VIEW to the U transcribed
Season 1, Episode #12
Professor Loren Martin

Podcast Interview with Professor Martin recorded on December 8, 2017

[Theme music]

Loren Martin (LM): Think about this: in terms of pain, chronic pain conditions, it cost the Canadian economy an estimated 50 to 60 billion dollars…a year. But in terms of funding for pain research, our main funding body would be the Canadian Institute of Health Research, I think we maybe make up one percent of their total budget. In terms of pain, it's become de-prioritized because it's sort of thinking that no one dies from pain, but there is obviously a certain quality of life aspect to it, and there's loss of productivity and healthcare.

In terms of where we are going, I think there's more of an effort for crosstalk, and I really want to emphasize that because I think that's really what we need more of. Because those basic science people, what we can do is we can help develop better therapeutics in these type of things, but if we are sort of in our silo, it may not have any applicability in people.

[Theme music fades in]

Carla DeMarco (CD): Pain, empathy and stress are at the forefront of this researcher's work, but he is also emphasizing the importance of funding for basic research and also how there should be "more crosstalk" and productive connections between basic researchers and clinical researchers.

Today on View to the U, Professor Loren Martin, a faculty member in U of T Mississauga's Department of Psychology, discusses the various aspects related to his work, and we touch on several areas, including his chronic pain research lab at UTM, how gender factors into pain studies, the placebo effect, and some of the challenges that emerging scholars face.

Also, coming to light in this interview is that size matters ... when it comes to a pill size, that is. But also, so does the color and price of that pill. I also attempt to debunk some so-called pain-related myths, which actually all turn out to be true. And in honour of UTM's 50th celebration, Loren reflects on the growth of the campus, and a look to more collaboration across disciplines.

Hello, and welcome to View to the U: An Eye on UTM Research. I'm Carla DeMarco at U of T Mississauga. View to the U is a monthly podcast that will feature UTM faculty members from a range of disciplines who will illuminate some of the inner workings of the science labs, and enlighten the social sciences and humanities hubs at UTM.

[Theme music fades out]

Professor Loren Martin has been a faculty member in UTM's Department of Psychology since 2015. In that same year, he was also awarded the prestigious Designation of Canada Research Chair in Translational Pain Research. His work aims to understand the interplay between stress and pain, how pain might negatively affect such things as learning and memory, and the biological mechanisms behind empathy and pro-social behaviors.

Your research spans several areas, including chronic pain, stress, social interactions and empathy. I'm just wondering if you could provide a broad overview of your work, and perhaps some specific projects you've worked on over your time. I know that you started first at McGill University in your academic career, but now you're here at UTM. I'm curious about what kinds of projects you have undertaken, or you could tell me a little bit more about your research.

LM: Sure. I was initially trained as a Behavioural Neuroscientist, and I was interested in memory and how synaptic changes ... and by synaptic changes, I mean the connections between neurons, how they change when we learn something, and how that occurs in the brain. About eight years ago now, I made the move to McGill and I decided, strategically, to work with someone in the pain field. I did this because there's a lot of overlap between the basic mechanisms, underlying memory, and underlying pain.

In the memory field, when we have changes in connections between neurons, our brain cells, and these connections are enhanced, that's a good thing. That usually results memory and these types of things. When that happens in the spinal cord, it's a bad thing because those changes now result in the neurons of the spinal cord now conveying more pain information.

CD: Like more intense pain?

LM: It can be an intensity thing, but it can also be a persistent thing. That's really one of the underlying theories for chronic pain. I strategically made this decision because I still have an interest in memory, and there is a lot of overlap, as I mentioned, between memory and pain. There is a theory in the pain field that a lot of chronic pain is because we can't forget the pain. I did this specifically because no one is really studying it.

When I was at McGill, I started looking at how memory changes in response to pain. This was my way of trying to merge the two fields. We did that, and actually have a very interesting story in which, if you experience pain in a particular environment, you have a memory for that pain within that environment, and upon revisiting that environment, your pain sensitivity changes. It actually becomes stronger. You become more sensitive, because we have a specific memory for that.

We do more than that. We also study how ... because pain is not only one thing, it's not only the biology that's important for pain signalling. Pain signalling is influenced by our thoughts, our feelings, who are friends with, who we interact with. We still look at it today in my lab how our friends change our pain. If we are friends and we are in pain together, how has our pain perception changed? What we find largely ... and this is true for both animals, we do a lot of work in animals, but we also do some work in people is that if we are in pain with our friends, our pain perception becomes heightened. We feel more pain.

If we are in pain with a stranger, there is sort of a blunted response. Our pain sensitivity doesn't change. I used that at McGill, and I use that today in my lab as a model of empathy. If we are in pain together, how does your pain influence my pain? Can I feel your pain? That is something that we've become very interested in first at McGill, but also we're interested in the biology. It really spans the social dynamics, psychology if you want to consider memory, and the pure biology. What changes when we injure ourself? That can be due to an inflammation, that can be due to a nerve injury.

What changes biologically that results in more pain signalling? That's really what chronic pain ends up being. If we've undergone an injury, and then this pain signalling is persistent. It's always there, and it's

not going away. We are looking at why it doesn't go away.

CD: I know from hearing you speak before that you talked very much about context, like what situation you're in when you're with your friends, or by yourself, it's about the context.

LM: It very much is. We've done some sort of conditioning experiments, and this work was started at McGill in which a very mild pain stimulus can be given to a mouse in a particular context, an environment. Then, we can take that mouse ... and it works the same for people, we can put that mouse or person back into that same context and their pain sensitivity has changed because their pain system has now been conditioned. What we do know is that it's stress. When you've experienced pain in a particular environment and you revisit that, there's a certain level of stress. For a long time now, we've known that stress can modulate pain both up and down. It's still being worked out, but stress can either reduce pain, or it can heighten pain.

CD: One of the studies I read that you were quoted in ... I think it was in Los Angeles Times where they were talking about this whole thing about putting your hand in the cold water. I remember years ago these two guys were trying to put their hands in an ice bucket to see who could hold their hand in there the longest. They each had their own thing, and it was just sort of, to me, that machismo, see who could hold it in the longest, but I almost felt like looking at them, they held their hands in there for really long time but trying to outdo each other. I just thought maybe there's something factoring in that they are able to turn off that pain receptor, I don't know.

LM: There are a number of studies like that. There was actually a study published earlier this year showing that macho guys actually make really poor pain subjects, specifically for that reason because they don't like to show their pain. It's not necessarily that they are not perceiving or feeling their pain, so they are actually, if they are included in a pain studies, they're skewing the results quite a bit.

A while back, there was another study published in which the males and females were looked at in terms of whether or not it was holding their hands in cold water, something like that. There was a big sex difference depending on whether or not the opposite sex was testing a participant. So, if it was a male participant being tested by a female experimenter, their pain threshold was very different. They were able to withstand cold water much more because they didn't want to show any form of pain in front of the female experimenter.

The opposite was true for females. They showed more pain if there was a male tester. We've shown that that also holds truth for mice. This was published in 2014 in one of the *Nature* Journals, and if the tester is a male, mice show less pain. If the tester is a female, they show normal levels of pain.

CD: That is fascinating.

LM: Yeah, and apparently it's not even true for pain research, because we did a few other experiments in which we show that it's also true for anxiety, and it all links back to stress. There was, this past year, at the Society for Neuroscience Conference, showing that the same male/female difference for antidepressant medication. When you give an antidepressant medication to a mouse, if the effects of the antidepressant medication are stronger if given by a male, as opposed to a female.

What it really boils down to is there is a big evolutionary mechanism for this, and maybe there might be some sort of predatory thing with mouse versus human. The gender thing becomes really interesting because mice are stressed by male humans, but not females.

A lot of it has to do with our axial secretions, because if you present just axial secretions that are predominately ... so, all of those sweat hormones that are found more in men versus women, the mice become more stressed. If you present to a mouse the axial secretions of a woman that are not really found in men, it doesn't do anything to them. It was a pretty remarkable finding at the time, and it really maybe suggests that a lot of the findings in the pre-clinical literature may actually be skewed.
CD: That is really incredible. I know that you also mentioned at the talk that you gave here about how colour also affects people's either perception of pain, but also you had mentioned some study that had to do with pills: one being a blue pill, one being a red pill. I was wondering if you could talk a little bit about that as well.

LM: Sure. Something the pharmaceutical industry has known for quite a while is that the color of a pill you take has a big placebo response. Take the color blue, for instance. If you take a blue pill and maybe it doesn't even have any active ingredients, you will sleep better and longer than if that pill were red. It's thought that under normal circumstances, we typically associate blue with very calm and soothing and relaxing. So, it makes sense that a lot of sleep tablets are actually blue, and they're not red.

Red evokes these emotions of rage, and maybe hostility and these types of things. That's why it's thought that it has this very, very counter effect. That blue effect holds true worldwide, except in Italy. If you know anything about the Italian culture, and this is not really proven, but the National Men's Soccer Team is the Azzurri ... the Italian blue. It's thought that for an Italian man, blue actually evokes excitement and all of the things normally associated with Italian soccer.

It really ties back to your beliefs and what we have been exposed to and conditioned to, but it's not only colour because in terms of placebo responses ... we think the size of the pill matters. If you take a larger pill, it has a better effect that some of the smaller pills. The price of the pill matters. If I were to give you a pill, and I said, "This is a really good analgesic. It costs so much money," or, "I will give you this pill, it's also a really good analgesic. It costs considerably less." You take both pills, you will have more of an analgesic response, so less pain, with a more expensive pill.

If I list all of the side effects associated with those pills, you will have more side effects with the more expensive pill. It's thought because we sort of associate price and cost of pills with more potency, so it's going to work better. If those two pills have the same side effects, we assume that that more expensive pill is going to work better, so it may do something more for our pain, but it's also going to do other things in terms of the side effects.

CD: I'm thinking every single brand out there, there's always the store brand, the generic one ... so, are we then sort of then fooled into thinking because we paid more…? I guess it's that same idea, though.

LM: It's exactly the same idea. For brand names versus generics, even though there is *nothing* different in terms of the chemical structure and what those drugs do, the brand name usually works better.

CD: Oh, really?

LM: It does. Not because it does anything pharmacologically different, but because we assume-

CD: We think.

LM: We think it must be better, because it's not just this generic brand.

CD: Is there a way to recondition ourselves into thinking…?

LM: I don't know. I really don't know. Even in terms of advertising, this is actually really interesting ... pharmaceutical companies put a lot of money towards their advertising campaigns, and the amount of money they put towards the advertisement, the placebo response is larger. This wasn't my study, but it was from my old lab at McGill. They've found that over time, the placebo effect has increased. If you look at all the studies, and these are just pain clinical trials not necessarily looking at the placebo study, but looking at all of those clinical trials in which an active drug was tested against a placebo, and then looking at the response of only that placebo group, the placebo response has increased from the 70's all the way up until whenever it was analyzed, maybe 2015, but only in North America.

CD: Why do you think that is, though?

LM: Probably because the placebo effect in North America is more in our face. There's more advertising, and it's only sort of a theory at this point, but especially in the U.S. Most of the studies are from the U.S., a little bit from Canada. There is a lot put towards advertising, and not to say that there isn't in Europe, but I think there's more emphasis on media and sort of awareness of either the drug, or even we're just more cognizant of the Placebo Effect.

At UTM, I teach a pain course and the last couple of lectures, we talked about the placebo effect. One of the first things I did was asked who believes in the placebo effect, and this was a 100-student course, everyone put up their hand. Whereas maybe in the 80's and 90's ... and this is not a real thing, but now people are now starting to realize the placebo effect can be real powerful.

CD: I know that you mentioned, too, there was another study that you had done where you were talking about if you put a metal rod that was a certain temperature ... if it was red, people indicated they felt more pain.

LM: If you place a metal rod in which there is a certain level of pain associated, so it's a cooled metal rod to minus 20 degrees Celsius, you touch that against your skin, it's going to be painful. If you are looking at a red light versus a blue light, you will report more pain when looking at the red light and being touched with that metal rod. It's thought that because we also associate the color red with things that are dangerous, things that might be hot ... so, our brain is pretty clever, and it wants us to avoid any of those situations.

In that context, pain can be pretty adaptive in that if you touch something that's hot, your pain system is going to become activated to tell you to get out of that situation. It's thought that that's what our brain is trying to do, because again, we've been conditioned to certain things even though we never really think about these things.

CD: One of the studies that I read about on our site was about some of the studies you've done related to empathy.

LM: In terms of empathy, what we really study, and what we're really interested in are the fundamental mechanism. Because we are a neuroscience lab, we like to study things on a fundamental, very basic level. A lot of those studies involve mouse subjects in which we will use a very, very mild pain stimulus and maybe have two mice, which they are in pain, and we observe what happens to their pain behaviour. When you're in pain ... when we put a mouse in pain with its buddy, so a mouse from the same cage, those mice experience more pain together.

It's thought that that's a very primitive form of empathy called "Emotional Contagion." We can do the same thing with people, so in that cold water experiment when you put the hands in the cold-water bath with your friend, there is more pain. We don't find that with the strangers. What we're doing now is we're looking to see what *blocks* empathy in strangers. That relates back to ... and this is a finding at McGill that we are stressed by people, and I guess mice, are stressed by individuals whom they don't know.

There's a certain degree of unfamiliarity, which breeds a certain level of stress. That is sufficient enough to block this very, very primitive form of empathy, and we call it "Emotional Contagion." And one of my grad students right now is looking at the neural mechanisms of what happens. There is a brain area, right at the front of our brain, called the medial prefrontal cortex. What we find is that there is a significant amount of stress receptors that become activated when those individuals don't know each other. If we block only those receptors, we now can reveal this emotional contagion. So, it really seems like ... and I should point out that that medial prefrontal cortex is highly implicated in human empathy.

It seems like it's very relatable to the human experience of empathy, but now we're just trying to boil it down to a very fundamental, targeted mechanism of empathy. Because, if at some point there were a way to target that system, we could maybe create more empathic individuals and break down barriers in these type of things. I guess that would be the long-term implications.

CD: I know one of the studies I was reading, the E-A ... something like that-

LM: EGFR?

CD: EGFR. That was a study related to looking into turning off that receptor? Can you describe that? I found that one really interesting, and I thought I'd have to ask you more about this.

LM: This was EGFR. This was a very long process. This actually started when I was at McGill. I joined the lab at McGill in December of 2010. I injected my first mouse for EGFR in December 2010. It was just published in August 2017.

CD: Seven years.

LM: Seven years I spent on that paper, but that paper started actually really interesting, because we identified that gene. EGFR is a protein, it's a receptor, but all proteins and receptors are made by genes. We identified the gene, specifically EGFR, as being associated with a specific chronic pain condition in people. We knew that this gene played a role in chronic pain in people, and then it became my job to figure out how does it work.

Through a lot of very, very long studies, what we show is that in various models of chronic pain ... we use mouse, we use rats, we even use fruit flies, to show in all of those species, EGFR plays a role in

sensory and specifically pain transduction. In any model of chronic pain, when there is inflammation, when there is nerve injury, the EGFR receptor becomes more active. It becomes more active in our peripheral nervous system, more or less.

What we find is that the EGFR feeds in, or there's a certain level of crosstalk with a receptor that we already know is involved in pain. That's how we believe it's sort of modulating the pain system. What was really interesting with that study is that it wasn't only the EGFR that was implicated, there were also molecules that bind to and activate the EGFR. Within that family of receptors, there are I want to say maybe eight or nine molecules that will activate the EGFR. But, it was only one ligand in particular, one molecule in particular, that was important for conveying the pain information.

What we're going to try to do now is ... because when you inhibit that receptor, there are a lot of side effects. They're not very bad side effects ... it's like dermatitis and skin rash, and these type of things, but we think a better strategy may actually be going after the ligand itself, going after the molecule so that if we can prevent that ligand from binding to the EGFR by maybe lowering its levels, it may be a pretty effective pain strategy for chronic-pain treatment.

CD: Is the ultimate intention would be to move away from ... I know the opiod crisis has become a big issue, so then this would be a way to sort of move away from that kind of treatment?

LM: Yeah, there's a big push right now to develop novel pain killing drugs that lack addictive and overdose potential, so the opioids. Targeting this system would be one way because there is no addiction potential, there is no overdose potential with any of these drugs. They do have side effects of their own, as I mentioned skin rash and these type of things, but that can either usually be controlled, or it's better than some of the adverse side effects that are associated with opiates such as the overdose and addictive potential.

CD: One of the things I'll sort of throw at you because I wanted to maybe debunk some myths that we have about pain. I sort of thought about this when I saw you speak, but do you think that people actually have different thresholds for pain? I hear people talk about this, and so I'm wondering do you think is actually something that happens?

LM: It's a big thing. It’s a big thing for a variety of reasons, but in the pain field there are *huge* individual differences in whether or not that is mediated by our genes, is one. In terms of genes, there are now over 400 pain genes that have been identified; genes that have some role or function in altering pain, and even our sex, our gender, has a very big effect on our pain perception with women being typically more sensitive than males. Going back to the pain gene, people with red hair are more sensitive to pain. This was identified by my old boss at McGill a number of years ago, and it has to do again with the gene, with a specific gene.

CD: That's so amazing. The other myth I was thinking about, maybe you touched on this a little bit, but I remember a long time ago there was different theories about people doing dentistry without using Novocaine, because they had put something to pinch the person's ear, they couldn't then feel the pain in their mouth. I know that there is this whole theory if you have a migraine, if you pinch between your thumb and your finger ... is there any truth to some of things?

LM: There actually is.

CD: Yeah?

LM: Yeah. There is something, and it's a very real phenomenon called "Condition Pain Modulation," which basically amounts to pain inhibits pain. If you have pain in a particular part of your body, and then I were to induce or inflict pain in another part of your body, sure you might have pain in that second site, but the original pain is going to be less.

CD: So, it's like redirecting it or something.
LM: Yeah, there is a certain level of modulation, and a lot of that modulation comes from the brain. It's the brain modulating any of that pain coming through the spinal cord and these types of things. Interestingly, fibromyalgics ... people that have fibromyalgia don't have that capability because they have deficient system to modulate any incoming pain signals.

CD: Fibromyalgia is a chronic pain condition.

LM: It is a chronic pain condition, it has a certain degree of inflammation. It's usually associated with pain in the tissue joints. There are specific diagnostic criteria for it, but the primary criteria is widespread pain, bodily pain, for three months or more.

CD: The other thing I wanted to ask you about was what have been some of the more surprising findings that you have come across over the course of your-

LM: As a student, especially being in the memory field, studying mostly animals ... even though I worked with an anesthesiologist, and so there was a clinical component to that. We were studying how memory becomes inhibited when you are exposed to general anesthetics. There was a huge clinical component to it.

But something that I was unaware of was the lack of translation between what happens in a mouse and what happens in a person. That, I think, for the past five to seven years, has been the biggest surprise to me: how far we still have to go in terms of developing new drugs, and new treatments, because most of the work happens in animals in terms of drug development and these types of things.

In a lot of cases, those drugs don't work in people. Why? That, I think, to me, has been really eye opening. A lot of my work, we really try to do both in the same studies. We try to ... for the empathy stuff, it was great to show this in mice, but does it have any applicability in people? If it *does*, then it becomes a little bit more relevant in terms of drug development. That was really surprising. You can start off with about 10,000 compounds and maybe come up with one drug, but it's like 9,999 failures to yield one drug. In terms of surprise, that was the biggest thing, I think.

To be honest with you, I don't think I would have been exposed to that if I weren't in the pain field, because a lot of our conferences and places that we present our data, it's not only the very basic researchers who are sort of studying one specific thing, because they're very integrative. You have to sort of take a global perspective of everything from the pre-clinical to the human experimental, to the clinical side of things.

It becomes very, very apparent in the pain field, especially there's a big push to have more crosstalk between the basic scientist and the clinical researchers to figure out the problem and what we can do, because it's great and it's nice to show things in animals. But, if it has no relevance to people or the

patients you intend to treat, then it's all for naught.

CD: I know from speaking to someone here that it takes so long to get something even to clinical trial, right?

LM: Oh, yes.

CD: You don't want to take any chances, but it's like all these studies ... to get to the clinical point, have been in the works for years, right?

LM: For years. Even, in terms of academics, academics will work on maybe you'll have your favourite receptor, things that you'll work on, maybe you'll spend 20 years on that. Maybe your body of work is enough to convince a pharmaceutical company that they should now invest their own resources into that. At the preclinical stage, they still want to convince themselves that this a real thing that they should be studying. They will spend a good six to eight years just studying it again, and then the whole clinical trial business can take another six to eight years, again.

The whole process for drug development, once the pharmaceutical company gets a hold of this, could be a good 14, 16 years ... it's a long time, even in terms of knowledge translation. So, just aside from the pharmaceutical companies and these types of things, it sort of estimated that if you're a psychologist and you are doing things and you find something that is very important in your line of work, it takes on average, 17 years for that finding in the lab to make it into clinical practice. What's the hold up?

CD: I'm thinking as you're talking about ... even if you wanted to do some sort of collaboration, as you said, you might be focusing on one specific receptor, but maybe you want to bring someone on board whose doing this other thing. I just think that would lend itself to this whole other level of complexity.

LM: It really becomes hard in terms of looking at specific receptors, that's really why people will study one main thing and then maybe look at how that one main thing maybe interacts with something else. There are only so many interactions, which you can do. Even people doing large genome sequencing type of things where they may now find your one molecule that you might interested in, now participates in maybe a host of other molecules. Modulation changes other things. I mean, you're going to pick one route to study at a time. You can't study everything at a time, especially if you just think of science at the preclinical level, everything is so controlled. Everything is so controlled because you want to make sure that A affects B, maybe B affects C, and then does A affect C? There are only so many iterations-

CD: Yeah, it's got to be so targeted [background]

LM: It does have to be targeted, which doesn't mean that maybe later on you can now study how A affects D or E, but to do it all together, it becomes very complex.

CD: You mentioned the translational pain, and I was thinking that must be very much tied into your Canada Research Chair, right?

LM: It is.

CD: Okay.

LM: The entire Canada Research Chair was written specifically with translation in mind.

As a new faculty, I can tell you it's been hard to set up both aspects because you only have so many resources in which you can focus on. So I made a concerted effort, at least for the first three years, to focus on getting the preclinical stuff up and running. We are now ready, at least, to start exploring some of the human side of things. We are also doing that through a few collaborations in which we have now on campus, with some people within our department, because we have expertise for measuring biological markers and these types of things.

Within the psychology department, there are faculty that are interested in biological markers, but just don't have the expertise to do it. We are trying to lend our expertise in terms of collaboration that way.

CD: I always like to ask people what do you think is the biggest impact of your work?

LM: I think the biggest impact ... and I don't know whether or not I would say impact *yet*, but at least where I think we have the potential to make the biggest impact is our effort to communicate and work with the clinical scientist, because we really are making a concerted effort to try and bridge the gap between what happens in our animal models, and what happens clinically. A couple of months ... and this is not only this next conference, but I will be going to the American Pain Society Conference in which – and it always happens to me – in which I am always involved in these symposiums.

Usually the symposiums are three faculty members, and in this instance, they are from different institutions: one's in the States, I'm in Canada, and one's in Europe. I'm always the token basic scientist because we ask relevant questions to the clinical scientists. This symposium, in particular, is on how social factors modulate our pain perception. In terms of pain, for the clinical scientist, it's a *huge* component of what they are interested in, what they study. The basic scientists, not so much.

There's not a lot in which they actually focus on on social factors, even psychological factors. Most basic scientists, because we use animal models, and what our animal models are good for ... they are good for studying biology, but we now know from a lot of clinical research that pain is *much more* than just biology, its psychological factors and its social factors, and it's really that interaction. I think that's probably our biggest impact, is really our ... I would say ability, but at least our *effort* to try and study and understand that.

CD: I'm curious about what do you think is the next goal you're aiming for, or where your field is headed in terms of pain and these studies?

LM: It's challenging, I think, for pain research in particular. I know everyone within their own fields sort of says, "We need more funding, and we need to do a better job," and these types of things.

But, think about this: in terms of pain, chronic pain conditions, it costs the Canadian economy an estimated 50 to 60 billion dollars, a year. But in terms of funding for pain research, and so our main funding body would be the Canadian Institutes of Health Research, I think we maybe make up one percent of their total budget.

In terms of pain, it's become deprioritized, because it's sort of the thinking that well, no one dies from pain, but there's obviously a certain quality of life aspect to it, and there's loss of productivity and healthcare costs, and all of these things are not even taken into account. We submitted a paper, and our opening sentence was, "25 percent of the world suffers from chronic pain at some point in their life." Which is actually a true statistic, the reviewer, probably not from the pain field, didn't believe us.

CD: Like you were just making it up.

LM: We're just making it up. It becomes really hard to convince people. In terms of where we are going, I think, and this is not only me, but there is more of an effort for crosstalk. I really want to emphasize that, because I think that's really what we need more of. I think that's-

CD: You're saying more with the basic science people with the clinical people.

LM: Yeah, exactly. Those basic science people, what we can do is we can help develop better therapeutics and these types of things, but if we are sort of in our silo, it may not have any applicability to people.

[Interlude music]

CD: Coming up: UTM @ 50.
Though he's still relatively new to UTM, having started here in 2015, Loren reflects on the growth and changing demographic of the campus, and the collaborations he would like to see develop on the horizon.

CD: I understand you've been here since 2015. It's been a couple of years, but I'm just wondering what kinds of changes you've seen since you've been here, and also maybe what do you foresee either for your field or your department coming up?

LM: In terms of changes, there's a lot of construction. There's a lot of things happening, it's just that it's a really slow process in terms of the way that the university works and construction projects work, and things like that. That's been slow. I think that good changes are coming.

In terms of changes, I'd probably point to two things. I was a student at U of T downtown, on the St. George campus in the Physiology Department, and while I was a student, UTM was really known as more of a teaching campus and things like that.

I think *now* there's much more of an effort to promote research and there's going to be the new creation of the science building, and these types of things. I think that's something that I've seen from my time as a Ph.D. student to now.

Also, something that I've noticed is that especially within my department, and I think this is true for some other departments, is that the campus as a whole is getting pretty young in terms of faculty. There is a lot of new faculty being recruited. Just within my department since I've been here, I want to say there are maybe three or four new faculty members.

This year we are recruiting two more with maybe another one next year. It's quite a bit, which I think is a good thing. There is an effort towards improving the research capabilities of the campus as a whole, and

even in terms of recruiting younger faculty for teaching purposes and different things, even within our department. We have a teaching-only faculty, and we've recruited some of those individuals, and they've been fantastic additions to our department.

CD: I don't know if it's just my perception, but I also think there's been more of an effort made to think more strategically to have some of these clusters of researchers that their work would be tied in with each other that there could be some collaborations going on, right?

LM: You know, something, which I would like to see more of ... and I think there will be, is maybe more crosstalk and more cross-departmental collaborations, and sort of getting away from us versus them type of things. I know in biology they've recruited some new and really good people, and so it's sort of more cross-departmental collaborations. I think it's good for us, especially as new faculty. I mean, resources become pretty limited and pretty scarce, but I think if we can help each other out a little, it becomes better.

I am probably at that point maybe now within the next year, where I'm really going to start pursuing more collaborations because as a new faculty, you want to spend at least the first couple of years, few years, just focusing on and getting your own research program up and running. Then, at some point, it becomes beneficial to have these collaborations.

CD: Yeah, for sure. I just wanted to thank you so much for coming in today and speaking about your work.

LM: Thanks. Thanks for the invitation. It's been a pleasure.

CD: Thanks.

[Wrap-up music fades in]

CD: We shared a bit of a laugh at the end there. I was getting way to personal about my pain in labor, which occurred 13 years ago, but still fairly vivid in my mind. I am sure what a world-class researcher wants to do is sit and listen to my birth story. But that's how I roll. And this giggly display, it just might represent my giddiness at completing 12 podcasts in 12 months over the course of 2017!

A special thanks to my fantastic guest, Loren Martin, who actually made talking about pain, fun.

Thank you to the Office of the Vice-Principal, Research, and thank you to all my guests over the course of this year for helping me to make this project a reality.

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Thank you.

[Wrap-up music fades out]