[00:00:01.800] - Host
From the Faculty of Pharmaceutical Sciences at UBC, welcome to TRxending, a podcast dedicated to exploring trending topics across all areas of pharmaceutical sciences, including research, practice, education, and community. Our episodes are recorded on the traditional, ancestral, and unceded territory of the Musqueam people. We encourage you, wherever you're listening, to also take a moment to consider the land you are on. The history, the original caretakers, and your relationship to it. Diabetes is one of the most common chronic conditions on the planet, affecting an estimated 830 million people worldwide. For a long time, management has come down to 2 main pillars: lifestyle changes and insulin therapy. But groundbreaking new research is completely rewriting that playbook. Today, I'm joined by Dr. Nicole Krentz, an assistant professor at the UBC Faculty of Pharmaceutical Sciences, and a recent recipient of the prestigious Breakthrough T1D Career Development Award. She's here to break down how protecting the pancreas's insulin-producing beta cells could transform the future of care and reveal some surprising hidden ties between type 1 and type 2 diabetes. If you want to dive deeper into Nicole's work, we've dropped a link to the full story in the description below. Now let's get to it.

[00:01:18.840] - Nicole
Yeah, my name is Nicole Krentz, and I'm an assistant professor in the Molecular and Systems Pharmacology Group here in the Faculty of Pharmaceutical Sciences at UBC. So that means that I have 2 roles sort of as a faculty member. I have an active research lab, so this is where we receive funding from the government or foundations to try and study the underlying causes of diabetes. And then also through my research lab, I train undergraduate students, graduate students, and postdocs to become scientists and researchers. So that's my research side of my role. And then I also teach. In our faculty, we have both a bachelor's program, so a Bachelor of Pharmaceutical Sciences, where we train people to become scientists as well. And then we also have a PharmD program. That's where we train the pharmacists in BC.

[00:02:05.700] - Host
Great. Welcome to the podcast. Thank you for joining us.

[00:02:08.620] - Nicole
Thanks for having me.

[00:02:10.010] - Host
Yeah, no worries. So you briefly explained your research or your role in your research, but I was wondering if you could expand a little bit and just give a brief overview of the research itself, like what you're doing with diabetes right now.

[00:02:22.030] - Nicole
Yeah, so we're interested broadly in diabetes. So by that, I mean, how and why people develop diabetes. And we always start with the genetics. So each of us have 4 to 5 million small changes in our DNA. Most of those changes do nothing, but some of them will influence our risk of developing diseases like diabetes.

[00:02:42.550] - Host
Right.

[00:02:42.720] - Nicole
And so we start by looking at those small changes in DNA that have been identified in people with diabetes. We take them into the lab where we can study exactly what those small changes do to how our cells form during development and how they produce and react to insulin.

[00:03:00.260] - Host
Okay, okay, very interesting. So I think a lot of people know about diabetes, obviously, it's one of the most common diseases and it's very prominent in public literature, but beyond that, we know the body has trouble with insulin, but in layman's terms, I'm wondering if you could explain what is it actually breaking at the cellular level?

[00:03:21.610] - Nicole
So it depends a little bit on the form of diabetes. There's sort of 2 main types of diabetes, which we call type 1, which is an autoimmune destruction of the cells in your pancreas that make insulin. So your immune system mistakes those cells as foreign and attacks and destroys those cells. So you end up not able to produce your own insulin.

[00:03:41.910] - Host
Okay, right.

[00:03:42.460] - Nicole
That's type 1. Type 2 is more commonly associated with obesity, but that's not a true characteristic of the disease. It's what we call insulin resistance. So by that, we mean you can still make some insulin, but your body doesn't respond to that insulin well. And so, what happens when your pancreas makes and puts insulin into the blood, it tells your other cells, oh, we have a lot of glucose in our blood, we need to take that glucose up and use it. And insulin is that really key signal that tells your other cells to do that. So, what happens in type 2 diabetes is that you end up not responding as well to that level of insulin, and so, that's what's known as insulin resistance. So what is really similar between the 2 forms of diabetes is central to that is a defect in the cells of your pancreas that make insulin. Those are called beta cells. So even though sort of the mechanism behind how the disease develops, central to both forms is some sort of defect in your body's ability to make or respond to insulin.

[00:04:50.450] - Host
I see, I see. Okay, so through your research, looking at these beta cells, did you find that there's like more of a, between type 1 and type 2 than we previously thought?

[00:05:01.900] - Nicole
Yeah, for sure. For many, many years, we thought of type 2 diabetes as more of an obesity-related, insulin resistance-related disease, and that is true to some extent, but from the genetics, we're actually seeing a lot more similarities and sort of this key kind of defect in how your cells make insulin. So there has to be this kind of key defect in your beta cell's ability to make insulin, and that's common across type 1 across both diseases. It's just in type 1, your body destroys those cells, and in type 2, they just struggle to make enough insulin to overcome that insulin resistance.

[00:05:40.620] - Host
Right, right. And we often hear, like, I think with type 2, that it's largely dependent on lifestyle and diet, and if you're good with those, you can kind of mitigate it later in life, is kind of how a lot of people see it. I'm wondering how true is that, or if There's certain markers that you can find in the DNA even before we're born, if that connects you to a higher risk of diabetes too later in life.

[00:06:08.620] - Nicole
Absolutely. There's definitely been a stigma surrounding type 2 diabetes, that it is very much lifestyle-driven. If we actually look at the genetic component of type 1 diabetes, probably about 50% of the risk is in your genes.

[00:06:24.580] - Host
Oh, wow.

[00:06:25.000] - Nicole
In type 2 diabetes, the range is anywhere from 30 to 70%. So genetically, they both have very similar roots in kind of our genes and in the mutations that we carry. So it's definitely something as a field we're growing to understand and appreciate more, and I hope that reflects in reducing the stigma that there is around type 2 diabetes, because, you know, it's not purely a lifestyle environmental thing. There has to be a genetic component to it as well.

[00:06:52.960] - Host
Hmm.

[00:06:53.700] - Nicole
And there's also some really interesting stories around individuals developing type 2 diabetes at a very low age, BMI, so without the obesity.

[00:07:02.580] - Host
Right, right.

[00:07:03.330] - Nicole
So as a postdoc, I studied this one gene called PAX4, and there are mutations in individuals in East Asia where they have high rates of variants or nucleotide or small DNA changes in this particular gene. And what's interesting about that is that this population of individuals in East Asia tend to develop type 2 diabetes at a lower body mass index.

[00:07:31.100] - Host
Oh, interesting.

[00:07:31.750] - Nicole
And that is because this gene is really, really important for how your beta cells are made.

[00:07:36.320] - Host
I see, I see.

[00:07:36.720] - Nicole
So before they're even born, they're starting out at this higher risk because they've generated less of those cells that make insulin.

[00:07:44.520] - Host
Oh, okay, okay.

[00:07:45.160] - Nicole
So that's a really nice example of why we need to rethink kind of what we believe is the underlying cause of type 2 diabetes, because there's a discrepancy between obesity and type 2 diabetes. Not everyone who is obese develops type 2 diabetes. Not everyone with type 2 diabetes is obese. And so we really need to kind of disconnect that idea and that stigma surrounding type 2 diabetes.

[00:08:10.340] - Host
Yeah, for sure. Even my dad, like, he's healthy as a horse and he, like, exercises a lot, eats well, but he's also like, he doesn't have diabetes, but he's on the edge. I don't know if it's called prediabetes, but he's like always hovering. He's been that way for several years, but it's like, wow, it's kind of interesting to see someone that healthy on the outside still, you know, being at risk.

[00:08:31.730] - Nicole
Yeah, absolutely.

[00:08:33.190] - Host
So, um, I— on the flip side, you mentioned that type 1 diabetes is 50%, um, like genetic. So what would the other 50% be like?

[00:08:41.910] - Nicole
Also potentially some sort of environmental trigger.

[00:08:44.800] - Host
Oh, okay, okay, I did not know that.

[00:08:46.030] - Nicole
So there are, um, there is sort of a model of type 1 diabetes where there's some sort of like viral

infection or some sort of environmental trigger that is necessary to kind of push you over into type 1 diabetes. So there could be a genetic component, but quite often with autoimmune diseases, there might be like a secondary environmental trigger that kind of pushes you over into a full autoimmune attack.

[00:09:12.230] - Host
Oh, okay, okay. So I think that's another stigma, 'cause I think a lot of people think it's just purely genetic.

[00:09:16.240] - Nicole
Exactly.

[00:09:16.980] - Host
I thought that coming in here, so.

[00:09:18.560] - Nicole
Yeah, and I think it's important also to recognize for both types that the environmental stimulus is often not something we have control over.

[00:09:26.840] - Host
Right, right.

[00:09:27.480] - Nicole
So if it's a viral infection, That's something that we have control over. If it's changes in your environment or in our lifestyle, that's also something often we don't have control over.

[00:09:38.700] - Host
Yeah, for sure. So I guess you're just dealt the cards you're dealt with.

[00:09:42.230] - Nicole
Yes.

[00:09:42.790] - Host
And that's where you come in, trying to help people cure and fix this disease. So yeah, you mentioned one key area of your research is looking at how the immune system is destroying these insulin-producing beta cells of the pancreas. I'm wondering if we could completely reverse this kind of beta cell destruction, what would that mean for people currently relying on daily injections with type 1 diabetes?

[00:10:06.500] - Nicole
Yeah, so I think the long-term goal of type 1 diabetes is to completely replace the requirement on insulin. So the way that we treat type 1 diabetes right now is by daily injections of insulin. And that's because your body doesn't make the insulin naturally, so you need to supplement with insulin injections. What we would like to see long-term is no longer needing an insulin injection. So there's kind of 2 ways you could do this. You could either prevent the disease from occurring, so somehow preventing the immune system from misrecognizing your pancreas as foreign, Right. and prevent that kind of autoimmune attack, and there's a lot of really cool research on that side of things, on how do we dampen that immune response. The other way is to replace those lost beta cells. So there's a lot of exciting research in that space, including a clinical trial here in Vancouver, where we can take human embryonic stem cells, we can coax them into insulin-producing cells in a dish, and then transplant those cells back into a patient.

[00:11:15.430] - Host
Oh, wow.

[00:11:15.610] - Nicole
So basically replacing the cells that have been lost. And that's been a really exciting area of research

for the past sort of 10, 15 years, and we're starting to see clinical trials clinical trials and success stories with that approach.

[00:11:28.680] - Host
Oh, wow, that's great. And so, is this like a novel approach to other diseases as well, or is it just kind of like within the realm of diabetes?

[00:11:35.370] - Nicole
Yeah, that's a great question. I think going back to both type 1 and type 2 diabetes being caused by the loss or dysfunction of a single cell type, which is a pancreatic beta cell, makes it unique in the way that we can now treat that through stem cell therapies. So because it's caused by the loss of one cell type, that makes it uniquely positioned to be treated with a stem cell therapy. There are not many diseases where a single cell type is affected.

[00:12:05.260] - Host
Right, I see, I see. Hopefully the progression continues and we can tackle this disease better in the future. So your research delves into the pancreas, right? I'm wondering if there's other things about organs that still surprise you or the scientific community today, just what you've noticed in your research or Yeah, I love this question because this is actually what made me want to be a scientist.

[00:12:33.110] - Nicole
So I'm really fascinated with how we develop in utero. So one of my first kind of research opportunities as an undergraduate was in Dr. Pamela Hoodless's lab at BC Cancer Agency, and she's interested in how organs form. And so through that work, I got to see how quickly organs form in a 24-hour period, how much changes, how complex it is, how intricate it is. And I was really fascinated by the fact that this, for the most part, occurs normally, right?

[00:13:08.720] - Host
Right, right.

[00:13:09.340] - Nicole
Even though we have 4 to 5 million changes in our DNA, we still form our organs correctly.

[00:13:15.970] - Host
True, yeah.

[00:13:16.630] - Nicole
Right? And that's a really fascinating thing to me, the fact, like, it's just such a beautiful process and so complex and intricate, and yet It occurs correctly every single time and very quickly.

[00:13:28.000] - Host
That's true, yeah, yeah.

[00:13:28.970] - Nicole
And so from that co-op experience, that undergraduate research experience, I completely changed my degree focus into developmental biology. I was just completely hooked with this fascinating process, and then chose to do my PhD in a developmental biology lab. So I think what I always find myself gravitating towards are studies and questions kind of surrounding that area. Like, how do we normally form? How do our bodies normally form? What happens when that goes wrong?

[00:13:59.440] - Host
Right, right.

[00:14:00.080] - Nicole
And how does that lead to disease? That's really sort of central to all of my research focus.

[00:14:04.400] - Host

Yeah, it's very interesting. I mean, the whole thing from when you start as an embryo all the way up.

[00:14:10.330] - Nicole
Yeah.

[00:14:10.580] - Host
I mean, it's kind of crazy.

[00:14:12.030] - Nicole
Yeah, I mean, it's 9 months in humans or it's 19 to 21 days in mice, and, you know, a lot has to happen in that small amount of time.

[00:14:20.590] - Host
Yeah, for sure, for sure.

[00:14:21.480] - Nicole
I'm also someone who loves puzzles, so I kind of feel like developing a puzzle— Yeah, it's like a kind of giant jigsaw puzzle. So it really fits my personality to study development.

[00:14:32.360] - Host
Yeah, sure. You mentioned previously stem cell research. And I think recently, correct me if I'm wrong, but this is also becoming intertwined with multi— what's it called?

[00:14:45.510] - Nicole
Multi-omics.

[00:14:46.180] - Host
Multi-omics, thank you. Which is big data for biology, as I understand it.

[00:14:51.160] - Nicole
Yep.

[00:14:52.160] - Host
So how does this How does this change the way we look for cures compared to how we did things like 20 years ago or in the past?

[00:14:58.150] - Nicole
It allows us to work at a much larger scale and at a faster pace. So by that I mean, you know, maybe 50 years ago we sort of had to go one gene at a time, one protein at a time, and that was really successful but slow because we can only focus on one thing at a time. Sort of with the advent of these omics or large datasets for biology, we can discover things faster, and we can understand how the mechanisms occur faster as well, just because it allows us to look at a larger number of genes at one time. And especially in the genetic side of things, the way we discovered the genetics of diabetes before was really like one gene at a time. So one single patient at a time.

[00:15:48.830] - Host
Right.

[00:15:49.050] - Nicole
And with these omics datasets, we're now sequencing millions of people, With and without diabetes, and we've identified over 600 different genes that potentially could influence risk.

[00:16:00.150] - Host
I see.

[00:16:00.420] - Nicole
So it's really going from sort of one-at-a-time approach to sort of large number of genes.

[00:16:04.230] - Host
Right, right, right. Does AI have like a part in this, like in analyzing all the datasets?

[00:16:09.290] - Nicole
Yeah, absolutely. I mean, AI and artificial intelligence has been used for several years now in the genetics. It just speeds up our ability to identify different genes.

[00:16:21.350] - Host
Right, exactly.

[00:16:22.090] - Nicole
It helps us sort of hypothesize why there might be defects. We can use AI for that. Yeah, so it also has sped up. I think the sort of downside of the data, large datasets, is that there's just so much data.

[00:16:38.490] - Host
Yeah.

[00:16:38.600] - Nicole
So prioritizing it can be difficult.

[00:16:40.770] - Host
True, yeah.

[00:16:41.090] - Nicole
And that's something AI can also help with.

[00:16:43.250] - Host
Do you run into issues with, like, clean data? Because I know data can be, you know, good quality or bad quality. And cleaning, it's often half the battle. I know for other industries anyway, I don't know about biology, but is that kind of the same kind of deal?

[00:16:56.730] - Nicole
Yeah, we're actually quite lucky, I think, in the diabetes space that people have been very collaborative. So people share their clean data very readily across, and there's also different platforms that you can use to look at all the datasets that have been generated across labs, across institutions, across countries.

[00:17:15.810] - Host
Very cool.

[00:17:17.120] - Nicole
So definitely it's a problem, But we work really hard to bring that data together in a way that is effective for everyone.

[00:17:26.410] - Host
Good. I guess it's becoming normalized too that we need clean datasets, so people are— when they initiate a dataset, they're thinking about this now versus like 10, 20 years ago, they're like, here's the data, we're just dumping it.

[00:17:38.770] - Nicole
No, exactly. It's very collaborative, so we all try to help each other out and put sort of the best datasets forward.

[00:17:44.680] - Host
Great, that's good to hear.

[00:17:45.480] - Nicole
Because we're working as a community to help people living with diabetes, so it is our responsibility to share this data openly and help it contribute to discovery.

[00:17:56.070] - Host
Yeah, for sure, for sure. So you've worked at a number of top institutions around the world. I'm wondering, in your experience, like visiting, visiting these top institutions and working at them, is there a trending topic in global pharmaceutical sciences right now that you're particularly excited in, or that the community is excited in?

[00:18:16.270] - Nicole
Oh, I think in diabetes, everything right now is GLP-1s. or GIP medications. So those are things like Ozempic for the treatment of type 2 diabetes. Oh, yes, yes, right. Or Mounjaro for the treatment of obesity. I particularly find this story really fascinating, both therapeutically because of the potential to impact the lives of so many people living with diabetes or with obesity, but it's also a really nice success story on the basic science side of things. So we've been studying GLP-1 and GIP for 4 decades now, Right. And we've been really focusing on these small little hormones. They're called the incretin hormones.

[00:18:57.280] - Host
Okay.

[00:18:57.760] - Nicole
They're produced in our gut and secreted into our blood, and they help control our blood glucose levels and overall metabolism.

[00:19:06.260] - Host
Okay.

[00:19:06.600] - Nicole
So this is something that we've known for 40 years. We've been studying how these hormones work.

[00:19:11.630] - Host
Right.

[00:19:12.260] - Nicole
And then sort of the past 10 years, we've seen the therapeutic payoff of all of that foundational science. So as a, basic scientists, I find it a really exciting success story because I like to think that the research I'm doing now in 30, 40 years could potentially lead to a new therapy for diabetes as well.

[00:19:31.660] - Host
Right, right, right. Yeah, it's very interesting. And then you hear about it everywhere now.

[00:19:36.400] - Nicole
Yeah, it's been really life-changing for people, especially I would say on the obesity side of things. We're now understanding more and more that there's food noise in individuals with obesity. That means that they struggle with that sort of noise of needing to eat food. And what the Mounjaro or these drugs can do is just dampen that noise. So no longer do they spend all day thinking about, when am I gonna eat? What am I gonna eat? And that can be really life-changing for someone who has had that sort of noise in the back of their head for many, many years.

[00:20:12.850] - Host
Yeah, yeah.

[00:20:14.150] - Nicole
And it shows that it's not about willpower. Again, going back to that stigma around obesity, it's not about willpower. There's this really strong, that is being caused by our brain to tell us to eat. And these

drugs can act and kind of dampen that signal.

[00:20:30.250] - Host
Yeah, very interesting. So yeah, moving on, can you describe a time in the lab where you found something in the genetic code that completely changed how you viewed diabetes risk, like the aha moment?

[00:20:42.020] - Nicole
Yeah, I think that goes back to the story I was telling you about where I researched in my postdoc. This was a collaboration with a group in Singapore, and so they had discovered through their clinic that individuals were coming in with diabetes at a very lean body mass index.

[00:20:59.520] - Host
Right.

[00:21:00.710] - Nicole
And so they sequenced these individuals and found that there was a particular mutation within this gene called PAX4. And because I'm a developmental biologist, I knew about PAX4. It's super essential for how our beta cells form. If you remove it in the mice, they don't make beta cells. So when I was offered this project as a postdoc, I was like, oh, this is cool. This is definitely going to be an issue in development. And so we worked in collaboration with this group in Singapore, and we made stem cells from either patients that carry that mutation, or we made our own stem cells with those mutations, and we tried to make beta cells out of them, and we showed that they don't form beta cells as well.

[00:21:40.840] - Host
Oh, okay.

[00:21:41.540] - Nicole
So what that told us is the reason individuals who carry this mutation develop diabetes at a lower rate BMI is because they're starting with less insulin-positive cells. So they're already kind of starting—

[00:21:54.770] - Host
Right.

[00:21:55.160] - Nicole
From birth with less ability to respond and make insulin. And so then when you add environmental triggers on top of that, that means they develop diabetes at a lower BMI. So I think that was a really cool aha moment for me because it tells me, you know, as a developmental biologist, I always think it always comes back to development, but that was a true story of this does affect development. And it also was interesting to know that there are different kind of ancestry-specific mutations.

[00:22:26.990] - Host
Right, right.

[00:22:27.510] - Nicole
So that also reminds me that we need to make sure we're studying diverse groups of people, because we might be missing out on a whole bunch of mutations or small changes in our DNA that are influential.

[00:22:40.060] - Host
Yeah, for sure, for sure. There's a lot of— we've been talking about type 1, type 2 diabetes, and kind of the stigmas, but there's a lot of myths too. I'm wondering if you could talk about myth-busting for a minute, and what are some of the things that you find yourself correcting people in terms of diabetes when you talk to people outside the lab?

[00:23:00.280] - Nicole

Yeah, I think there's sort of this misconception between type 1 and type 2 diabetes that one is good and one is bad.

[00:23:07.580] - Host
Well, neither are good.

[00:23:10.050] - Nicole
And neither are bad, right? They just differ in how we manage them. So type 1 diabetes, we manage with insulin, Type 2 diabetes, we can manage with pills and other drugs. But central to both is this underlying issue in how insulin is made and how we respond to insulin. So even though they are definitely separate diseases, there's commonality and similarity between the two.

[00:23:36.040] - Host
Right, right.

[00:23:37.320] - Nicole
And that's often something I find when I talk to people whose loved ones or individuals themselves who have diabetes, sort of a misconception behind it.

[00:23:47.010] - Host
Right, right, for sure.

[00:23:47.810] - Nicole
There's a lot more similarity between the different forms of diabetes than there are differences.

[00:23:52.310] - Host
Yeah, yeah, for sure, and your research points to that. So looking to the future, do you think one day we might be able to predict a person's risk for diabetes during infancy or even earlier? You talked about the genetic code or markers. Is that something that we can look forward to?

[00:24:08.620] - Nicole
Absolutely, there's a lot of research right now looking into what's called a personalized or a genetic risk score, so that's where we would look at someone's genes, and I mentioned there's 4 to 5 million small changes in your DNA. Most of those don't confer any risk, but we now have an idea of a number of those changes that would increase your risk of diabetes. So if we look at someone's genetics, we can sort of calculate their individual risk by the presence and the number of these markers that they have.

[00:24:42.830] - Host
Okay, okay.

[00:24:43.330] - Nicole
And so that's definitely something that's in kind of early stages of research, but is showing a lot of promise. And the next step will be to roll that out into the clinic.

[00:24:55.700] - Host
Do you think that'll take a while? Or— and before you mentioned 30, 40 years, but I mean, I have hopes that it'll speed up now. Like, I don't know, it's a different time now, especially with AI and everything.

[00:25:07.130] - Nicole
Yeah, I think the science is there. I think the struggle might be how we roll that out practically in the clinic, because it does require an expert. I'm not an expert in genetics, and we can't expect every doctor or pharmacist to be an expert in those things.

[00:25:24.720] - Host
Yeah, for sure.

[00:25:25.180] - Nicole
So we would need to train people to really interpret this data.

[00:25:29.350] - Host
I see.

[00:25:29.990] - Nicole
And so that will be, I think, the link that is difficult for us to navigate, is how do we get the data in a digestible form where clinicians can counsel patients on their potential risk?

[00:25:44.020] - Host
Right. I mean, the good news is I think this, uh, the whole personalized medicine branch is becoming much more, uh, known and popularized, I guess. Um, I see it because we were Faculty of Pharmaceutical Science. Not only do we see it in research, but in practice too.

[00:25:58.950] - Nicole
Yeah.

[00:25:59.140] - Host
I know the clinic is doing a bunch of, of research related to personalized medicine. Um, not at like the molecular or data or anything, but like looking at just like individualized patient care.

[00:26:10.350] - Nicole
Yeah, and there's definitely success stories, like the BRCA1 gene in breast cancer is a very strong sort of determinant of your risk of breast cancer.

[00:26:18.380] - Host
Right, right.

[00:26:18.870] - Nicole
And there are success stories of people having their genome sequenced, looking for that potential BRCA gene mutation, and then maybe taking some preventative steps to reduce their risk of developing breast cancer. So there's definitely a model there, it's just a matter of how we roll that out, and also the genetics of diabetes is a lot more complex. Right. It's not one single gene, it's a variety of genes. So how do we kind of interpret the overall risk?

[00:26:50.400] - Host
Right, exactly. So we mentioned that diabetes is very, very common. I'm sure someone listening here has, maybe have it, or at least has a family member who they know has it. I know I do. You probably do. So for someone listening who has a family history of diabetes or maybe It's in their family. What does your research offer in terms of hope or practical understanding for, say, the next decade or so?

[00:27:18.160] - Nicole
Yeah, I think there's sort of 2 ways in which my research could be beneficial to individuals living with diabetes. One side would be the genetics. So my lab is really interested in following up on how those different changes in our DNA impact our risk of developing of developing diabetes. And so that will just help us going back to the personalized medicine approach, it'll help us sort of clarify the ways in which those mutations can impact diabetes risk.

[00:27:52.040] - Host
Right.

[00:27:52.540] - Nicole
And therefore can kind of feed back into that risk score because we'll know how detrimental or impactful those small changes are. So that's one way sort of on the personal medicine side. The other way is that by understanding the underlying causes causes of diabetes, being in the Faculty of Pharmaceutical Sciences, I believe that has the potential for helping us develop new therapeutic

strategies.

[00:28:17.550] - Host
Right.

[00:28:17.970] - Nicole
So going back to those incretins, Ozempic and Mounjaro, those are rooted in basic science. So people that studied those hormones for decades, and then we realized the translational potential. So my goal is that what we're studying now in the lab could have a therapeutic potential, hopefully not in 40 years, hopefully sooner than that. So that's always goal whenever we're taking on projects in the lab is we like to think of how are we going to positively impact the lives of people living with diabetes. And through the BC Diabetes Research Network and through the Breakthrough T1D Canucks for Kids Center of Excellence, we spend a lot of time talking to individuals with diabetes and trying to ground our research in what will actually make an impactful change on their lives. So they often tell us, You know, we don't really care if there's a cure, we just want better treatments. We want ways that we can control our lives better and not rely on insulin injections constantly and blood pricks constantly.

[00:29:17.710] - Host
Yeah, exactly.

[00:29:18.780] - Nicole
So we always try to keep in mind what will impact the lives of people living with diabetes and ground our research in that philosophy first.

[00:29:29.020] - Host
Great, great, great. Speaking more about the future, I'm wondering what excites you within the realm of diabetes research that we haven't talked about yet? Or it could be pharmaceutical science research in general.

[00:29:41.730] - Nicole
Yeah, I'm interested to see what the next big discovery will be. I don't have a crystal ball, so I don't know what that would be. But, you know, in my career, when I started, stem cell research was just kind of getting off the ground. 15 years later, that's in clinical trials.

[00:30:01.120] - Host
True, yeah.

[00:30:01.660] - Nicole
And at the time it seemed impossible and like science fiction. So I'm really excited to see kind of what that next thing that seems impossible at this point, but in the next sort of 10, 15 years becomes possible and also in place in diabetes research.

[00:30:22.160] - Host
Yeah.

[00:30:22.740] - Nicole
I don't know what that is. If I did, I would, I don't know, I would have some sort of magic What do you call that?

[00:30:34.210] - Host
You mentioned crystal ball. Yes.

[00:30:37.160] - Nicole
If I knew what it was going to be, I would have some sort of crystal ball.

[00:30:40.470] - Host
I think we all want one of those.

[00:30:41.620] - Nicole
Yeah, and I would probably purchase a lottery ticket and fund all research with the funding if I had some sort of understanding of what was to come.

[00:30:52.110] - Host
Great. Well, thank you, Nicole, so much for being on the podcast, and thank you to our listeners. If you're interested in the podcast, please Please like and subscribe and leave a review. It really helps bring the podcast to more people. And we'll see you next time on What's Trending in Pharmaceutical Sciences.