Behavior and Health and co-directs the Johns Hopkins Center for Sleep-Related Symptom Science. During sleep, he explains, “the space between the cells in your brain actually expands and allows fluid to flow through and wipe away the detritus of the day—all the toxins, in a sense, that your brain puts out.”

The funny thing about cheating on sleep in order to get more things done during the day is that it’s actually counterproductive. When we’re sleep-deprived, our minds operate less efficiently.

Research has shown we’re less able to concentrate or to quickly recall information like a person’s name, and our brains have a tougher time retaining and retrieving memories.

You say this doesn’t sound like you? Perhaps that’s because people are, in fact, poor judges of how badly sleep-deprived they are. One University of Pennsylvania study published in 2003 found that by the end of a two-week study—when “performance was at its worst levels” on tests like matching up numbers with symbols—people getting four to six hours of sleep a night said they felt “only slightly sleepy.”

It’s possible to get away with some level of sleep deprivation because we seem to be able to compensate when it comes to logical tasks and even some complicated, rule-based tasks. But try to think creatively or innovatively, or to deal effectively with the unexpected, and results are not so good. This is particularly problematic when a person’s profession requires analysis and critical thinking. Take the practicing physician, says Gamaldo. “When we see a patient, it’s not like the patient goes, ‘This is what’s going on, and I’m going to give you some options for what it is.’ No, you have to take all that information, process it, retrieve from your knowledge and experience, and then spit out something.”

What’s more, sleep deprivation affects your prefrontal cortex, the area of your brain that deals in part with complex emotion and impulse control. That prefrontal cortex is critical when it comes to moderating the amygdala, the area of the brain that processes emotions and memories. So a lack of sleep...
can make us anxious and emotionally unstable. Serious sleep issues, like chronic insomnia, can lead to depression.

“The amygdala is specifically activated by either positive or negative emotional things,” explains Gamaldo. “The prefrontal lobe helps to sort of neutralize or balance it. I like to tell folks who are Freudians: Your amygdala is your emotional brain, or the id part of your personality. The prefrontal lobe is like the super ego that says: ‘Let’s think about this rationally.’” With sleep deprivation, however, that connection between the prefrontal cortex and the amygdala is affected. The cortex is less able to blunt negatively charged situations and emotions, meaning it’s more difficult to rationalize them. Think road rage, says Gamaldo.

Speaking of driving, the National Transportation Safety Board equates fatigue-impaired performance to alcohol-impaired performance. It cites a 2003 study published by Henry Ford Hospital’s Sleep Disorders Center in Detroit that found that losing two hours of sleep was similar to blowing a .05 percent on a breathalyzer. The AAA Foundation for Traffic Safety has estimated that nearly 17 percent of fatal car crashes studied in a nationally representative sample of accidents involved—though were not necessarily caused by—a sleepy driver. Fatigue has also played a role in numerous plane crashes. There are now rules regulating the number of hours airline pilots and commercial drivers must rest before going to work.

Michael Smith has been working in the field of sleep research for nearly 20 years. He’s an expert on the neurobehavioral causes, consequences, and treatments of sleep loss and on how the latter relates to pain. His research has shown that various types of sleep deprivation—from missing out on a few hours to chronic insomnia—can amplify a person’s pain and may even be a risk factor for developing pain that stretches out longer than the normal healing period.

Currently, he’s investigating the mechanisms by which sleep deprivation amplifies pain. One study involves disrupting the sleep of healthy people so that they get about four hours of sleep a night for two to three nights in a row. “One of the theories is that sleep deprivation causes inflammation,” says Smith. “And we know inflammation sensitizes your nociceptors, the neurons that fire your pain signals. So if sleep deprivation exacerbates or creates inflammation, then that might be one way it makes us more pain sensitive.”
In a separate study, Smith found that disrupted sleep might contribute even more to our sensitivity to discomfort. Women who experienced four hours of sleep with forced awakenings suffered an increase in spontaneous pain compared to women who simply slept for four hours. “This means that it’s not just the amount of sleep loss; we found that it was particularly bad if you’ve had this fragmented sleep,” says Smith.

Smith believes this may be a sign that sleep disruptions weaken our body’s natural pain regulating system, which functions in part by endogenous opioids, like endorphins. These chemicals are similar to morphine and relieve pain and provide pleasant feelings. (In turn, this may also mean that pain relief drugs like morphine may be less effective on sleep-deprived hospital patients.) “We are investigating whether sleep deprivation may down-regulate your ability to modulate pain using the opioid system,” he says.

As if being fuzzy-headed, emotionally vulnerable, and sensitive to pain isn't enough, it also seems that sleep deprivation can hit us around the waistline. Recent research has shown that not only do we crave calorie-dense foods when we're sleep-deprived, we're also less capable of fending off our amygdala's instant gratification urge to chow down, damn the calories! Hormones that help manage our appetite—leptin and ghrelin—end up working against us. Naturally occurring levels of the former, an appetite suppressant, and the latter, a hunger stimulant, can be thrown off-kilter when we don't sleep, causing us to eat more.

What about the argument that if you're up late, your body needs extra calories to keep going? That's true, but we still may be eating more than our body needs. Last year, a study out of the University of Colorado Boulder found that people who slept only five hours burned 5 percent more energy than test subjects who slept up to nine hours. However, they consumed 6 percent more calories—gobbling up more calories in after-supper snacks than at any of their individual meals. After five days, the short sleepers had gained almost two pounds.

“[Sleep deprivation] also affects things like our metabolism [and] the way we process and handle sugar,” says Smith.

Sleep deprivation affects your prefrontal cortex, the area of your brain that deals in part with complex emotion and impulse control.
Smith. “If you sleep deprive yourself even for four hours [a night] for about a week, you’ll get insulin resistance, which is a main feature of type 2 diabetes.”

Some of the most dramatic effects of fatigue occur in people who are chronically sleep-deprived, like shift workers. According to the Bureau of Labor Statistics, nearly 15 million Americans work at jobs with irregular schedules, like night shifts or rotating shifts. “There’s data that their life span is shorter,” says Gamaldo. Shift workers are at a potentially increased risk for colorectal cancer and gastrointestinal disorders. And although more research is needed, several studies have linked a higher risk of breast cancer with women who work mostly at night, like some nurses.

Scientists believe that being constantly exposed to light at night may be one of the possible culprits; this exposure can disrupt our body’s melatonin, an anti-carcinogenic hormone produced by our brain’s pineal gland that is secreted when it’s time to sleep. Johns Hopkins biologist Samer Hattar is one of those scientists concerned about nighttime light. While his research does not involve sleep deprivation per se, it nevertheless provides more reason to switch the lights off at a reasonable hour.

Hattar studies mice and their circadian clocks, the biological timekeeper that runs in all of us on a roughly 24-hour cycle tied to environmental light. In many ways, we are governed by these clocks—they dictate everything from our hormones to sleep and hunger cycles. Despite all the artificial light that surrounds us, these clocks are still most influenced by the sun.

Hattar and his lab have found that even abnormal exposure to light at night that doesn’t affect mice’s sleep patterns can still affect their mood and cognitive abilities. Mice on a cycle that alternated between light and darkness every three hours—a cycle used because it did not affect their circadian clocks—exhibited signs of depression and learning problems. “One of the things that excites me is that since light has such a major effect on us, does this irregular light schedule we are exposing ourselves to underlie a risk factor for neuropsychological diseases?” says Hattar. “Like depression or autism, we know these diseases have genetic susceptibility factors. Maybe these, under very defined light-dark environments and sleep-wake cycles, can be subdued and not as expressed as would happen if you put them under irregular light conditions.”

Hattar is hoping to investigate this theory further. His findings in mice, however, are cause for concern for more than just shift workers. With our fondness for squeezing in a few more minutes with tablets, televisions, and smartphones, we often forget that we’re getting more than just entertainment and information in the evening. We’re also getting a dose of artificial light that can help cheat us of sleep.

“You don’t want to be active 24 hours a day, and you don’t want to get light 24 hours a day;” says Hattar. “Light, like everything else, has a temporal domain to it.”
Better Bedtimes

If you’re making a conscious effort to get to bed earlier, here are some tips that may help the zzzz’s come faster.

1 / Exercise
Exercise, at least moderately, three to four times a week. Research indicates exercise improves sleep.

3 / Limit Caffeine
Don’t drink caffeinated beverages past 4 o’clock; that includes decaffeinated green tea, which still contains small amounts of caffeine. A glass of wine with dinner is fine, but avoid using it as a nightcap two or three hours before bedtime. Although alcohol may feel like it induces sleep, it can actually lead to rebound insomnia in the middle of the night.

4 / Relax
Have a relaxation ritual roughly one hour before bed that includes shutting down electronic devices and—if possible—your television. It’s believed that the type of light produced by monitors may keep people awake. “[Research has] shown that light at night can lower your melatonin, especially blue-shifted white light, and that can delay your sleep onset,” says Johns Hopkins biologist Samer Hattar.

2 / Control Light
Make sure your bedroom is cool and dark; blackout curtains or a sleep mask may help if you’re particularly sensitive to light. This means you’ll want to expose yourself to sunlight in the morning, when it’s time to wake up—so make sure to crack the blinds or walk the dog in the sun with your coffee. “Caffeine is good,” says Charlene Gamaldo, medical director of the Johns Hopkins Center for Sleep at Howard County General Hospital and an associate professor of neurology at the Johns Hopkins School of Medicine. “It just needs to be at the appropriate point in the day that’s in line with your sleep-wake pattern.”
Sights Unseen
[MEMOIR]

Nearly two miles above sea level, Rosemary Mahoney is given pragmatic advice on how to know where she is: “When we feel the ground coming different under our feet, we know where we find ourselves.” At that moment, Mahoney is blindfolded and being led around Lhasa, Tibet, by two blind teenage girls, Yangchen and Choden. When Mahoney asks Yangchen how she knows it’s cloudy outside, you can almost hear the implied “duh” in the young woman’s tone: “I do not feel the sun on my nose.”

Mahoney is a travel writer fond of solitary excursions to unusual and sometimes remote places. The above scene comes from For the Benefit of Those Who See (Little, Brown, 2014), her latest adventure, for which she tries to rob herself of her most trusted tool: sight. The book is informed by two teaching stints, one at Braille Without Borders, a training center for the blind in Lhasa, in 2005; the other at its sister facility, the International Institute for Social Entrepreneurs in Trivandrum, India, in 2009.

Throughout her story, Mahoney tries to understand sightless reality, and she does it with such blunt tenderness that it lends her writing a shambolic glee. Though she alludes to secondary sources—philosophical considerations of blindness, medical accounts of sight being restored to blind patients—it’s her experiences that make For the Benefit of Those Who See so thoughtful. In her introduction she confesses that she once considered blindness worse than death; later, she speculates that her students have a fundamental connection to the world that she doesn’t. In between, For the Benefit of Those Who See documents what happens when a well-trained observer begins to hear, smell, and touch the world as individually as she sees it. // BRET MCCABE
Linda Bunyard is a dietitian with the Johns Hopkins Weight Management Center. Her work focuses on nutrition counseling, behavior modification, and healthy nutrition.

Field Guide to the Natural World of Washington, D.C.
Howard Youth
A guide to the natural wonders of the nation’s capital. Naturalist Howard Youth offers a tour of the city’s flora and fauna as he hikes through Rock Creek Park and along the Potomac and Anacostia rivers. Drawings by Carnegie artist Mark A. Klingler and photography by Robert E. Mumford Jr. illustrate the journey.

Bipolar Disorder: A Guide for Patients and Families
Francis Mark Mondimore, MD
A completely updated third edition of Johns Hopkins psychiatrist Francis Mark Mondimore’s best-selling book. Initially published in 1999, this splendidly written, compassionate, and comprehensive text has sold more than 150,000 copies in paperback alone. The simplicity of style makes this an exceptionally valuable book for individuals with bipolar disorder and their families.

Confronting Chronic Pain: A Pain Doctor’s Guide to Relief
Steven H. Richeimer, MD, with Kathy Steligo
A comprehensive guide to help people who suffer from chronic pain. Steven Richeimer, an associate professor of anesthesiology and psychiatry at the University of Southern California, answers fundamental questions about what causes persistent pain and how to get relief from physical, social, and emotional suffering.

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My worst day

I’m feeling the stitches pull apart, and the incision at the top of my head is opening up. The titanium mesh plate is visible to the eye. I go back to the plastic surgeon, and he stitches me up in his office and says, “This should hold it.” A couple of weeks later, it’s opening up again.

What I did to get better

Thanks to a diagnosis from the hospital’s head of reconstructive surgery, it turned out that my head was rejecting the plate. So I had one surgery to remove the plate, and then for six months I had to wear a protective hat before I could have another surgery to put a new plate in. In total, it took five surgeries to figure this all out and for me to finally heal.

Me now

I know complications can occur with any surgery, and this was an unusual case, but the key was getting better communication and coordination across the disciplines within the hospital. I’m now co-chair of Johns Hopkins’ Patient and Family Advisory Council so that I can be a voice and a support for other patients. I have also obtained my Health and Wellness Coach certification and launched my own coaching and consulting business. As for my head, I get an MRI scan every six months to make sure the tumor hasn’t grown back. So far, so good.

Charlene Rothkopf

You might think Charlene Rothkopf’s bleakest health hour came the day doctors told her she had a golf ball–sized tumor on her brain. An executive vice president of human resources for a Fortune 500 company, Rothkopf, then 56 years old, stepped into management mode. She found a specialist at the Johns Hopkins Hospital to remove the tumor, updated her will, and sat down with her husband and two grown children to discuss contingencies. Bad days indeed. But more were still to come. Doctors successfully removed the tumor, but postop complications sent Rothkopf into multiple surgeries over the next two years.
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