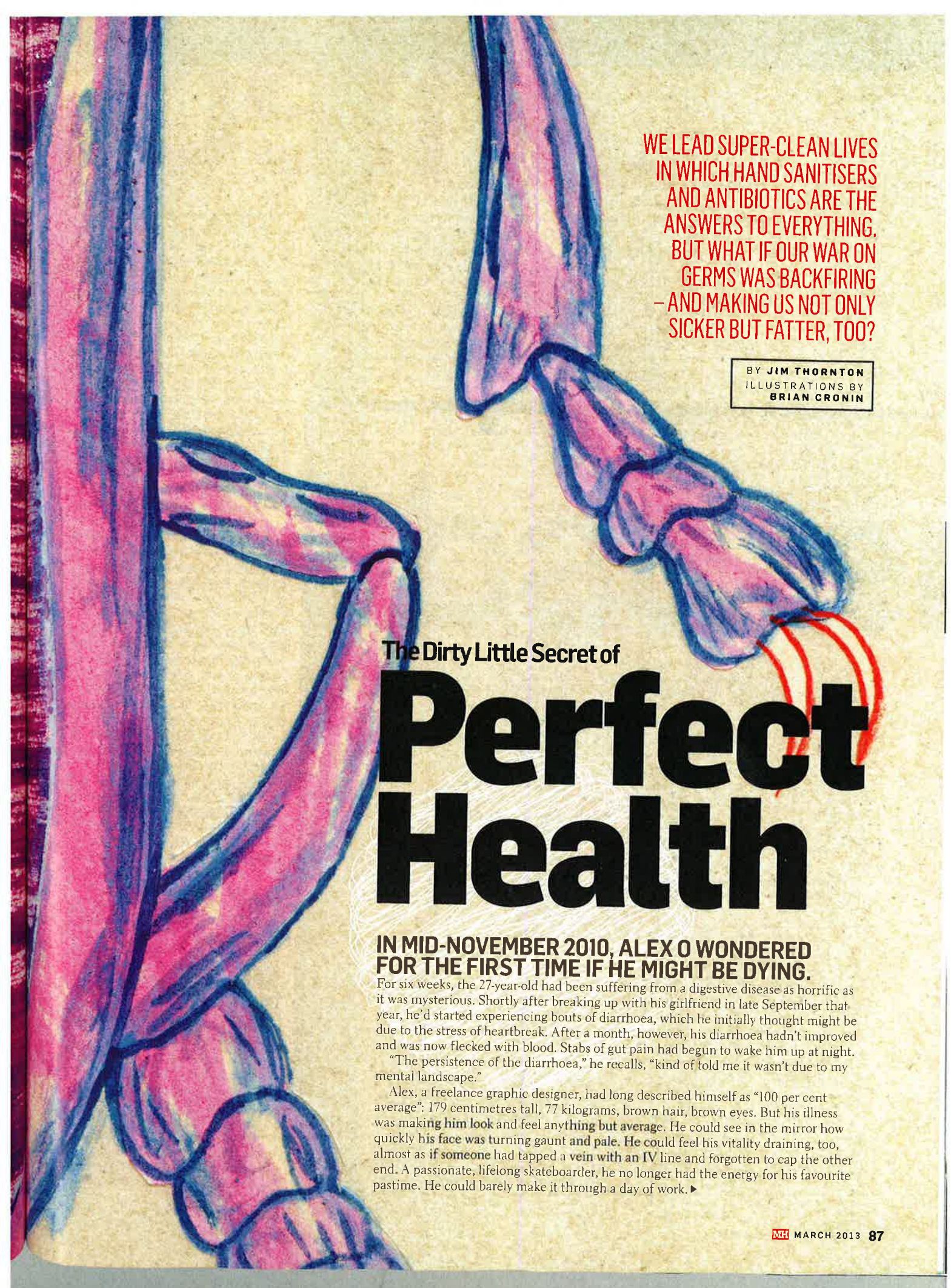

WE LEAD SUPER-CLEAN LIVES IN WHICH HAND SANITISERS AND ANTIBIOTICS ARE THE ANSWERS TO EVERYTHING. BUT WHAT IF OUR WAR ON GERMS WAS BACKFIRING – AND MAKING US NOT ONLY SICKER BUT FATTER, TOO?

BY **JIM THORNTON**
ILLUSTRATIONS BY **BRIAN CRONIN**

The Dirty Little Secret of

Perfect Health

IN MID-NOVEMBER 2010, ALEX O WONDERED FOR THE FIRST TIME IF HE MIGHT BE DYING.

For six weeks, the 27-year-old had been suffering from a digestive disease as horrific as it was mysterious. Shortly after breaking up with his girlfriend in late September that year, he'd started experiencing bouts of diarrhoea, which he initially thought might be due to the stress of heartbreak. After a month, however, his diarrhoea hadn't improved and was now flecked with blood. Stabs of gut pain had begun to wake him up at night.

"The persistence of the diarrhoea," he recalls, "kind of told me it wasn't due to my mental landscape."

Alex, a freelance graphic designer, had long described himself as "100 per cent average": 179 centimetres tall, 77 kilograms, brown hair, brown eyes. But his illness was making him look and feel anything but average. He could see in the mirror how quickly his face was turning gaunt and pale. He could feel his vitality draining, too, almost as if someone had tapped a vein with an IV line and forgotten to cap the other end. A passionate, lifelong skateboarder, he no longer had the energy for his favourite pastime. He could barely make it through a day of work. ▶

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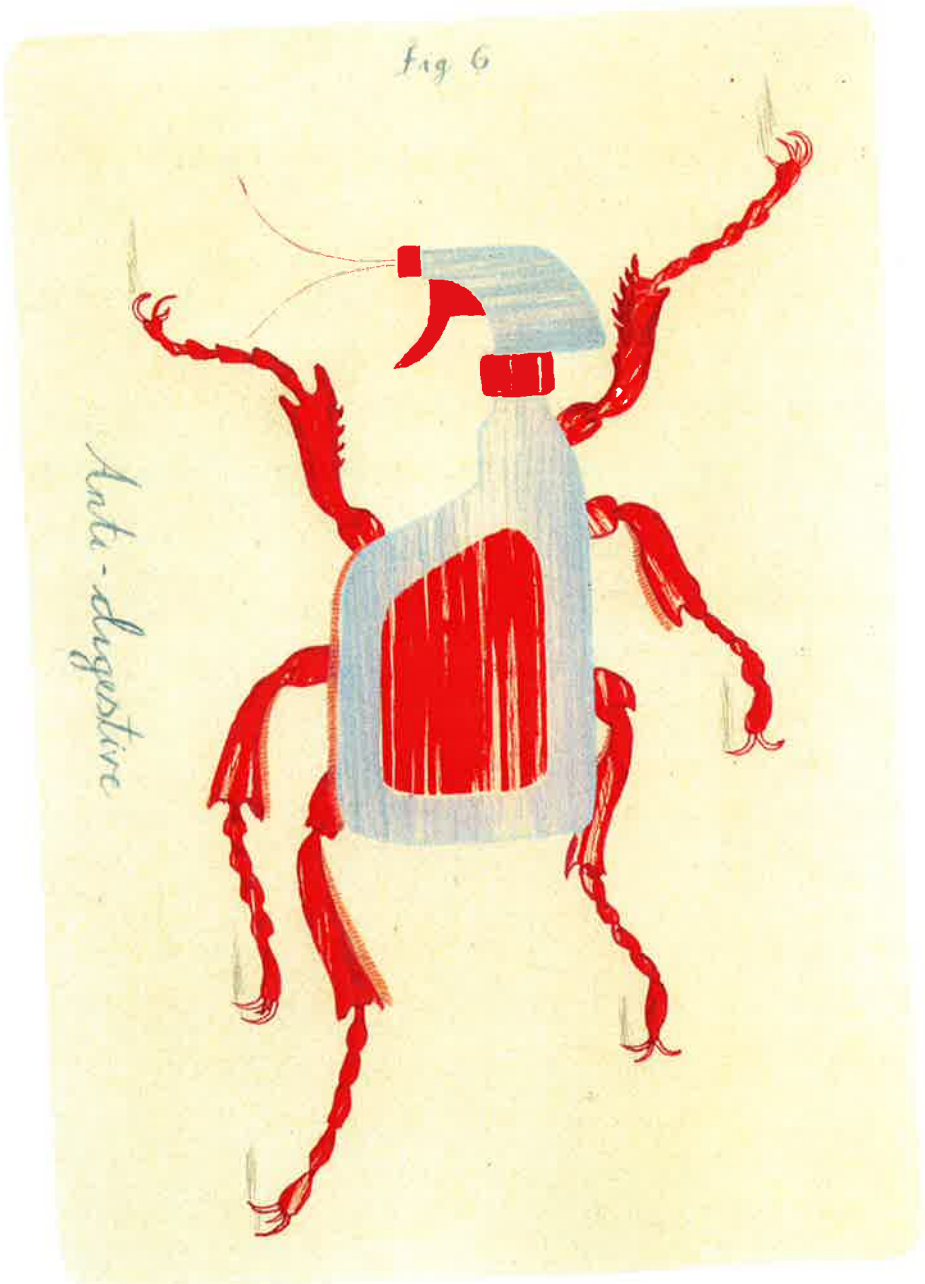
Alex's family doctor had tried everything he could think of, including diet changes and a week-long course of antibiotics. Nothing worked. The doctor finally referred Alex to a gastroenterologist, who ordered tests for three potential culprits: cancer, HIV/AIDS and a gut infection caused by a bacterium called *Clostridium difficile*, or *C. diff*, for short. First identified as a cause of intestinal infection in the Seventies, this rod-shaped bacterium inhabits the digestive tract and in some people doesn't produce symptoms. It can be harmless, provided its population remains under control.



Considering the alternatives, Alex found himself hoping his problem was an over-production of *C. diff*. "When the gastroenterologist explained what he was testing for, he didn't rate one possibility as more likely than the others, because he didn't want to give me false hope," he says. "I remember waiting for the HIV/AIDS test, in particular, which he'd ordered because I was so anaemic. There was one night when I thought that it probably was HIV and that I might die from it. It's really awful to say, but physically and mentally I was in so much pain I almost wished I were dead."

Eventually, the doctors ruled out cancer and HIV. Alex was so relieved when the tests came back positive for an infection that the news struck him as more curious than dire. What he didn't know then was that eliminating this stomach bug is one of the most difficult battles faced by infectious-disease specialists today – and one they often lose.

Each year in the US, *C. diff* kills more people than HIV does. Here, 30 people are known to have died in Sydney and Melbourne, according to Professor Thomas Borody, medical director of the Sydney-based Centre for Digestive Diseases (CDD). "But it's not a reportable disease, so we don't know how many Australians are carrying it," he says. "There's a groundswell in the medical community to make it reportable, so we're hoping that from sometime in 2013, that'll be the case." (See "Australia set for *C. Diff* epidemic", page 92.)

TAKE A QUICK look at yourself in a full-length mirror. What stares back is first and foremost a human being: a massive assortment of human cells organised into human tissues and human organs.

If this conventional description seems reasonable to you, brace yourself for a fundamental shift in self-concept: for every one cell in our bodies, at least 10 microbes – from bacteria to fungi to viruses – piggyback atop and within us. Thanks to powerful new investigative tools such as next-generation gene sequencers, scientists continue to uncover an astonishing diversity of species. To date they've been concentrating on bacteria. This is partly

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because these are our most common fellow travellers, and partly because technologies for sampling viruses, fungi and other such organisms are still being refined.

The figures are nothing short of staggering. Up to 100,000,000,000,000 (that's 100 trillion) individual bacterial cells from thousands of different species colonise everything from the mucous membranes of your nostrils to the lining of your urethra, and a myriad of body niches in-between. An infinitesimal pittance of these bacteria consists of hostile invaders; their numbers, for the most part, are held in check. A slightly larger share is made up of transients – bugs whose populations rise and fall depending on your environmental exposure. The vast majority, however, are permanent residents called "commensals", which are beneficial bugs whose lives have co-evolved with ours since ancient times. This collective assortment is known as the human microbiome.

A single square centimetre of skin, for example, hosts 10,000 bacteria perched on the outside surface. Lightly scrape your fingernail across the same small area and you'll unearth 50,000 more.

Skin, of course, is an ecological desert compared with your body's truly prime real estate. "Most microbes prefer rich environments where there's a lot of food," says microbiologist Dr George Weinstock, associate director of the Genome Institute in the US. "And the gut is obviously where the nutrients are." Some estimates suggest that up to four kilograms of microorganisms colonise the food highway that begins at the average guy's mouth and ends at his bum. Revolting, yes, but also crucial.

"The more we learn, the more we recognise how many vital contributions our commensals provide," says Dr Lita Proctor, project director of the $175 million Human Microbiome Project, which was launched in 2007 by the National Institutes of Health in the US.

FOR EVERY ONE CELL IN OUR BODIES, AT LEAST 10 MICROBES – FROM BACTERIA TO FUNGI TO VIRUSES – PIGGYBACK ATOP AND WITHIN US

Start with the role they play in activating, training and maintaining our immune systems. For example, when skin commensals detect harmful bacteria, they trigger their human host to recruit inflammatory and immune cells to aid in the defence. Likewise, new research on mice suggests that when commensals in the gut detect flu viruses, they may use white blood cells to send warning signals to the lungs, sparking a counterattack from respiratory immune cells.

Our microbiome also helps us digest components of plant-based foods, such as dietary fibre and polysaccharides (the long-chain carbohydrates in starch) that we can't break down on our own. Researchers have even discovered that the intestines of Japanese people carry bacteria that help digest seaweed.

In the ultimate example of human-bacterial symbiosis, each cell in our bodies contains mitochondria, organelles that take energy stored in simple sugars, fatty acids and amino acids and release it in a form that powers everything we do. Our mitochondria are so essential that you might think they've always been absolutely 100 per cent human. But, in fact, ancestors of today's mitochondria were once bacteria that were living independent lives. Serendipitous infection of the ancestors of humans led eventually to the merger of invader and host. We've remained inseparable ever since.

bacteri

Another contribution comes courtesy of the extraordinary number of metabolic by-products our microbiome produces. Bacteroides in the colon produce vitamin K. One common skin resident, *Propionibacterium acnes*, breaks down sebum, an oily substance produced by our sebaceous glands, creating a natural skin moisturiser. Other commensals alter the acid levels of their preferred habitat, making these areas less hospitable to destructive microbes.

When pathogens attempt a hostile takeover, good bugs release natural antibiotics known as bacteriocins to halt their advance. *Lactobacillus salivarius* in our mouths, for example, secretes a toxin lethal to listeria monocytogenes – the bug behind deadly foodborne infections. Another bodyguard, the skin commensal *Staphylococcus epidermidis*, produces a peptide that kills other dangerous staph germs.

Finally, the sheer enormity of friendly bacteria guard us against dangerous bugs by way of a process known as "colonisation resistance". It's analogous to an apartment complex that's jam-packed with good tenants who don't ask for much and always pay their rent on time. When microbial thugs – *C. diff*, for example – come looking for a place to grow, those tenants help ensure they don't find any vacancies. At least not usually – and not without help.

ON NEW YEAR'S DAY 2011, ALEX CALLED HIS FATHER to ask for a ride to the hospital. A week before, he'd completed his fifth course of antibiotics, this time with a more powerful, broad-spectrum drug. For 10 days he'd religiously swallowed four pills a day, in the process killing virtually everything inside his digestive tract.

Once the pill supply ended, his gastroenterologist prescribed probiotic capsules, which contain several strains of live bacteria common in a healthy gut. The hope was that these bugs might jumpstart the repopulation of a more normal gut microbiome. And this, in turn, could prevent *C. diff* from running wild again.

Alex realised within a few days that this latest strategy, like all of those preceding it, was failing. By New Year's Day he was in a world of hurt. "As a skateboarder," he says, "I'd developed a pretty high threshold for pain. Over the years, I've broken fingers, an ankle, a wrist and my arm." But these injuries were nothing compared with the agony now stabbing at his core. ▶

The diarrhoea was also the worst he'd ever experienced. What his body was producing, Alex recalls, had no resemblance whatsoever to normal human waste. "It was bright red and completely liquefied," he says. "It looked like exorcism blood." By the time his father got him to the hospital, he was so anaemic the emergency department doctors debated giving him a full blood transfusion. In three months he'd lost 12kg. He was gaunt to the point of emaciation and the pain had kept him awake for days.

The docs prescribed the strong painkiller oxycodone and administered multiple units of saline. As soon as the gastroenterologist arrived, he immediately started yet another round of antibiotics, this time using vancomycin, the most powerful agent yet. For the next month, Alex rarely left home.

"I could go to work for maybe three hours," he says. "I was so sick at many points that I couldn't do much more than immediately return home and lie around. I'd get these occasional survival pangs of hunger, then I'd eat a little."

Vancomycin in pill form, alas, proved no more effective than the other antibiotics Alex had taken. His doctor next tried liquid vancomycin, which he hoped might work better. This had to be refrigerated, which bound Alex even closer to home. He began despairing that he'd ever lead a normal life again.

ria

The liquid drug failed, too. His gastroenterologist searched the medical literature, desperate to find something – anything – that might give his young patient a chance against the relentless enemy ruining him from within. The search led to Dr Alexander Khoruts, a gastroenterologist who'd reported nearly too-good-to-be-true success at treating recurrent *C. diff* infection. The intervention sounded both bizarre and, frankly, disgusting. But it had worked for dozens of patients.

What's more, a referral to Khoruts wouldn't even require Alex to leave his hometown of Minneapolis. An associate professor of medicine at the University of Minnesota, Khoruts' office was just blocks away from Alex's apartment.

THE ONLY TIME IN OUR LIVES WHEN OUR BODIES ARE thought to be completely sterile is during the nine months we spend in the womb. Throughout gestation, researchers have found, the composition of microbes in the mother's vagina undergoes dramatic changes in preparation for the newcomer's passage through the birth canal. "Infants are like microbe magnets," says Proctor, "and we know babies pick up a huge part of their microbiome during vaginal delivery." Researchers refer to this as vertical transmission because it's handed down from one generation to the next.

But birth is only the start. In the first 2-3 years of life, we continue to add and subtract new populations in many ways. We pick up some new germs through skin-to-skin contact with parents and siblings. We add others during the transition to solid foods, crawling explorations of the natural world, taste-testing virtually anything we can cram into our infant mouths, encounters with animals and insects, and increased exposure to more and more humans and their microbes.

"Your immune system needs to be educated," says Dr Julia Segre, a skin researcher at the National Human Genome Research Institute, "and the best way to do this is to be exposed to lots of different microbes that can do the teaching."

What's worrisome is that we may be flunking ourselves. Evidence continues to mount that the younger we are when our antibiotic exposure starts, the more serious and lasting are the problems caused to the "good bug" populations in our bodies.

Typically, the drugs are prescribed for ear infections, bad colds, sore throats and the like. And antibiotics can sometimes prevent serious escalation of an illness – stopping strep throat, for example, from turning into rheumatic fever. Still, experts believe, the drugs are wildly over-prescribed and may be dispensed more to placate worried parents than to help their kids.

Patients have long assumed that antibiotics may not always help, but they're unlikely to hurt, either. In other words, "better safe than sorry".

"We've been damaging our flora for the past 60 years with antibiotics," says Borody. "But unfortunately, they are a necessary evil for some infections. We can't blame GPs though; they are judicious. But it's still a problem and we have to keep using them until we get a better therapy."

Of course, one well-known consequence is the emergence of antibiotic-resistant bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA), which made its first appearance in 1961. In 2005, the Centres for Disease Control estimated that MRSA caused 278,000 hospitalisations and contributed to 17,000 deaths in the US. Its targets aren't limited to the sick and immune-compromised: news accounts worldwide have documented athletes in peak shape succumbing to MRSA through skin exposure in changing rooms and gyms and via contact during sport.

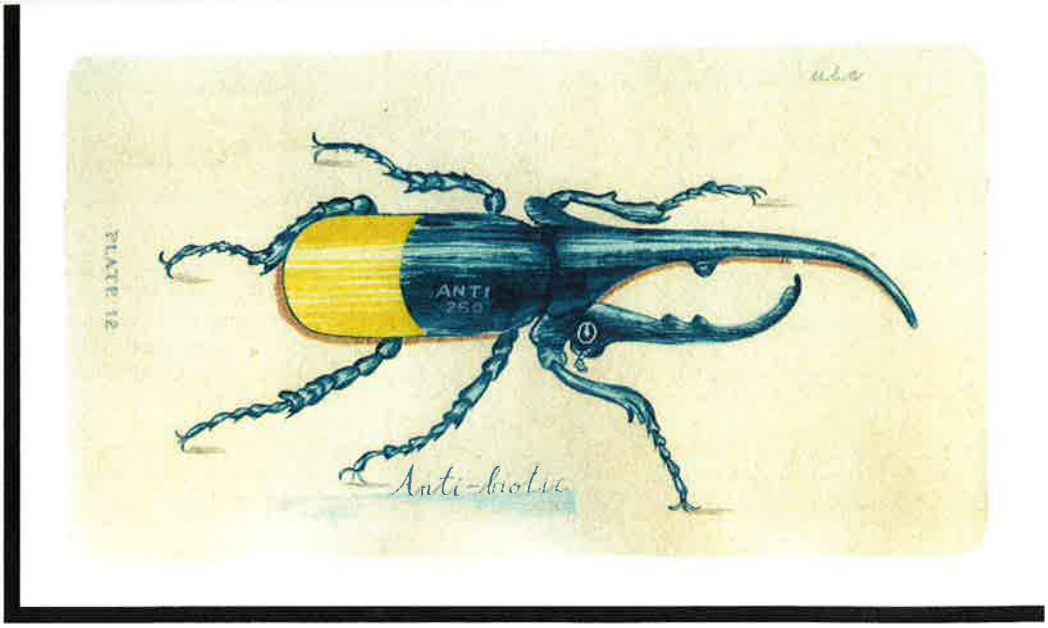
Another deadly germ, *Escherichia coli* O157:H7, has followed an eerily similar trajectory. It was first identified as a foodborne pathogen in 1982, after contaminated hamburgers triggered severe bloody diarrhoea in dozens of diners. Since then, this virulent cousin of our "normal" *E. coli* intestinal residents has led to many deaths and high-profile recalls of tainted foods: in 2011 in the US, there was a 10-state outbreak linked to lettuce and, in 2009, 30 states were hit by a contagion traced to cookie dough. In 2012, Australian beef mince was implicated in a recall in the US after it tested positive for *E. coli* O157:H7 in South Carolina. Closer to home, last year in Western Australia, a Greek salad stocked in Woolworths, IGA and independent supermarkets was recalled due to contamination, while an apricot yoghurt was also taken off the shelves.

But even if you dodge the sickness bullet, there's a good chance you will gain weight – and believe it or not, antibiotics may be one of the culprits here, too.

SINCE AT LEAST the early Fifties in the US, low-dose antibiotics have been a routine additive in livestock feed, a practice known by the acronym STAT, for sub-therapeutic antibiotic therapy. Antibiotics are used in Australia for the same reason and are administered according to guidelines developed by the Australian Veterinary Association.

"Most non-farmers assume that this is to prevent some disease in the herd," says Khoruts. "It's not. The real reason is the discovery that antibiotics make animals fatten up more quickly."

By 1954, researchers at the US Naval Medical Research Unit had heard



about STAT. They also knew of several small human studies that showed that antibiotics helped premature infants and undernourished children gain weight. Very little, however, had been published on weight effects in adults.

Strep infections can quickly spread through military ranks and US navy researchers had demonstrated that giving antibiotics prophylactically at the first sign of an outbreak could reduce the number of people who fell ill. Might these drugs also be boosting the weight of robustly healthy young men?

To find out, they randomised six 55-man companies of navy recruits into three groups. Every morning for the next seven weeks, each man was given a yellow capsule containing either an antibiotic (penicillin or chlortetracycline) or a placebo; the recruits didn't know which one they were taking.

By the end of week seven, all three groups had gained weight. But those on antibiotics had gained significantly more – on average, 2.2kg in the chlortetracycline group and 1.85kg in the penicillin group, versus only 1.2kg in the placebo group. This antibiotic-enhanced fattening may not have reached the level seen in antibiotic-fed farm animals, but then again, most men of that era hadn't received their first antibiotic doses as young as weaned calves and piglets had – a distinction that's no longer true today. "Can using antibiotics to treat our kids for ear infections be setting them up for obesity in adulthood?" asks Weinstock. "And if so, how?"

One intriguing possibility centres on the gut bacteria *Helicobacter pylori*. This bug hit the medical radar big time in 2005 after Australian doctors Barry Marshall and Robin Warren proved that it caused most stomach ulcers; the doctors won the Nobel Prize in Medicine for their work. But *H. pylori* isn't all bad – quite the opposite, actually. When it's present in healthy numbers, *H. pylori* reduces the stomach's production of ghrelin, the so-called hunger hormone. In so doing, it may not only dampen appetite signals in the brain, but also decrease fat storage in adipose tissue. So *H. pylori* could be a natural ally against gluttony.

Until the beginning of the 21st century, believe researchers, *H. pylori* was the most common bacterial species in the human stomach. But then, suddenly and without warning, it began disappearing. "By the turn of the 21st century," says Dr Martin Blaser, a professor of medicine and microbiology at New York University, "fewer than six per cent of children in the US, Sweden and Germany were carrying the organism." Many factors, he concedes, could be playing a role in *H. pylori*'s rapid demise, but antibiotics are his prime suspects. A single course of the antibiotic given for ear infections, for instance, could wipe out the entire *H. pylori* population in up to half of young patients.

Emerging research suggests that damage to the gut microbiome may be partly to blame for metabolic syndrome, a cluster of conditions including high blood sugar, high triglycerides and a large waist circumference. Left untreated, the syndrome increases the risk of heart disease, stroke and type 2 diabetes. In a fascinating study published in the journal *Diabetologia*, French researchers showed that the blood concentrations of a specific bacterial gene accurately predicted which of 3000-plus people would go on to develop diabetes 6-9 years later. The same gene concentrations also predicted which normal-weight patients would go on to develop abdominal obesity.

Another component of metabolic syndrome is inflammation in fat cells. For reasons not yet understood, inflammation appears to change the way these cells store and mobilise fat. "This is the basis for one leading hypothesis about a microbiome role in metabolic syndrome," says Weinstock. "Certain bacteria overgrowing in the gut may increase an inflammatory response in adipose tissue, ramping up fat storage and weight gain."

The troubling extinction of *H. pylori* in so many people has been impossible for scientists to miss. But what about other, less visible members of our microbial ecology? Researchers continue to discover never-before-seen genes with each successive round of sequencing. Could the tag team of modern hygiene and indiscriminate antibiotic use be eradicating critical commensals before we even learn of their existence, let alone what roles they serve?

"The most important factor in modern allergic and metabolic diseases might not be the decreased sampling of microorganisms in the food, air, water and soil," says Blaser, "but instead could reflect the loss of our ancestral microorganisms. Antibiotics kill the bacteria we do want as well as those we don't."

KHORUTS DID NOT INVENT THE FAECAL MICROBIOTA TRANSPLANT, which was first described in the medical literature in 1958. But over the past few years, he has become one of the most accomplished practitioners and enthusiastic proponents of the procedure. Also known euphemistically as "human probiotic infusion", or HPI, Khoruts concedes that, regardless of nomenclature, most people greet the concept with disgust.

The notable exceptions are those who are too sick to care.

Such was the case with Khoruts' first faecal transplant patient, a 63-year-old woman infected with *C. diff* who came to him as her last hope. "By this point," he says, "her life was ruined. She'd lost 27kg and I knew she was going to die. I gave her every antibiotic combination I could think of and not one of them helped." ▶

GUT REACTION

NAMING THE BACTERIA IN YOUR STOMACH COULD HELP DOCTORS HEAL YOU

You know your blood type, right? How about your bug type? A study in the journal *Nature* found that your individual microbiome is dominated by one of three bacterial genuses – *bacteroides, prevotella* or *ruminococcus* – swimming around in your stomach. More than a mere scientific curiosity, the finding could lead to a new era of personalised medicine. "The three gut types can explain why the uptake of medicines and nutrients varies from person to person," says study co-author Dr Jeroen Raes.

Knowing your type, in other words, might lead to diets and drug-delivery systems customised for you and your bacteria. That's the best case; however, the reality is likely to be much more complicated, says Weinstock.

Still, if such links do emerge, he adds, another study gives hope for self-determination: University of Pennsylvania researchers showed that switching from a meat-based diet to a plant-based diet, or vice versa, may alter your type. "Unlike blood type, you are not necessarily locked into your bacteria type forever," says Weinstock.

germs

AUSTRALIA SET FOR *C. DIFF* EPIDEMIC

Professor Borody, who has performed faecal microbiota transplantation (FMT) on more than 2500 patients in Australia, says we are in the beginning stages of a *C. diff* epidemic. As we travel more, and Australia continues to grow as an tourist destination, so too will the number of infected people. "The only way it can be introduced is through international travellers," he explains. "People eat infected foods, come here and get diarrhoea."

The Centre for Digestive Diseases is the only location in Australia that performs FMT, but Borody is hoping that will soon change. "We've done more FMTs than the rest of the world put together," he says. "We can cure almost 100 per cent of patients who have *C. diff* and there hasn't been one published adverse effect of FMT yet." Further evidence for the efficacy of FMT came in a study published in the *New England Journal of Medicine* in January, which found the procedure provided an almost universal cure for people with *C. diff* infection, compared with a 30 per cent cure rate with vancomycin.

To protect yourself from ingesting *C. diff*, avoid prepackaged deli meats such as turkey and pork when overseas, as up to 40 per cent have been shown to carry the epidemic strain. Borody also warns that taking too many antacids may also "significantly predispose" you to catching the bacteria. "They switch off acid to make us feel better and stop us getting heartburn," he says. "But at the same time, it's the acid which tends to sterilise our stomach."

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If anything, her condition worsened, and Khoruts suspected he knew why. One of the unique traits of *C. diff* is its ability to hunker down during hard times. It does this by forming seed-like spores that place it in near-suspended animation. Because of this, any antibiotic treatment has an inherent limitation: it effectively kills off active *C. diff*, as well as most of the "good guy" commensal species active in the gut. But *C. diff* spores aren't doing anything active, so antibiotics have no target to attack. As soon as a patient stops taking the drugs, the spores "hatch" and *C. diff* returns in overwhelming numbers.

"Our first and best barrier against *C. diff* is our natural bacteria," explains Khoruts. "As long as that microbial world is balanced and intact, it's very difficult to become infected. But when antibiotics suppress or disrupt our natural bacteria, it creates room for *C. diff* to proliferate."

Normally, if you need to restore the balance of good bacteria in your gut, you can pop probiotic capsules. This wasn't really an option for Khoruts' first HPI patient. A single probiotic capsule contains, at most, billions of live bacteria from only a handful of species; she needed trillions of individual microbes from hundreds of species.

To date, doctors have found only one way to accomplish this: after approval by the university's institutional review board, Khoruts secured an 85-gram sample of faeces from the patient's husband, placed it in a blender with saline solution and created a "special smoothie". After filtering and screening this for transmissible diseases, it was ready for transplant by colonoscope.

Based on the scattered case reports he'd read, Khoruts was guardedly optimistic that the transplant would help. What he didn't expect was how *much* it helped – and how quickly. Within a matter of days, the horrible affliction that had tormented the woman for a year was gone. Not everyone responds that quickly and sometimes it takes more than one try for the microbial "graft" to take.

Alas, this is exactly what happened with Alex. Khoruts offered two options. Alex could go back on vancomycin to again exterminate everything in his gut and then try a second transplant. Or he could opt for a stalemate: to remain on vancomycin "essentially forever". The *C. diff* would stay dormant, but his natural gut microbiome would be permanently wiped out.

"I told Khoruts that I absolutely did not want to be on antibiotics for the rest of my life," says Alex. "I said I was willing to have as many transplants as I needed to eliminate this bug."

Luckily, he needed only one more. Within two days of the second transplant, Alex sensed that something truly different was happening inside him. By the 10th day, he had his first solid elimination in nearly nine months, which he now jokingly refers to as his "proud father's stool".

"I wanted to take pictures," he recalls, "and send them to my parents, saying, 'Look what I did!'"

Alex, who feels "insanely lucky" to have received such innovative medical care, has suffered no recurrent symptoms since the graft "took". If anything, he feels even better than before, in large part because he now takes his health – and that of his microbiome – to heart.

C. DIFF HAS AN ABILITY TO HUNKER DOWN IN HARD TIMES, MAKING IT VERY HARD TO WIPE OUT

LIVING THE LIFE BACTERIAL

HOW TO STAY ON GOOD TERMS WITH YOUR GERMS

1 Skip antibacterial soaps.
The active ingredient, triclosan, has been linked to hormone disruption in animals and bacterial resistance to antibiotics. The University of Michigan found that using plain soap prevented infectious illness just as effectively as using triclosan products.

2 Nix antibiotic ointment for nicks.
Overuse of creams containing neomycin, a common antibiotic, may be leading to resistant strains of MRSA, reports *Emerging Infectious Diseases*. Clean small wounds with soap and water, then bandage to prevent contamination by potential pathogens.

3 Go with yoghurt.
Yoghurt with live bifidobacterium promotes regularity and eases irritable bowel syndrome, studies suggest; other possible benefits are not proven. If yoghurt makes you gag, pop a probiotic supplement.

4 Ask not for antibiotics.
Most common infections – from bronchitis to stomach flu – are caused by viruses, not bacteria. "Besides being a waste of money," says Khoruts, "you're killing off normal bacteria that protect you."

IF THE ONLY USE for faecal transplants was to save the lives of *C. diff* sufferers, they would still be a boon to medicine. But researchers in The Netherlands think there's even more untapped potential in the treatment, which is why they're trying to find out whether transplants can help people suffering from metabolic syndrome. Khoruts thinks it's worth the gamble.

"This is really complex biology," he says, "and we can try to sort through this for the next hundred years, hoping to make sense of it all. Or we can take a shortcut with faecal transplants and see what they can fix and what they can't. Maybe that's just primitive surgeon-like thinking."

Throughout the history of medicine, such "primitive" thinking has led to the discovery of many treatments that we knew were effective long before we knew why. Preliminary data from the Dutch scientists suggest this might prove true here, too. "An abstract of early results shows that the transplants are improving insulin sensitivity, the fundamental defect in metabolic syndrome," says Khoruts.

Proctor has even heard of pioneering medical schools offering faecal banking programs. This way, patients facing chemo, radiation and similarly harsh interventions that wreak havoc with good bugs can restore the microbiome once treatment ends.

Khoruts suspects that it's only a matter of time before specially engineered lozenges make faecal transplants as easy as swallowing a pill. Other doctors share his optimism that someday we'll see a host of such interventions. Segre, for example, imagines different "prebiotic" skin creams that nourish and serve as ideal growth media for the legions of commensals standing guard across our hides.

Until such changes in medical treatment become mainstream, there's one change we can all make right now – and it involves our attitude.

"We've long been taught to consider microbes our enemies," says Segre. "We talk about them in the language of warfare: how best can we kill all these adversaries? But the vast majority of microbes living on and in us aren't our enemies. Our goal should be not to annihilate them, but to maintain a healthy balance. It's time to start having a more kind and loving relationship with our bacteria." ●