

Why is COVID-19 so much more dangerous to men? This Canadian geneticist has a personal stake in finding out

When Kamran Shazand tested positive for the novel coronavirus, his DNA became a clue in a global mystery

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Kamran Shazand is a Canadian geneticist who lives in Tampa, where he leads a research institute at the Shriners Hospital for Children. Tristan Wheelock/The Globe and Mail

On Saturday March 21, Kamran Shazand dove into the 50-metre pool at his fitness club in Tampa, Fla., as he does every week to swim laps. Usually, the 60-year-old can breaststroke his way through half an hour, non-stop. But that day, every two laps, he was out of breath.

That night, Dr. Shazand, a Canadian geneticist who had recently moved from Toronto to Tampa to head up a new research institute at the Shriners Hospital for Children, shivered under three sweaters and a heap of blankets while his temperature shot up to 42 degrees Celsius.

From Canada his wife, Mehran, could see him struggling for breath on their video chat. She implored him to go to the hospital, worried he had come down with the novel coronavirus that had begun its spread across North America. Dr. Shazand was sure of it. He suspected it the moment he lost his breath in the pool. Still, he assured her, most people come out of it just fine, only a small percentage get really sick: “I’m not going to be in that two or three per cent,” he said.

Looking back, three months deeper into the global pandemic, there were signs that Dr. Shazand’s infection could lead to a rough road ahead: His age, an underlying condition of hypertension, his A+ blood type, and remarkably – a risk factor he shares with half the people on the planet – being male.

All over the world, reports are piling up to suggest that COVID-19 hits men harder than women. While the new coronavirus seems to infect both sexes equally, statistics show that it’s men who suffer the most severe outcomes. They are more likely than women to be hospitalized, more likely to end up in intensive care and significantly more likely to die.

The lopsided toll the new coronavirus takes on men is now considered a major clue in the international hunt under way to find which genes make people vulnerable or resistant to COVID-19. As it happens, the genetic traits involved in manhood also affect the immune system, particularly in the the clash with this virus. Studies so far have also honed in on genes linked to blood pressure, blood type and even Alzheimer’s disease, suggesting the full genetic story may be a surprising one – with a different risk profile for each person.

Canada, teaming up with projects in the United Kingdom and the United States, hopes to contribute the fully decoded genomes of 10,000 COVID-19 patients to better understand the genes behind the disease – a global mission that’s aiming for 100,000 genomes in all. It’s a \$40-million, federally funded effort that, if successful, will dwarf any previous attempt to collect and store the DNA of Canadians. In the same way that the Second World War spurred the wider use of antibiotics, and the Cold War fuelled the Space Race, the 2020 pandemic is speeding the construction of population DNA databases as a new tool for public health.

“Once we build this [database] it won’t just be relevant for this virus, or its second wave, but for the virus that comes after it,” says project leader Stephen Scherer, a senior scientist at The Hospital for Sick Children and The University of Toronto. “This is our moon shot.”

David Maslove, the clinician scientist at Queen’s University and ICU doctor at Kingston General Hospital who is co-ordinating the cross-country DNA collection, says the effort has to be global in order to garner big enough numbers to find clear links between genes and the severity of an infection: “If we

can pinpoint the genes involved, it can lead us to new treatment ideas, and it can inform the efforts to help protect people who are genetically susceptible ... and help them function more like people who are resistant.”

One of the pandemic’s great challenges is predicting how an infection will play out. Some people have no symptoms. Some suffer nothing more than a sore throat. Then, there are others, like Dr. Shazand – whose DNA will be Canada’s first contribution to the cause – who end up in an epic fight for their lives.

Up then Down

Dr. Shazand believes he knows how he got it. The week before his ill-fated swim, he supervised the move of his lab and its fragile equipment from the children’s hospital to a new space at the University of South Florida. Aware of the risks with so many people involved, he had pre-ordered masks and other personal protective equipment, but it never arrived. “I looked all over the place for masks and stuff, but the stores were all sold out,” he says.

Regret dogged him as his condition grew worse and, with his family on the other side of a closed border, fear as well: “It was scary being alone, that first week was a real nightmare.”

His head ached, his heart raced and he grew increasingly dizzy. He wished the hospital had admitted him after he tested positive, but staff sent him home to try to get better on his own. By mid-week, after dosing himself with Tylenol, aspirin and cold baths, his fever fell. Perhaps he’d beaten the virus after all, he told Mehran.

Then, a day later, his temperature spiked again, and his breath fell short. “You have to go to the hospital now!” Mehran told him. “And he said, ‘I don’t feel like I can go. I would have to go down the stairs and leave food for the cat.’ It took him an hour to get everything organized to leave.”

On Friday, March 28, Dr. Shazand somehow managed to drive himself to Tampa General Hospital. Gasping for breath, his heart thumping more than 150 beats a minute, this time he was admitted straight into the intensive care unit and put on oxygen immediately.

“After that, two doctors came to talk to me about my feelings about resuscitation, and whether I had a will,” says Dr. Shazand, “That was not a very good discussion, it was quite depressing. I tried to address their questions as much as I could, but I don’t really remember anything else.”



A researcher at the Beijing Advanced Innovation Center for Genomics runs a COVID-19 test this past May. Genetic variation in humans can play a decisive role in who gets sick from a particular illness, and who doesn't. WANG ZHAO/AFP via Getty Images

Running in the Family

People tend to think of infections as something you simply catch by chance, and that the ability to fight it off depends on whether you're run down or well rested. But increasingly, research shows that "catching it" has a whole lot to do with the genes you inherit. "It's quite counterintuitive when you first discover how strongly our susceptibility to infection is shaped by our genes, but it really is much more strongly inherited from parent to child than heart disease or cancer," says Kenneth Baillie, an ICU doctor and the University of Edinburgh scientist leading the genome project in the UK.

The landmark study on this topic comes from Denmark, where researchers explored causes of death among 1,000 people who had been adopted and compared these to the conditions that killed their biological and adopted parents. They found that "if your biological parents died young of an infection," Dr. Baillie says, "then you're six times more likely to die of an infection yourself. It makes sense when you consider how humans have evolved: We've been in a

constant arms race with bugs that cause infections since the very beginning of our evolution, and because of that those bugs exert a very strong pressure on gene mutations.”

People who have the right genetic mutations to fend off a certain pathogen live to pass their genes on to the next generation. It's estimated that 10 per cent of Europeans have high resistance to the AIDS virus because they inherited a gene variant that protected their ancestors against a viral plague in the Middle Ages.

And signs abound that resistance and susceptibility genes also play a role in COVID-19. Dr. Baillie points to reports in which the new coronavirus has caused severe infection in several members of the same family, but not among those related only by marriage – suggesting the blood relatives share an innate vulnerability.

In April, researchers from King's College London reported that genes are 50-per-cent responsible for determining which symptoms people with COVID-19 will experience. Based in part on the study of 2,600 twins in the U.K. (including identical twins who share the same DNA), the symptoms linked partly to genes include fever, delirium, shortness of breath, diarrhea and the loss of taste and smell.

Dr. Maslove, who together with Dr. Baillie had been hunting for the genes behind sepsis since 2016, says other clues come from the range of patients who end up in ICU – “a lot of them have hypertension, but not all of them, a lot of them have diabetes, but not all of them, so there's a lot of room here for a genetic explanation.”

Papa, please

The morning after Dr. Shazand was admitted to Tampa General, ICU staff called Mehran. “They told me that his respiration is now 50 [breaths per minute], and that's way high. It should be around 14 to 16. His lungs were working hard to get oxygen, but they could not.”

Mehran, along with Dr. Shazand's eldest daughter, Sophie, a Toronto school teacher, began tapping a network of doctors among their family and friends in Europe, the U.S. and Iran for input.

“In March, we didn't really know anything about treating this in North America,” Sophie said. “It came down to everyone telling me to beg everyone to intubate him as quickly as possible.” Tampa doctors agreed it would be best not to wait – but Dr. Shazand was reluctant.

Somewhere he'd read that COVID-19 patients put on ventilators have low survival odds. He also didn't like the idea that he would first have to be knocked out and put into a coma with fentanyl and propofol – powerful drugs that made him think of Michael Jackson and Prince and overdose deaths. But by week's end, he could barely put two words together taking a deep breath. Sophie told him, "Papa, please ask to be intubated."

On March 29, Dr. Shazand agreed to be put to sleep. Doctors threaded a tube down his throat and into his windpipe through which a ventilator pumped the oxygen he so desperately needed into his lungs.

With electronic access to his medical files, his family constantly tracked his condition in real time, seeing what the doctors were seeing – and fretting. Lung x-rays revealed widespread inflammation. His kidneys were failing, and he had to be put on dialysis. Acid levels in his blood climbed dangerously high.

Sophie, Mehran, Dr. Shazand's younger daughter Carole and sister, Soussan, a biochemist in Montreal, convened daily for several video conferences to discuss treatment options. Early on they asked doctors to give him remdesivir, the antiviral drug that was then showing promise in clinical trials. But the kidney problems ruled him out as a candidate and at that point, the hospital was not part of the trial, says Mehran: "The doctor told me: 'This isn't China, we can't just give him an experimental drug without FDA approval.'"

Doctors treated him instead with two antibiotics, a corticosteroid and the anti-inflammatory hydroxychloroquine. That week, U.S. President Donald Trump was touting the drug as a "game changer," although three large studies since have concluded the medication, traditionally used to treat malaria and auto-immune disorders, offers no benefit to COVID-19 patients.

This was apparent for Dr. Shazand by his fourth day on the ventilator. "They called and told us he is very, very sick," Mehran recalls, "and I knew it, because I had access to all his lab work and it looked terrible."

One of the nurses suggested the family record audio messages she could play for Dr. Shazand during a scheduled "sedation vacation," when his anaesthesia levels would be briefly lowered, in part to ensure he could regain a certain level of consciousness.

"There were four of us [on the recording] and we all talked," Mehran says. "You're amazing," they told him, "You're great fighter ... you can do this ... we have so many plans, so you have to come back."

The nurse texted them afterward to say that she'd played the message three times and he'd moved both his arms and raised his eyebrows as a few tears fell. Indeed, he would remember their voices later – "I remember I was crying and

trying to talk but my tongue wouldn't move. I was looking at the ceiling, I was seeing this weird light and people walking around me and I was imagining myself in a plane ... and a lady asked me, 'Do you know where you are?' But I couldn't answer her, and I just went back into the darkness."

COVID-19 case data by sex, select countries

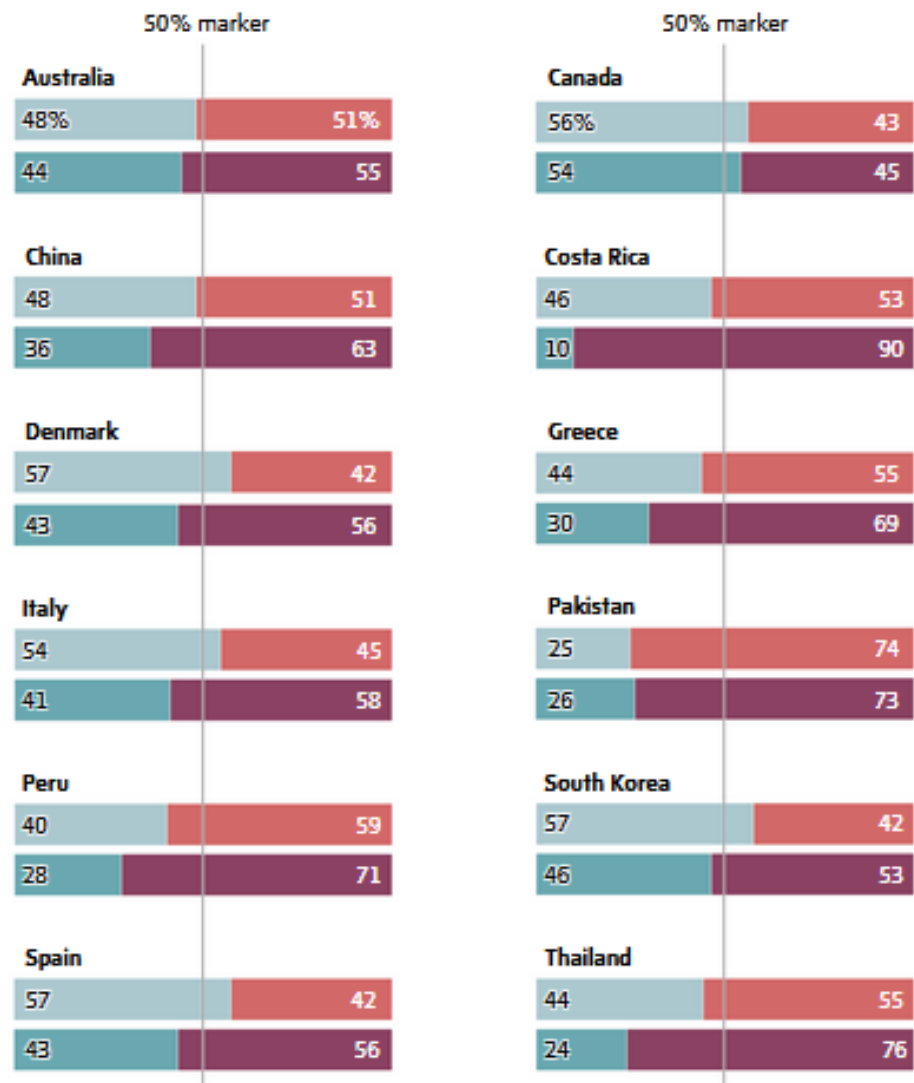
As of June 10, 2020

FEMALE

- Confirmed
- Deaths

MALE

- Confirmed
- Deaths



The X File

Reports that the new coronavirus kills men more than women emerged from the earliest days of the outbreak in Wuhan, China. But the mysterious trend has since been documented in Italy, the U.K, the U.S., Denmark, South Korea, Spain, Thailand, Greece and more.

In Canada, where women make up more than half the fatalities from COVID-19, the country seems like an outlier. But most cases here have stemmed from long-term care homes in Ontario and Quebec, where elderly women make up 70 per cent of residents; 80 per cent of the staff who care for them are also women. But a May report from Toronto's University Health Network offers a different picture. Its study of more than 194,000 Ontario residents who tested for the virus between January and April found that while men were less likely than women to be tested, men had higher rates of infection, hospitalization, ICU admissions and deaths (7.3 per cent vs 5.6 per cent).

The question no one can yet answer is why.

There's speculation that men suffer from more heart disease and other conditions, or are more vulnerable due to social factors and risky behaviours – being more likely to smoke and drink, and perhaps less likely to follow rules around physical distancing or hygiene. After all, with a long history of hunting, warring and riding motorbikes without helmets, men have always recorded lower life expectancies than women.

Yet as the trend holds up across age groups, countries and cultures, another hypothesis is also gaining ground: That in humanity's eternal war with nasty pathogens, biology has handed males the shorter stick. For starters, estrogen, the prime hormone of females, boosts immunity. Testosterone, the same male hormone tagged for fuelling risky behaviour, suppresses the immune system. And in the battle against COVID-19 in particular, men may be the genetic underdogs. The gene known as ACE2, which controls how easily this coronavirus can invade a human cell, happens to be housed on the X chromosome. Women have two copies of the X but men have only one.

As one of the two chromosomes that determines biological sex in people, women receive an X from each parent. Men inherit the male-sex determining Y chromosome from their fathers and a single X chromosome from their mothers. This leaves men with no back up or alternate copy of any gene on the X, including the one that encodes ACE2.

Short for angiotensin-converting enzyme 2, ACE2 is a receptor that sits on the surface of cells where its normal job is to receive chemical messages. These

chemicals bind to the receptor and prompt the cell to release molecules which, among other things, lower blood pressure and inflammation. But the spiked coat of the COVID-19 virus can also stick to it like Velcro, and once it latches on, it opens ACE2 like a key in a lock and hijacks the cell's machinery to copy itself and go forth to infect other cells.

The theory is that if a man has the type of ACE2 gene that offers an unlocked door on his cells, the COVID-19 virus can barge right in – and unlike women, there is no other version of the gene to counter the effect.

“Just by virtue of the way male biology works with the X chromosome, men are only going to have one kind of ACE2 and that might be, hypothetically speaking, less advantageous than a woman because she could have two different kinds of doors [including] some that are locked up,” says William Gibson, a clinical geneticist at the University of British Columbia.

Dr. Gibson, lead author on a recent paper exploring links between ACE2 gene types and male vulnerability to COVID-19, notes that the SARS virus also breaks into human cells through the ACE2 portal – and it also proved more lethal to men. During the 2003 SARS epidemic, which infected 8000 people and killed nearly 800 worldwide, men had a death rate of 22 per cent while the rate among women was 13 per cent.

In genomic terms, the X is also no pipsqueak. It totes about 1,000 genes and harbours more related to the immune system than any other chromosome. This includes genes that help to sound an early alarm when a microbe invades, and others that tailor defensive attacks with specific antibodies.

In 2019, a study in Human Genomics linked the lone X of men to higher rates of bacterial, fungal, parasitic and viral infections. And this spring, a U.S. study titled The Lethal Sex Gap – COVID-19, found that although immune responses decline in both sexes over time, the changes – resulting in lower antibody production and more inflammation – “were significantly greater in magnitude in men” and may explain why the new coronavirus has been particularly deadly among older males.

THE X FACTOR

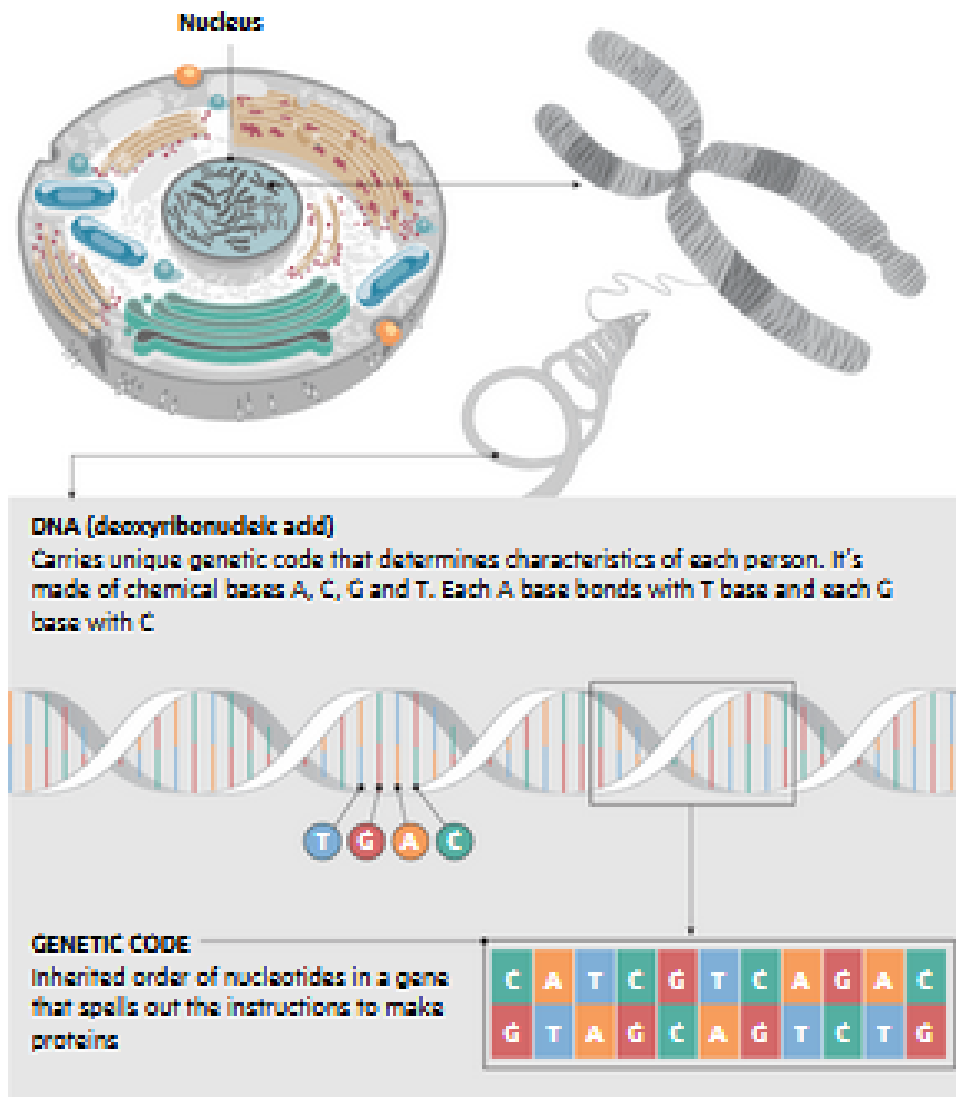
Passed from parents to offspring, chromosomes contain genes that define the characteristics of each living creature. The X chromosome has more immune-related genes than any other chromosome. Research finds this makes men's immune systems less robust than women's.

HUMAN CELL

Nucleus of every cell contains 46 chromosomes. Each parent contributes 23 chromosomes, one of them being a sex chromosome.

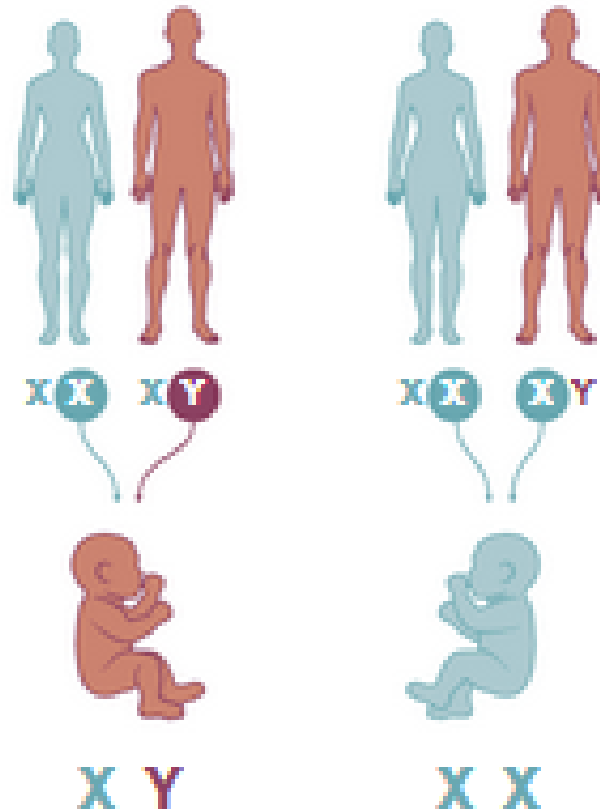
CHROMOSOMES

They are comprised of one tightly coiled molecule of DNA which contains genes. Genes encode the recipe for proteins that control the function of a cell.



SEX CHROMOSOMES

Referred to as the X and Y chromosomes, they determine the sex of the offspring. The mother's egg carries an X, while the father's sperm carries either an X or Y.



Generally, XY corresponds to male and XX to female, though the child's gender identity may turn out differently as they develop.

WHY MEN ARE SUSCEPTIBLE?

Studies show that having only one X chromosome leaves males more vulnerable to pathogens – viruses, bacteria, fungi, parasites – and more susceptible to infection.

MURAT YÜKSELİR / THE GLOBE AND MAIL, SOURCE:
NATIONAL HUMAN GENOME RESEARCH INSTITUTE; GRAPHIC NEWS

Ten Beats

Not long after his 'sedation vacation,' Dr. Shazand's immune system spiralled into a catastrophic overreaction. Described as a 'cytokine storm,' it's now known as a hallmark syndrome of COVID-19 and one that takes the life of many patients. Fighting to beat the pathogen, immune cells can overproduce a relentless army of cytokine proteins that rampage through the bloodstream, attacking healthy tissues as well as the virus, causing widespread inflammation, blood clots and organ failure.

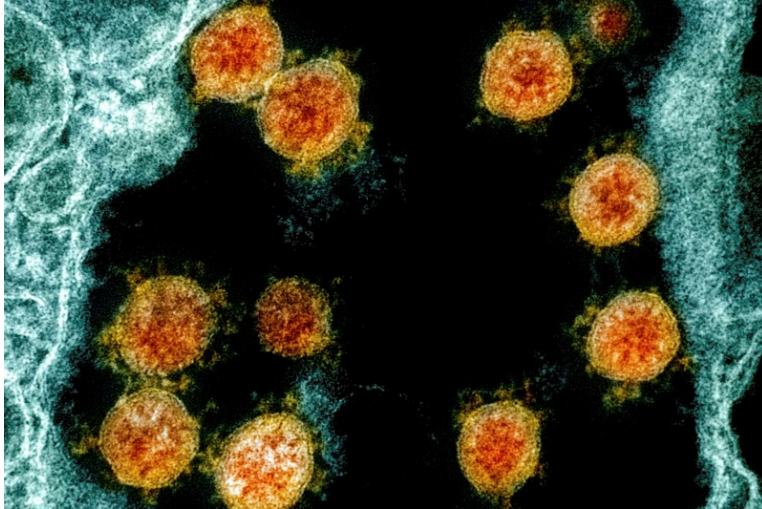
Dr. Shazand was in the grip of that storm when ICU staff told Sophie her father might die that night. His lungs and liver had quit. His kidneys had given up. His heartbeat had dropped to 10 beats a minute. "Basically, only his brain was working," Sophie recalls. "The staff were sounding so defeated already. Some of them were talking about what to do with his body after death."

But in a last-ditch effort to save him, his doctors recommended the compassionate release of a \$1,500- per-dose immune-suppressing drug called Tocilizumab, a medication usually used to treat rheumatoid arthritis. "We said, 'Yes, please go ahead and give it.' "

Within 24 hours, Dr. Shazand's inflammatory markers came down, and his fever as well. The family cheered – but the celebration was short-lived. The next day, on April 2, the numbers jumped back up – "and they called us to say they don't have another dose for him."

Over the long-distance negotiations, a hospital official explained to Sophie "that they had only one dose and with the new protocol she had received, my dad was not going to be the recipient of that one dose."

Sophie argued that her father was so very deserving of it, that he'd worked for years at the Ontario Institute for Cancer Research, that he wouldn't be in Florida at all were it not for his desire to work on rare childhood diseases at a non-profit hospital. But the official told her they had to follow hospital protocol: "All I knew," says Sophie, "is that my Dad was not going to receive it."



SARS-CoV-2, the virus that causes COVID-19, is shown in orange under an electron microscope. Coronaviruses are so named because their circular shape and protruding spikes resemble crowns, or 'corona' in Latin. National Institutes of Health via AP

From Head to Toe

At first, COVID-19 looked like its cousin, SARS – a severe respiratory disease. But the new coronavirus has craftier ways of spreading itself and unleashing an eclectic array of symptoms – stealing taste and smell, turning toes blue, triggering strokes, blood clots, diarrhea and cytokine storms.

None of this surprises Josef Penninger. While studying fly hearts in 1989, the molecular immunologist was among the first to discover the ACE2 protein that the COVID-19 virus uses to invade a human cell. Now the director of the Life Sciences Institute at UBC, Dr. Penninger says COVID-19 is bound to have many faces. “[It] affects so many tissues because ACE2 is all over the place, in the brain, in the lung, in the kidney, in the gut, in the blood vessels, in the heart. Late-stage COVID-19 is not a lung disease, it’s multiorgan failure disease,” he says.

The virus doesn’t just use ACE2 as a doorway, it kicks the door in, robbing the body of the crucial chemicals ACE2 normally makes to keep a body healthy says Dr. Penninger. “That’s why I believe SARS and COVID-19 became so dangerous – they hit a system which keeps multiple organs in balance, so lung injury becomes very severe, the blood vessels leak, there’s massive inflammation, and the plasma spills out into the lung.”

Of course, not everyone suffers the same way with a COVID-19 infection, and the ACE2 gene may play a lead role in that. Dr. Penninger says the gene not only varies between people, but within the same person, in response to diabetes, say, or age. Children, for instance, have few ACE2 receptors in their noses according to a recent study in the *Journal of the American Medical Association*,

which may be why children tend to have low COVID-19 infection rates – the virus has fewer doorways to break through.

Researchers are also exploring whether ACE2 gene mutations might also help to explain why COVID-19 cases and deaths are disproportionately higher among minority groups. The suspicion is that certain ACE2 variants, along with socio-economic inequalities, make some ethnic groups genetically susceptible to the disease. University of Iowa researchers, for instance, have found African Americans and European Americans tend to have different levels of ACE2 expression, which may impact their differing rates of disease. The thinking is that the higher the level of ACE2, the more severe the infection.

Dr. Penninger and his team now have a drug that mimics ACE2 in Phase 2 clinical trials in Europe. In development since SARS, it's designed to reduce inflammation and blood pressure like the real ACE2, but also act as a decoy that destroys the new coronavirus as it binds to the fake instead of a human cell. Results are still months away, but Dr. Penninger says that given on compassionate grounds to an Austrian woman dying of COVID-19, the drug helped to save her life: "She lives and is fully recovered."

The Art of the Deal

While Dr. Shazand hovered near death, his network of loved ones kicked into high gear to find him a second dose of the drug they hoped would save his life. "It was the most frightening part of the care," says Mehran, "not knowing what to do, or how to get this drug to him, trying to think of whatever we could do to get it."

Sophie explored a raft of options – from obtaining the drug from her sister-in-law at a city hospital to buying it from Costco and driving it to Florida – but all of them involved time she feared her father didn't have. In the end, she called the hospital back and pleaded once more for them to immediately give her father the dose they had and that she would replace it within three days.

"By that point, the lady said, 'I remember your Dad's wife saying you guys had some pretty good contacts with pharmaceutical companies,' and I said, 'Yes we do – what do you need?' And she said the hospital had no remdesivir, that only hospitals approved for testing [the experimental antiviral] can get their hands on it."

Sophie quickly reached out to her Aunt Soussan in Montreal to work her pharmaceutical contacts to help the hospital become one of the remdesivir testing sites. But even before the deal was sealed, the hospital official let her know that her father would be given the second dose after all.

“It completely brought all his inflammatory markers down,” Sophie says. “At the end of the day, I think of all the people who cannot articulate what they want, cannot advocate, all those muted voices ... if you don’t push a little bit, what then?”

Within 48 hours of taking the drug, Dr. Shazand was brought out of his coma, able to breath on his own for the first time in eight days.



Stephen Scherer is shown in 2018 at the Toronto lab of The Centre for Applied Genomics, which he leads. Dr. Scherer is also head of Canada’s Personal Genome Project, and Dr. Shazand sought his help answering the genetic mysteries surrounding his illness. Fred Lum/The Globe and Mail

The Numbers Game

Last year, before COVID-19 broke out, Dr. Shazand contributed his DNA to Canada’s Personal Genome Project. The open database of whole genomes and medical information was created in 2012 to speed disease-gene discovery and the development of treatments tailored to a person’s genes.

The online PGP resource is now the template for the database researchers plan to build with the 10,000 genomes, medical histories and lifestyle details of Canadian COVID-19 patients. Dr. Scherer, who also heads the PGP, predicts that Canadians will be willing to take part, that the desire to learn about their own susceptibility to a killer virus, and protect those most at risk, will trump privacy concerns people traditionally have around sharing their DNA.

“If we get big enough numbers, we should be able to pinpoint any gene that’s involved in COVID,” Dr. Scherer says. “We’ll be able to look for people who are the outliers, those who are resistant to the disease and those who were hit in the most adverse way.”

A month after leaving the hospital, Dr. Shazand called Dr. Scherer, who also directs The Centre for Applied Genomics and heads up the PGP. He told him how close he’d come to death while battling the novel coronavirus and that he’d be happy to have his genome put under the proverbial microscope.

A preliminary search has so far turned up no rare quirks among some of the genes suspected to play a role in COVID-19. Dr. Shazand’s ACE2, for example, carries no unusual mutation. Though it’s not yet clear which forms of the gene might help or hinder the virus’s ability to infect someone.

Meanwhile, the list of human genes implicated in COVID-19 continues to grow. Researchers in the U.K. recently reported that people who carry two copies of a gene that dramatically ups the risk of Alzheimer’s Disease also face a higher chance of catching COVID-19 – and twice the risk of suffering severely from it.

Dr. Shazand does not carry that gene. But he does have blood type A+, which a June report in the *New England Journal of Medicine* found is associated with a 45-per-cent higher risk of acquiring COVID-19 compared to people with other blood types. The study was based in part on finding a cluster of genes in common after sequencing the whole genomes of more than 1,900 patients who suffered severe infections in Spain and Italy.

Studying whole genomes, says Dr. Maslove, is an approach that says, “we don’t really know what genes are involved, let’s look at all of them – and maybe we find something that we didn’t know to look for.” It could also lead doctors to treatments for COVID-19 they wouldn’t otherwise consider trying, he says, including medications already on the market.

Healing

Nearly three months after he was unhooked from a ventilator, Dr. Shazand’s comeback is a long and lonely road. He’s still in Tampa, thousands of miles away

from his family. It still hurts him to breath deeply, his liver is damaged and his resting heart rate is so high, he worries that walking around the block will trigger a heart attack.

“My recovery is and will be very slow,” he says. Dr. Shazand lost 35 pounds during his two weeks in the hospital. He woke to discover he didn’t even have the strength to hold his iPhone – “I didn’t realize how heavy it was.”

The disease humbled him, he says, taking him down so completely when he thought that as a fit man – a regular swimmer and tennis player who never smoked and rarely drank – he would be more resistant. “Sure, I have hypertension, but who doesn’t at 60 years of age?” he says. “But I’m guessing it’s not really about your body health or age, it really is your genetics that drives a good part of the infection.”

“That’s my message to everyone – Don’t think you’re invincible ... take this seriously.”

One of the few COVID-19 patients with the expertise to look at his own genome, Dr. Shazand has spotted suspicious mutations in the immune system genes of his X chromosome. It’s not possible to understand at this point what they mean, he says, but he’s delighted to be one the first Canadians to contribute his DNA to the international gene hunt.

In the end, Dr. Shazand says he takes some pride in knowing he beat the virus. But he realizes the victory has much to do with the relentless fight his loved ones waged from afar, which only adds to the pain that he’s been unable to see them yet: A dozen weeks after he was first infected, Dr. Shazand continued to test positive for the virus .

“It’s dead virus,” he explains. Genetic remnants can be picked up with a swab of his nasal cavity but, it’s believed, they are incapable of causing infection. Blood tests show he now has antibodies against the COVID-19 virus. Even so, he doesn’t want to take the chance of exposing others to the disease. He is waiting until July to fly back to his family: “You know, I think they suffered more than myself actually, because they were awake.”