Science Will Win Podcast
Season 5
Episode 2 Transcript

Amanda Lopez: (00:05)
We get a lot of unsolicited advice which kind of makes you cringe 'cause it's like I've had eczema all my life. Do you think a bar of moisturized soap is gonna help me? Like you don't think I've tried this?

Ron Gamble: (00:22)
Welcome back to another episode of Science Will Win. I'm your co-host Dr. Ron Gamble. I'm a theoretical astrophysicist STEM educator and science communicator and I'm joined here by my partner in crime and in life,

Raven Baxter: (00:36)
Dr. Raven Baxter, also known as Raven, the Science Maven. I'm a molecular biologist and science educator.

Ron Gamble: (00:44)
We've partnered with Pfizer in a whole production team to learn everything we can about some of the most exciting medical technologies today. Raven and I will be taking turns, leading episodes this season and diving deep into cutting edge innovations in disease treatment and prevention. For this episode, I'm leading.

Raven Baxter: (01:09)
Alright, Ron, what you got for me?

Ron Gamble: (01:12)
Well, as you know, I'm a person who lives with chronic illness. For me that means living with a hidden illness, right? Dysautonomia? Mm-hmm Dysautonomia is a disease that impacts millions of people all over the world in various forms. The symptoms can vary from person to person, but essentially it's a malfunction of your autonomic nervous system that controls all of the things that you don't have to think about. Like your heart rate and pain responses, which can feel like your nervous system is having a flu. And that's just the tip of the iceberg. So that feeling that you've tried everything, it's familiar and I sympathize with anyone facing challenges today, finding treatment options and navigating their healthcare, including someone you've heard at the start of this episode. You'll hear more from her later.

Raven Baxter: (02:02)
I understand the frustration and stress of it all, but I've done a lot of research into ways we can improve the standard of care for chronic illnesses and I'm looking forward to talking more about that today.

Ron Gamble: (02:13)

That's exactly where we're going. But before we get into the science of it all, I'd like to start with an analogy. Imagine this, it's the seventh inning and the bases are loaded. The smell of popcorn and hot dogs wafts through the air. Are you getting the picture here?

Raven Baxter: (02:31)
It sounds like you've transported us to... a baseball game.

Ron Gamble: (02:40)
It's definitely a baseball game. It's America's pastime. You know, go Yankees.

Raven Baxter: (02:45)
Go sports!

Ron Gamble: (02:47)
I promise I'm going somewhere with this, right? So just hear me out. In baseball, the best shortstop in the game can catch any ball that comes their way, but they can only catch the balls that come between second and third base. Now, imagine if you had the shortstop with an equally good third baseman and an equally good first baseman, right? Even better. What if you had all of these abilities in one superhuman baseball player like a Jackie Robinson? Now that one person could catch all the balls that are hit.

Raven Baxter: (03:22)
And that sounds great, but also impossible!

Ron Gamble: (03:26)
Maybe for baseball. But what if I told you that scientists today are working on a therapeutic modality, A single entity that can harness a similar power and engage multiple distinct targets. Something that can interact with multiple disease causing cellular or molecular pathways. In other words, they can catch all the baseballs, they're called multi-specific antibodies. And I'm gonna tell you all about them. How they work, how they're made, and their potential to treat diseases in unprecedented ways. We're looking toward a new frontier of highly specialized treatment options in medicine and hopefully a better standard of care for patients.

Raven Baxter: (04:08)
I like the sound of that. I mean I of all people love antibodies. I mean, I did make that whole song about “antibodyodys” and I'm sure many Science Will Win listeners are familiar with the purpose that antibodies serve fighting off foreign substances or antigens in the body. Multi-specific antibodies are really cool because they take into account all of the knowledge that we've acquired about how antibodies work and then we build new types of antibodies and incorporate different parts of antibodies to generate something that is like a tool that can tackle many different things that once, just like how you explained that baseball analogy. Ron, I'm really excited for you to learn about multi-specific antibodies, because it's a wild world out there and there's so much to learn.

Ron Gamble: (04:57)
I'm glad you said that. Okay. I did some light reading about these antibodies. First off, the antibodies we're talking about are laboratory engineered therapeutic antibodies. So, not the ones naturally produced by your immune system. Like you said, scientists capitalized on knowledge and research of the body's natural immune process to create laboratory made molecules engineered to target certain molecular and cellular processes that are dysregulated by disease.

Raven Baxter: (05:29)
Look at you! I see you're out of your physics bag. You're getting into your molecular biology bag.

Ron Gamble: (05:36)
I am a scientist! So, like regular antibodies, they inhibit a dysregulated function by binding to a target or a protein involved in the dysregulation. Multi-specific refers to the ability of one antibody to bind to or engage multiple targets. In other words, they do more than one thing. That's why they're referred to as multi-specific or multifunctional. I also think it's important to understand what antibodies look like. And I know you know a lot about that, right?

Raven Baxter: (06:10)
Oh my gosh, yes. Okay, so antibody, yada yada yadies... Just kidding. Antibodies are really cool if we talk about antibody structure. You ever done the YMCA dance? Ron? Yeah, Okay. And like how do you spell "Y" with your body when you're doing the "Y"part,

Ron Gamble: (06:29)
You put your hands up.

Raven Baxter: (06:30)
Put your hands up. Okay, imagine you're standing and doing that dance. You're gonna keep your legs together and your hands up to make the letter "Y." That's how antibodies look. And so, you have the arms which are literally like your arms, and then you have the stem, which is your feet together, and they're "Y" shaped proteins. They have heavy and light chains. Those are made up of amino acids. And the reason why they're called chains is because amino acids are strung together to create proteins. And so, the heavy and light chains comprise the arms that form the binding sites. And then the stems are also heavy chains, and they're connected to the arms by a bond that brings everything together. So, you can think of like your chest area as that bond.

Ron Gamble: (07:18)
Right? So that's what I imagined when I was reading through this multi-specific antibodies utilize this very structure that you described in a pretty cool way. And I didn't even know this... to learn more, we spoke to a scientist at Pfizer to break it all down.

Laird Bloom: (07:35)
My name is Laird Bloom. I'm a senior director in the biomedicine design department at Pfizer.

Ron Gamble: (07:41)
Laird leads a group that designs and engineers antibodies for therapeutic use. He manages scientists in the lab to help optimize antibody discovery and development. His work spans many departments at Pfizer including inflammation, immunology, oncology, cardiovascular and more.

Raven Baxter: (07:59)
I can't wait to hear from Laird. And isn't it cool how integral antibody engineering is to almost every field, every type of disease --- antibodies are extremely important to our bodily functions.

Ron Gamble: (08:11)
I thought it was also cool. So, we asked him to describe how certain multi-specific antibodies are made.

Laird Bloom: (08:17)
What we do with a bispecific is we've engineered the “Y” so that two different half “Y”s can come together and put two different arms onto the same stem. Essentially with a Tri- Specific we can graft on a third arm in several different places. We can stick it on the top of one of the ends of the “Y”, we can stick it at the bottom of the end of the stalk. And a big part of the field now is trying to figure out what the best way to attach the different pieces would be. But essentially, it's all kind of built around that core “Y”-shaped antibody.

Raven Baxter: (08:46)
Okay, first of all, I love bispecific antibodies. I even have several, I bought several of the same kind bispecific antibody hair clips. They're really cute. Okay. Like first of all, who looked at a regular antibody and said cute, but what if it were a multitasker? Okay, with main character energy, I feel like bispecific antibodies are like the double-sided tape of immunology and one end grabs one thing and then the other end grabs the other thing and then suddenly your immune system is running like a, it's like a buddy cop movie where it's like the Rock and Kevin Hart come together to fight crime.

Ron Gamble: (09:31)
Such a nerd. I love it! To put you on blast, you got more than just the hair clips. Like you have the antibody like stuffed animals around your office in the house, like they're everywhere.

Raven Baxter: (09:41)
Now why you gotta tell 'em all my business?

Ron Gamble: (09:44)
I mean I think that's cool, but also like this is, this is what's going on.

Raven Baxter: (09:49)
Yes. The woman who made the literal song about antibodies has a lot of antibody things around the house. Yes. You got me!

Ron Gamble: (09:58)
Alright Raven, I do wanna share more of what I learned. I feel like I'm impressing you...A regular therapeutic antibody or monoclonal antibody typically has identical protein chains on its arms that form its binding regions, multi-specific antibodies, add variety. Those bi-specifics you heard layered mention bring together two distinct antibody halves to make a whole, and can engage with two different targets as a result. Then, tri-specifics go further and add on an additional arm, meaning they can engage three targets. Scientists are even working on tetra-specifics.

Raven Baxter: (10:38)
I'm so impressed. Yes, you're right. But Ron, like I'm wondering how did scientists even think of this? Did you find out why there was a decision to combine antibody components in the first place?

Ron Gamble: (10:51)
I did. I got you. You know, I'm like a science history buff. The idea of drug combination in general has existed for decades, as you know. But the notion of a two in one antibody, that didn't come up until the 1960s, and as Laird explains, it started in the oncology space.

Laird Bloom: (11:08)
There was a clear case where a combination of drugs would not do the job. You had to have the two pieces tied together if you were gonna kill a tumor cell with the immune system. And so, I think the necessity of that combination was a big factor in convincing the community that bi-specifics could be made to work. And so, I think that sort of snowballed from there.

Ron Gamble: (11:27)
While the push for multi-specific antibodies began in oncology, scientists have long dreamed of using them to treat other diseases. To find out more, we called up another Pfizer scientist who has been invested in this technology for years.

Tom Wynn: (11:42)
I'm Tom Wynn, I am vice president of discovery in Pfizer's inflammation immunology research unit, which focuses on developing innovative treatments for autoimmune inflammatory and fibrotic diseases. You can think about inflammatory bowel disease as one area where we have multiple monoclonal antibodies that are used to treat patients. Maybe 20 to 30% of patients may benefit from a specific therapeutic antibody. It blocks their key driver of disease. There are many different drivers of disease and disease is heterogeneous, meaning this patient's disease doesn't necessarily look exactly like this other patient's disease. So, they each respond to different therapeutics in different ways. What we're trying to do with multi-functionals is attack two or three core drivers of disease. So, the hope is that we'll reduce patients' experience from switching from one medicine to another so that they stay on a single highly effective medicine much longer because these therapeutics may target several different mechanisms of disease in one molecule.

Raven Baxter: (12:48)
You know, Ron, we both struggle with chronic illnesses and one of the things that the patient community is really yearning for are more treatment options and just to have a better quality of life in general. And so, if we have therapeutics that are this specific and adaptable, I think that could also translate into great solutions for patients.

Ron Gamble: (13:10)
Right? So back in the earlier days of Laird and Tom's careers, this all seemed like a far-off possibility. Sure, the idea of multi-specific antibodies sounded great, but scientists needed to experiment and figure out if it was actually possible. Here's how Laird explained his team's role in that.

Laird Bloom: (13:29)
The first encounter was with a cancer project, and the idea was that a multi-specific antibody could grab onto a cancer cell on one end and grab onto an immune system cell that could kill the cancer cell with the other and bring those two together and actually kill the tumor very specifically. And this was an idea that had been kicking around for a while. We partnered with a company that was a specialist in doing that, and we had the opportunity to build a bispecific antibody that was directed against tumors and see that firsthand. That was quite an exciting experience.

Ron Gamble: (14:00)
And the technology has come a long way, allowing scientists to envision advanced capabilities beyond bi-specific antibodies.

Laird Bloom: (14:09)
Once people started to see that this could be done, then they got kind of very creative about what else you could do. I mentioned trying to bring two different cells together, bring the cancer cell together with a cell that can kill that cancer cell that's for a bispecific. Adding on a third and a fourth element might be adding some specificity to that cancer cell. We know that as you look deeply into various disease processes, there are lots of subtypes of cells within a disease process and most of the time, only one small subset is the cell type you really wanna target. And the rest you'd rather leave alone. And as we get better and better at making multi-specifics possibility of honing in specifically on those cell types that we want to hit and avoid any others, that's very exciting to think we can do that better. That's true for cancer, that's true for autoimmunity as well. Think about two-factor authentication when you're trying to get onto a website, you have your pin and you have your password and you can't get there unless you have both pin and password. That's the kind of thing we're doing with a tri-specific to make sure we have just the right cell type that we're trying to kill has the pin and the password essentially on it.

Raven Baxter: (15:10)
Yeah, I mean it's just so fascinating to hear about all this work because there is so much research that has to be done to even make this possible. Like, for example, what happens when

you switch off something, right? What is the impact of that? Or is there a downstream effect to joining two things together? There is just so much knowledge that you need to have as a foundation for this kind of work. And so, I am just blown away.

Ron Gamble: (15:36)
Yeah, they're trying to engineer a like super antibody here, right? So, they're trying to get something that can have some hyper-specific set of marching orders and allow them to identify specific cell types, you know, while ignoring others. So, you know, that's very difficult, but that's where the technology is going, and I think they're gonna make smarter more tactical antibodies.

Raven Baxter: (16:00)
So, Ron, I wanna know how close we are to getting these multi-specific antibodies into humans

Ron Gamble: (16:07)
Today, the FDA has approved over a dozen bispecific antibodies to treat cancer and the work on tri-specific and even tetra-specific antibodies continues. But this momentum isn't limited to oncology. Scientists hope to bring multi-specifics to the inflammatory and immunology space.

Raven Baxter: (16:26)
This is so exciting. I am intrigued. I wanna know what will it take to get drugs that use multi-specific antibodies into the lives of more patients, particularly for diseases that have never been treated in this way before?

Ron Gamble: (16:40)
Right. So, I did more reading and more investigating. At Pfizer, the process of scaling up and bringing a multi-specific antibody drug through to regulatory approval begins with finding drug targets, which I know you know about. That's the job of Tom Wynn, who we heard from before. He and his team start by thinking a lot about the disease process and patient needs.

Tom Wynn: (17:05)
We think of an important disease that has unmet medical need. Can we look at existing human data, both foundational data from patients that have active disease and more importantly, can we look at data from various clinical trials and determine, okay, what pathways are effectively served by a given therapeutic and what pathways and drivers of disease are still not met? Can we find targets in those other pathways that we combine with that initial therapeutic and build a better therapeutic that has multi-functionality? So that's sort of the stepwise process. Identify the disease with unmet medical need, Understand the pathogenesis of the disease, understand the function of current treatments, and can we build something better with multi-functionality.

Ron Gamble: (17:51)
And by something better, he means

Tom Wynn: (17:53)
Really, the key word is homeostasis.

Ron Gamble: (17:55)
Quick pause here for a little vocab check: homeostasis. Raven, you got this one?

Raven Baxter: (18:01)
I do. Homeostasis is the regulation and balance of our body's internal systems.

Ron Gamble: (18:07)
That's right. Now, back to Tom.

Tom Wynn: (18:10)
You really need to target those different cell types and the mechanisms they're introducing that drive disease. So, we'd like to get rid of all of them, all of those pathways that lead to inflammation from the different cell types. How can we best do that with multi-functionals that target different aspects of those different cell types? How do we take it from a disease state, back to that homeostatic healthy setpoint.

Raven Baxter: (18:32)
This is drug discovery research, and it can be very intense, especially you think about how many different cell types, and then different drugs, different targets, different mechanisms, and then it becomes like a factorial of testing things. I used to work in high throughput drug discovery, and we would test tens of thousands of combinations of applications of drugs at a time. And so, you have to understand like these sound like some really big questions to answer -- because they are.

Ron Gamble: (19:03)
They are really big questions. Laird says that in his opinion, Tom's job is the hardest part of the whole process. But with the advancement of systems immunology and AI technology, scientists have an increased ability to interpret large data sets and build mathematical models. Something that I know about. These models reveal important properties for the drug, like how much of it you need to have or how tightly and quickly it needs to bind. They can also use machine learning to predict how well a molecule will bind and how viscous it will be. So, listeners may remember this part of the process from last season's episode on AI software. All of this helps scientists discern how effective a drug will be and how to dose it.

Raven Baxter: (19:52)
Alright, Ron, I see you're well on your learning journey.

Ron Gamble: (19:54)
Right?

Raven Baxter: (19:55)
Okay. Did you figure out how they turn an antibody into an actual drug in terms of dosing?

Ron Gamble: (20:02)
I read that too. So that's when we really get into the development process. Here's a quick overview. First, scientists take all the data they've gathered on disease targets and enter the lab to start generating antibodies.

Laird Bloom: (20:17)
So, we have methods in a test tube where we can search a collection of 10 billion or more antibodies and find the ones that will bind to that target. So that's our initial phase is getting antibodies either from an immunized animal or from our human libraries.

Ron Gamble: (20:31)
Scientists then test those antibodies to see how well they work. Once that's done and they've assembled the ideal antibody molecule, the next step is to test them in cell systems. They look to see if these molecules can perform predictably and reliably in their intended context. And if all goes to plan,

Laird Bloom: (20:51)
We can concentrate it up, we can store it for a long time, it doesn't degrade. We can put it into a, a small volume that might be appropriate for a syringe to administer to a patient. And, uh, it fits the numerical criteria that our, our modelers came up with. That said, if you know, we have to bind this tightly and last this long to be effective, if it meets those criteria, then we feel we're ready to go.

Raven Baxter: (21:14)
But clinical trials don't happen immediately.

Ron Gamble: (21:18)
Right. Every compound has to go through extensive safety testing before scientists can get permission to do clinical tests. I think that's another thing you know about.

Raven Baxter: (21:28)
It's a lengthy process, but if things go well, if science wins...sorry, I couldn't help myself.

Ron Gamble: (21:37)
Then it's really time to part-ay!

Laird Bloom: (21:40)
If we can put all the pieces together and, and it does everything we want, then it's a big celebration. You know, we'll have a town hall meeting, we'll have cake...

Tom Wynn: (21:48)
But the big celebration usually was when we get that therapeutic into the first patient. Drug development is very challenging. There's lots of things that make it complicated and so successes need to be celebrated. But most importantly it's successes in patients.

Raven Baxter: (22:05)
Yeah, I mean, like, I've been there before, and it is so rewarding. Drug discovery is really intense. I mean, there can be situations where you never really quite know what's gonna work until it works. And then it's like you found a needle in a haystack of possibilities. And so, imagine finding that needle in a haystack after months and months, even years of work, you and your team. Endless hours in the lab. Boom, we got it. Like, yeah! We're popping champagne, dude.

Ron Gamble: (22:33)
It's lit! The same thing happens in physics. It's you celebrate the wins, you pop the champagne, and then you get back to work because there's always more science to do. Okay, Raven, well I hope you liked my little information flex, but I think it's time to shift towards the patient perspective on all of this.

Raven Baxter: (22:54)
I loved the information flex. You did a really great job, Ron, and I'm really excited to get into the patient perspective.

Ron Gamble: (23:02)
I am too! Now this patient is someone who has participated in clinical trials and has firsthand experience navigating treatment options. They're also someone whose life could potentially be directly impacted by multi-specific antibodies.

Raven Baxter: (23:19)
Ooh, tell me everything!

Ron Gamble: (23:23)
I will. Well, I'd like you to meet my new friend, Amanda.

Amanda Lopez: (23:28)
My name is Amanda Lopez, and I live in Texas.

Ron Gamble: (23:31)
I really connected with Amanda on so many levels. We both deal with a chronic condition. We were raised by single parents, and we grew up with our Spanish speaking Abuelitas.

Raven Baxter: (23:42)
Wow. I can't believe y'all have so much in common.

Ron Gamble: (23:45)
So Raven, it's a very small world. Right? When we sat down to talk, it felt like we were having a real patient-to-patient conversation. The condition Amanda deals with is eczema, also known as atopic dermatitis. Pfizer is actually testing multi-specific antibodies on this disease, which is potentially great news for Amanda who has lived with eczema for almost her entire life.

Raven Baxter: (24:10)
Yes, eczema. I have eczema. Um, I'm actually sitting here literally with my ointment on hand and it's just, whew. It can be very disturbing to your quality of life.

Ron Gamble: (24:25)
Absolutely. I've seen this firsthand with you and my family members who struggle with eczema to better understand the potential for multi-specific antibodies to improve the standard of care of eczema. We're also going to hear from a scientist working to cure it.

Emma Guttman: (24:43)
My name is Emma Guttman and I'm the chair of the Department of Dermatology at the Icahn School of Medicine at Mountain Sinai.

Ron Gamble: (24:50)
Dr. Guttman co-founded the International Eczema Council, the largest organization for key opinion leaders in Eczema. Before hearing more from our patient, Amanda, we asked Dr. Guttman to give us a primer on eczema from the clinical perspective, an atopic dermatitis 1 0 1 class, if you will.

Emma Guttman: (25:09)
So atopic dermatitis or eczema, first of all, is the most common, uh, inflammatory disease we have in humans. It involves about 7% of the adults in the United States, a large number and up to 15% of the children. The majority of patients will not have severe disease. They will be mild. But we have a moderate to severe atopic dermatitis in about a third of the patients. And it's important to differentiate these because in patients with mild disease, we typically can apply creams and that will be enough. Whereas the people that have moderate to severe disease most of the time will need to have a either an oral or a biologic. So basically systemic treatment.

Ron Gamble: (25:53)
The implications of a severe case of eczema can be quite serious.

Emma Guttman: (25:57)
When you have severe disease, many times you'll have infections, you'll have open wounds, and also patients with severe disease will have more systemic inflammation. And now we know that having inflammation in the circulation for a long time is not good for you. Later on in life, it predispose you to have cardiovascular disease and so on.

Ron Gamble: (26:23)
And it's not just the physical effects Raven. symptoms of eczema can disrupt patients' lives in all sorts of ways,

Emma Guttman: (26:29)

Starting from school. They cannot focus, they don't sleep well at night. They itch, the itch is, uh, waking them in the middle of the night, then later on university, they don't perform well. And that tells us how important it is to bring them closer to a cure or disease control.

Raven Baxter: (26:48)
So, does a cure feel like a possibility within reach? And how close are we?

Ron Gamble: (26:54)
I would say it's hard to say for certain. Eczema is quite complicated to treat as we know.

Emma Guttman: (27:00)
There is no one size fits all. It's a very heterogeneous disease that has multiple facets. And in order to treat it successfully, we can either personalize treatment to different patients or different aspects or have treatments that target more than one cytokine pathway that will be able to, uh, treat more holistically more of the patient populations in eczema.

Ron Gamble: (27:25)
Fun fact, cytokines are proteins that act like chemical messengers, creating different pathways or networks of signaling interactions between cells. These pathways can be linked to cellular activities like immune response.

Emma Guttman: (27:38)
Sometimes we see in the more severe patients, they may have responded initially, well, after a while they don't respond, and we need to either increase the dose or switch it around.

Ron Gamble: (27:49)
What we do know is that major progress is being made, particularly in treatment technology. Multi-specific antibodies are a part of that.

Emma Guttman: (27:58)
The previous immune suppressants were targeting all the pathways, all the cells. You'll have a lot of off target effects, right? You'll be, uh, having side effects. Whereas when you target specifically, you work in a more meaningful way, sometimes, sometimes even better. So, we are fortunate now that we live in an era that we understand much more of atopic dermatitis and its pathways. And, uh, in recent years, we've, we've seen approval of biologic treatments that target different cytokines in the disease.

Raven Baxter: (28:31)
It sounds like this all goes back to the power of targeting multiple disease pathways, like what Tom and Laird spoke about earlier.

Ron Gamble: (28:40)
Exactly.

Emma Guttman: (28:41)
I view that as the future of atopic dermatitis treatment, because that will allow us to target more of the patients successfully. Since each one of them may show more of one pathway versus the others. I think our, our near-term goal is to treat eczema safely and effectively. But in the future, ideally I would also like to have a treatment that we give it for some time, then we stop it, and ideally either the disease doesn't come back at all or maybe we need to give the treatment every six months, every year, very infrequently.

Ron Gamble: (29:18)
Dr. Guttman says that developing a cure for eczema requires collaboration from academics, pharmaceutical companies, and even the people who have the condition.

Emma Guttman: (29:28)
So, we need the engagement of the community, including the patient community.

Raven Baxter: (29:32)
Hmm. A patient voice. Yes. Okay.

Ron Gamble: (29:36)
Which brings us back to my conversation with Amanda, who I should add is also an ambassador for the National Eczema Association.

Raven Baxter: (29:46)
Well, what are you waiting for? Let's hear from her.

Ron Gamble: (29:49)
Well, first I was curious about how having eczema affected Amanda's childhood.

Amanda Lopez: (29:55)
I had the symptoms of eczema when I was born, but I wasn't diagnosed with it till like, as a toddler. There's different kinds of eczema. Wow. I've had more than one. When my parents, when their divorce was official, um, we ended up having custody with my mother and you know, for the most part she raised us. But I am a middle child.

Ronald Gamble:
Me too.

Amanda Lopez:
And the only female, and I have a bunch of brothers. So I would see my mom like really have to struggle to take mm-hmm . Take care of me because I was the only one that was sick. And so I, in a sense, I felt like even though I was getting my mother's attention in that way, I didn't want my mother's attention that way because she was so stressed out. Like, you know, taking me to the doctor and then making sure I had my medicines and, you know, I saw the toll that it took on her.

Amanda Lopez: (30:47)
And so I told her, I was like, mom, just let you can let me. And that's what I was telling you. I I started giving myself shots at eight because I was like, I'll just do it. You know, like I'll put my own lotions on, I I'll give myself my own bleach baths and whatever, oatmeal baths. And so, she basically just bought what I needed or what I asked for and I took it from there. You know, like I, I didn't wanna see my mom stressed out like that. And so, I really feel for, you know, the parents that have children that have like, especially the severe kind and um, it's, it's very heartbreaking. And I remember like, we were at a grocery store standing in line, and I had a flare and the person behind us was waiting and just, just like staring at both my mom and I and basically like judging us. Like, you know, 'cause you, you look at this child's that looks sick and the first thing people are thinking or like, what is a mom doing about it? You know? Right. And it's so I hated that for my mom. I really did. It was so sad.

Ron Gamble: (31:55)
I think that further kind of compounds the journey, right? It's, you know, what you were doing and you were trying to help out your mom. I grew up in a single parent home too. Anybody who knows that life like you, you know, you're trying to help out your parents. And I think that is like, that's really commendable, right?

Amanda Lopez: (32:12)
That was more of like, I wanted to take control of my condition and be able to manage it. And again, I think that kind of just carried over into adulthood, 'cause now it's like I do so much advocacy, and I am trying to raise, you know, education about eczema and the treatments and also just having that support in the community. 'Cause I didn't have that. Right.

Raven Baxter: (32:38)
That story is little heartbreaking. First of all, that's a lot of pressure for a child to take on. And imagine growing up as a child, knowing that you're being judged and knowing that it's because of something that you barely have any control over.

Ron Gamble: (32:52)
So, it's very interesting to see how eczema impacted Amanda's family. Mm-hmm. You know, even though she was the only one with it, it's, I mean, I can vibe with having something you can't control. You're wondering, “well why me?” You're trying to figure out what works, what doesn't work, you know? So, it's something that is definitely hard to deal with for sure. But now in her forties, Amanda described how she still has to be diligent about keeping on top of her eczema symptoms.

Amanda Lopez: (33:20)
It always carries over to adulthood, right? Like when you're a child, some of those traumas carry over if they're not addressed. I think mostly for me it's like the coverup, you know, making sure that I don't have somebody to say something or comment or gimme some unsolicited advice. Like, “oh, try this cream”. It, it calls for a lot of cumbersome type of daily routine treatments and

regimens. 'Cause if you don't stay on top of it, yeah, it can go from bad to worse and mm-hmm. Super quickly in a matter of like days. And that, I think that's the most difficult part, is the management. Especially if you don't know what your culprits are.

Ron Gamble: (34:02)
When Amanda references culprits, she's talking about things that trigger her eczema symptoms.

Amanda Lopez: (34:08)
At one time my culprits were like sugar. So, I stayed away from sugar for a while and then your body's always changing, right? So, when I got into my adulthood, when I got married and went through a divorce, that all increased my stress level and anxiety. And so that's when I started noticing more flares. So, then I started to say, okay, like this is my culprit distress. You know, it was just a vicious cycle of that itch scratch cycle. Rest should be sacred, sleep should be sacred. Especially people who have like chronic conditions that keep 'em up at night, such as eczema. But as soon as I get to bed and I'm like, okay, it's time to wind down. And I'm just like, “oh, why am I so itchy? Why am I so scratchy?” Yeah. And then I wake up and it's just like I've tore my skin up and then there's blood on the sheet. Oh wow. So it, it can be pretty shocking when you wake up sometimes. 'cause you see the aftermath and you're like, “wow, I, I really tore up my skin.” And so you feel guilty because you know, you're trying to listen to your body, listen to your skin, listen to what it's asking for. I have this mental battle, like what am I doing to betray my body?

Raven Baxter: (35:18)
There's a lot of emotions that can come with having such a potentially visible skin condition. I cannot imagine how stressful it is though, just managing even the emotions tied to this.

Ron Gamble: (35:31)
Eczema is an inflammatory condition and stress does cause inflammation. So, you can kind of see the endless back and forth cycle, right? Eczema symptoms and triggers can fluctuate throughout a patient's life. And coming to terms with that has been frustrating for Amanda. She recalled receiving results from an allergy test a few months back, doctors told her that many of the foods she'd spent her entire life enjoying had become allergens for her.

Amanda Lopez: (35:57)
Like, I can't eat spinach. Like, that's all I eat. Or like veggies, here's, that's crazy here I'm trying to be like healthy. And they're like, no, no, you can't have that. I was like, you're taking my life away from me. That's what I eat.

Ron Gamble: (36:11)
W-T-F.

Amanda Lopez: (36:12)
Beans, I'm Hispanic. Um, right. Nuts, seeds, all kinds, you know, that was a very low moment for me. . Wow. And so I know. And so when I went to the grocery store I cried a little bit 'cause I

was like, okay, can't have that. Can't have that, can't have that. And then so when I started filling my basket, I looked at my basket and I said, okay, let me just at least be grateful for the things that I can have.

Ron Gamble: (36:35)
Allergies or food triggers aren't the single drivers of eczema to be clear. But this has been a notable part of Amanda's eczema experience.

Raven Baxter: (36:45)
Listen, I'm literally going through this right now. I had a severe anaphylactic allergic reaction to a fiber supplement and I said, this is so crazy. So I went to an allergist, I did a skin test, extensive skin prick test, and they tested for a lot of food allergies, and I found out that I am allergic to most food. It was actually mind-boggling. And now that I am very aware of that, now I'm using that as a tool to avoid unnecessary inflammation. And I'm taking into account like, okay, if I eat this food, is my skin gonna flare up? Like, what are the consequences of introducing new inflammation into my body?

Ron Gamble: (37:34)
I mean, I know exactly what you're talking about. I all of a sudden became allergic to nuts and seeds in my thirties. Like, where did this come from? But like, it's again, like your body doing things behind the scenes. Yeah. And we're trying to figure that out. It's very frustrating.

Raven Baxter: (37:49)
I really feel for her though, because it's kind of like a grieving process. All of the foods that you enjoy, right? They taste good. It fills a special place in your heart. Especially if it's something that, you know, is like a family dish. Right? And then imagine being allergic to a lot of those ingredients. I mean, I feel for Amanda, I get it. Now, I know her eczema journey is still developing, but has she found anything that works? Like, what does treatment look like for her?

Ron Gamble: (38:23)
Well,

Amanda Lopez: (38:24)
This is the reason why I'm part of advocacy, is because I'm 43 years old and I still struggle with finding effective treatment options. As many great technology and biologics that are out there that work for others have yet to work for me. So I'm still searching, I'm still on the lookout. And, um, in the meantime, I just try to stick with simple treatments that I know that my body is able to handle and likes at the time.

Raven Baxter: (38:56)
Wow. So, no treatment has worked.

Ron Gamble: (38:59)

Right. Amanda told me that she's pretty much done it all. Light therapy, acupuncture, steroid creams, oral medications, a clinical trial – nothing has made a major difference in improving her physical symptoms.

Amanda Lopez: (39:13)
Like I said, I'm 43 years old. Right. . So, um, I, you know, and I'm still patiently waiting, I'm still patiently waiting, but I, I, I know we're on the right track

Ron Gamble: (39:22)
And that's actually why I was so eager to talk with Amanda about multi-specific antibodies and the potential she sees in the future of eczema care.

Amanda Lopez: (39:33)
I have faith in it and I know something will come up. Whether it be that kind of treatment approach with all the, the science coming together. This is a really exciting time because this is like the time that I, I, I don't ever remember that there was so many different treatments coming down the pipeline. Wow. It was always just, oh, use this cream or use this, you know, steroid and use this antibiotic and then that was it. Everything I've tried has been such a fail, but you know, again, all I can do is try.

Raven Baxter: (40:11)
Well Ron, I know it will still be some time before multi-specific antibody drugs could be available for eczema patients. But I must say, I'm feeling optimistic, and it sounds like Amanda is too.

Ron Gamble: (40:23)
I'd say so the future remains open-ended for patients like Amanda and that keeps scientists like Tom Wynn going,

Tom Wynn: (40:32)
I'm just super excited about getting these into patients, testing them, then seeing whether or not this hypothesis of a targeting multiple pathways and drivers of disease really pays off for patients. I think we're just at the beginning.

Raven Baxter: (40:51)
Ron, like I gotta say, you taught me a lot today and my journey with eczema is still very new and I learned so much about the condition and I'm especially excited that there are developments on the frontier that gives me a lot of hope. And I'm sure it gives a lot of other eczema sufferers hope as well.

Ron Gamble: (41:11)
Well, that's all for today's episode. I'm glad you enjoyed my little, our, our fun facts and all the science. So, Raven, how'd I do?

Raven Baxter: (41:19)

You did amazing, Ron, I'm so proud of you. I hope you enjoyed it.

Ron Gamble: (41:23)
It was definitely an information flex.

Raven Baxter: (41:26)
Yes, I'm looking forward to the next time you do this. But speaking of next time, next week, I am taking the reins again to drop some knowledge on a modality that is near and dear to my heart. Molecular glues! The rest of the season has a lot in store, new labs, new stories from researchers and patients, and new breakthroughs.

Ron Gamble: (41:47)
Can't wait. We'll see y'all then. Science Will Win is produced by Acast Creative studios and hosted by me, Dr. Ron Gamble.

Raven Baxter: (42:00)
And me! Dr. Raven Baxter. Please take a minute to rate, review, and follow. Science Will Win wherever you get your podcasts. It helps new listeners to find the show.

Ron Gamble: (42:11)
To learn more about what we talked about this week, you can head over to Pfizer social media accounts. We'll also drop a link down in the show notes. Special thanks to all of our guests and the Pfizer research and development teams, and thank you for listening.