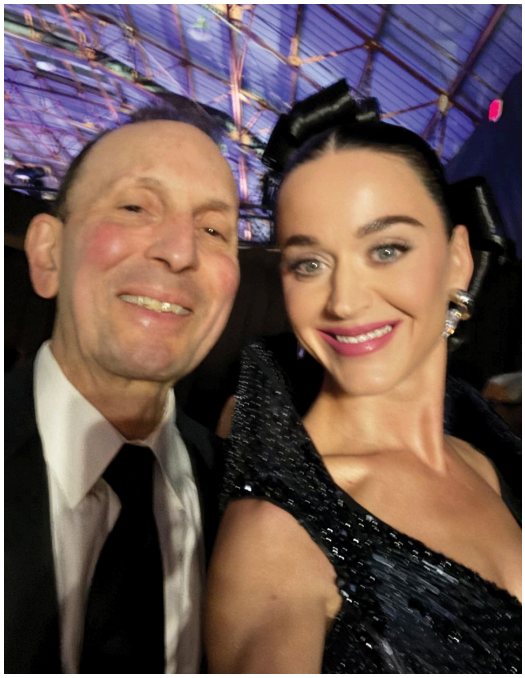
Dr. Ozempic

I was just a baby scientist when I discovered the hormone that made Ozempic possible. I had no idea how life-changing—and world-changing—that breakthrough would be

BY DANIEL DRUCKER, AS TOLD TO COURTNEY SHEA | JULY 2, 2025

Listen to this article

It's not often that I find myself in a room with some of the world's biggest celebrities. But there I was, this past April, dressed in a tux and receiving the Breakthrough Prize in Life Sciences for my role in the discovery of GLP-1, a metabolic hormone that is also the active ingredient in Ozempic. It's wild to think that something I started working on in a lab in Boston in the 1980s—my first real job after medical school—led me to the so-called Science Oscars. Unlike most other science galas, where you're lucky to get a decent meal, this ceremony is very swanky. It's held in Los Angeles and attended by bold-face names from the tech world (Mark Zuckerberg, Jeff Bezos, Sergey Brin) and from Hollywood (Gwyneth Paltrow, Jodie Foster, Lizzo). Per strict instructions from my daughter-in-law, I got selfies with Paris Hilton and Katy Perry.



I received the award alongside fellow GLP-1 experts Joel Habener, Jens Juul Holst, Lotte Bjerre Knudsen and Svetlana Mojsov. It was presented to us by Will.i.am and Lauren Sánchez, who made a joke about how a lot of people in the room owed us a debt of gratitude. That got a big laugh.

Over the course of the evening, attendees kept coming up to me to share the success they'd had with GLP-1 medications. Ozempic has become a catch-all name, like Kleenex or Band-Aid, but there are other popular brands too, like Wegovy and Mounjaro. Of course, the impact these medications have had and the potential they hold for the future extend far beyond the elites gathered in that ballroom. Still, it felt good to be acknowledged at such a high-profile event. In my acceptance speech, I thanked everyone for showing their support at a time when science is under attack. Given the rise of Robert Kennedy Jr. and the MAHA movement, the war on higher education, and persistent anti-vax sentiments, science needs all the star power it can get.

A lot of kids realize they want to be a doctor the first time they put a Band-Aid on their teddy bear. I know this because I used to interview candidates for the University of Toronto's medical program, and most applicants told some version of the same story. That was never me. I grew up loving regular kid things: riding my bike, games, music. I wasn't the type who thought about career goals. It was only in high school that I gravitated toward the sciences and mathematics—I liked the

certainty of numbers and data. With English, you might get a bad mark because the teacher didn't agree with your interpretation of Shakespeare, and I'd always wonder, *How do you know what a playwright was thinking 400 years ago?*

Related: *Meet the artists, professors, scientists and other luminaries ditching the US and moving north*

I did my undergrad in science at the University of Ottawa and was accepted to U of T's medical school. Being a doctor seemed cool—they were the tech bros of that time—but I had no real idea what it entailed. There were no doctors or scientists in my family or in my parents' circle of friends. My first semester on campus, I was flipping through a massive course catalogue and landed on endocrinology. Learning about the thyroid gland and steroid hormones sounded interesting, so I chose the class as an elective. I went on to have two influential mentors: Dr. Gerard Burrow, a professor recruited from Yale, and Dr. Charles Hollenberg, chair of the department of medicine at U of T. Both were endocrinologists and passionate about their work. They encouraged me to consider research alongside my plans to be a practising physician.

As a scientist, I'm not much of a believer in fate, but it does seem like the universe had a plan for me

After graduating, I interned in internal medicine at Johns Hopkins, in Baltimore. My med school sweetheart, Cheryl, remained in Toronto to pursue her training in dermatology, so I knew I'd be coming back—if I didn't, I was pretty sure she would find another option. We got married in 1981 and had our first son in 1983. The plan was to move to Boston for a couple of years so we could do our research fellowships at Harvard and Massachusetts General Hospital, then come home, where I would take over Dr. Burrow's thyroid clinic at Toronto General Hospital.

Drucker and his wife, Cheryl Rosen, moved to Boston from Toronto as young doctors to pursue their research. Photo by Hudson Taylor

As a scientist, I’m not much of a believer in fate, but it does seem

like the universe had other plans. I showed up to my first day of work in Boston and was told that thyroid research in the Habener lab at Harvard was winding down—my new focus was going to be the glucagon gene. I was devastated. The whole reason I'd chosen Boston was because I was going to be the thyroid guy. The only thing I knew about glucagon was that it was a hormone and it played an important role in regulating blood sugar. But junior scientists weren't inclined to challenge their superiors, so I accepted the sudden change of course, unaware of the impact it would have on both my life and medical history.

Related: *How chemist and computer scientist Alán Aspuru-Guzik brought self-driving laboratories from Harvard to Toronto*

At the time, nobody was thinking in terms of scientific posterity. Lab work is notoriously tedious—for every great day, there are months when it feels like nothing is happening. When I arrived in Boston, the Habener lab had already identified the genetic sequence for glucagon, which counteracts insulin by raising blood-sugar levels. My job was to figure out the potential of a newly identified hormone related to glucagon called "glucagon-like peptide-1," or GLP-1. I conducted a series of experiments where I incubated the GLP-1 peptide with different types of cells to see if they would respond. I tried fibroblasts—nothing happened. Next I tried pituitary cells, which control many endocrine systems—zip. It was only when I incubated GLP-1 with islet cells from the pancreas that they responded by producing more insulin. Progress!

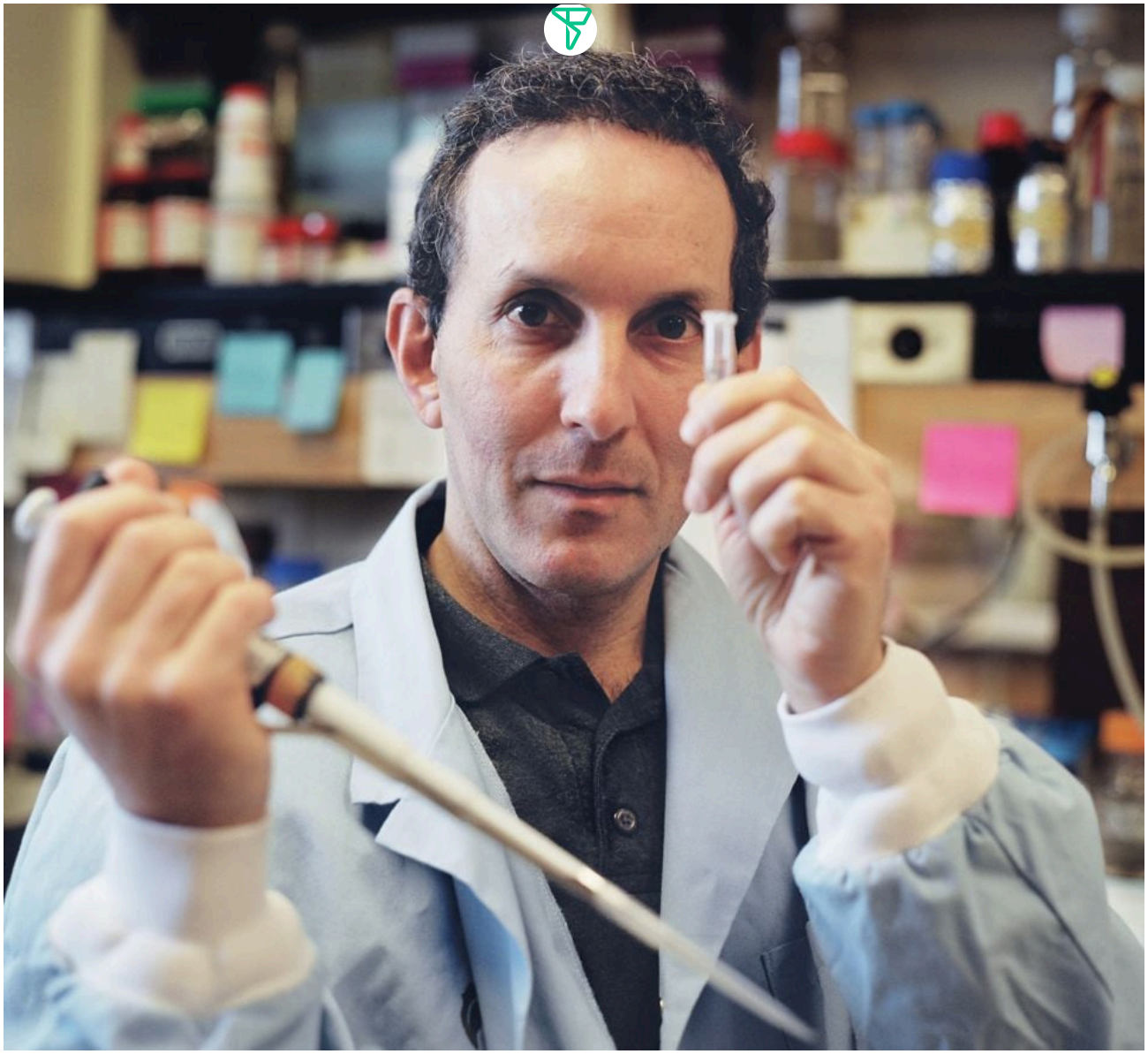
The possible link to diabetes wa[illegible]nmediately clear—people with Type 2 diabetes have insufficient insulin, or their insulin doesn't work properly, which is what causes high blood sugar, but we were still in the early stages of discovery. In the Canadian made-for-TV movie on Frederick Banting and Charles Best, they're running their experiments, and then there's this big moment when the blood sugar is lowered in dogs. *Eureka! We've discovered insulin!* Actual discovery science entails asking a series of questions, and most of the answers don't end up being all that interesting. When you do get a fascinating answer, the reward is that you get to ask another question. If someone ever makes a movie about the Ozempic story, they'll need to figure out a way to ratchet up the drama.

There was one morning that was plenty dramatic, though. Several weeks after the islet discovery, I came into the lab to find that my notebooks had disappeared. I rushed to my boss's office in a panic. *Someone stole my work!* I asked if we needed to call security, but my boss calmly explained that nothing was missing: he had taken my notebooks to the hospital's patent lawyers. I was relieved and more than a little flattered, but I knew that the odds of a patented discovery turning into a drug were about one in 10,000. Besides, I was just starting out. As a young father and husband, I was mostly excited to publish some scientific papers, compete for a real faculty job, and ultimately be able to afford to add mushrooms and peppers when we ordered pizza.

After I'd spent three years in Boston, U of T offered me an assistant professor position that included my own lab at the

Banting and Best Diabetes Cent so my family and I moved back to Toronto. I started my clinical endocrinology practice at Toronto General and worked my butt off writing grants and getting funding.

Over the ensuing years, it became clear that insulin secretion was just one of many things GLP-1 did, so my colleagues and I worked to firm up those discoveries and move the research toward an actual drug. It's a lot of hoops. When Banting and Best figured out that their purified extract lowered blood glucose in dogs, they walked across the street to Toronto General and said, "Why don't you try this in patients and see if it works?" Today, you have to conduct years of safety testing, first on animals and then, if you're lucky, in human trials.



When Drucker began his research more than 40 years ago, he knew next to nothing about the hormone that would dominate his work

Pharmaceutical companies were paying attention to the work on GLP-1, although a few initial investors backed out because of side effects observed in the early trials, including nausea, vomiting and diarrhea. My colleagues spent a lot of time adjusting doses and figuring out the ideal titration process, which is the rate at which you gradually introduce some drugs. The biggest challenge was that GLP-1 deteriorated quickly in humans, making it difficult to maintain effective drug levels. We needed something that would

last longer. The solution came from an unlikely source: the Gila monster. This venomous desert-dwelling lizard metabolizes at an extremely slow rate—an adult requires just three or four meals a year—and it secretes a compound called exendin-4 in its venom that is nearly identical to GLP-1 but longer-lasting. With the help of Bob Murphy, the senior herpetology curator at the Royal Ontario Museum, we obtained a Gila from a zoo in Utah and flew it to Toronto.

Big Pharma companies were skittish about giving humans a hormone mimetic from a venomous lizard—understandable, I suppose. A small biotech firm called Amylin Pharmaceuticals stepped up, and I periodically advised them during the drug development process. By 2004, they were ready to submit a Type 2 diabetes treatment called Byetta (synthetic exendin-4) to the FDA for approval. The following April, I was at the Amylin offices in San Diego as we waited for the agency's notification of approval of the world's first GLP-1 medicine. After what felt like forever, the fax came through, and there it was: APPROVED. Let's just say it was a fun evening at the bar. All the pharma giants—Eli Lilly, Novo Nordisk, Sanofi, Bristol Myers Squibb—were working to develop long-acting GLP-1-mimicking drugs, but it was a little start-up that got the very first approval. That was a huge deal, and Amylin ended up being bought by Bristol Myers for $5.3 billion a few years later. For me, the approval was the validation of 20 years of relentless effort. This thing was actually going to have a life outside the lab.

As patients began taking GLP-1 medications for diabetes, we started seeing significant weight loss. This wasn't a surprise: years earlier, my lab and other groups had done studies that showed GLP-1 suppressed appetite in mice and rats, so to see it in humans was more like a proof of concept. We knew there would be even more weight loss if we upped the dosage, but that came with complications—stronger doses meant stronger side effects, so that was something to work through. A few different GLP-1 medicines came on the market, but when Ozempic was released in 2017, we really started to see the magic. In some trials, up to 40 per cent of people taking the drug—2.4 milligrams once a week, delivered via injection—were losing 20 per cent of their body weight. The drug had been approved for Type 2 diabetes, but as word started getting out, it was being prescribed off-label for weight loss.

One of the best things about the results we were seeing was that they validated obesity as a biological phenomenon, not simply a behavioural one. For years, I saw people in my clinical practice who had tried everything to lose weight—diet, exercise, behavioural therapy—and none of it was consistently successful. To be able to prove to the world that obesity was a treatable medical problem and not some sort of moral failing was a relief, plus the medical community could finally recommend something that could really help their patients.

The first time I came across GLP-1 medication in the wild, so to speak, was at my golf club. It was early in the pandemic, and a bunch of us were social-distancing outside and grabbing a bite to

eat. One of my fellow golfers, who had always been overweight, turned to the group and asked if anyone wanted to share a pizza. Everyone looked at him like, *We've been having lunch with you once a week for decades—you've never asked us to share - anything*. He explained that he was taking "that drug Drucker's been working on" and wasn't very hungry. Around the same time, one of my family members went on GLP-1 medication, and the result was transformative. There was weight loss, but it was so much more than that. She told me how she used to dread going to the grocery store because she'd spend two hours in the aisles obsessing over ingredient labels and calories. On the drug, she breezed through this once excruciating process in 20 minutes—no incessant, obsessive internal dialogue; no concern that she would overeat.

GLP-1 medications slow the rate at which the stomach empties itself—people stay fuller longer, so they eat less often. But the drugs also work on receptors in the brain to eliminate or significantly quiet obsessive ruminating, also known as food noise. For a lot of people, their relationship with food is largely practical. They might enjoy certain meals more than others or have a favourite ice cream flavour, but in most cases they'll eat when they're hungry and stop when they're full. On the other end of the spectrum, you have those who are, by and large, ruled by food. If they're struggling with their weight, they're fighting an often insurmountable battle, both physically and psychologically. It's true that GLP-1 medications can rob some people of the pleasure of food and, in rare cases, lead to depression. But I've heard from

many others about the relief they feel at being able to reclaim their mental energy.

When Oprah Winfrey went public about using GLP-1 medication in 2023, she made a comment along the lines of, “Oh, this is why thin people are thin,” meaning they’re not actually more disciplined or virtuous, they’re just not experiencing the same temptations and triggers. Her admission was a big deal. Until then, nobody had wanted to say that they were taking the drugs, in part because of the public outcry. When the GLP-1 drug semaglutide—marketed for weight loss as Wegovy—had gone mainstream a couple of years earlier, there was a huge spike in demand that coincided with pandemic supply chain problems. Suddenly, people with Type 2 diabetes who had been using the medication for years could no longer access it.

I’m not suggesting there isn’t a hierarchy of need, but it was unfortunate that people using the drug for weight loss were demonized. Around this time, there were also a lot of horror stories being broadcast. You could get booked on CNN if you had suffered negative side effects, such as chronic diarrhea and bleeding. These incidents are unfortunate and serious but quite rare. They overshadowed the incredible results we were seeing and continue to see: last year, the largest safety study to date on medical therapy for obesity showed a 20 per cent reduction in heart attacks, strokes and death among people with obesity and heart disease who are taking semaglutide.

I've never heard a criticism of GLP-1 medications that I wasn't already anxious about. I'm the guy who asks himself, *What could go wrong?*

Of course, there are also people who aren't obese, who are 160 pounds but would feel more comfortable at 140, using the drug. I don't have a problem with that as long as it's done in consultation with a doctor. And I think that should be something you pay for, the same way you would pay for a holiday or to redo your walk-in closet. If we're talking about someone who is 110 pounds but wants to be 100, that's more worrisome. *Does this person have an eating disorder? Is the medicine facilitating body image distortion?* These are the thoughts that keep me up at night. I've never heard a criticism of GLP-1 medications that I wasn't already anxious about. I worry about patients like the ones I used to see at SickKids—teens with Type 1 diabetes who didn't want to take their insulin regularly because it increased their appetite and made them gain weight—and I wonder what access to GLP-1 medicines could mean for them. I worry about misuse. I'm the guy who looks at drug safety, and many other situations in life, and asks himself, *What could go wrong?*

At the same time, to paraphrase Wayne Gretzky, I am always trying to look ahead to where the puck is going. At the moment, we are seeing progress in research exploring whether GLP-1 medications could be used to treat Alzheimer's. The data is mixed but promising. Similar work is being done studying GLP-1

medicines for Parkinson's disea. CAMH is one of many mental health facilities investigating the potential of GLP-1 as a treatment for addiction, which makes sense given the results we have seen with food noise. Could there be a similar effect on alcohol, smoking and drug addictions? Gambling? We don't know yet, but we are asking the questions, and we're excited to learn the answers.

Working in diabetes and endocrinology at U of T, you're very aware that these are the hallowed halls where insulin was discovered a century ago. That makes the bar for achievement incredibly high, but it also reminds us of the impact discovery science can have on the world—if we get our priorities straight. Canada is the second lowest of any G7 country for science funding per capita. You have a crisis like the pandemic, and suddenly everyone wants to know why Canada doesn't really have a domestic drug industry, but you can't put the horse in front of the cart. Supporting discovery science means investing in the questions as opposed to the answers. When I walked into the lab in Boston 40 years ago, the funding wasn't to go and find a treatment for obesity, Type 2 diabetes or whatever else the GLP-1 hormone may help treat. The question was, *What is this glucagon gene? How does it work? And what might it do?* And now here we are.

I don't think I'll ever get used to turning on the TV or riding the subway and seeing ads for Ozempic plastered everywhere. But I do

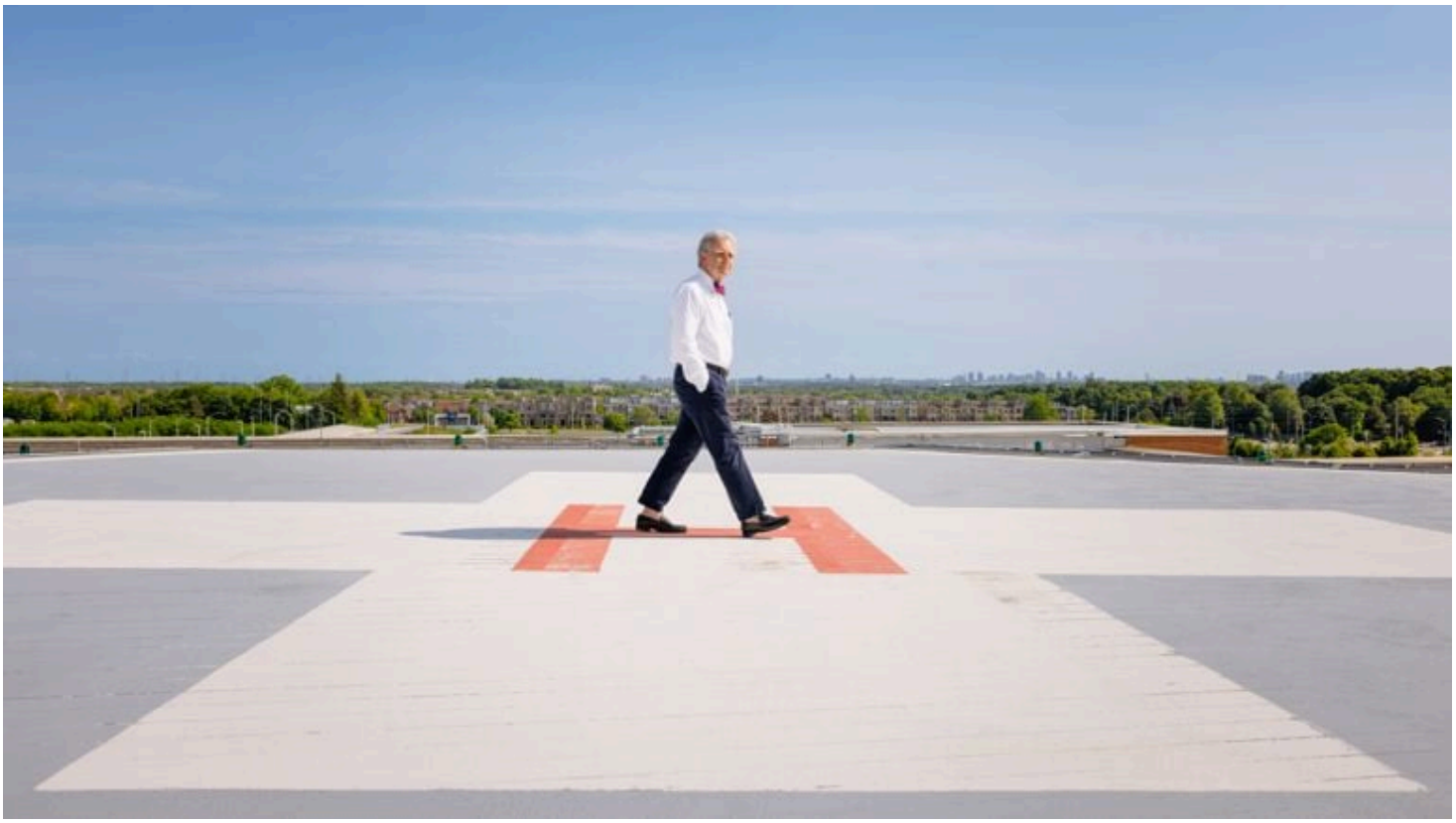
hope the drugs become even mo mainstream, because that should reduce the stigma. A common criticism is that people who use these drugs to manage weight are “taking the easy way out,” as if they are cheating and haven’t earned their results. Would you ever look at someone with rheumatoid arthritis or colon cancer and say, “Why aren’t you working harder on your health?” It’s prejudice. We all deserve medical treatment that can significantly decrease our chances of dying.

I would love to see insurance companies cover GLP-1 medications for people at risk of serious health problems associated with obesity. Accessibility and affordability have been significant barriers, but with new pill versions coming out soon, perhaps as early as next year, that will change. People often ask me if GLP-1 drugs are a cure for obesity, if this is “the end of fat.” I would never say that. The scientific community has a responsibility to continue examining root causes. Fifty years ago, there were all these Malthusian predictions that starvation was the greatest challenge facing humanity. Now, 30 per cent of the Canadian population is obese, and even the poorest countries in the world are affected. Why is that? Our genetics haven’t changed. Is it pollution? Is it a virus? Is it our food supply? Is it the way our cities are structured, that we drive everywhere and are obsessed with our devices? It’s almost certainly a multi-factor answer. We can’t just throw up our hands and put everyone on Ozempic.

Could accessibility to GLP-1 medications change the kinds of bodies we idealize? That’s something to ponder when you’re walking through the Art Gallery of Ontario and looking at

paintings from the 17th and 18th centuries, back when most people couldn't afford to eat as much as they needed and a full figure was a sign of wealth. Aesthetic ideals are based on what's unavailable to most people, not on what's healthy. Today's society worships thinness. Would that change if weight loss became readily available? It's an interesting question but one I'll leave to the sociologists. I've got enough on my docket. I want to golf, spend time with my wife and grandchildren, and continue the work that has been so meaningful to me. I'm a pretty low-key guy—my friends tell me I should be more excited. I *am* excited, and I'm proud of the things I have accomplished, but I also have a long list of priorities and obligations beyond science. For instance, Thursday is garbage day.

This story appears in the July 2025 issue of Toronto Life *magazine. To subscribe,* ***click here.*** *To purchase single issues,* ***click here.***



CITY NEWS

“I knew they would look after me”: How this GTA doctor ended up at his own hospital as a patient

DEEP DIVES

The Chatbot Will See You Now: I traded my human therapist for an AI equivalent

SHOPPING

Fall for fashion must-haves in Bloor-Yorkville this autumn

LIFE

Go behind the groundbreaking work at The Princess Margaret

CITY NEWS

Is Eugene Levy our greatest good luck charm?

Please let Mark Carney continue to be right about the Blue Jays