Symposium 2025 podcast

Korey: Welcome everybody to the 2025 Research Symposium by Global Brands for Eczema Research. Thank you to today's sponsors: the Pierre Fabre Eczema Foundation, Novartis, Codex Labs, and Kenview for making the event possible.

Our mission is to reduce suffering for children impacted by moderate to severe eczema through breakthroughs and research and family support. And our vision is a future without childhood eczema, We fund research studies on eczema prevention, and many of our grantees are talking about their funded projects today so we're super proud of that.

So with that, I am happy to introduce our first speaker today, Dr. Carina Venter. She's an allergy specialist, dietician and Associate Professor of Pediatrics at University of Colorado, Denver School of Medicine. She was in the first class of GPER grantees in 2024, and she'll be talking about her really interesting research on the relationship between dietary fat intake and skin lipidomics in children with eczema. Basically telling us if we eat a lot more fat in our diet, does that somehow also improve these beneficial fats that are in the skin? It's never really been studied. And so we're really excited to have Dr. Venter present her novel research today.

Dr. Venter: thank you for that introduction. And thank use so much, for funding my research. The question really in the back of my mind is can we feed the skin?

Today we will look at the association between nutrition and eczema, and can dietary changes lead to improvement in eczema.

Sphingolipids is a word for, really, the skin barrier fatty acids, a special type of fat that makes up a big part of the skin's outer layer. And so the skin lipids of children with eczema is clearly is disturbed. So can we bring that skin layer back to balance by changing the diet?

When we talk about healthy eating, we always hear, perhaps, that saturated fats are not good for the heart, but when it comes to the skin, the skin needs all these fats to be healthy. It needs the saturated fats, the full fat milk. It needs the mono and polyunsaturated fats: olive oil and the fats that we get in nuts and seeds. And the Omega-3 fatty acids, you know, from fatty fish and in most plant oils. And that's another important message that we need to get across, is that really

when it comes to feeding the skin, there's not one particular good or baddy. The skin needs all kinds of fats to be healthy. And, our data beautifully supports that.

But, I have to admit that I didn't actually know that you can eat sphingolipids in food. And, when I asked the question about whether eating these things would actually affect the skin, nobody knew. And I couldn't even find animal data to look at that. Today, I am going to show you that we have answered those questions to a great extent.

So to study the association between nutrition and eczema, we asked 20 children between one and ten to collect a skin sample from their skin using sticky tape. 10 children with eczema and 10 children had no eczema. And we also measured food intake over the past month, to see what the children's diets look like. It's a small sample, so we didn't find any significant differences in fat intake between children with and without eczema. But the children with eczema for every single fat we studied, had lower intake.

And so then when we actually compared what was going on in the skin, the children with eczema handled fatty acids completely differently and had very strong positive correlations between eating any fat and restoring the skin barrier. It's like the skin of the child with eczema is crying out for fat to repair like a sponge. So you give a fat to a child with eczema and you drive the fats in the skin that's going to repair the skin barrier.

But in the children without eczema, there wasn't this desperate, positive effect seen between eating fat and driving the fats in the skin that produces barrier repair.

And so we want fats in the diets of children with eczema. Focus on olive oil, avocado, nut butters. Nut butters, it's not just there for preventing food allergy, they have a very profound effect. on the gut microbiome. Nut butters increase bifidobacteria in the gut, but nut butters also give us the fat that helps us to restore the skin barrier.

What I say to the families is: feed this child full fat yogurts and full fat milks, they do such a great job of feeding the skin barrier.

We talk about beef, egg, liver, not everybody's cup of tea, but it is one of the important foods when it comes to the skin barrier.

We talk a lot about Omega-3 fatty acid rich foods, chia seeds and flax seeds in children that has got an aversion to fish, to help calm the skin inflammation.

We talk about the fact that omega six are not baddies. You know, fatty acids like tahini, seed butters and nut butters help to strengthen the skin lipids.

And then really to focus more on soy intake. soy is a very good source of ceramides and we saw a significant effect between ceramide intake and ceramide in the skin. Most children actually do like fried tofu, or alternate between soy yogurts and cow's milk based yogurts, if they can have cow's milk.

Remember it's small numbers, but I still think that the data is exciting and I always think, can the dietary advice I provide lead to harm? And I don't think any of that will lead to any harm, and most likely some beneficial effects.

So with that, I'd like to thank you for your time and I'm happy to take any questions.

Korey: Terrific. Thank you so much. What a great presentation. I think parents really wanna know what to feed their kids who have eczema, and this is some really practical, useful information: lots of fats.

But we do know that kids with eczema usually have lots of dietary constraints, and so how do we navigate that?

Dr. Venter: The easy answer is they need to see a dietician. Second best answer is, with every additional food in the infant diet you bring risk of food allergy down. And, improve the gut microbiome.

So first I say to the parents, yes, your child's not eating 200 foods. We've got five, but we are gonna get to six and we are gonna get to seven, every food counts. Some children cannot stand the taste of avocado. but then, use avocado oil in cooking.

It is definitely true that lots of children with eczema have food constraints but there is always an alternative to get to as healthy as possible diet.

Korey: Any questions in the room?

Jasmine: Are artificially hydrogenated fatty oils or acids beneficial or non-beneficial to people with eczema?

Dr. Venter: I haven't studied the hydrogenated fats. My gut feeling would be no. I think as we are learning more about ultra processed foods and all these artificial things, they just never seem to do any good.

Angela: I wonder if the typical nutritionist would support higher dietary fat?

Dr. Venter: I don't know. I think the whole allergy message is sometimes difficult for the nutrition world. But, we are training dieticians to be more able to deal with food allergies. But yeah, I think there's always a battle between allergy and nutrition.

Korey: And a question about what the mother eats in pregnancy and the effect on later development of allergy in the child…

Dr. Venter: There's no need for mothers to avoid allergens, but we really don't know the answer. And so I say to mothers, do as you do, if you like nuts, eat them. If you don't then don't eat them. I don't think we have a straight answer,

And breastfeeding is particularly complicated because between 25 and 50% of mothers who eat the allergen will not secrete it into their breast milk. We don't understand why it happens. So what you eat may not necessarily be what you're, parting onto your baby.

Dr. Insel: Yeah. I will point out that In the escape study. They're introducing egg and peanut, in the third trimester and then continuing during breastfeeding. And hopefully we'll get an answer but it's gonna take a couple years.

Dr. Venter: Yeah.

Korey: Wonderful.

Well, without further ado, I'm happy to introduce our next speaker, Dr. Theodora Karagounis. She's Assistant Professor of Dermatology at New York University Grossman School of Medicine. She is a 2024 GPER Award recipient, and her talk will be on *Staph aureus* bacteria gut colonization, and its relationship to microbiome maturity and eczema.

Dr. Karagounis: Thank you Korey, for that introduction. I'm gonna talk to you about how early life gut colonization by *Staph aureus* may be contributing to atopic dermatitis.

Just to start us off with a little bit of background, the gut microbiome is a collection of bacteria that lives inside our gut, and early in life there's this drastic change in those bacteria in the gut. It starts out as few and relatively not that many different species. then there's this rapid increase in the diversity and

number of bacteria there. Diversity really increases in those first two years of life and then plateaus.

Now, when this gut microbiome development process is interrupted, it's associated with a number of diseases and particularly with atopic diseases. So now it turns out, in the first two months of life, up to 70% of children have *staph aureus* in their stools, or their gut, and then it tends to go away.

So why are we focusing on *Staph aureus*? It's a type of bacteria that is well known to contribute to atopic dermatitis when found on the skin. Eczema children who have *Staph aureus* in the skin tend to have worse disease. It tends to bloom in the setting of flares and there's been a lot of excellent research showing how this happens with *Staph aureus* producing toxins that break down the skin or cause itch.

So putting that all together, we wanted to ask, does *Staph aureus* gut colonization contribute to pediatric atopic dermatitis? And to do that, we set up two cohorts of children, aged 6 months to 13 years of age, and we collected swabs from various body sites: the nose, the skin, the rectum: and we checked to see if *Staph aureus* was present.

When we look at the rectum, we see this really striking difference where over 50% of children with atopic dermatitis had *Staph aureus* detected in the rectum versus only two of our healthy controls.

To validate these data, we looked at a second cohort of children. age 6 to 14 months. And we looked in the stool of these children to see if they had *Staph aureus* or not. Again, we found despite this younger cohort, near 80% of the children with atopic dermatitis had *Staph aureus* in their stool versus approximately 50% of the healthy controls. So what these data tell us is that gastrointestinal *Staph aureus* is indeed associated with pediatric atopic dermatitis.

So next we wanted to understand how *Staph aureus* in the gut may be contributing to atopic dermatitis. And a simple question we could ask is: *Staph aureus* being transmitted between the gut and the skin? 'cause we know if it's on the skin, we know it exacerbates disease.

So to do this, we looked to see if there's certain mutations present that we could track. And you can think of tracking these mutations as a fingerprint, a way to identify a certain bacteria and where it came from. These mutations are exclusively shared between the skin lesion and the rectum and they're not found

in the nose. Providing genetic evidence of direct transmission of these bacteria between the skin lesion and the rectum, suggesting that the gastrointestinal tract does act as a reservoir that exchanges *Staph aureus* with the skin.

But we still wanted to understand: what are the factors leading to this *Staph aureus* colonization? To know how we could potentially intervene early on to decrease this burden of *Staph aureus*. And as I mentioned, in this first cohort, we found that these children had decreased diversity of their microbiomes in the ones we saw *Staph aureus.* In our second cohort we didn't really find any differences. These children were younger and that's a time of really rapid microbiome development with a lot of noise in the data.

So we wanted to sequence additional samples and we were able to obtain 260 additional samples with the support of GPER. And we've processed over half the samples to date. And in the children that have *Staph aureus* we anticipate that we'll see less gut microbiome maturity or development, or lower diversity compared to those without. And our main question is: how early does this happen?

So takeaways is: children with eczema are more likely to have *Staph aureus* in their gut. and we think it can potentially act as a reservoir for skin colonization. (In mouse models, we found that mice who are colonized with *Staph aureus* in the gut have increased overall immune activation, potentially contributing to skin inflammation). *Staph aureus* in the gut is linked to a disrupted gut microbiome, and we're trying to unravel the early events that lead to that. And more work is needed to understand what factors lead to *Staph aureus* living in the gut.

So with that, there are many people to thank. I especially wanna thank Korey and Global Parents for Eczema Research as a whole for funding this research. And I wanna thank our study participants as well as all our collaborators who allowed this work to happen.

Thank you.

Korey: Great presentation. Dr. Karagounis.

So should we get rid of *Staph aureus* in the gut?

Dr. Karagounis: I wouldn't necessarily think of just getting rid of *Staph aureus* altogether, with example an antibiotic. 'cause we know that disrupts the gut microbiome and overall worsens training of the immune system. But I would

think of it more in how can we better either introduce nutrients that would help the immune system to continue increasing in diversity and not necessarily allowing *Staph aureus* to persist or to overgrow there.

Korey: Okay, terrific.

And let's move on to our next presentation. I'm pleased to introduce Dr. Richard Insel, he's a Research Professor in the Department of Pediatrics at University of Rochester School of Medicine and Dentistry. And he'll be presenting a really, interesting finding, the title of his talk kind of says it all, Gone Missing the Lack of Bifidobacterium in Infant Gut and its Relationship to Childhood Allergic Disease. So, Dr. Insel take it away

Dr. Insel: Thank you GPER for the opportunity to present the My Baby Biome study. This is the largest study in the United States of the early infancy gut microbiome. And it involved infants ages 4 to 12 weeks of age. And there were over 435 samples collected.

The families were asked to donate a stool sample during that period of time.Those stool samples were analyzed in two ways. First, what's there? What are the gut bacteria in those stool samples? And second, how do those bacteria function?

In addition, the families were asked to fill out questionnaires and specifically about allergic disease.

About 34% of the babies were born by C-section, which is representative of what does occur in the United States. 87% of the babies were receiving some breast milk, of whom a little bit more than half were exclusively being breastfed.

There was a deficit of a particular bacteria called *Bifidobacterium*. Now this finding was really surprising to us because very early on in infancy, bifidobacterium has historically predominated in the gut microbiome. And the reason it has predominated is because there is a naturally occurring prebiotic in breast milk that can support the growth and function of *Bifidobacterium*.

So the third most common component in breast milk are a group of sugars called human milk oligosaccharides, HMOs. And they are over 150 of these sugar structures. Any single woman will express about 20 to 40 of these, and these sugars will promote the outgrowth of *Bifidobacteria*. *Bifidobacteria* can

promote healthy immune function and immune development, as well as healthy gut barrier function, obviously important for the prevention of allergic disease.

The first finding I wanna show you from this study was quite striking. Although 24% of the infants had an abundance of *Bifidobacteria*, there was another 24% of infants who had no detectable *Bifidobacteria*, and that included not just 35% of C-section births, but 19% of vaginal birth infants. In addition, another 52% of infants had decreased *Bifidobacteria*.

When we look at the whole gut microbiome, we identified three distinct community states designated as community states one, two, and three. All of the community states were dominated by vaginal birth. Although community state three had a disproportionate number of C-section births. In addition, breastfeeding was occurring in all the community states, but there was an excess of exclusive breastfeeding and community state one.

When we look at the bacterial content of these community states, there really were two take home messages. One was, there was a distinct difference in the abundance of *Bifidobacterium*. Community state one had a predominance of *Bifidobacterium breve*, community state two had a predominance of *Bifidobacterium longum*, and community state three, that lacked the *Bifidobacteria* had about a predominance of *Proteobacteria* and *Clostridia*. These are pathobionts, microbes that promote a pro-inflammatory state and can even be invasive.

Now what's the meaning of this? Well, this really comes through when we look at the gene abundance of different groups of genes. And community state one had more genes that can utilize those sugars, those human milk oligosaccharides, and was more able to convert them to beneficial metabolites. Much more than community state two and community state three.

In contrast, there is pretty much very little HMO utilization genes in this community state two. We find some interesting findings here where community state three *does* use those HMOs. However, what's using those HMOs is not *Bifidobacterium*, but are those pathobionts that promote that pro-inflammatory state.

And this is brought home by the second take home here. And that is when we look at factors that promote the pathogenicity of those microbes, what we find is a predominance of those in community state three and community state two, the a paucity of those in community state one.

Last, about 30% of the infants had developed allergic disease. Approximately 20% had developed eczema. And what we found was that community state two, as well as community state three had a threefold increased relative risk of the development of allergic disease compared to community state one, with the high abundance of *Bifidobacteria*.

When we look at antibiotic use, we know that antibiotic use in infancy increases risk of allergic disease. That risk we found was very comparable to what we saw with community state two and community state three.

Last, I'll just point out that *Bifidobacterium breve*, which predominated in community state one, was associated with almost a fivefold decreased risk of allergic disease.

So in summary, in early infancy we're defining a healthy foundational state, which is associated with decreased risk of allergic disease, associated with an abundance of *Bifidobacterium* and specifically *Bifidobacterium* that can metabolize those human milk oligosaccharides in breast milk, and convert them to beneficial metabolites.

In addition, the outgrowth of *Bifidobacterium* prevents the outgrowth of pathobionts and pathogens and creates what we call colonization resistance. And those metabolites not only improve healthy immune function, but also promote healthy gut barrier function.

In contrast, a dysbiotic state with decreased *Bifidobacteria*, in some cases outgrowth of pathogens and pathobionts and those microbes, are not using the human milk oligosaccharides and not generating those beneficial metabolites. Unfortunately that was occurring in about three quarters of the infants.

I just wanna thank again GPER for this opportunity and thanks for your attention.

Korey: Thank you, Dr. Insole, fascinating finding that so many children in the US are missing these critical bacteria.

Why do you think we are seeing this depletion in bifidobacteria in US infants?

Dr. Insel: We don't know for sure. A lot of the thinking is: antibiotic use during pregnancy, antibiotic use prior to pregnancy. We know that *Bifidobacteria* are exquisitely sensitive to antibiotics, and in the United States as well as in Europe, we do use antibiotics in pregnancy to try to prevent group B strep infections in

the offspring. And so a lot of mothers are receiving antibiotics in that late third trimester. That's the leading thought.

Korey: And this window of late pregnancy, early childhood, the perinatal period seems to be key for antibiotics.

Dr. Insel: There's good data to show that antibiotic use late during that third trimester for group B strep does decrease *Bifidobacterium*. What has not been shown is whether that has adverse health outcomes, but we do know it drops, abundance of *Bifidobacteria* in the offspring. And then I just wanna point out that all C-section deliveries are associated with the use of antibiotics.

Korey: Do we need to change how we're addressing group B strep so that we're not giving every mother antibiotics for that,

Dr. Insel: Yeah, I will note in Europe the approach is different. They only screen high risk mothers. Whether that leads to less effects on *Bifidobacteria* really hasn't been well studied. But there's no question that interpartum antibiotics have decreased risk of group B strep infections in the offspring.

Korey: Would introducing a probiotic be helpful to support healthy microbiome diversity?

Dr. Insel: Feeding *Bifidobacterium* and specifically *Bifidobacterium longum* subspecies infantis to breastfeed infants promotes colonization at a very early age and generation of those beneficial metabolites. That's in the breastfeeding state, and that's because of those HMOs having a prebiotic effect to promote the outgrowth. So by breastfeeding, you're feeding those microbes, and that promotes their growth and function.

Korey: Great. So does it make sense to really be breastfeeding as long as you can?

Dr. Insel: Absolutely There's good data on that, at least breastfeeding through the first six months, but the longer the better and stopping breastfeeding early, very early, we find it really changes the gut microbiome quite dramatically. It sort of accelerates it prematurely. So breastfeed, and keep at it as long as possible.

Korey: Okay. Terrific.

Well, thank you for that presentation and we'll move to our next speaker.

Dr. Insel: Thank you.

Korey: Dr. Derek Chu, is Assistant Professor of Medicine at McMaster University and a 2025 GPER grantee. And his presentation, a really innovative study, that's just getting underway, looking at skin health and allergen exposure strategy to prevent the atopic march.

And with that, Dr. Chu please take over.

Dr. Chu: Thanks so much Korey and GPER for your support.

Eczema is one of the very first signs of what often is a progression of multiple allergic diseases such as food allergy, asthma and allergic rhinitis, that all impact quality of life. And, as we've just heard from our colleagues, the importance of the microbes and the progression of eczema over time.

And that goes along with many risk factors for the development of eczema that we've identified through various studies.

So for one, infants that bathed more than once a week had an increased risk of developing eczema compared to those that bathed less.

And that those that were exposed to antimicrobials or antibiotics or other disrupting agents to our bacterial balance even through, say, wet wipes that we frequently use on baby, or even on pacifiers, had a much increased risk of developing food allergy.

And machine dishwashing with detergents and rinse aids and so on compared to handwashing alone also had an increased risk of developing eczema and allergy.

And lastly, Environmental exposure to allergens on disrupted skin, such as eczema, at increased risk of developing allergies to these environmental allergens, whether that be food, and potentially other things, that would cause hay fever, as well as asthma.

The problem, however, is that this is all what we call observational data. They're patterns that we are seeing, but we have no direct study that has checked if we fix all these different factors, can we actually prevent eczema from developing in the first place? To do that, we need what's called a randomized controlled trial.

So we're putting together a study called SHAPE, where we'll ask study participants to either follow recommendations that will protect the skin barrier or conversely, following their usual care practices. And we'll collect how frequently eczema develops in this year-long pilot study.

We'll make sure that all the study components are in place, similar to what would be done for a dress rehearsal before a major production. And if feasibility is demonstrated, we'll then go on to a very large randomized control trial to definitively test the role of this intervention package to prevent eczema.

So, for example, looking at the timing of baby's first bath. Often babies are born with a natural protective layer called the vernix caseosa. That might actually be very important. But does it make a difference? We need to test it directly.

Bathing baby more frequently might increase the chance of eczema. So if individuals can bathe weekly or less, there'll be less of a problem.

Those soaps that we use are highly disruptive to our natural moisturizing factors as well as the healthy microbes on our skin, and may open up the possibility of having abnormal colonization such as *Staph* or others that could contribute to the development of eczema and allergy.

And the next would be microbial disruptions, whether that be those wet wipes, for instance, that we put on pacifiers and so on, and detergents and rinse aids may have an important effect in disrupting the microbiome and colonization as well.

And then lastly, limiting environmental allergen exposure.

So this lays the groundwork for a potentially transformative, family centered and non-drug approach to preventing eczema in early infancy rRather than a pharmaceutical or a doctor.

So we're really excited, we're just in trial startup mode. So with that, very grateful for all your support in making this trial happen. Thank you.

Korey: Thank you, Dr. Chu. I think this is an incredibly innovative and exciting study. You've taken our best evidence about what we know might be causing eczema and allergies and really put it to test.

With that said, if I'm a parent with a young child who I think might develop eczema are these strategies I could implement right now?

Dr. Chu: I'm sure some parents are. And part of what our study is trying to assess is: how frequently are they? So our control group, they'll be asked to do what they normally would do to get an understanding of what are parents doing routinely nowadays.

Korey: Terrific.

So part of your study is to really see how those different exposures influence later eczema development exactly. And when might we have an answer?

Dr. Chu: In just about a year, so that might be Q1 / 2, 2027.

Korey: Terrific.

Well, I think this has been an amazing discussion and presentation today. And one of the things I take away from it is there's this critical window in very early life where we have the chance to maybe correct some problems with the immune system that lead to eczema. And so we really need to understand how we can reshape the gut and the skin in this critical period so that we can prevent eczema.

We do fund research on eczema and allergy prevention, and our next grant cycle is coming out in early 2026, so please look for that. If you're interested, sign up for our newsletter at www.gper.org/newsletter. And we'll see you next year,