

ALSO BY BILL BRYSON

The Lost Continent
The Mother Tongue
Neither Here Nor There
Made in America
Notes from a Small Island
A Walk in the Woods
I'm a Stranger Here Myself
In a Sunburned Country
Bryson's Dictionary of Troublesome Words
Bill Bryson's African Diary
A Short History of Nearly Everything
A Short History of Nearly Everything: Special Illustrated Edition
The Life and Times of the Thunderbolt Kid
Shakespeare: The World as Stage
Bryson's Dictionary for Writers and Editors
At Home: A Short History of Private Life
At Home: A Short History of Private Life: Illustrated Edition
One Summer
The Road to Little Dribbling: Adventures of an American in Britain

CONTENTS

Cover

Also by Bill Bryson

Title Page

Copyright

Dedication

[1 How to Build a Human](#)

[2 The Outside: Skin and Hair](#)

[3 Microbial You](#)

[4 The Brain](#)

[5 The Head](#)

[6 Down the Hatch: The Mouth and Throat](#)

[7 The Heart and Blood](#)

[8 The Chemistry Department](#)

[9 In the Dissecting Room: The Skeleton](#)

[10 On the Move: Bipedalism and Exercise](#)

[11 Equilibrium](#)

[12 The Immune System](#)

[13 Deep Breath: The Lungs and Breathing](#)

[14 Food, Glorious Food](#)

[15 The Guts](#)

[16 Sleep](#)

[17 Into the Nether Regions](#)

[18 In the Beginning: Conception and Birth](#)

[19 Nerves and Pain](#)

[20 When Things Go Wrong: Diseases](#)

[21 When Things Go Very Wrong: Cancer](#)

[22 Medicine Good and Bad](#)

[23 The End](#)

[*Acknowledgments*](#)

[*Notes on Sources*](#)

[*Bibliography*](#)

[*Illustration Credits*](#)

[*Illustrations*](#)

[*About the Author*](#)

1 HOW TO BUILD A HUMAN

How like a god!

—WILLIAM SHAKESPEARE

LONG AGO, WHEN I was a junior high school student in Iowa, I remember being taught by a biology teacher that all the chemicals that make up a human body could be bought in a hardware store for \$5.00 or something like that. I don't recall the actual sum. It might have been \$2.97 or \$13.50, but it was certainly very little even in 1960s money, and I remember being astounded at the thought that you could make a slouched and pimply thing such as me for practically nothing.

It was such a spectacularly humbling revelation that it has stayed with me all these years. The question is, was it true? Are we really worth so little?

Many authorities (for which possibly read “science majors who don't have a date on a Friday”) have tried at various times, mostly for purposes of amusement, to compute how much it would cost in materials to build a human. Perhaps the most respectable and comprehensive attempt of recent years was done by Britain's Royal Society of Chemistry when, as part of the 2013 Cambridge Science Festival, it calculated how much it would cost to assemble all the elements necessary to build the actor Benedict Cumberbatch. (Cumberbatch was the guest director of the festival that year and was, conveniently, a typically sized human.)

Altogether, according to RSC calculations, fifty-nine elements are needed to construct a human being. Six of these—

carbon, oxygen, hydrogen, nitrogen, calcium, and phosphorus—account for 99.1 percent of what makes us, but much of the rest is a bit unexpected. Who would have thought that we would be incomplete without some molybdenum inside us, or vanadium, manganese, tin, and copper? Our requirements for some of these, it must be said, are surpassingly modest and are measured in parts per million or even parts per billion. We need, for instance, just 20 atoms of cobalt and 30 of chromium for every 999,999,999½ atoms of everything else.

The biggest component in any human, filling 61 percent of available space, is oxygen. It may seem a touch counterintuitive that we are almost two-thirds composed of an odorless gas. The reason we are not light and bouncy like a balloon is that the oxygen is mostly bound up with hydrogen (which accounts for another 10 percent of you) to make water—and water, as you will know if you have ever tried to move a wading pool or just walked around in really wet clothes, is surprisingly heavy. It is a little ironic that two of the lightest things in nature, oxygen and hydrogen, when combined form one of the heaviest, but that's nature for you. Oxygen and hydrogen are also two of the cheaper elements within you. All your oxygen will set you back just \$14 and your hydrogen a little over \$26 (assuming you are about the size of Benedict Cumberbatch). Your nitrogen (2.6 percent of you) is a better value still at just forty cents for a body's worth. But after that it gets pretty expensive.

You need about thirty pounds of carbon, and that will cost you \$69,550, according to the Royal Society of Chemistry. (They were using only the most purified forms of everything. The RSC would not make a human with cheap stuff.) Calcium, phosphorus, and potassium, though needed in much smaller amounts, would between them set you back a further \$73,800. Most of the rest is even more expensive per unit of volume, but fortunately only needed in microscopic amounts. Thorium costs over \$3,000 per gram but constitutes just 0.0000001 percent of you, so you can buy a body's worth for thirty-three cents. All the tin you require can be yours for six cents, while zirconium

and niobium will cost you just three cents apiece. The 0.000000007 percent of you that is samarium isn't apparently worth charging for at all. It's logged in the RSC accounts as costing \$0.00.*¹

Of the fifty-nine elements found within us, twenty-four are traditionally known as essential elements, because we really cannot do without them. The rest are something of a mixed bag. Some are clearly beneficial, some may be beneficial but we are not sure in what ways yet, others are neither harmful nor beneficial but are just along for the ride as it were, and a few are just bad news altogether. Cadmium, for instance, is the twenty-third most common element in the body, constituting 0.1 percent of your bulk, but it is seriously toxic. We have it in us not because our body craves it but because it gets into plants from the soil and then into us when we eat the plants. If you are from North America, you probably ingest about eighty micrograms of cadmium a day, and no part of it does you any good at all.

A surprising amount of what goes on at this elemental level is still being worked out. Pluck almost any cell from your body, and it will have a million or more selenium atoms in it, yet until recently nobody had any idea what they were there for. We now know that selenium makes two vital enzymes, deficiency in which has been linked to hypertension, arthritis, anemia, some cancers, and even, possibly, reduced sperm counts. So, clearly it is a good idea to get some selenium inside you (it is found particularly in nuts, whole wheat bread, and fish), but at the same time if you take in too much you can irremediably poison your liver. As with so much in life, getting the balances right is a delicate business.

Altogether, according to the RSC, the full cost of building a new human being, using the obliging Benedict Cumberbatch as a template, would be a very precise \$151,578.46. Labor and sales tax would, of course, boost costs further. You would probably be lucky to get a take-home Benedict Cumberbatch for much under \$300,000—not a massive fortune, all things

considered, but clearly not the meager few dollars that my junior high school teacher suggested. That said, in 2012 *Nova*, the long-running science program on PBS, did an exactly equivalent analysis for an episode called “Hunting the Elements” and came up with a figure of \$168 for the value of the fundamental components within the human body, illustrating a point that will become inescapable as this book goes on, namely that where the human body is concerned, the details are often surprisingly uncertain.

But of course it hardly really matters. No matter what you pay, or how carefully you assemble the materials, you are not going to create a human being. You could call together all the brainiest people who are alive now or have ever lived and endow them with the complete sum of human knowledge, and they could not between them make a single living cell, never mind a replicant Benedict Cumberbatch.

That is unquestionably the most astounding thing about us—that we are just a collection of inert components, the same stuff you would find in a pile of dirt. I’ve said it before in another book, but I believe it’s worth repeating: the only thing special about the elements that make you is that they make you. That is the miracle of life.

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We pass our existence within this warm wobble of flesh and yet take it almost entirely for granted. How many among us know even roughly where the spleen is or what it does? Or the difference between tendons and ligaments? Or what our lymph nodes are up to? How many times a day do you suppose you blink? Five hundred? A thousand? You’ve no idea, of course. Well, you blink fourteen thousand times a day—so much that your eyes are shut for twenty-three minutes of every waking day. Yet you never have to think about it, because every second of every day your body undertakes a literally unquantifiable number of tasks—a quadrillion, a nonillion, a quindecillion, a

vigintillion (these are actual measures), at all events some number vastly beyond imagining—without requiring an instant of your attention.

In the second or so since you started this sentence, your body has made a million red blood cells. They are already speeding around you, coursing through your veins, keeping you alive. Each of those red blood cells will rattle around you about 150,000 times, repeatedly delivering oxygen to your cells, and then, battered and useless, will present itself to other cells to be quietly killed off for the greater good of you.

Altogether it takes 7 billion billion billion (that's 7,000,000,000,000,000,000,000,000, or 7 octillion) atoms to make you. No one can say why those 7 billion billion billion have such an urgent desire to be you. They are mindless particles, after all, without a single thought or notion between them. Yet somehow for the length of your existence, they will build and maintain all the countless systems and structures necessary to keep you humming, to make you you, to give you form and shape and let you enjoy the rare and supremely agreeable condition known as life.

That's a much bigger job than you realize. Unpacked, you are positively enormous. Your lungs, smoothed out, would cover a tennis court, and the airways within them would stretch nearly from coast to coast. The length of all your blood vessels would take you two and a half times around Earth. The most remarkable part of all is your DNA (or deoxyribonucleic acid). You have a meter of it packed into every cell, and so many cells that if you formed all the DNA in your body into a single strand, it would stretch ten billion miles, to beyond Pluto. Think of it: there is enough of you to leave the solar system. You are in the most literal sense cosmic.

But your atoms are just building blocks and are not themselves alive. Where life begins precisely is not so easy to say. The basic unit of life is the cell—everyone is agreed on that. The cell is full of busy things—ribosomes and proteins, DNA, RNA, mitochondria, and much other cellular arcana—but none

of those are themselves alive. The cell itself is just a compartment—a kind of little room: a *cell*—to contain them, and of itself is as nonliving as any other room. Yet somehow when all of these things are brought together, you have life. That is the part that eludes science. I kind of hope it always will.

What is perhaps most remarkable is that nothing is in charge. Each component of the cell responds to signals from other components, all of them bumping and jostling like so many bumper cars, yet somehow all this random motion results in smooth, coordinated action, not just across the cell but across the whole body as cells communicate with other cells in different parts of your personal cosmos.

The heart of the cell is the nucleus. It contains the cell's DNA—three feet of it, as we have already noted, scrunched into a space that we may reasonably call infinitesimal. The reason so much DNA can fit into a cell nucleus is that it is exquisitely thin. You would need twenty billion strands of DNA laid side by side to make the width of the finest human hair. Every cell in your body (strictly speaking, every cell with a nucleus) holds two copies of your DNA. That's why you have enough to stretch to Pluto and beyond.

DNA exists for just one purpose—to create more DNA. A DNA molecule, as you will almost certainly remember from countless television programs if not school biology, is made up of two strands, connected by rungs to form the celebrated twisted ladder known as a double helix. Your DNA is simply an instruction manual for making you. A length of DNA is divided into segments called chromosomes and shorter individual units called genes. The sum of all your genes is the genome.

DNA is extremely stable. It can last for tens of thousands of years. It is nowadays what enables scientists to work out the anthropology of the very distant past. Probably nothing you own right now—no letter or piece of jewelry or treasured heirloom—will still exist a thousand years from now, but your DNA will almost certainly still be around and recoverable, if only someone could be bothered to look for it.

DNA passes on information with extraordinary fidelity. It makes only about one error per every billion letters copied. Still, because your cells divide so much, that is about three errors, or mutations, per cell division. Most of those mutations the body can ignore, but just occasionally they have lasting significance. That is evolution.

All of the components of the genome have one single-minded purpose—to keep the line of your existence going. It's a slightly humbling thought that the genes you carry are immensely ancient and possibly—so far anyway—eternal. You will die and fade away, but your genes will go on and on so long as you and your descendants continue to produce offspring. And it is surely astounding to reflect that not once in the three billion years since life began has your personal line of descent been broken. For you to be here now, every one of your ancestors had to successfully pass on its genetic material to a new generation before being snuffed out or otherwise sidetracked from the procreative process. That's quite a chain of success.

What genes specifically do is provide instructions for building proteins. Most of the useful things in the body are proteins. Some speed up chemical changes and are known as enzymes. Others convey chemical messages and are known as hormones. Still others attack pathogens and are called antibodies. The largest of all our proteins is called titin, which helps to control muscle elasticity. Its chemical name is 189,819 letters long, which would make it the longest word in the English language except that dictionaries don't recognize chemical names. Nobody knows how many types of proteins there are within us, but estimates range from a few hundred thousand to a million or more.

The paradox of genetics is that we are all very different and yet genetically practically identical. All humans share 99.9 percent of their DNA, and yet no two humans are alike. My DNA and your DNA will differ in three to four million places, which is a small proportion of the total but enough to make a

lot of difference between us. You also have within you about a hundred personal mutations—stretches of genetic instructions that don't quite match any of the genes given to you by either of your parents but are yours alone.

How all this works in detail is still largely a mystery to us. Only 2 percent of the human genome codes for proteins, which is to say only 2 percent does anything demonstrably and unequivocally practical. Quite what the rest is doing isn't known. A lot of it, it seems, is just there, like freckles on skin. Some of it makes no sense. One particular short sequence, called an Alu element, is repeated more than a million times throughout our genome, including sometimes in the middle of important protein-coding genes. It is complete gibberish, as far as anyone can tell, yet it constitutes 10 percent of all our genetic material. No one has any idea why. The mysterious part was for a while called junk DNA but now is more graciously called dark DNA, meaning that we don't know what it does or why it is there. Some is involved in regulating the genes, but much of the rest remains to be determined.

The body is often likened to a machine, but it is so much more than that. It works twenty-four hours a day for decades without (for the most part) needing regular servicing or the installation of spare parts, runs on water and a few organic compounds, is soft and rather lovely, is accommodatingly mobile and pliant, reproduces itself with enthusiasm, makes jokes, feels affection, appreciates a red sunset and a cooling breeze. How many machines do you know that can do any of that? There is no question about it. You are truly a wonder. But then so, it must be said, is an earthworm.

And how do we celebrate the glory of our existence? Well, for most of us by eating maximally and exercising minimally. Think of all the junk you throw down your throat and how much of your life is spent sprawled in a near-vegetative state in front of a glowing screen. Yet in some kind and miraculous way our bodies look after us, extract nutrients from the miscellaneous foodstuffs we push into our faces, and somehow

hold us together, generally at a pretty high level, for decades. Suicide by lifestyle takes ages.

Even when you do nearly everything wrong, your body maintains and preserves you. Most of us are testament to that in one way or another. Five out of every six smokers won't get lung cancer. Most of the people who are prime candidates for heart attacks don't get heart attacks. Every day, it has been estimated, between one and five of your cells turn cancerous, and your immune system captures and kills them. Think of that. A couple of dozen times a week, well over a thousand times a year, you get the most dreaded disease of our age, and each time your body saves you. Of course, very occasionally a cancer develops into something more serious and possibly kills you, but overall cancers are rare: most cells in the body replicate billions and billions of times without going wrong. Cancer may be a common cause of death, but it is not a common event in life.

Our bodies are a universe of 37.2 trillion cells operating in more or less perfect concert more or less all the time.^{*2} An ache, a twinge of indigestion, the odd bruise or pimple, are about all that in the normal course of things announces our imperfectability. There are thousands of things that can kill us—slightly more than eight thousand, according to the *International Statistical Classification of Diseases and Related Health Problems* compiled by the World Health Organization—and we escape every one of them but one. For most of us, that's not a bad deal.

We are not perfect by any means, goodness knows. We get impacted molars because we have evolved jaws too small to accommodate all the teeth we are endowed with. We have pelvises too small to pass children without excruciating pain. We are hopelessly susceptible to backache. We have organs that mostly cannot repair themselves. If a zebra fish damages its heart, it grows new tissue. If you damage your heart, well, too bad. Nearly all animals produce their own vitamin C, but we can't. We undertake every part of the process except,

inexplicably, the last step, the production of a single enzyme.

The miracle of human life is not that we are endowed with some frailties but that we aren't swamped with them. Don't forget that your genes come from ancestors who most of the time weren't even human. Some of them were fish. Lots more were tiny and furry and lived in burrows. These are the beings from whom you have inherited your body plan. You are the product of three billion years of evolutionary tweaks. We would all be a lot better off if we could just start fresh and give ourselves bodies built for our particular *Homo sapien* needs—to walk upright without wrecking our knees and backs, to swallow without the heightened risk of choking, to dispense babies as if from a vending machine. But we weren't built for that. We began our journey through history as unicellular blobs floating about in warm, shallow seas. Everything since then has been a long and interesting accident, but a pretty glorious one, too, as I hope the following pages make clear.

*¹ The RSC calculations were done in British pounds and have been converted here into U.S. dollars at the rate that prevailed in the summer of 2013 of £1 = \$1.57.

*² That number is of course an educated guess. Human cells come in a variety of types, sizes, and densities and are literally uncountable. The figure of 37.2 trillion was arrived at in 2013 by a team of European scientists led by Eva Bianconi from the University of Bologna in Italy and was reported in the *Annals of Human Biology*.

2 THE OUTSIDE: SKIN AND HAIR

Beauty is only skin deep, but ugly goes clean to the bone.

—DOROTHY PARKER

I

IT MAY BE slightly surprising to think it, but our skin is our largest organ, and possibly the most versatile. It keeps our insides in and bad things out. It cushions blows. It gives us our sense of touch, bringing us pleasure and warmth and pain and nearly everything else that makes us vital. It produces melanin to shield us from the sun's rays. It repairs itself when we abuse it. It accounts for such beauty as we can muster. It looks after us.

The formal name for the skin is the cutaneous system. Its size is about two square meters (approximately twenty square feet), and all told your skin will weigh somewhere in the region of ten to fifteen pounds, though much depends, naturally, on how tall you are and how much buttock and belly it needs to stretch across. It is thinnest on the eyelids (just one-thousandth of an inch thick) and thickest on the heels of our hands and feet. Unlike a heart or a kidney, skin never fails. "Our seams don't burst, we don't spontaneously sprout leaks," says Nina Jablonski, professor of anthropology at Penn State University, who is the doyenne of all things cutaneous.

The skin consists of an inner layer called the dermis and an outer epidermis. The outermost surface of the epidermis, called the stratum corneum, is made up entirely of dead cells. It is an

arresting thought that all that makes you lovely is deceased. Where body meets air, we are all cadavers. These outer skin cells are replaced every month. We shed skin copiously, almost carelessly: some twenty-five thousand flakes a minute, over a million pieces every hour. Run a finger along a dusty shelf, and you are in large part clearing a path through fragments of your former self. Silently and remorselessly we turn to dust.

Skin flakes are properly called squamae (meaning “scales”). We each trail behind us about a pound of dust every year. If you burn the contents of a vacuum cleaner bag, the predominant odor is that unmistakable scorched smell that we associate with burning hair. That’s because skin and hair are made largely of the same stuff: keratin.

Beneath the epidermis is the more fertile dermis, where reside all the skin’s active systems—blood and lymph vessels, nerve fibers, the roots of hair follicles, the glandular reservoirs of sweat and sebum. Beneath that, and not technically part of the skin, is a subcutaneous layer where fat is stored. Though it may not be part of the cutaneous system, it’s an important part of your body because it stores energy, provides insulation, and attaches the skin to the body beneath.

Nobody knows for sure how many holes you have in your skin, but you are pretty seriously perforated. Most estimates suggest you have somewhere in the region of two to five million hair follicles and perhaps twice that number of sweat glands. The follicles do double duty: they sprout hairs and secrete sebum (from sebaceous glands), which mixes with sweat to form an oily layer on the surface. This helps to keep skin supple and to make it inhospitable for many foreign organisms. Sometimes the pores become blocked with little plugs of dead skin and dried sebum in what is known as a blackhead. If the follicle additionally becomes infected and inflamed, the result is the adolescent dread known as a pimple. Pimples plague young people simply because their sebaceous glands—like all their glands—are highly active. When the condition becomes chronic, the result is acne, a word of very uncertain derivation. It

appears to be related to the Greek *acme*, denoting a high and admirable achievement, which a faceful of pimples most assuredly is not. How the two became twinned is not at all clear. The term first appeared in English in 1743 in a British medical dictionary.

Also packed into the dermis are a variety of receptors that keep us literally in touch with the world. If a breeze plays lightly on your cheek, it is your Meissner's corpuscles that let you know.* When you put your hand on a hot plate, your Ruffini corpuscles cry out. Merkel cells respond to constant pressure, Pacinian corpuscles to vibration.

Meissner's corpuscles are everyone's favorites. They detect light touch and are particularly abundant in our erogenous zones and other areas of heightened sensitivity: fingertips, lips, tongue, clitoris, penis, and so on. They are named after a German anatomist, Georg Meissner, who is credited with discovering them in 1852, though his colleague Rudolf Wagner claimed that he in fact was the discoverer. The two men fell out over the matter, proving that there is no detail in science too small for animosity.

All are exquisitely fine-tuned to let you feel the world. A Pacinian corpuscle can detect a movement as slight as 0.00001 millimeter, which is practically no movement at all. More than this, they don't even require contact with the material they are interpreting. As David J. Linden points out in *Touch*, if you sink a spade into gravel or sand, you can feel the difference between them even though all you are touching is the spade. Curiously, we don't have any receptors for wetness. We have only thermal sensors to guide us, which is why when you sit down on a wet spot, you can't generally tell whether it really is wet or just cold.

Women are much better than men at tactile sensitivity with fingers, but possibly just because they have smaller hands and thus a more dense network of sensors. An interesting thing about touch is that the brain doesn't just tell you how something feels, but how it *ought* to feel. That's why the caress of a lover feels wonderful, but the same touch by a stranger

would feel creepy or horrible. It's also why it is so hard to tickle yourself.

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One of the most memorably unexpected events I experienced in the course of doing this book came in a dissection room at the University of Nottingham in England when a professor and surgeon named Ben Ollivere (about whom much more in due course) gently incised and peeled back a sliver of skin about a millimeter thick from the arm of a cadaver. It was so thin as to be translucent. “That,” he said, “is where all your skin color is. That’s all that race is—a sliver of epidermis.”

I mentioned this to Nina Jablonski when we met in her office in State College, Pennsylvania, soon afterward. She gave a nod of vigorous assent. “It is extraordinary how such a small facet of our composition is given so much importance,” she said. “People act as if skin color is a determinant of character when all it is is a reaction to sunlight. Biologically, there is actually no such thing as race—nothing in terms of skin color, facial features, hair type, bone structure, or anything else that is a defining quality among peoples. And yet look how many people have been enslaved or hated or lynched or deprived of fundamental rights through history because of the color of their skin.”

A tall, elegant woman with silvery hair cut short, Jablonski works in a very tidy office on the fourth floor of the anthropology building on the Penn State campus, but her interest in skin came about almost thirty years ago when she was a young primatologist and paleobiologist at the University of Western Australia in Perth. While preparing a lecture on the differences between primate skin color and human skin color, she realized there was surprisingly little information on the subject and embarked on what has become a lifelong study. “What began as a small, fairly innocent project ended up taking over a big part of my professional life,” she says. In 2006, she

produced the highly regarded *Skin: A Natural History* and followed that six years later with *Living Color: The Biological and Social Meaning of Skin Color*.

Skin color turned out to be more scientifically complicated than anyone imagined. “Over 120 genes are involved in pigmentation in mammals,” says Jablonski, “so it is really hard to unpack it all.” What we can say is this: skin gets its color from a variety of pigments, of which the most important by far is a molecule formally called eumelanin but known universally as melanin. It is one of the oldest molecules in biology and is found throughout the living world. It doesn’t just color skin. It gives birds the color of their feathers, fish the texture and luminescence of their scales, squid the purply blackness of their ink. It is even involved in making fruits go brown. In us, it also colors our hair. Its production slows dramatically as we age, which is why older people’s hair tends to turn gray.

“Melanin is a superb natural sunscreen,” says Jablonski. “It is produced in cells called melanocytes. All of us, whatever our race, have the same number of melanocytes. The difference is in the amount of melanin produced.” Melanin often responds to sunlight in a literally patchy way, resulting in freckles, which are technically known as ephelides.

Skin color is a classic example of what is known as convergent evolution—that is, similar outcomes that have evolved in two or more locations. The people of, say, Sri Lanka and Polynesia have light brown skin not because of any direct genetic link but because they independently evolved brown skin to deal with the conditions of where they lived. It used to be thought that depigmentation probably took perhaps ten thousand to twenty thousand years, but now thanks to genomics we know it can happen much more quickly—in probably just two or three thousand years. We also know that it has happened repeatedly. Light-colored skin—“de-pigmented skin,” as Jablonski calls it—has evolved at least three times on Earth. The lovely range of hues humans boast is an ever-changing process. “We are,” as Jablonski puts it, “in the middle

of a new experiment in human evolution.”

It has been suggested that light skin may be a consequence of human migration and the rise of agriculture. The argument is that hunter-gatherers got a lot of their vitamin D from fish and game and that these inputs fell sharply when people started growing crops, especially as they moved into northern latitudes. It therefore became a great advantage to have lighter skin, to synthesize extra vitamin D.

Vitamin D is vital to health. It helps to build strong bones and teeth, boosts the immune system, fights cancers, and nourishes the heart. It is thoroughly good stuff. We can get it in two ways—from the foods we eat or through sunlight. The problem is that too much UV exposure damages DNA in our cells and can cause skin cancer. Getting the right amount is a tricky balance. Humans have addressed the challenge by evolving a range of skin tones to suit sunshine intensity at different latitudes. When a human body adapts to altered circumstances, the process is known as phenotypic plasticity. We alter our skin color all the time—when we tan or burn beneath a bright sun or blush from embarrassment. The red of sunburn is because the tiny blood vessels in the affected areas become engorged with blood, making the skin hot to the touch. The formal name for sunburn is erythema. Pregnant women frequently undergo a darkening of the nipples and areolae, and sometimes of other parts of the body such as the abdomen and face, as a result of increased production of melanin. The process is known as melasma, but its purpose is not understood. The flush we get when angry is a little counterintuitive. When the body is poised for a fight, it mostly diverts blood flow to where it is really needed—namely, the muscles—so why it would send blood to the face, where it confers no obvious physiological benefit, remains a mystery. One possibility suggested by Jablonski is that it helps in some way to mediate blood pressure. Or it could just serve as a signal to an opponent to back off because one is really angry.

At all events, the slow evolution of different skin tones

worked fine when people stayed in one place or migrated slowly, but nowadays increased mobility means that lots of people end up in places where sun levels and skin tones don't get along at all. In regions like northern Europe and Canada, it isn't possible in the winter months to extract enough vitamin D from weakened sunlight to maintain health no matter how pale one's skin, so vitamin D must be consumed as food, and hardly anyone gets enough—and not surprisingly. To meet dietary requirements from food alone, you would have to eat fifteen eggs or six pounds of swiss cheese every day, or, more plausibly if not more palatably, swallow half a tablespoon of cod liver oil. In America, milk is helpfully supplemented with vitamin D, but that still provides only a third of daily adult requirements. In consequence, some 50 percent of people globally are estimated to be vitamin D deficient for at least part of the year. In northern climes, it may be as much as 90 percent.

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As people evolved lighter skin, they also developed lighter-colored eyes and hair—but only pretty recently. Lighter-colored eyes and hair evolved somewhere around the Baltic Sea about six thousand years ago. It's not obvious why. Hair and eye color don't affect vitamin D metabolism, or anything else physiological come to that, so there seems to be no practical benefit. The supposition is that these traits were selected for as tribal markers or because people found them more attractive. If you have blue or green eyes, it's not because you have more of those colors in your irises than other people but because you simply have less of other colors. It is the paucity of other pigments that leaves the eyes looking blue or green.

Skin color has been changing over a much longer period—at least sixty thousand years. But it hasn't been a straightforward process. “Some people have de-pigmented; some have re-pigmented,” Jablonski says. “Some people have altered skin tones a lot in moving to new latitudes, others hardly at all.”

Indigenous populations in South America, for instance, are lighter-skinned than would be expected at the latitudes they inhabit. That is because in evolutionary terms they are recent arrivals. “They were able to get to the tropics quite quickly and had lots of gear, including some clothing,” Jablonski told me. “So in effect they thwarted evolution.” Rather harder to explain have been the KhoeSan people of southern Africa. They have always lived under a desert sun and have never migrated any great distance, yet have 50 percent lighter skin than would be predicted by their environment. It now appears that a genetic mutation for lighter skin was introduced to them sometime in the last two thousand years by outsiders—but who these mysterious light-skinned outsiders were and how they came to be in southern Africa are unknown.

The development in recent years of techniques for analyzing ancient DNA means that we are learning more all the time and much of it is surprising—and some is confusing and some disputed. Using DNA analysis, in early 2018 scientists from University College London and Britain’s Natural History Museum announced to widespread astonishment that an ancient Briton known as Cheddar Man had had “dark to black” skin. He seems also to have had blue eyes. Cheddar Man was among the first people to return to Britain after the end of the last ice age some ten thousand years ago. His forebears had been in Europe for thirty thousand years, more than sufficient time to have evolved light skin, so if he was truly dark-skinned, it would be a real surprise. However, other authorities have suggested that the DNA used in the analysis was too degraded and our understanding of the genetics of pigmentation too uncertain to allow any conclusions about the color of Cheddar Man’s skin and eyes. If nothing else, it was a reminder of how much we have still to learn.

“Where skin is concerned, we are still in many ways at the very beginning,” Jablonski told me.

Skin comes in two varieties: with hair and without. Hairless skin is called glabrous, and there isn't much of it. Our only truly hairless parts are lips, nipples and genitalia, and the bottoms of our hands and feet. The rest of the body is covered with either conspicuous hair, called terminal hair, as on your head, or vellus hair, which is the downy stuff you find on a child's cheek. We are actually as hairy as our cousins the apes. It's just that our hair is much wispier and fainter. Altogether we are estimated to have five million hairs, but the number varies with age and circumstances, and is only a guess anyway.

Hair is unique to mammals. Like the underlying skin, it serves a multitude of purposes: it provides warmth, cushioning, and camouflage, shields the body from ultraviolet light, and allows members of a group to signal to each other that they are angry or aroused. But some of these features clearly don't work so well when you are nearly hairless. In all mammals, when they are cold, the muscles around their hair follicles contract in a process known formally as horripilation but more commonly as getting goose bumps. In furry mammals, it adds a useful layer of insulating air between the hair and the skin, but in humans it has absolutely no physiological benefit and merely reminds us how comparatively bald we are. Horripilation also makes mammalian hair stand up (to make animals look bigger and more ferocious), which is why we get goose bumps when we are frightened or on edge, but of course that doesn't work very well for humans either.

The two most enduring questions with respect to human hair are when did we become essentially hairless and why did we retain conspicuous hair on the few places we did? As to the first, it isn't possible to state categorically when humans lost their hair, because hair and skin aren't preserved in the fossil record, but it is known from genetic studies that dark pigmentation dates from between 1.2 and 1.7 million years ago. Dark skin wasn't necessary when we were still furry, so that would strongly suggest a time frame for hairlessness. Why we retained hair on some parts of our bodies is fairly

straightforward with respect to the head but not so clear elsewhere. Hair on the head acts as a good insulator in cold weather and a good reflector of heat in hot weather. According to Nina Jablonski, tightly curled hair is the most efficient kind “because it increases the thickness of the space between the surface of the hair and the scalp, allowing air to blow through.” A separate but no less important reason for the retention of head hair is that it has been a tool of seduction since time immemorial.

Pubic and underarm hair are more problematic. It is not easy to think of a way that armpit hair enriches human existence. One line of supposition is that secondary hair is used to trap or disperse (depending on theory) sexual scents, or pheromones. The one problem with this theory is that humans don't seem to have pheromones. A study published in 2017 in *Royal Society Open Science* by researchers from Australia concluded that human pheromones probably don't exist and certainly play no detectable role in attraction. Another hypothesis is that secondary hair somehow protects the skin beneath it from chafing, though clearly a lot of people remove hair from all around their bodies without a notable increase in skin irritation. A more plausible theory, perhaps, is that secondary hair is for display—that it announces sexual maturity.

Every hair on your body has a growth cycle, with a growing phase and a resting phase. For facial hair a cycle is normally completed in four weeks, but a scalp hair may be with you for as much as six or seven years. A hair in your armpit is likely to last about six months, a leg hair for two months. Removing hair, whether through cutting, shaving, or waxing, has no effect on what happens at the root. We each grow about twenty-five feet of hair in a lifetime, but because all hair falls out at some point, no single strand can ever get longer than about three feet. Hair grows by one third of a millimeter a day, but the rate of hair growth depends on your age and health and even the season of the year. Our hair cycles are staggered, so we don't usually

much notice as our hair falls out.

II

IN OCTOBER 1902, police in Paris were called to an apartment at 157 rue du Faubourg Saint-Honoré, in a wealthy neighborhood a few hundred yards from the Arc de Triomphe in the 8th arrondissement. A man had been murdered and some works of art stolen. The murderer left behind no obvious clues, but luckily detectives were able to call upon Alphonse Bertillon, a wizard at identifying criminals.

Bertillon had invented a system of identification that he called anthropometry but that became known to an admiring public as Bertillonage. The system introduced the concept of the mug shot and the practice, still universally observed, of recording every arrested person full face and in profile. But it was in the fastidiousness of its measurements that Bertillonage stood out. Subjects were measured for eleven oddly specific attributes—height when seated, length of left little finger, cheek width—which Bertillon had chosen because they would not change with age. Bertillon's system was developed not to convict criminals but to catch recidivists. Because France gave stiffer sentences to repeat offenders (and often exiled them to distant, steamy outposts like Devil's Island), many criminals tried desperately to pass themselves off as first-time offenders. Bertillon's system was designed to identify them, and it did that very well. In the first year of operation, he unmasked 241 fraudsters.

Fingerprinting was actually only an incidental part of Bertillon's system, but when he found a single fingerprint on a window frame at 157 rue du Faubourg Saint-Honoré and used that to identify the murderer as one Henri Léon Scheffer, it caused a sensation not just in France but around the world. Quickly, fingerprinting became a fundamental tool of police work everywhere.

The uniqueness of fingerprints was first established in the

West by the nineteenth-century Czech anatomist Jan Purkinje, though in fact the Chinese had made the same discovery more than a thousand years earlier and for centuries Japanese potters had identified their wares by pressing a finger into the clay before baking. Charles Darwin's cousin Francis Galton had suggested using fingerprints to catch criminals years before Bertillon came up with the notion, as did a Scottish missionary in Japan named Henry Faulds. Bertillon wasn't even the first to use a fingerprint to catch a murderer—that happened in Argentina ten years earlier—but it is Bertillon who gets the credit.

What evolutionary imperative led us to get whorls on the ends of our fingers? The answer is that nobody knows. Your body is a universe of mystery. A very large part of what happens on and within it happens for reasons that we don't know—very often, no doubt, because there are no reasons. Evolution is an accidental process, after all. The idea that all fingerprints are unique is actually a supposition. No one can say for absolute certain that no one else has fingerprints to match yours. All that can be said is that no one has yet found two sets of fingerprints that precisely match.

The textbook name for fingerprints is dermatoglyphics. The plow lines that make up our fingerprints are papillary ridges. They are assumed to aid in gripping, in the way tire treads improve traction on roads, but no one has ever actually proved that. Others have suggested that the whorls of fingerprints drain water better, make the skin of the fingers more stretchy and supple, or improve sensitivity, but again no one really knows what they are there for. Similarly, no one has ever come close to explaining why our fingers wrinkle when we have long baths. The explanation most often given is that wrinkling helps them to drain water better and improves grip. But that doesn't really make a great deal of sense. Surely the people who most urgently need a good grip are those who have just fallen in water, not those who have been in it for some time.

Very, very occasionally, people are born with completely

smooth fingertips, a condition known as adermatoglyphia. They also have slightly fewer sweat glands than normal. This would seem to suggest a genetic connection between sweat glands and fingerprints, but what that connection is has yet to be determined. As cutaneous features go, fingerprints are frankly pretty trivial. Far more important are your sweat glands. You might not think it, but sweating is a crucial part of being human. As Nina Jablonski has put it, “It is plain old unglamorous sweat that has made humans what they are today.”

Chimpanzees have only about half as many sweat glands as we have, and so can't dissipate heat as quickly as humans can. Most quadrupeds cool by panting, which is incompatible with sustained running and simultaneous heavy breathing, especially for furry creatures in hot climates. Much better to do as we do and seep watery fluids onto nearly bare skin, which cools the body as it evaporates, turning us into a kind of living air conditioner. As Jablonski has written, “The loss of most of our body hair and the gain of the ability to dissipate excess body heat through eccrine sweating helped to make possible the dramatic enlargement of our most temperature-sensitive organ, the brain.” That, she says, is how sweat helped to make you brainy.

Even at rest we sweat steadily, if inconspicuously, but if you add in vigorous activity and challenging conditions, we drain off our water supplies very quickly. According to Peter Stark in *Last Breath: Cautionary Tales from the Limits of Human Endurance*, a man who weighs 155 pounds will contain a little over forty-two quarts of water. If he does nothing at all but sit and breathe, he will lose about one and a half quarts of water per day through a combination of sweat, respiration, and urination. But if he exerts himself, that rate of loss can shoot up to one and a half quarts per hour. That can quickly become dangerous. In grueling conditions—walking under a hot sun, say—you can easily sweat away ten and a half to twelve and a half quarts of water in a day. No wonder we need to keep

hydrated when the weather is hot.

Unless the loss is halted or replenished, the victim will begin to suffer headaches and lethargy after losing just three to five quarts of fluid. After six or seven quarts of unrestored loss, mental impairment starts to become likely. (That is when dehydrated hikers leave a trail and wander into the wilderness.) If the loss gets much above ten and a half quarts for a 155-pound man, the victim will go into shock and die. During World War II, scientists studied how long soldiers could walk in a desert without water (assuming they were adequately hydrated at the outset) and concluded that they could go forty-five miles in 80-degree heat, fifteen miles in 100-degree heat, and just seven miles in 120-degree heat.

Your sweat is 99.5 percent water. The rest is about half salt and half other chemicals. Although salt is only a tiny part of your overall sweat, you can lose as much as three teaspoonfuls of it in a day in hot weather, which can be a dangerously high amount, so it is important to replenish salt as well as water. Sweating is activated by the release of adrenaline, which is why when you are stressed, you break into a sweat. Unlike the rest of the body, the palms don't sweat in response to physical exertion or heat, but only from stress. Emotional sweating is what is measured in lie detector tests.

Sweat glands come in two varieties: eccrine and apocrine. Eccrine glands are much the more numerous and produce the watery sweat that dampens your shirt on a sweltering day. Apocrine glands are confined mostly to the groin and armpits (technically the axilla) and produce a thicker, stickier sweat.

It is eccrine sweat in your feet—or more correctly the chemical breakdown by bacteria of the sweat in your feet—that accounts for their lush odor. Sweat on its own is actually odorless. It needs bacteria to create a smell. The two chemicals that account for the odor—isovaleric acid and methanediol—are also produced by bacterial actions on some cheeses, which is why feet and cheese can often smell so very alike.

Your skin microbes are exceedingly personal. The microbes

that live on you depend to a surprising degree on what soaps or laundry detergents you use, whether you favor cotton clothing or wool, whether you shower before work or after. Some of your microbes are permanent residents. Others camp out on you for a week or a month and then, like a wandering tribe, quietly vanish.

You have about 100,000 microbes per square centimeter of your skin, and they are not easily eradicated. According to one study, the number of bacteria on you actually rises after a bath or shower because they are flushed out from nooks and crannies. But even when you try scrupulously to sanitize yourself, it isn't easy. To make one's hands safely clean after a medical examination requires thorough washing with soap and water for at least a full minute—a standard that is, in practical terms, all but unattainable for anyone dealing with lots of patients. It is a big part of the reason why every year some two million Americans pick up a serious infection in the hospital (and ninety thousand of them die of it). “The greatest difficulty,” Atul Gawande has written, “is getting clinicians like me to do the one thing that consistently halts the spread of infections: wash our hands.”

A study at New York University in 2007 found that most people had about 200 different species of microbes on their skin, but the species load differed dramatically from person to person. Only four types appeared on everyone tested. In another widely reported study, the Belly Button Biodiversity Project, conducted by researchers at North Carolina State University, sixty random Americans had their belly buttons swabbed to see what was lurking there microbially. The study found 2,368 species of bacteria, 1,458 of which were unknown to science. (That is an average of 24.3 new-to-science microbes in every navel.) The number of species per person varied from 29 to 107. One volunteer harbored a microbe that had never been recorded outside Japan—where he had never been.

The problem with antibacterial soaps is that they kill good bacteria on your skin as well as bad. The same is true of hand

sanitizers. In 2016, the Food and Drug Administration banned nineteen ingredients commonly used in antibacterial soaps on the grounds that manufacturers had not proved them to be safe over the long term.

Microbes are not the only inhabitants of your skin. Right now, grazing in the divots on your head (and elsewhere on your oily surface, but above all on your head) are tiny mites called *Demodex folliculorum*. They are generally harmless, thank goodness, as well as invisible. They have lived with us for so long that according to one study their DNA can be used to track the migrations of our ancestors from hundreds of thousands of years ago. At their scale, your skin to them is like a giant crusty bowl of cornflakes. If you close your eyes and use your imagination, you can almost hear the crunching.

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One other thing the skin does a lot, for reasons not always understood, is itch. Although a great deal of itching is easily explained (mosquito bites, rashes, encounters with poison ivy), an awful lot of it is beyond explanation. As you read this passage, you may feel an urge to scratch yourself in various places that didn't itch at all a moment ago simply because I have raised the matter. No one can say why we are so suggestible with respect to itches or even why in the absence of obvious irritants we have them at all. No single location in the brain is devoted to itching, so it is all but impossible to study neurologically.

Itching (the medical term for the condition is pruritus) is confined to the outer layer of skin and a few moist outposts—eyes, throat, nose, and anus primarily. No matter how else you suffer, you will never have an itchy spleen. Studies of scratching showed that the most prolonged relief comes from scratching the back but the most pleasurable relief comes from scratching the ankle. Chronic itching occurs in all kinds of conditions—brain tumors, strokes, autoimmune disorders, as a side effect of

medications, and many more. One of the most maddening forms is phantom itching, which often accompanies an amputation and provides the miserable sufferer with a constant itch that simply cannot be satisfied. But perhaps the most extraordinary case of unappeasable suffering concerned a patient known as M., a Massachusetts woman in her late thirties who developed an irresistible itch on her upper forehead following a bout of shingles. The itch became so maddening that she rubbed the skin completely away over a patch of scalp about an inch and a half in diameter. Medications didn't help. She rubbed the spot especially furiously while asleep—so much so that one morning she awoke to find a trickle of cerebrospinal fluid running down her face. She had scratched through the skull bone and into her own brain. Today, more than a dozen years later, she is reportedly able to manage the scratch without doing severe damage to herself, but the itch has never gone away. What is most puzzling is that she has destroyed virtually all the nerve fibers in that patch of skin, yet the maddening itch remains.

Probably no mystery of the outer surface causes greater consternation, however, than our strange tendency to lose our hair as we age. We have about 100,000 to 150,000 hair follicles on our heads, though clearly not all follicles are equal among all people. You lose, on average, between fifty and a hundred head hairs every day, and sometimes they don't grow back. About 60 percent of men are "substantially bald" by the age of fifty. One man in five achieves that condition by thirty. Little is understood about the process, but what is known is that a hormone called dihydrotestosterone tends to go slightly haywire as we age, directing hair follicles on the head to shut down and more reserved ones in the nostrils and ears to spring to dismaying life. The one known cure for baldness is castration. Ironically, considering how easily some of us lose it, hair is pretty impervious to decay and has been known to last in graves for thousands of years.

Perhaps the most positive way to look at it is that if some

part of us must yield to middle age, the hair follicles are an obvious candidate for sacrifice. No one ever died of baldness, after all.

* “Corpuscle,” from the Latin, meaning “little body,” is a somewhat vague term anatomically speaking. It can signify either unattached, free-floating cells, as in blood corpuscles, or it can signify clumps of cells that function independently, as with Meissner’s corpuscles.

like health and diet), so the numbers are much closer to being equal—though it should also be noted that 85 percent of our own cells are red blood cells, which aren't true cells at all, because they don't have any of the usual machinery of cells (like nuclei and mitochondria), but are really just containers for hemoglobin. A separate consideration is that bacterial cells are tiny, whereas human cells are comparatively gigantic, so in terms of massiveness, not to mention the complexity of what they do, human cells are unquestionably more consequential. Then again, looked at genetically, you have about twenty thousand genes of your own within you, but perhaps as many as twenty million bacterial genes, so from that perspective you are roughly 99 percent bacterial and not quite 1 percent you.

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Microbial communities can be surprisingly specific. Although you and I will each have several thousand bacterial species within us, we may have only a fraction in common. Microbes are ferocious housekeepers, it seems. Have sex and you and your partner will perforce exchange a lot of microbes and other organic material. Passionate kissing alone, according to one study, results in the transfer of up to one billion bacteria from one mouth to another, along with about 0.7 milligrams of protein, 0.45 milligrams of salt, 0.7 micrograms of fat, and 0.2 micrograms of “miscellaneous organic compounds” (that is, bits of food). But as soon as the party is over, the host microorganisms in both participants will begin a kind of giant sweeping-out process, and within only a day or so the microbial profile for both parties will be more or less fully restored to what it was before they locked tongues. Occasionally, some pathogens sneak through, and that is when you get herpes or a head cold, but that is the exception.*¹

Luckily, most microbes have nothing to do with us. Some live benignly inside us and are known as commensals. Only a tiny portion of them make us ill. Of the million or so microbes that have been identified, just 1,415 are known to cause disease in humans—very few, all things considered. On the other hand, that is still a lot of ways to be unwell, and together those 1,415 tiny, mindless entities cause one-third of all the deaths on the planet.

As well as bacteria, your personal repertoire of microbes consists of fungi, viruses, protists (amoebas, algae, protozoa, and so on), and archaea, which for a long time were thought to be just more bacteria but actually represent a whole other branch of life. Archaea are very like bacteria in that they are quite simple and have no nucleus, but they have the great benefit to us that they cause no known diseases in humans. All they give us is a little gas, in the form of methane.

It's worth bearing in mind that all these microbes have almost nothing in common in terms of their history and genetics. All that unites them is

tininess. To all of them, you are not a person but a world—a vast and jouncing wealth of marvelously rich ecosystems with the convenience of mobility thrown in, along with the very helpful habits of sneezing, petting animals, and not always washing quite as fastidiously as you really ought to.

II

A VIRUS, IN the immortal words of the British Nobel laureate Peter Medawar, is “a piece of bad news wrapped up in a protein.” Actually, a lot of viruses are not bad news at all, at least not to humans. Viruses are a little weird, not quite living but by no means dead. Outside living cells, they are just inert things. They don’t eat or breathe or do much of anything. They have no means of locomotion. We must go out and collect them—off door handles or handshakes or drawn in with the air we breathe. They do not propel themselves; they hitchhike. Most of the time, they are as lifeless as a mote of dust, but put them into a living cell, and they will burst into animate existence and reproduce as furiously as any living thing.

Like bacteria, they are incredibly successful. The herpes virus has endured for hundreds of millions of years and infects all kinds of animals—even oysters. They are also terribly small—much smaller than bacteria and too small to be seen under conventional microscopes. If you blew one up to the size of a tennis ball, a human would be five hundred miles high. A bacterium on the same scale would be about the size of a beach ball.

In the modern sense of a very small microorganism, the term “virus” dates only from 1900, when a Dutch botanist, Martinus Beijerinck, found that the tobacco plants he was studying were susceptible to a mysterious infectious agent even smaller than bacteria. At first he called the mysterious agent *contagium vivum fluidum* but then changed it to “virus,” from a Latin word for “toxin.” Although he was the father of virology, the importance of his discovery wasn’t appreciated in his lifetime, so he was never honored with a Nobel Prize, as he really should have been.

It used to be thought that all viruses cause disease—hence the Peter Medawar quotation—but we now know that most viruses infect only bacterial cells and have no effect on us at all. Of the hundreds of thousands of viruses reasonably supposed to exist, just 586 species are known to infect mammals, and of these only 263 affect humans.

We know very little about most other, nonpathogenic viruses because only the ones that cause disease tend to get studied. In 1986, a student at the State University of New York at Stony Brook named Lita Proctor decided to look for viruses in seawater—which was considered a highly eccentric thing to do because it was universally assumed that the oceans have no viruses except perhaps for a transient few introduced through sewage outfall pipes and the

like. So it was a slight astonishment when Proctor found that the average quart of seawater contains up to 100 *billion* viruses. More recently, Dana Willner, a biologist at San Diego State University, looked into the number of viruses found in healthy human lungs—somewhere else that viruses were not thought to lurk much. Willner found that the average person harbored 174 species of virus, 90 percent of which had never been seen before. Earth, we now know, is aswarm with viruses to a degree that until recently we barely suspected. According to the virologist Dorothy H. Crawford, ocean viruses alone if laid end to end would stretch for ten million light-years, a distance essentially beyond imagining.

Something else viruses do is bide their time. A most extraordinary example of that came in 2014 when a French team found a previously unknown virus, *Pithovirus sibericum*, in Siberia. Although it had been locked in permafrost for thirty thousand years, when injected into an amoeba, it sprang into action with the lustiness of youth. Luckily, *P. sibericum* proved not to infect humans, but who knows what else may be out there waiting to be uncovered? A rather more common manifestation of viral patience is seen in the varicella-zoster virus. This is the virus that gives you chicken pox when you are small, but then may sit inert in nerve cells for half a century or more before erupting in that horrid and painful indignity of old age known as shingles. It is usually described as a painful rash on the torso, but in fact shingles can pop up almost anywhere on the body surface. A friend of mine had it in his left eye and described it as the worst experience of his life. (The word, incidentally, has nothing to do with the tiles of a roof. Shingles as a medical condition comes from the Latin *cingulus*, meaning a kind of belt; as a roofing material, it is from the Latin *scindula*, meaning a stepped tile. It is just by chance that they ended up in English with the same spellings.)

The most regular of unwelcome viral encounters is the common cold. Everyone knows that if you get chilled, you are more likely to catch a cold (that is why we call it a cold, after all), yet science has never been able to prove why—or even, come to that, *if* that is actually so. Colds unquestionably are more frequent in winter than in summer, but that may only be because we spend more time indoors then and are more exposed to others' leakages and exhalations.

The common cold is not a single illness but rather a family of symptoms generated by a multiplicity of viruses, of which the most pernicious are the rhinoviruses. These alone come in a hundred varieties. There are, in short, lots of ways to catch a cold, which is why you never develop enough immunity to stop catching them all.

For years, Britain operated a research facility called the Common Cold Unit, but it closed in 1989 without ever finding a cure. It did, however, conduct some interesting experiments. In one, a volunteer was fitted with a

device that leaked a thin fluid at his nostrils at the same rate that a runny nose would. The volunteer then socialized with other volunteers, as if at a cocktail party. Unknown to any of them, the fluid contained a dye visible only under ultraviolet light. When that was switched on after they had been mingling for a while, the participants were astounded to discover that the dye was everywhere—on the hands, head, and upper body of every participant and on glasses, doorknobs, sofa cushions, bowls of nuts, you name it. The average adult touches his face sixteen times an hour, and each of those touches transferred the pretend pathogen from nose to snack bowl to innocent third party to doorknob to innocent fourth party and so on until pretty much everyone and everything bore a festive glow of imaginary snot. In a similar study at the University of Arizona, researchers infected the metal door handle to an office building and found it took only about four hours for the “virus” to spread through the entire building, infecting over half of employees and turning up on virtually every shared device like photocopiers and coffee machines. In the real world, such infestations can stay active for up to three days. Surprisingly, the least effective way to spread germs (according to yet another study) is kissing. It proved almost wholly ineffective among volunteers at the University of Wisconsin who had been successfully infected with cold virus. Sneezes and coughs weren’t much better. The only really reliable way to transfer cold germs is physically by touch.

A survey of subway trains in Boston found that metal poles are a fairly hostile environment for microbes. Where microbes thrive is in the fabrics on seats and on plastic handgrips. The most efficient method of transfer for germs, it seems, is a combination of folding money and nasal mucus. A study in Switzerland in 2008 found that flu virus can survive on paper money for two and a half weeks if it is accompanied by a microdot of snot. Without snot, most cold viruses could survive on folding money for no more than a few hours.

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The two other forms of microbe that commonly lurk within us are fungi and protists. Fungi for a long time were a kind of scientific bewilderment, classified as just slightly strange plants. In fact, at a cellular level, they aren’t very like plants at all. They don’t photosynthesize, so they have no chlorophyll and thus are not green. They are actually more closely related to animals than to plants. It wasn’t until 1959 that they were recognized as quite separate and given their own kingdom. They essentially divide into two groups—molds and yeasts. By and large fungi leave us alone. Only about three hundred out of several million species affect us at all, and most of those mycoses, as they are known, don’t make you really ill, but rather cause only mild discomfort or irritation, as with athlete’s foot, say. A few, however, are much nastier than

that, and the number of nasty ones is growing.

Candida albicans, the fungus behind thrush, until the 1950s was found only in the mouth and genitals, but now it sometimes invades the deeper body, where it can grow on the heart and other organs, like mold on fruit. Similarly, *Cryptococcus gattii* was for decades known to exist in British Columbia in Canada, mostly on trees or in the soil around them, but it never harmed a human. Then, in 1999, it developed a sudden virulence, causing serious lung and brain infections among a scattering of victims in western Canada and the United States. Exact figures are impossible to come by because the disease is often misdiagnosed and, remarkably, is not reportable in California, one of the main sites of occurrence, but something over three hundred cases in western North America have been confirmed since 1999, with about a third of victims dying.

Rather better reported are figures for coccidioidomycosis, which is more commonly known as valley fever. It occurs almost entirely in California, Arizona, and Nevada, infecting about ten thousand to fifteen thousand people a year and killing about two hundred, though the actual number is probably higher because it can be confused with pneumonias. The fungus is found in soils, and the number of cases rises whenever soils are disturbed, as with earthquakes and dust storms. Altogether fungi are thought to be responsible for about a million deaths globally every year, so hardly inconsequential.

Finally, protists. A protist is anything that isn't obviously plant, animal, or fungus; it is a category reserved for all those life-forms that don't fit anywhere else. Originally, in the nineteenth century, all single-celled organisms were called protozoa. It was assumed that all were closely related, but over time it became evident that bacteria and archaea were separate kingdoms. Protists is a huge category and includes amoebas, parameciums, diatoms, slime molds, and many others that are mostly obscure to all but people working in biological fields. From a human health perspective, the most notable protists are those from the genus *Plasmodium*. They are the evil little creatures that transfer from mosquitoes into us and give us malaria. Protists are also responsible for toxoplasmosis, giardiasis, and cryptosporidiosis.

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There is, in short, an astounding array of microbes all around us, and we have barely begun to understand their effects on us, for good and ill. A most arresting illustration of that arose in 1992 in the north of England in the old mill town of Bradford, West Yorkshire, when Timothy Rowbotham, a government microbiologist, was sent to try to track down the source of an outbreak of pneumonia. In a sample of water he took from a storage tower, he found a microbe unlike anything he or anyone else had ever seen before. He

interpretation. First, Fleming deserves credit for perceiving the significance of the mold; a less alert scientist might simply have tossed the whole lot out. Moreover, he dutifully reported his discovery, and even noted the antibiotic implications of it, in a respected journal. He also made some effort to turn the discovery into a usable medicine, but it was a technically tricky proposition—as others would later discover—and he had more pressing research interests to pursue, so he didn't stick with it. It is often overlooked that Fleming was a distinguished and busy scientist already. He had in 1923 discovered lysozyme, an antimicrobial enzyme found in saliva, mucus, and tears as part of the body's first line of defense against invading pathogens, and was still preoccupied with exploring its properties. He was hardly foolish or slapdash, as is sometimes implied.

In the early 1930s, researchers in Germany produced a group of antibacterial drugs known as sulfonamides, but they didn't always work well and often had serious side effects. At Oxford, a team of biochemists led by the Australian-born Howard Florey began searching for a more effective alternative and in the process rediscovered Fleming's penicillin paper. The principal investigator at Oxford was an eccentric German émigré named Ernst Chain, who bore an uncanny resemblance to Albert Einstein (right down to the bushy mustache) but had a far more challenging disposition. Chain had grown up in a wealthy Jewish family in Berlin but had decamped to England with the rise of Adolf Hitler. Chain was gifted in many fields and considered a career as a concert pianist before settling on science. But he was also a difficult man. He had a volatile temperament and slightly paranoid instincts, though it seems fair to say that if there was ever a time when a Jew might be excused paranoia it was the 1930s. He was an unlikely candidate to make any discoveries because he had a pathological fear of being poisoned in a lab. Despite his dread, he persevered and found to his astonishment that penicillin not only killed pathogens in mice but had no evident side effects. It appeared to be the perfect drug: one that could devastate its target without wreaking collateral damage. The problem, as Fleming had seen, was that it was very hard to produce penicillin in clinically useful quantities. Under Florey's command, Oxford gave over a significant amount of resources and research space to growing mold and patiently extracting from it tiny amounts of penicillin.

By early 1941, they had just enough to trial the drug on a policeman named Albert Alexander, who was a tragically ideal demonstration of how vulnerable humans were to infections before antibiotics. While pruning roses in his garden, Alexander had scratched his face on a thorn. The scratch had grown infected and spread. Alexander had lost an eye and now was delirious and close to death. The effect of penicillin was miraculous. Within two days, he was sitting up and looking almost back to normal. But supplies quickly ran short. In desperation the scientists filtered and reinjected all they could from

Alexander's urine, but after four days the supplies were exhausted. Poor Alexander relapsed and died.

With Britain preoccupied by World War II and the United States not yet in it, the quest to produce bulk penicillin moved to a U.S. government research facility in Peoria, Illinois. Scientists and other interested parties all over the Allied world were secretly asked to send in soil and mold samples. Hundreds responded, but nothing they sent proved promising. Then, two years after testing had begun, a lab assistant in Peoria named Mary Hunt brought in a cantaloupe from a local grocery store. It had a "pretty golden mold" growing on it, she recalled later. That mold proved to be two hundred times more potent than anything previously tested. The name and location of the store where Mary Hunt shopped are now forgotten, and the historic cantaloupe itself was not preserved: after the mold was scraped off, it was cut into pieces and eaten by the staff. But the mold lived on. Every bit of penicillin made since that day is descended from that single random cantaloupe.

Within a year, American pharmaceutical companies were producing 100 billion units of penicillin a month. The British discoverers found to their chagrin that the production methods had been patented by the Americans and that they were now required to pay royalties to make use of their own discovery.

Alexander Fleming didn't become famous as the father of penicillin until the closing days of the war, some twenty years after his serendipitous discovery, but then he became very famous indeed. He received 189 honors of all types from around the world, and even had a crater on the moon named for him. In 1945, he shared the Nobel Prize in Physiology or Medicine with Ernst Chain and Howard Florey. Florey and Chain never enjoyed the popular acclaim they deserved, partly because they were much less gregarious than Fleming and partly because his story of accidental discovery made better copy than their story of dogged application. Chain, despite sharing the Nobel Prize, became convinced that Florey had not given him sufficient credit, and their friendship, such as it was, dissolved.

As early as 1945, in his Nobel acceptance speech, Fleming warned that microbes could easily evolve resistance to antibiotics if they were carelessly used. Seldom has a Nobel speech been more prescient.

IV

THE GREAT VIRTUE of penicillin—that it scythes its way through all manner of bacteria—is also its elemental weakness. The more we expose microbes to antibiotics, the more opportunity they have to develop resistance. What you are left with after a course of antibiotics, after all, are the most resistant microbes. By attacking a broad spectrum of bacteria, you stimulate lots of

defensive action. At the same time, you inflict unnecessary collateral damage. Antibiotics are about as nuanced as a hand grenade. They wipe out good microbes as well as bad. Increasing evidence shows that some of the good ones may never recover, to our permanent cost.

Most people in the Western world, by the time they reach adulthood, have received between five and twenty courses of antibiotics. The effects, it is feared, may be cumulative, with each generation passing on fewer microorganisms than the one before. Few people are more aware of this than an American scientist named Michael Kinch. In 2012, when he was director of the Yale Center for Molecular Discovery in Connecticut, Kinch's twelve-year-old son, Grant, developed severe abdominal pains.

"He'd been at the first day of a summer camp and he'd eaten some cupcakes," Kinch recalls, "so we thought at first it was just a combination of excitement and overindulgence, but the symptoms got worse." Eventually, Grant ended up in Yale New Haven Hospital, where a number of alarming things happened quickly. It was found that he had a ruptured appendix and that his intestinal microbes had escaped into the abdomen, giving him peritonitis. Then the infection developed into septicemia, which meant it had spread to his blood and could go anywhere in his body. Dismayingly, four of the antibiotics Grant was given didn't have any effect on the marauding bacteria.

"That was really astounding," Kinch recalls now. "This was a kid who had been on antibiotics just once in his life, for an ear infection, and yet he had gut bacteria that were resistant to antibiotics. That shouldn't have happened." Fortunately, two other antibiotics did work and Grant's life was saved.

"He was lucky," Kinch says. "The day is fast approaching when the bacteria inside us may not be resistant to two-thirds of the antibiotics we hit them with, but to all of them. Then we really are in trouble."

Today Kinch is the director of the Center for Research Innovation in Business at Washington University in St. Louis. He works in a once derelict, now stylishly renovated telephone factory that is part of a neighborhood salvation project undertaken by the university. "This used to be the best place in St. Louis to score crack," he says with a hint of ironic pride. A cheerful man of early middle years, Kinch was brought to Washington University to foster entrepreneurship, but one of his central passions remains the future of the pharmaceutical industry and where new antibiotics will come from. In 2016, he wrote an alarming book on the matter, *A Prescription for Change: The Looming Crisis in Drug Development*.

"From the 1950s through the 1990s," he says, "roughly three antibiotics were introduced into the U.S. every year. Today it's roughly one new antibiotic every other year. The rate of antibiotic withdrawals—because they don't work anymore or have become obsolete—is twice the rate of new

introductions. The obvious consequence of this is that the arsenal of drugs we have to treat bacterial infections has been going down. There is no sign of it stopping.”

What makes this much worse is that a great deal of our antibiotic use is simply crazy. Almost three-quarters of the forty million antibiotic prescriptions written each year in the United States are for conditions that cannot be cured with antibiotics. According to Jeffrey Linder, professor of medicine at Northwestern University, antibiotics are prescribed for 70 percent of acute bronchitis cases, even though guidelines explicitly state that they are of no use there.

Even more appallingly, in the United States 80 percent of antibiotics are fed to farm animals, mostly to fatten them. Fruit growers can also use antibiotics to combat bacterial infections in their crops. In consequence, most Americans consume secondhand antibiotics in their food (including even some foods labeled as organic) without knowing it. Sweden banned the agricultural use of antibiotics in 1986. The European Union followed in 1999. In 1977, the Food and Drug Administration ordered a halt to the use of antibiotics for purposes of fattening farm animals, but backed off when there was an outcry from agricultural interests and the congressional leaders who supported them.

In 1945, the year that Alexander Fleming won the Nobel Prize, a typical case of pneumococcal pneumonia could be knocked out with forty thousand units of penicillin. Today, because of increased resistance, it can take more than twenty million units per day for many days to achieve the same result. On some diseases, penicillin now has no effect at all. In consequence, the death rate for infectious diseases has been climbing and is back to the level of about forty years ago.

Bacteria really are not to be trifled with. They not only have grown steadily more resistant but have evolved into a fearsome new class of pathogen commonly known, with scarcely a hint of hyperbole, as superbugs. *Staphylococcus aureus* is a microbe found commonly on human skin and in nostrils. Generally it does no harm, but it is an opportunist, and when the immune system is weakened, it can slip in and wreak havoc. By the 1950s, it had evolved resistance to penicillin, but luckily another antibiotic called methicillin had become available and it stopped *S. aureus* infections in their tracks. But just two years after methicillin's introduction, two people at the Royal Surrey County Hospital in Guildford, near London, developed *S. aureus* infections that would not respond to methicillin. *S. aureus* had, almost overnight, evolved a new drug-resistant form. The new strain was dubbed Methicillin-resistant *Staphylococcus aureus*, or MRSA. Within two years, it had spread to mainland Europe. Soon after that, it leaped to the United States.

Today, MRSA and its cousins kill an estimated 700,000 people around the world annually. Until recently a drug called vancomycin was effective against MRSA, but now resistance has begun to emerge to it. At the same time, we are facing the formidable-sounding carbapenem-resistant Enterobacteriaceae (CRE) infections, which are immune to virtually everything we can throw at them. CRE kills about half of all those it sickens. Luckily, so far, it doesn't usually infect healthy people. But watch out if it does.

Yet as the problem has grown, the pharmaceutical industry has retreated from trying to create new antibiotics. "It's just too expensive for them," Kinch says. "In the 1950s, for the equivalent of a billion dollars in today's money, you could develop about ninety drugs. Today, for the same money, you can develop on average just one-third of a drug. Pharmaceutical patents last only for twenty years, but that includes the period of clinical trials. Manufacturers usually have just five years of exclusive patent protection." In consequence, all but two of the eighteen largest pharmaceutical companies in the world have given up the search for new antibiotics. People take antibiotics for only a week or two. Much better to focus on drugs like statins or antidepressants that people can take more or less indefinitely. "No sane company will develop the next antibiotic," Kinch says.

The problem needn't be hopeless, but it does need to be addressed. At the current rate of spread, antimicrobial resistance is forecast to lead to ten million preventable deaths a year—that's more people than die of cancer now—within thirty years, at a cost of perhaps \$100 trillion in today's money.

What nearly everyone agrees is that we need a more targeted approach. One interesting possibility would be to disrupt bacteria's lines of communication. Bacteria never mount an attack until they have assembled sufficient numbers—what is known as a quorum—to make it worthwhile to do so. The idea would be to produce quorum-sensing drugs that wouldn't kill all bacteria but would just keep their numbers permanently below the threshold, the quorum, that triggers an attack.

Another possibility is to enlist bacteriophages, a kind of virus, to hunt down and kill harmful bacteria for us. Bacteriophages—often shortened to just phages—are not well known to most of us, but they are the most abundant bioparticles on Earth. Virtually every surface on the planet, including us, is covered in them. They do one thing supremely well: each one targets a particular bacterium. That means clinicians would have to identify the offending pathogen and select the right phage to kill it, a more costly and time-consuming process, but it would make it much harder for bacteria to evolve resistance.

What is certain is that something must be done. "We tend to refer to the antibiotics crisis as a looming one," Kinch says, "but it is not that at all. It's a

taps of Morse code. And out of this bare and neutral information it creates for you—quite literally creates—a vibrant, three-dimensional, sensually engaging universe. Your brain is you. Everything else is just plumbing and scaffolding.

Just sitting quietly, doing nothing at all, your brain churns through more information in thirty seconds than the Hubble Space Telescope has processed in thirty years. A morsel of cortex one cubic millimeter in size—about the size of a grain of sand—could hold two thousand terabytes of information, enough to store all the movies ever made, trailers included, or about 1.2 billion copies of this book. Altogether, the human brain is estimated to hold something on the order of two hundred exabytes of information, roughly equal to “the entire digital content of today’s world,” according to *Nature Neuroscience*.^{*1} If that is not the most extraordinary thing in the universe, then we certainly have some wonders yet to find.

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The brain is often depicted as a hungry organ. It makes up just 2 percent of our body weight but uses 20 percent of our energy. In newborn infants, it’s no less than 65 percent. That’s partly why babies sleep all the time—their growing brains exhaust them—and have a lot of body fat, to use as an energy reserve when needed. Your muscles actually use even more of your energy, about a quarter, but you have a lot of muscle; per unit of matter, the brain is by far the most expensive of our organs. But it is also marvelously efficient. Your brain requires only about four hundred calories of energy a day—about the same as you get in a blueberry muffin. Try running your laptop for twenty-four hours on a muffin and see how far you get.

Unlike other parts of the body, the brain burns its four hundred calories at a steady rate no matter what you are doing. Hard thinking doesn’t help you slim. In fact, it doesn’t seem to confer any benefit at all. An academic at the University of California at Irvine named Richard Haier used positron

emission tomography scanners to find that the hardest-working brains are usually the least productive. The most efficient brains, he found, were those that could solve a task quickly and then go into a kind of standby mode.

For all its powers, nothing about your brain is distinctively human. We use exactly the same components—neurons, axons, ganglia, and so on—as a dog or hamster. Whales and elephants have much larger brains than we have, though of course they also have much larger bodies. But even a mouse scaled up to the size of a human would have a brain just as big, and many birds would do even better. It also turns out that the human brain is a little less imposing than we had long assumed. For years, it was written that it has 100 billion nerve cells, or neurons, but a careful assessment by the Brazilian neuroscientist Suzana Herculano-Houzel in 2015 found that the number is more like 86 billion—a pretty substantial demotion.

Neurons are not like other cells, which are typically compact and spherical. Neurons are long and stringy, the better to pass on electrical signals from one to another. The main strand of a neuron is called an axon. At its terminal end, it splits into branch-like extensions called dendrites, as many as 400,000 of them. The tiny space between nerve cell endings is called a synapse. Each neuron connects with thousands of other neurons, giving trillions and trillions of connections—as many connections “in a single cubic centimeter of brain tissue as there are stars in the Milky Way,” to quote the neuroscientist David Eagleman. It is in all that complex synaptic entanglement that our intelligence lies, not in the number of neurons, as was once thought.

What is surely most curious and extraordinary about our brain is how largely unnecessary it is. To survive on Earth, you don't need to be able to write music or engage in philosophy—you really only need to be able to outthink a quadruped—so why have we invested so much energy and risk in producing mental capacity that we don't really need? That is just one of the many things about your brain that your brain won't tell you.

As the most complex of our organs, the brain not surprisingly has more named features and landmarks than any other part of the body, but essentially it divides into three sections. At the top, literally and figuratively, is the cerebrum, which fills most of the cranial vault and is the part that we normally think of when we think of “the brain.” The cerebrum (from the Latin word for “brain”) is the seat of all our higher functions. It is divided into two hemispheres, each of which is principally concerned with one side of the body, but for reasons unknown the wiring is crossed, so that the right side of the cerebrum controls the left side of the body and vice versa. The two hemispheres are connected by a band of fibers called the corpus callosum (meaning “tough material” or literally “calloused body” in Latin). The brain is wrinkled by deep fissures known as sulci and ridges called gyri, which give it more surface area. The exact pattern of grooves and ridges in brains is distinctive to each individual—as distinctive as your fingerprints—but whether it has anything to do with your intelligence or temperament or anything else that defines you is unknown.

Each hemisphere of the cerebrum is further divided into four lobes: frontal, parietal, occipital, and temporal—each broadly specializing in certain functions. The parietal lobe manages sensory inputs like touch and temperature. The occipital lobe processes visual information, and the temporal lobe principally manages auditory information, though it also helps with processing visual information. It has been known for some years that six patches on the temporal lobe, known as face patches, become excited when we look at another face, though which parts of my face excite which of your patches is still largely uncertain, it seems. The frontal lobe is the seat of the higher functions of the brain—reasoning, forethought, problem solving, emotional control, and so on. It is the part responsible for personality, for who we are. Ironically, as Oliver Sacks once noted, the frontal lobes were the last parts of the brain to be

deciphered. “Even in my own medical student days, they were called ‘the silent lobes,’ ” he wrote in 2001. That’s not because they were thought to lack functions but because those functions do not reveal themselves.

Beneath the cerebrum, at the very back of the head about where it meets the nape of the neck, is the cerebellum (Latin for “little brain”). Although the cerebellum occupies just 10 percent of the cranial cavity, it has more than half the brain’s neurons. It has a lot of neurons not because it does a great deal of thinking but because it controls balance and complex movements, and that requires an abundance of wiring.

At the base of the brain, descending from it rather like an elevator shaft connecting the brain to the spine and the body beyond, is the oldest part of the brain, the brain stem. It is the home of our more basic operations: sleeping, breathing, keeping the heart going. The brain stem doesn’t get a lot of attention in the popular consciousness, but it is so central to our existence that “brain-stem death” is the fundamental measure of deadness in humans in the United Kingdom.

Scattered through the brain rather like nuts in a fruitcake are many smaller structures—hypothalamus, amygdala, hippocampus, telencephalon, septum pellucidum, habenular commissure, entorhinal cortex, and a dozen or so others—which are collectively known as the limbic system (from the Latin *limbus*, meaning “peripheral”). It’s easy to go a lifetime without hearing a word about any of these components unless they go wrong. The basal ganglia, for instance, play an important part in movement, language, and thought, but it is only when they degenerate and lead to Parkinson’s disease that they normally attract attention to themselves.

Despite their obscurity and modest dimensions, the structures of the limbic system have a fundamental role in our happiness by controlling and regulating basic processes like memory, appetite, emotions, drowsiness and alertness, and the processing of sensory information. The concept of the limbic system was invented in 1952 by an American neuroscientist,

Paul D. MacLean. Not all of today's neuroscientists agree that the components form a coherent system. Many think they are just lots of disparate parts connected only by the fact that they are concerned with bodily performance rather than with thinking.

The most important component of the limbic system is a little powerhouse called the hypothalamus, which isn't really a structure at all but just a bundle of neural cells. The name describes not what it does but where it is: under the thalamus. (The thalamus, meaning "inner chamber," is a kind of relay station for sensory information and is an important part of the brain—there isn't any part of the brain that isn't important, obviously—but is not a component of the limbic system.) The hypothalamus is curiously unimposing. Though only about the size of a peanut and weighing barely a tenth of an ounce, it controls much of the most important chemistry of the body. It regulates sexual function, controls hunger and thirst, monitors blood sugar and salts, decides when you need to sleep. It may even play a part in how slowly or rapidly we age. A large measure of your success or failure as a human being is dependent on this tiny thing in the middle of your head.

The hippocampus is central to the laying down of memories. (The name comes from the Greek for "sea horse" because of its supposed resemblance to that creature.) The amygdala (Greek for "almond") specializes in handling intense and stressful emotions—fear, anger, anxiety, phobias of all types. People whose amygdalae are destroyed are left literally fearless, and often cannot even recognize fear in others. The amygdala grows particularly lively when we are asleep, and thus may account for why our dreams are so often disturbing. Your nightmares may simply be the amygdalae unburdening themselves.^{*2}

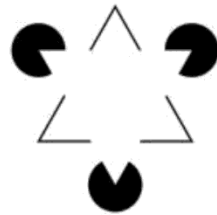
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Considering how exhaustively the brain has been studied, and for how long, it is remarkable how much elemental stuff we still

before. It's just that the foam reflects light in a different way. You get the same effect with crashing waves on a beach—greeny-blue water, white foam—and lots of other phenomena. That is because color isn't a fixed reality but a perception.

You have probably at some time or other encountered one of those illusion tests that require you to stare for fifteen or twenty seconds at a red square, then shift your vision to a blank sheet of paper, and for a few moments you will see a ghostly square of greenish blue on the white paper. This “afterimage” is a consequence of tiring some of the photoreceptors in your eyes by making them work extra intently, but what is relevant is that the greenish-blue color is not there and has never existed anywhere but in your imagination. In a very real sense, that is true of all colors.

Your brain is also extraordinarily good at finding patterns and determining order in chaos, as these two well-known illusions show:



In the first illustration, most people see only random smudges until it is pointed out to them that the picture contains a dalmatian dog; then suddenly for nearly everyone the brain fills in the missing edges and makes sense of the whole composition. The illusion dates from the 1960s, but no one seems to have kept a record of who first created it.

The second illustration does have a known history. It is

called a Kanizsa triangle, after the Italian psychologist Gaetano Kanizsa, who created it in 1955. There is of course no actual triangle in the picture, except for the one your brain puts there.

Your brain does all these things for you because it is designed to help you in every way it can. Yet paradoxically it is also strikingly unreliable. Some years ago, a psychologist at the University of California at Irvine, Elizabeth Loftus, discovered that it is possible through suggestion to implant entirely false memories in people's heads—to convince them that they were traumatically lost in a department store or shopping mall when they were small or that they were hugged by Bugs Bunny at Disneyland—even though these things never happened. (Bugs Bunny is not a Disney character and has never been at Disneyland.) She could show many people pictures of themselves as a child in which the image had been manipulated to make them look as if they were in a hot-air balloon, and often the subjects would suddenly remember the experience and excitedly describe it, even though in each case it was known that it had never happened.

Now, you might think that you could never be that suggestible, and you would probably be right—only about one-third of people are that gullible—but other evidence shows that we all sometimes completely misrecall even the most vivid events. In 2001, immediately after the 9/11 disaster at the World Trade Center in New York, psychologists at the University of Illinois took detailed statements from seven hundred people about where they were and what they were doing when they learned of the event. One year later, the psychologists asked the same question of the same people and found that nearly half now contradicted themselves in some significant way—put themselves in a different place when they learned of the disaster, believed that they had seen it on TV when in fact they had heard it on the radio, and so on—but without being aware that their recollections had changed. (I, for my part, vividly recall watching the events live on television in New Hampshire, where we were then living, with two of my

children, only to learn later that one of those children was in fact in England at the time.)³

Memory storage is idiosyncratic and strangely disjointed. The mind breaks each memory into its component parts—names, faces, locations, contexts, how a thing feels to the touch, even whether it is living or dead—and sends the parts to different places, then calls them back and reassembles them when the whole is needed again. A single fleeting thought or recollection can fire up a million or more neurons scattered across the brain. Moreover, these fragments of memory move around over time, migrating from one part of the cortex to another, for reasons entirely unknown. It's no wonder we get details muddled.

The upshot is that memory is not a fixed and permanent record, like a document in a filing cabinet. It is something much more hazy and mutable. As Elizabeth Loftus told an interviewer in 2013, “It’s a little more like a *Wikipedia* page. You can go in there and change it, and so can other people.”

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Memories are categorized in many different ways, and no two authorities seem to use quite the same terminologies. The most frequently cited divisions are long-term, short-term, and working (for duration) and procedural, conceptual, semantic, declarative, implicit, autobiographical, and sensual (for type). Fundamentally, however, memories come in two principal varieties: declarative and procedural. Declarative memory is the kind you can put into words—the names of state capitals, your date of birth, how to spell “ophthalmologist,” and everything else you know as fact. Procedural memory describes the things you know and understand but couldn’t so easily put into words—how to swim, drive a car, peel an orange, identify colors.

Working memory is where short-term and long-term memories combine. Say you are presented with a mathematical problem to solve. The problem resides in short-term memory—

you won't, after all, need to remember the problem months from now—but the skills necessary to make the computation are kept in long-term memory.

Researchers also sometimes find it useful to distinguish between recall memory, which is what you can remember spontaneously—the kinds of things you know when you do a general knowledge quiz—and recognition memory, which is where you are a bit hazy on the substance but can recall the context. Recognition memory explains why so many of us struggle to remember the contents of a book but can often recall where we read the book, the color or design of the cover, and other seeming irrelevancies. Recognition memory is actually useful because it doesn't clutter the brain with unnecessary details but does help us to remember where we can find those details if we should need them again.

Short-term memory is really short—no more than half a minute or so for things like addresses and phone numbers. (If you can still remember something after half a minute, it is no longer technically a short-term memory. It's long term.) Most people's short-term memory is pretty abysmal. Six random words or digits is about all that most of us can reliably retain for more than a few moments.

On the other hand, with effort we can train our memories to perform the most extraordinary stunts. Every year the United States has a national memory championship, and the feats performed there are truly astounding. One memory champion could recall 4,140 random digits after looking at them for just thirty minutes. Another was able to remember twenty-seven randomly shuffled decks of cards in the same time period. Yet another could recall a single deck of cards after thirty-two seconds of study. That may not be the most worthwhile use of the human mind, but it is certainly a demonstration of its incredible powers and versatility. Most of the memory champions, by the way, are not spectacularly intelligent. They just are motivated enough to train their memories to do some extraordinary tricks.

It used to be thought that every experience is stored permanently as memory somewhere in the brain but that most of it is locked away beyond our power of immediate recall. The idea arose principally from a series of experiments in Canada from the 1930s to the 1950s by the neurosurgeon Wilder Penfield. While carrying out surgical procedures at the Montreal Neurological Institute, Penfield discovered that when he touched a probe to patients' brains, it often evoked powerful sensations—vivid smells from childhood, feelings of euphoria, sometimes a recollection of a forgotten scene from very early life. From this it was concluded that the brain records and stores every conscious event in our lives, however trivial. Now, however, it is thought that the stimulation was mostly providing the sensation of memory and that what the patients were experiencing was more like a hallucination than a recalled event.

What is certainly true is that we retain a great deal more than we can easily summon to mind. You may not recollect much of a neighborhood you lived in when you were small, but if you went back and walked around it, you would almost certainly remember very particular details you hadn't thought about for years. With sufficient time and prompting, we would probably all be astonished at how much we have stored away inside us.

The person from whom we learned a good deal of what we know about memory was, ironically, a man who had very little of it himself. Henry Molaison was an amiable and good-looking young man of twenty-seven in Connecticut who suffered from crippling episodes of epilepsy. In 1953, inspired by the efforts of Wilder Penfield in Canada, a surgeon named William Scoville drilled into Molaison's head and removed half of the hippocampus from each side of his brain and most of the amygdalae. The procedure greatly reduced Molaison's seizures (though it didn't entirely eliminate them) but at the tragic cost of robbing him of the ability to form new memories—a condition known as anterograde amnesia. Molaison could recall