Michael Roesselin:

We've got people tuned in from Scotland and England and all over the U.S. and Puerto Rico, and we're talking about a new topic today, and I think everybody in our audience knows Kiran, but he's the founder of Microbiome Labs and formulator for all of their products. He speaks in a million different countries on a lot of microbiome-related topics around the world. He's done, I don't know, I'm going to just throw out a ballpark number, around 50 webinars and interviews and podcasts with us over the years, and he's everybody around here's favorite microbiologist.

As we discussed on The Rebel Health Spotlight interview we recently did, you can tell the story, but you've recently shifted your focus and there's really two projects that you're working on. One of them is related to the skin, so I'm curious, we'll just start with, why did you switch focus to the skin? You're a microbiome guy, you're a gut guy. Now, you're on the skin. How did that happen?

Kiran Krishnan:

Yeah, great question. I had some focus on the skin while at Microbiome Labs as well, but really looking at the gut-skin access in that case. How can we improve the skin by improving the gut? That was the focus there because everything I was doing within Microbiome Labs was around the gut itself. That's when we developed that product called SereneSkin, which we knew impacted certain aspects of the gut, especially short-chain fatty acid production, inflammation, and so on, which then affects the skin.

Now, as more data started to come out, especially one study that I'll talk about today called The Baltimore Longitudinal Study of Aging, what the data is starting to show that the skin microbiome itself plays a very important role not only in skin health, but then overall health, systemic health as well, and for conditions distal to the skin, so things that are outside of your normal eczema, psoriasis, and aging and other things. We're talking about chronic disease that's inside the body being affected by the microbiome on the skin, and [inaudible 00:02:21]-

Michael Roesselin:

Wait, wait, wait. Hold on-

Kiran Krishnan:

... [inaudible 00:02:21].

Michael Roesselin:

... hold on. This is backwards.

Kiran Krishnan:

Yeah.

Michael Roesselin:

I brought this up before you came on saying that we had this conversation. I just want to pause for a second that we had this conversation and it's been "common knowledge, I'll put in quotes, amongst functional medicine, integrative health, and conventional medicine that problems of the skin or dysfunction of the skin or unhealthy skin is a symptom of dysfunction somewhere else and that that's the way the relationship works. Did you just say that that can be backwards?

Kiran Krishnan:

Yeah, that's the crazy thing about it. We used to think that the skin was a reflection of the inside, and so, of course, if your inside is unhealthy, your skin is going to be unhealthy as well. Now we know that that's a bi-directional highway where an unhealthy outside of the skin, which is driven by a dysfunction in the microbes of the skin, also drives an unhealthy inside, and then that unhealthy inside can drive an unhealthy outside and then back and forth. Like most things in the body, it's cyclical. There is a cyclical connection and it becomes perpetual in that cycle can perpetuate over and over again. What started to become clear to me even when we were doing the gut-skin focus is we were dabbling in understanding the skin microbiome at that time, but we didn't have enough data that existed in the outside world to really, truly grapple with the skin microbiome.

We knew that there was some connection between what's happening in the gut, what your skin looks like, and then what happens on the skin and what your guy and the rest of your body looks like. That's what we'll unpack today is this issue of leaky skin, and that's a relatively new term for most people. We did a lot of pioneering in the world of leaky guy, starting back in 2012, 2013, and really dissected it into this problem called endotoxemia, which I'm sure people have heard me talk a lot about. Then, fortunately now, lots of practitioners are talking about endotoxemia, which is the actual pathology of leaky gut, how leaky gut leads to disease is through endotoxemia.

Now, what we're going to talk about is leaky skin and how that impacts both skin and can impact systemically as well. Now, we have to pay attention to all these different biomes, the biome in your skin and the biome in your gut, but that's also not surprising because we know anytime we disrupt ecosystems in our body, we're going to lead ourselves to disease. We want to make sure we're supporting all the ecosystems. This became kind of a new, exciting frontier, and I met a young lady named Isabel Vitale, who was also very interested in this topic, so there was a great synergy there. We went on this adventure to explore doing something for leaky skin and the skin biome.

Michael Roesselin:

Leaky skin, yes, that sounded so weird just to... Oh, somebody's saying mine sounds lower than Kiran's. People have been saying that lately, and my audio is turned up a hundred percent.

Kiran Krishnan:

Maybe mine is just really loud and the relativeness of it is [inaudible 00:05:45]-

Michael Roesselin:

I don't know. I can talk louder, but there's a baby in the house, so I can't shout. Everybody says, "Fine here," so I don't know. I turned it... It's full blast and I will just do my best. "Sound is fine," so who knows? Okay, anyways, bi-directional highway, which is like everything else. I think that the idea now that the body works and an X makes Y makes Z linear fashion doesn't really apply-

Kiran Krishnan:

Right.

Michael Roesselin:

... anywhere, and that there is no independent system and this one does this one and this one does this. That is not surprising, and when you say, "leaky skin," it's not like I have blood coming out of my arms-

Kiran Krishnan:

Right.

Michael Roesselin:

... so I'm curious what you mean by leaky. What's leaking? Where is it going exactly? How does that happen?

Kiran Krishnan:
I have a number of slides. Do you want me to run through some slides?

Michael Roesselin:
Sure, yeah. We always love slides.

Kiran Krishnan:
Yeah, we've created some slides just for this, so-

Michael Roesselin:
Oh, okay.

Kiran Krishnan:
... in many occasions, the Rebel Health Tribe Community is the first to see these slides, right? So-

Michael Roesselin:
Yeah, yeah.

Kiran Krishnan:
... I remember a number of occasions where we've done that where the [inaudible 00:07:03]-

Michael Roesselin:
Yeah, we are the Guinea pig.

Kiran Krishnan:
... [inaudible 00:07:04] exactly. If you guys-

Michael Roesselin:
Yeah, I remember your-

Kiran Krishnan:
... can make sense, then-

Michael Roesselin:
... presentation on the guy microbiome immune, I believe you went on to teach that a lot of different places at a lot of different conferences, and we were the first ones to get that one and that if you guys haven't seen that, it's easily the best thing I've ever seen that combines gut microbiome, immune, and how that all works together. We are grateful to be the recipients of the first slides, so I will get out of the way.

Kiran Krishnan:

Well, awesome. Well, thank you so much and thank you for everyone that's jumped on. I think we're 166 people now, which is awesome, so this is relevant to everybody. Whether you have a primary concern of skin and how the skin appears or how it's health, what conditions you're dealing with on the skin or not, this is relevant because what we're seeing now is that skin is a significant and independent driver of chronic disease, not only skin aging and skin-related disease, but other diseases that are peripheral to the skin itself. This is what we'll unpack for you.

Let's look at our skin ecology. We've got about 20 square feet of skin in our bodies, which represents one of the largest organ systems that we have in our system. There's a lot of surface area there on that skin. We have around 1.5 trillion resident bacteria. You can have up to a thousand different species of microbes on the skin, so for every skin cell, you've got somewhere between 30 to 35 microbial cells. It's a very complex ecosystem, a highly dense ecosystem as well. The most common genres are things like proprionibacterium, corynebacterium, and staphylococci. There's other ones on there as well, but those will come into play a little bit as we talk a little bit more, so keep those in mind.

In addition, we have both this mix of transient microbes and resident microbes on the skin. Now, transient microbes, of course, are not surprising because our skin is outward-facing. It's in the environment. It gets exposed to every place we go, everything we lay on, every person we hug and rub onto. All of those things create an exposure to transient microbes. Now, as it turns out, transient microbes will last on your skin anywhere from a few hours to a few days. The resident microbes are there almost permanently until they're shifted in a significant way, so that's an important thing.

Now, a key thing is that transient microbe exposure can determine in part what the resident microbes look like because certain transient microbes elicit an effect called quorum sensing. This is a very important component of all of this. Quorum sensing is the ability of microbes to reach each other's signatures and then affect each other's population by producing compounds or eliciting your immune system to deal with the microbes that are present. Some transient microbes can end up on your skin, read the environment, and then produce effects that change what the resident microbes look like. That's the important concept that people need to keep their mind on,

When we look at the ecosystem of different parts of the body, one of the interesting things about the skin is, everyone knows this, is different parts of your skin have different texture, different environments and all of that. Thereby, it also changes the types of microbes that you have on those skin. For example, if you look at just the face, the face is a very sebaceous area, which means there's a lot of sebaceous glands that produce a lot of oil. It's high oil levels, there's a lot of cracks and crevices. The pores get filled with oil, so it becomes anaerobic, so you've got a lot of anaerobic environments on the face itself.

Then, because of that, you tend to have a dominance of a type of bacteria called propionibacteria. That's a phylum of bacteria that have lots of different genus and species within it, but propionic bacterium tend to be liophilic. They like fatty acids and they can also live well in an anaerobic environment. Your face tends to have a lot of propinioibacterium. Compare that to the ecology, for example, of the arms or the back of the legs. These are the dry areas of your skin, so there's less oil being produced, which makes sense. Of course, you don't want to have super oily arms and legs all the time. Because of that and because these areas tend to be more in contact with surfaces throughout the day, you also end up with a higher diversity of microbes in these areas.

You have a mix of some propionibacterium, you have staphylococcus, micrococcus, corynebacterium, streptococcus, and so on, so you tend to have higher diversity on these dry areas of your skin. That's just to help delineate there's differences in different parts of the skin because of what that skin looks like, what it's exposed to, how much sebaceous glands there are, how much oil is being produced and so on. A lot of that will dictate the different effects within that skin microbiome as well.

Now, like our gut microbiome, we're constantly disrupting our skin microbiome. There's lots and lots of things that affect the skin microbiome, age being one of them. As you age, your skin microbiome will start to change, and part of that change and the acceleration of that change is what ages the skin, and we'll unpack that a little bit. Gender, so your hormone levels can affect what your skin microbiome looks like. Genetics, of course, and genetics I would say probably has the least effect on what your skin microbiome looks like. The environment, the level of pollutants in your area, the

type of pollutants in your areas, are you in an area that's very wet? There's a lot of mold in your area. Are you near a power plant? You've got different types of pollutants there. Do you live near a farm where they're spraying a lot of stuff? Those are the kinds of things that can affect your skin microbiome based on your ecosystem around you.

The climate that you're in, are you in a dry environment like Arizona or Nevada? Or are you in a very high, moist environment like in Costa Rica where I just was near the rainforest? If you have high humidity, that changes what your ecosystem and your skin can look like. The use of cosmetics, the type of diet you have, hormones, as I mentioned earlier, immune function, how healthy is your immune system> That, in part, goes back to the gut-skin access. Lifestyle, so the choices you make, the types of skincare products you utilize. Then, gut health as well. Of course, there is that gut-skin access, which has an impact on your skin ecosystem.

What we do know is ecological disruptions of the skin is the primary insult that sets a cascade of processes going that ends up disrupting skin cell function and then thereby disrupting immune function in the skin as well. This becomes a root cause driver of skin aging, so skin aging does start with a disruption of the skin ecosystem. It's not just about getting old. It's not just about the chronological age. It's not just about what's happening on the inside. There is a disruption of the skin microbiome over time as well, and that allows the aging process of the skin to occur.

Again, I promise you we'll unpack how that happens, but one of the key things that people need to understand is this core tenet of leaky skin. What is this relationship? Let me move my little window here so I can see the text. The core tenet here is that as the skin microbiome changes, it alters the relationship between the host cell, which is all the skin cells, whether it's the dermis layer or the epidermis layer or the squamous cells or the mucosal cells within the skin, whether it's all of those cells, what is that relationship like to the microbes and, thereby, to the immune system? There's a constant influence. We know that the immune system of the host can modulate the microbial community by producing things like antimicrobial compounds, by attacking certain microbes, by expressing things like pathogen-associated receptors, pathogen-associated lymphoid tissues, all of these things that can be activated that can control microbes on the skin itself.

The function of the immune system plays an important role in that, but the host cell acts as a middleman there modulating some of this function because the microbes are sitting on the host cell. The host cell, then, has to translate to the immune system what is present in that system, and part of how the host cell detects what kind of microbes are there is based on what kind of metabolic activities the microbes are undertaking. Dysfunctional microbes are reproducing more toxins and compound and gases and things like that, and then the host cell perceive that and then send signals to the immune system that's around that goes, "Hey, there's something going on with the microbes here that's not good." Then, the immune system can respond to it.

There's a very clear connection between the microbial community, the host cell metabolism, and immune activation, and then the function of the host immune system as well. The microbes can also directly impact host immune response by compounds that they produce. Beneficial microbes can actually activate the immune cells against dysfunctional microbes, or dysfunctional microbes can produce induction compounds that can activate the immune system and create more inflammation. There's lots of levels of communication that occur. The specifics of all of this is less important.

The important thing to note is that there are these three parts that are occurring on the skin. You've got the microbial community, your own cells, and your immune system, and all of the communication and relationship that occurs between those three parts. When things start to fall apart, you'll start to see that there's a dismantling of what the microbes are doing, how that affects the skin cell, and then how that affects the immune system's response to all of that. That's the part I really want you to get, that there's these parts and they start to create an effect on one another through a cascade of dysfunction, which starts with a change in the microbial community. That's a part to remember. Now, where is this-

Michael Roesselin:

Hold on, hold on a second. That's-

Kiran Krishnan:

... yeah.

Michael Roesselin:

... not much different than the gut, any of that.

Kiran Krishnan:

Exactly. Yeah, it-

Michael Roesselin:

That's a very similar conversation that we've had about the gut a lot of times, the microbes talking to the immune cells, which cause back-and-forth in different activities or actions and the immune cells responding to the microbes. It's just a back-and-forth conversation. I think this one's just a little tough. We don't think about it because we don't see this. We see the skin as an actual barrier, that nothing goes in or out of this, and that there is no transfer in or out of this, that the immune cells are on the inside of this skin and the bugs would be on the outside of this skin. The skin isn't actually cells itself like the gut lining is cells and the little fingers. We view this as like leather, I think.

Kiran Krishnan:

Totally. Yeah, it's we don't-

Michael Roesselin:

So [inaudible 00:18:41]-

Kiran Krishnan:

... [inaudible 00:18:41] an active system.

Michael Roesselin:

... [inaudible 00:18:42] yeah. That's what I'm getting at. It doesn't look like or appear to us like it's an active system. I just wanted to point out the clear similarities between what you just went through and what's on that other slide deck about the gut immune microbiome thing, so go ahead.

Kiran Krishnan:

Yeah, and then the scientific word for that is crosstalk, so there's this crosstalk between host cells, immune cells in the microbial community, and in a balanced ecosystem, that crosstalk is very favorable for all three of them. It's favorable for the microbial community, it's favorable for the host cell and its function, and it's of course favorable for the immune system as well. Now, normally that crosstalk gets dismantled by the microbial community being adjusted. Then, the host cell and the immune system are getting different messages, and those different messages elicit a different response. Those different responses tend to be unfavorable overall, so that's exactly right.

I think there's so many things you'll see that are analogous to the gut that actually makes it super exciting because now we know that the skin is much more of an active ecosystem. It's an active system. It's a dynamic barrier in that it is acting as an absolute barrier to preventing things from going in and preventing things from coming out, but there's a lot of, at the microbial level, communication happening that is really important as well. Now, a lot of the ideas around this

thought of a leaky skin and how dysfunction in the skin can lead to chronic disease came from this scientific revolution of this study called The Baltimore Longitudinal Study of Aging. Now, what was unique about this study, it was one of the first aging-related study that looked at aging from a longitudinal perspective.

What does that mean? Well, that means that normally in aging studies, what they do is they take an age population, they take a cohort of people that are in their 70s and 80s and they compare a number of biological function to a cohort of people in their 30s. Then, they look at the differences in biological function to try to discern changes that occur over time. There's a problem with that in that there's some inaccuracies because the people that are in their 70s and 80s went through very different things than what the people in their 30s are going through right now. The environment's different. The ecosystems, the food system is different. The political system is different. Everything's different, so their impact of what's impacting their biology has been quite a bit different than the things that are impacting the 30-year-olds, so you don't get a very clear picture.

These researchers, back in I think it's 1958, decided that, "Hey, let's take individuals in their 20s and 30s and let's just follow them and measure all kinds of outcomes and changes in their biology over time and try to understand what drives those changes, whether they're negative or positive in these individuals." Each person is their own control over a very long period of time, so you get more information that's far more accurate and more interesting in terms of trends and what affects age and what affects skin in this case.

Well, skin wasn't the focus on this. It was just age, but then what this study revealed, as they were trying to figure out what markers really predicted biological age over chronological age and predictive risk factors for death and disease, what they found, which is what you see here, is weathered or unhealthy skin was emerging as a major risk factor for almost every single age-related disease from Parkinson's to type 2 diabetes. The assumption, even in this case, was that weathered skin or aged skin was a reflection of an unhealthy inside. What they figured out is it turns out that skin is a predictor of the development of disease states down the road. It became one of the most effective predictors in all of the outcomes that they measured among these individuals. This is where the skin part came into the picture.

Keep in mind, this was not a skin-focused study. These researchers figured out over a almost 60-year study that, "Wait a minute, skin and the aging of skin was the best predictor of death, disability, and mortality in individuals." Individuals that had skin that was biologically 10 years older than what their chronological age had had significantly higher risk of developing chronic disease and dying from that disease, and they were able to trace a pathology how dysfunctional skin can lead to systemic inflammation. Aged skin drives chronic disease risk. This is a new paradigm you guys are hearing, well, some of the first to hear this.

Then, let's talk about some known skin conditions that are driven by skin microbiome dysbiosis. One that's really well-known is eczema and atopic dermatitis. Lots and lots of people suffer with this. In fact, lots of babies suffer with eczema right off the bat because they're born with a dysfunctional microbiome, a dysfunctional skin microbiome, and right off the bat, you start to see eczema symptoms being introduced into these children, and then, of course, throughout their lives as well. This is driven by an increase in the type of pathogenic bacteria called staphylococcus aureus. Now, your skin microbiome in the dry areas of the skin especially have staphylococci in it, which is staphylococcus as a genus, but the type of staphylococci we should see predominantly is staph epidermidis, which is the friendly microbe. That's a good commensal skin microbe.

Compare that to if you have a higher level of staph aureus. It's going to trigger inflammatory responses in the skin. It's going to trigger a dismantling of collagen and elastin fiber, and also a dismantling of something called the ceramide system in the skin. Now, the ceramide system is a very important system. It's a good place to bring this up because the ceramide system is a fatty acid matrix that exists in the [inaudible 00:25:03] part of the epidermal layer of the skin. Now, what that ceramide layer does is it creates a fatty acid matrix that prevents moisture from escaping because water and oil don't mix. Water, moisture stays within the skin. The skin maintains moisture over time, and it also doesn't allow pathogens and toxins to move through because most toxins will get captured in the oil and won't actually leak through.

Most pathogens are not lipophilic, so they don't go well through the oil matrix as well. That oil matrix, that ceramide layer, is incredibly important as a barrier from things going through from the outside that shouldn't go through, and then loss of things like moisture from the inside. Now, pathogenic organisms like staphylococcus aureus can break down that ceramide layer and disrupt the barrier function of the skin. That increases immunological activity because you've got lots of things leaking through now, and that immunological activity will create the characteristic lesions that you see in eczema and even atopic dermatitis, and eczema is essentially atopic dermatitis. That is a really, really important root cause driver.

Now, with eczema, you can put all these anti-inflammatories on there. That's going to further disrupt that skin. It may give you temporary relief, but you will not alleviate eczema or atopic dermatitis until you change that skin microbiome and you reduce staph aureus, you increase epidermidis, and increase other beneficial skin microbes. Now, the other one, psoriasis, this is another very common issue. Psoriasis is this disrupted balance of low diversity on parts of the skin where you should have high diversity. You get an increase in this corynebacterium and an increase, again, in staphylococcus and streptococcus, typically the pathogenic versions.

Very similar pathology, they break down the ceramides. They induce inflammatory responses because now your barrier is dysfunctional. Your host cells in that region are signaling to your immune cells that go, "Hey, the microbial ecosystem's different. They're producing toxins. They're doing all kinds of things that they're not supposed to do. Let's recruit immune cells to the area." Immune cells come in and start damaging all of the skin cells. Those are the lesions that you see as a result of the immune damage that's occurring. This is very similar to when you disrupt the microbiome in the top part of the mucosa of the gut, and that causes a recruitment of your intestinal epithelium to recruit immune cells to that area, and the immune cells damage that whole area with inflammation. That damage leads to leaky gut and things like colitis or Crohn's, which are inflammatory conditions in the lining of the gut.

Psoriasis, atopic dermatitis, eczema are inflammatory conditions on the skin surface that are mediated by your immune system, but that immune system is triggered by a change in the microbes in the region, and then the signaling from the host cell. Acne is similar to that as well. Acne is driven by a significant increase in a commensal bacteria on the skin called cutibacterium acnes. Now, it's normal to have cutibacterium acnes on the skin.

Certain versions of cutibacterium acnes can be beneficial as well, but when you have too much cutibacterium acnes, that can get into the pores. It can trigger an inflammatory response from your immune system, which then forms a lesion in the pores, so you get clogged pores by a lot of oil production. You get an inflammation of the sebaceous gland. You get inflammation in that pore from the immune system coming in and attacking it. Now, you get an inflammatory lesion which looks like acne. That's driven largely by cutibacterium acnes.

Now, the other thing that's also now well-known, another condition that's driven by dysbiosis in the skin is aging, so the characteristics of aging. Characteristics of aging, when you look at the changes in the skin, the physiological changes, now we know visually we see things like fine lines, wrinkles, dryness, sagging of the skin, hyperpigmentation and heterogeneous pigmentation. Those are the things we identify as aged or weathered skin. What's actually happening from a biological standpoint is a lowered level of sebum production. Your sebaceous glands are no longer producing enough oils for your skin, so you have lowered level of sebum production. You have lower hydration of the skin because of the breakdown of the ceramide layer, so the skin dries out and you get an increase in immunological function.

That drying out of the skin, the increase in immunological production, and that lowered level of sebum production leads to cellular senescence, which are the pigment cells in the area. The melanocytes become senescent, so they overproduce pigment. The breakdown of collagen and elastin fiber, so you lose elasticity of the skin and you end up with fine lines and wrinkles. Then, of course, you lose the tautness of the skin as you lose structure and the skin start to sag as well. Then, the skin, the epidermis starts to thin because those cells are becoming senescent. They're not turning over enough, so you lose the thickness and the regeneration of the skin, so not only do they become thin, they become dull-looking as well because the cells aren't turning over. They're dying and you've got deeper and deeper layers of dead skin.

All of the things that we see as age-related functions are actually things that are a result of a dismantling of aspects of biology of the skin, and all of that starts with microbes changing on the skin. Now, like we talked about earlier, it's a cyclical thing because as you get microbial dysbiosis on the skin, you start seeing lowered sebum production, lowered hydration, increased immune function, lower sebaceous, sorry, lower ceramide matrix and so on. Those changes further change the microbiota on the skin, and that further change causes more of those biological functions to occur, so it keeps going cyclical This is why skin ages more and more and more over time.

You start to see a flip of microbes on the skin. You start to see an increase of corynebacterium instead of propionibacterium because, remember, propionibacterium are the ones that like the oil, like the anaerobic environments. As you're reducing the oil, as you're reducing the anaerobic environments, you get an overgrowth of corynebacterium, which is a hallmark of skin aging. That corynebacterium increases inflammation and increases the dismantling of the sebaceous production and also the ceramide layer, which then causes more of the age-related dysfunction. It is a cyclical thing.

Further aging, further loss of barrier dysfunction driven by more microbial imbalance, and then overall, there's an inability of the skin to protect its host from negative stimuli like UV radiation, like blue light irradiation that we're all getting right now from our screens, like chemical and environmental triggers that are present as well. Not only do you lose the barrier function of your skin, the skin starts to age, but then the skin also loses its resilience, the ability of the skin to bounce back. That's why you get frown lines and smile lines because those lines occur when your skin increases when you make those expressions, but when you release the expression, the skin bounces back. It's more resilient. You lose that resilience, so now you've got those lines there permanently.

You used to be able to run around when you were younger, get exposed to the sun, and maybe get burned a little, but your skin recovers, but now you're probably more sensitive to the sun. We know most people as they age, they can spend less and less time in the sun before their sun gets really burned really badly, and it takes longer to recover from it. All of that is that loss of resilience on your skin. All of these conditions driven by skin dysbiosis. Age and the loss of resilience in the skin is also driven by skin dysbiosis.

Let's talk about some of the effects of a balance microbiome on the skin and how that results in features of the skin, and then, how that changes in an imbalanced state. Balanced skin, number one, when you have a balanced microbiome on the skin, you prevent the overgrowth of pathogens, thereby reducing toxin production and reducing the recruitment of immune cells to that region. What does that look like on the skin? It provides resilience to the skin with low levels of inflammation and a high tolerance from negative stimuli because now your skin is thick. It can repair itself effectively. It's not dealing with an imbalance of microbes that are constantly dismantling the skin barriers and skin layers, the ceramide layer, the collagen, the elastin, and so on."

You have resilient skin, your youthful skin. It's resilient to negative stimuli outside in the environment. You've got a microbiome on the skin that produces adequate protease enzymes, and these protease enzymes help turn over the stratum corneum of the skin. That's part of the cellular turnover that needs to happen. If that happens, then the skin can repair fast to damage. You can get abrasions on the skin. You can get negative stimuli from the sun. You can get any other damage to the skin that occurs, rubbing it on certain things, having a fabric that rubs on your skin all day long. All of those types of abrasive things that can damage the skin gets repaired relatively quickly. You may not even notice that it happened.

That turning over the skin also increases the glow of the skin because you have less dead cells on the skin and more functional live cells on the skin with better oil production. You have a thicker appearance of the skin. You have that fresh, glowing look that everybody wants to try to achieve, and your skin also is more resilient because it repairs faster. Now, you also have healthy balanced microbes that produce adequate lipase enzymes on the skin. Lipase enzymes are enzymes that break down fatty acids and regenerate the lipid layer.

This allows the skin's ceramide layer to keep breaking down and regenerating like the mucosal layer in the gut lining, you need to keep regenerating it because it's the thing that traps a lot of toxins and all of that. The ceramides are

trapping toxins, trapping pathogens. You need to break those down, allow those to leak off, and then create a new ceramide layer. The beneficial bacteria help do that, just like Akkermansia in the lining of the gut breaks down the mucosa in order to regenerate it.

The beneficial bacteria on the skin break down the lipid layer on the skin in order to generate it, so skin can maintain moisture. It acts as a strong barrier resistant to bacterial and toxin translocation. It's resistant to water loss, so you maintain that nice, healthy, glowing, moisturized skin. It provides proper function to collagen and elastin fibers. The skin is taut, it's useful, it can crease, but it'll bounce right back. The crease isn't permanent. This is exactly the hallmark of youthful, healthy skin. The production of lipase enzymes by beneficial bacteria is a critical part of this just as the production of protease enzymes was as well.

Now, the other thing is this good skin bacteria can also negate things that are being produced on the skin. For example, urease can be produced, free fatty acids that regulate sebum and manage the pH of the skin. Urease is being produced because some of the skin cells, sorry, some of the microbes on your skin can actually produce ammonia and urea as a metabolite of their fermentation and their metabolic process. That urea and that ammonia can actually increase the pH of the skin. When you increase the pH of the skin, you start to dismantle collagen and elastin fibers and the protein matrix of the skin. Maintaining the right pH of the skin is really important. Your beneficial bacteria will produce urease and will negate the production of ammonia gas as well, so it maintains a pH. At the same time, it creates free fatty acids, which are acidifying the skin to maintain the right pH.

Skin that has a good pH balance can prevent things like fungal overgrowth and yeast overgrowth on the skin. We know that when you increase the pH in the skin, you can increase the growth of fungus and yeast. In fact, certain fungus like Malassezia is an independent risk factor for developing Alzheimer's, for example. If you have a lot of fungal growth on your face, you can actually absorb and breathe in those fungus into your upper respiratory tract. That fungus can create a lot of inflammation in the central nervous system, which is now thought to be an independent risk factor for the development of Alzheimer's and dementia because of the toxigenic inflammatory response that that fungus creates inside the body from overgrowing on the skin. The pH balance is really important. It also preserves collagen and elastin function because those are proteins and proteins are very susceptible to an increased pH.

They can degenerate. They can also degenerate when your pH gets too acidic, but you would know your pH is too acidic on your skin because your skill will burn to begin with. Maintaining that pH balance is really important so you get that youthful composition, your skin can bounce back from things, and it also prevents a sagging of the skin, which is another result of collagen and elastin function. Now, the other thing of it is the effect of quorum sensing. This is really, really important because this is part of what maintains the ecosystem of the skin. Effective quorum sensing and the production of biofilms produces a stabilization of the ecosystem of the skin so it's less susceptible to constant changes from environmental stimuli. Some of this quorum sensing and the production of biofilms comes from the transient microbes that end up on your skin.

As it turns out, the spores are really great at that quorum sensing on the skin itself, just like they are in the gut. Remember, we encounter spores in the outside environment. In fact, in the natural air, in the desert dust, for example, that blows across most of the continents, you've got a high concentration of spores in there, and spores are naturally present on your skin, in your respiratory tract, and of course, on your gut and in your gut. As it turns out, on the skin, spores have a quorum-sensing function where they can detect the presence of dysfunctional microbes and they can increase immunological activity against those microbes to bring their levels down. They can also induce a production of biofilms to protect and maintain a healthy balance of microbes on the skin.

This healthy balance of microbes provides the resilience that your skin needs to reduce things like oxidative stress and damage that occurs to skin from negative stimuli. It also reduces the incidence of senescence. Senescence, I mentioned it earlier, but if you're not clear on what that is, senescence is when cells become zombie cells, if you will. They're not reproducing. They're not doing the things they're supposed to do. They instead get obsessed with a singular function. In the case of melanocytes, the cells that make your melanin, when they become senescent they just start producing lots

of pigment. They don't die off. They don't regenerate. They just sit there, produce lots of pigment, thereby getting those age spots and hyperpigmentation areas.

Then, senescence in those cells can actually propagate senescence in all of the neighboring skin cells as well. Now, you start getting all of these zombie cells all over your skin. An aged individual has lots and lots of zombie cells in their skin that's not turning over, that's not producing oils, that's not producing ceramides, that's not maintaining collagen and elastin fibers and all of that. That senescence becomes a big, big issue in aging overall. That's true for internally as well. We know cellular senescence is a big issue in internal aging within the body as well, so that quorum sensing from transient microbes on the skin becomes a really, really important part of balancing the microbiome of the skin.

Now, let's talk about dysbiosis on the skin, and then how that results to features on the skin. For one, if you don't have a balanced microbiome, you get pathogen overgrowth. These pathogens produce high toxins on the skin, and that causes the recruitment of immune cells because the host cells there are going, "Hey, we have too many pathogens producing these compounds. We need the immune cell to show up." That means your skin is going to be red. It's going to be sensitive to things. It's going to be irritated and it's going to be highly susceptible to conditions like eczema, acne, psoriasis and so on. You have sensitive skin that doesn't function properly the way it's supposed to. That can all be due to a pathogen overgrowth because you don't have the right balance of microbes on the skin.

You also now are lowering protease production. Remember, protease was really important to turn over skin cells in the stratum corneum. Now, we have low protease production. Skin doesn't turn over adequately. You get an accumulation of damaged skin cells. That leads to skin that looks very thin and dull, so there's no glow. There's no shine. The skin doesn't repair fast enough, so any damage leaves a mark much longer, can scar much easier. All of these things that are a hallmark of aging with the skin, that is all driven in part by this low protease production. This loss of ceramide in the lipid layer, this is due to lack of lipase production and, of course, the gut also plays a role here. Skin loses the moisture and becomes leaky. Microbes and toxins migrate through and drive inflation.

Now, what does this look like on the skin? This means you have dry, irritated, and red skin, which is, again, a hallmark of aging. You get yeast and fungal overgrowth on the skin, reduction of collagen and elastin function and concentration in the skin. That means your skin becomes thin. It can even have noxious color to it, noxious smell to it, and then it can have more wrinkles and more fine lines as a result of it. The last thing is the skin becomes very susceptible to oxidative damage because of the lack of diversity within the skin microbiome. It accumulates a lot of free radicals. Then, things like UV exposure and stimulants will drive more oxidative stress and oxidative damage. That leads to more senescent cells that are not replicating,. These are zombie cells. Eventually, it can eve lead to senescence to the point where you start triggering tumor formation in the case of melanocytes that become tumors eventually.

You get discoloration and hyperpigmentation of the skin, which is a normal thing, and then in rare cases, you can end up with melanoma and other skin-like cancer conditions. This is what a dysbiotic ecosystem on the skin can result like. If you look at the results, you get redness, sensitivity, irritation, loss of barrier function of the skin, high susceptibility to things like eczema, psoriasis, and atopic dermatitis. Overall, you get thin skin, dull skin, fine lines and wrinkles, discoloration, and hyperpigmentation. These are all the hallmarks of aged dysfunctional skin, and it's driven by dysbiosis that occurs in that skin.

Now, what can you do about it? This is where you really want to be able to change the microbiome of the skin and, then, thereby change features or characteristics of the skin. We did a couple of small-scale studies. We're doing some larger scale studies here. Here's participant one I'm going to use as an example. I'll show you two skin microbiome shift examples, and then the resulting change on the skin itself. If you look at this individual on their baseline microbiome analysis, now the reason why there's two of them is they're looking at two different layers of the skin, two different, sorry, areas of the skin itself. Now, if you look kat this individual, cutibacterium acnes, remember, that is the commensal bacteria that when it's at really high levels can lead to the formation of acne lesions.

If you look at this individual's skin microbiome, if you don't know anything about them, you've never seen a picture of them, just looking at this 59, almost 60% prevalence of cutibacterium acnes on this individual's skin, you would assume

that this individual may have acne, may likely have acne lesions. Now, when you also looked at staph epidermidis, this person's got a relatively low amount of that beneficial staphylococcus. They have corynebacterium, which is a age-related organism on the skin. In this case, though, all of that is trumped by this very high amount of cutibacterium acnes. Then, we use the SIV Biome-Balancing Serum on them, which we're going to talk about in a second, and in a matter of 14 days, we see a huge change in the amount of cutibacterium acnes. It goes from the number one most prevalent organism on their skin, on their face, to something that's number three with 13% prevalence on their skin.

Now, if you didn't know this individual, never saw a picture of them, you would say, "Just from this microbiome analysis, this individual has acne lesion," and from this microbiome analysis, they probably don't have acne. Then, when you look at the pictures, it absolutely translates to that. The before, this is with the almost 60% cutibacterium acnes. Look at all of those inflammatory lesions that the individual has. Now, that individual's inflammatory lesions are dramatically reduced. After shifting the skin microbiome, not doing much of anything else, we see a dramatic reduction in blemishes. We see a dramatic reduction in inflammation, and not only that, we see regulated oil levels, so he had reported that his skin was not oily as before, so it wasn't breaking down the oils and having an overactive sebaceous gland because of the inflammation. Also, the skin tone had improved.

You see lots of regions of redness and darkening of the skin, some of that hyperpigmentation spots that you tend to see, most of those tend to be gone now with this individual. You get more uniform color, almost no inflammatory lesions and just very few tiny lesions left. All of that is from simply changing the microbes on the skin. This result, this effect of reducing the lesions and improving the skin will be far more permanent as long as they maintain that balance of microbes on the skin. Another example here of somebody with elevated cutibacateriums. Now, this case, this individual only had 14% as a relative abundance, but they also had a lot of streptococcus, and this higher prevalence of streptococcus in combination with cutibacterium can drive lots of inflammation on the skin because, remember, streptococcus can be a pro-inflammatory microbe on the skin.

This individual had this combination of streptococcus/cutibacterium that was relatively high and likely has inflammation and maybe even acne lesions in the skin. You treat them for 14 days with the SIV Biome-Balancing Serum. We've reduced the streptococcus measurably, and at the same time, we've also reduced the cutibacterium. Now, you see that that combination of streptococcus/cutibacterium is reduced, so we would assume that their skin has improved. Inflammation is reduced, lesion count is improved as well. You see bacillus amyloliquefaciens, which is a good commensal transient microbe, is now present on the skin, and you see other bacilli as well. This individual's bacilli has actually taken hold and stayed longer than normally bacilli would, but I think that's in part in fighting against the streptococcus and increasing the growth of staphylococcus epidermidis.

Notice here in the top five microbes, this individual did not have staphylococcus epidermidis. That's the really commensal beneficial version of staph epidermidis. This is not even present in the top five. Now it shows up as 8.8, almost 9% of the abundance with the reduction in cutibacterium and with the reduction in streptococcus as well. This individual's skin should be improving quite dramatically and you see that. This is just in 14 days. You see all of these inflammatory lesions, even this inflamed area by the nostril itself, all of these inflammatory lesions, even the ones on the skin, are all gone in just 14 days. On top of that, you see all of these hyperpigmentation spots in these individuals. I don't know if you could see where I'm pointing with just the mouse, but there's lots of little hyperpigmentation spots all over this individual's face.

Michael Roesselin:

We can see the cursor.

Kiran Krishnan:

Oh, you can? Okay, awesome. These little spots here, these areas here on the skin, these have all dramatically improved and we have much more uniform color to the skin. Of course, you're not going to do anything about these

beauty marks, which are great to have anyway. They're pretty, these freckles. Those are completely different than any other physiology, but when you look at these hyperpigmentation spots, those are all gone as well. The other thing is the texture of the skin. If you look at this individual's... their skin right here on the left-hand side is a lot more pocked. Looks more like the surface of the golf ball. There's a lot more indentations and pocks all over the skin. Those indentations are also then colored with pigment.

You see a lot less of that here, a lot better uniformity and texture of the skin. We've reduced inflammation, we've reduced acne lesions, and we've improved skin tone and clarity in this individual in 14 days. That's a significant improvement, and the reason for that is we're making all of these microbiome changes, and these same microbiome changes is going to translate to improved barrier function, improved resilience, much more resilient to fine lines and wrinkles and damage from negative stimuli like UV radiation, like blue light radiation, and so on. It's a very, very profound change. We're not just spot treating the acne and bringing down those lesions, so that's a really important thing to think about.

Here's some other before-and-afters. After 30 days, you see all of this hyperpigmentation and the tonality of the skin. You see a significant fading and reduction of that. You see all of these acne lesions, so some are inflammatory lesions, some are non-inflammatory lesions. All of those dramatically reduced in just 30 days. You see this dysregulation of oil levels in different parts of the skin. You see a much more uniform level of oil production in the skin, and then, of course, improved tonality and texture as well, which is a little hard to see in this picture, but it's something that was reported quite significantly by this individual. We didn't ask them to smile or not smile in the before-and-after. It's just their mood. This is just how they felt before and after. She has a beautiful face in general, but now the skin is a lot healthy and a lot more youthful looking.

Here's another individual. In just 14 days, you see the reduction in lesions, both inflammatory and non-inflammatory lesions in this individual that are completely gone. The propensity of lesions are gone. Some of that hyperpigmentation is improved as well in their skin, and then the tone and texture of the skin. You see the skin is a lot less uniform in terms of its flatness, of its texture. You see an improvement of that texture in the skin overall as well.

Here's another individual with 21 days, and again, these aren't long periods of time. We're making these significant changes in two weeks, three weeks, 30 days. Yeah, people are seeing these dramatic changes. You see in this individual lots of changes, not only in terms of the inflammatory and non-inflammatory lesions, changing dramatically in just 14 days, but then you see the uniformity of the coloring of the skin as well. Some of these lines and wrinkles improving, all of that in just that 21-day period. It's really, really exciting to see.

Now, all of this is being achieved by this Spore-Based Biome-Balancing Serum. Now, the key to the Biome-Balancing Serum is choosing the right spores. In this case, it's a subtilis and coagulants combination, and then choosing skin-identical delivery system for it. This is where Isabelle's awesome formulation work came into play because she created this serum to be identical to some of the fatty acid profile of the skin, and then introducing the spores on that carrier has shown to be incredibly effective because the spores can penetrate a little bit deeper into the skin because of that carrier system. It builds resilience. It establishes a healthy skin foundation and microbiome. It supports the balance within the microbiome to make the skin more resilient and it, of course, delivers relief as well relatively quickly.

This is the first bioactivated skin serum that's proven so far to solve issues that are associated with an imbalance of the skin microbiome. We just initiated a 400-subject trial on this. We're going to get a lot of data coming out on that over the next five to six weeks. Morgan is managing that study for us, and so we'll get a lot of awesome data out of that, but so far, what we're seeing in the numerous case trials that we've done is that it's really, really affecting the skin. One of the core components of the technology is the quorum sensing. I mentioned earlier that bacillus is really good at quorum sensing. That's one of the things that we're taking advantage of with the bacilli being able to do that quorum sensing, identify the presence of dysfunctional microbes, and then alerting their immune system and also creating some chemistry that affects the growth of those dysfunctional microbes as well.

It's applicable to all, no contraindications. There's no what we call skin purging, which is like the Herxheimer reaction that you get in the gut when you do something that makes a dramatic change in the gut. You don't get that purging with this. This ease of use, it's once a day at night before you go to bed, before you put on your moisturizer, so after you wash your face. You put it on and you can use it on the body as well, almost anywhere on the body. It is adaptable to all of the different skin regions. We have people that use it on eczema and psoriasis on the body, truncal and body acne, face acne, and so on. It rebalances all of the different skin microbiomes, so super easy to use. Doesn't require a whole lot of things.

This is more for estheticians about the back bar and home use. At the end of the day, we're super excited to introduce this SIV because not only is it, of course, going to help your skin look better, improve things that you're dealing with on the skin like eczema, psoriasis, acne, other inflammatory conditions on the skin, it's, of course, going to slow down and perhaps even reverse some of the aging process that you might be concerned about. Ultimately, we now know that dysfunctional skin is leaky skin. Leaky skin is an independent risk factor for chronic health issues.

This may be a missing link to a lot of the things that you've already done. You've worked on the gut, you've worked on your diet. You've worked on all of these things. You may not still be getting as good as you want to be in your wellness journey. Maybe leaky skin is a part of it, so that may be something that you want to examine as well. That's the end of the slides. We can jump into the discussion, but thank you for letting me share these slides for the first time with The Rebel Health Tribe Group.

Michael Roesselin:

Thank you. The slides were great. I took a lot of notes. Just another reminder, please put the questions in the Q&A. There's questions in the chat. I think most of them have gotten into the Q&A. Now, we are going to have to book you for a very soon part two to do Q&A-

Kiran Krishnan:

Yes.

Michael Roesselin:

... because I'm at 9:10, 9:15 PM now. I have another thing that started nine minutes ago that I was supposed to be on that I already have pushed, so I have... No, it's not trough Microbiome Labs. We're going to have to bring you back on very soon to just do all the questions I think because we have 40 questions and there's-

Kiran Krishnan:

Oh, awesome.

Michael Roesselin:

... there's no way. There's no way, so what I'm doing right now is I am copying all of the questions, and those pictures are pretty dramatic.

Kiran Krishnan:

They are.

Michael Roesselin:

What are you doing with the 400 people? What is the study? What are you measuring or what's the situation?

Kiran Krishnan:

The way we recruited it is we wanted to recruit 400 people that had occasional non-cystic acne because we find that those are the people that tend to have very dramatic changes to their skin microbiomes because many of them have very high cutibacterium. Some of them have high streptococcus as well, and acne is one of those things that you could see, visually see changing in a significant amount, a very short amount of time, quite significantly if you do the right thing. Then, also, acne is a great surrogate condition for lots of other inflammatory conditions on the skin. That's the primary recruitment is 400 people who experience occasional intermediate non-cystic acne.

Now, 50 of those 400 people we're going to be doing skin microbiome analysis no matter what the results are for before and after. We want to see over a larger cohort of people what the skin microbiome changes look like, and so we're going to get lots of good microbiome data. We're going to get lots of good usage data. We're going to get data on acne, and some of these people are also going to have eczema, psoriasis, aging, all of those things, so we'll get good data on all of that, and-

Michael Roesselin:

Wow.

Kiran Krishnan:

... for those that aren't sure, so this is a separate company, sivcare.com is where you find it. We'll also be making it available through Rebel Health Tribe and making it accessible to you guys, so this is separate from Microbiome Lab.

Michael Roesselin:

Yeah, yeah. I put the link in the chat to the SIV, and we actually bundled it with the SereneSkin Probiotic, too. I'm sure that that combination would work well. We put everything on sale, the probiotics, the SIV, and the SIV bundle for everybody to be able to try it. The code, the link, all of that's right in the chat and we'll send it out with the recording. The questions are all over the place, so usually I'm pretty good at taking 40 questions and turning them into five questions and getting them all answered, but this, you opened a Pandora's box here. There are a ton of questions that I cannot... that people want to sign up for your studies, so I cannot-

Kiran Krishnan:

We'll be able to. You know-

Michael Roesselin:

... [inaudible 01:01:54].

Kiran Krishnan:

... yeah, Morgan is in the chat. I think she's on here. Morgan, if you want to put your email where people can reach you, this is a consumer-level study, so we may be able to actually include you in it, which would be interesting. I don't know the logistics of it. She's much more managing that than I am, so if you email her, maybe she'll be able to show you how to do that. I did see a question pop up about shipping. So far, we're shipping everywhere, so this-

Michael Roesselin:

This doesn't have... I'm not going to jinx anything and I'm not going to make any guarantees or promises, but because shipping things to Europe is a way bigger pain in the ass than I believed it would be when I moved over here, it's

impossible when it comes to supplements, especially certain countries. I don't think they're nearly as strict on topical skin-related products-

Kiran Krishnan:

Yeah.

Michael Roesselin:

... so as long as you don't tell them you're going to eat it, I think you should be fine, and don't eat it. There's probiotics for that and there's distributors of that over here, but I think that you're more likely to get this through. We're going to check our side with our distribution to see to make sure because we've paused shipments from our distribution to certain countries in Europe because of the customs and the taxes and the problems that arise, but I think this one will be able to get through. You guys have been shipping into Europe?

Kiran Krishnan:

We have, yeah. We haven't had any problems. We've been shipping it to Australia and New Zealand and all kinds of places. We haven't had any issues-

Michael Roesselin:

Oh, okay.

Kiran Krishnan:

... but again-

Michael Roesselin:

Well, those are-

Kiran Krishnan:

... topicals are less regulated.

Michael Roesselin:

... [inaudible 01:03:32] okay-

Kiran Krishnan:

Yeah-

Michael Roesselin:

... cool.

Kiran Krishnan:

... so we shouldn't have the same issue.

Michael Roesselin:

Okay, because Australia and New Zealand were impossible for supplements, too.

Kiran Krishnan:

They are [inaudible 01:03:40].

Michael Roesselin:

They're even impossible to set it up for retail, for like wholesale for-

Kiran Krishnan:

It is.,

Michael Roesselin:

... for distributors, so-

Kiran Krishnan:

Australia's the hardest-

Michael Roesselin:

... [inaudible 01:03:48].

Kiran Krishnan:

... yeah.

Michael Roesselin:

Yeah, yeah. We have a lot of Australian people in our community and that we've heard rants and rage for a long time about it, so Canada, yes, should be okay. We'll talk to Marianne on our side, and Marianne will talk to our shipping and make sure that everything's cool. Email Morgan about interest in wholesale. I saw a couple questions for wholesale. Email Morgan, ask her. We're not wholesaling it, just talk to them. Mary, I don't know when the questions will be answered, but I've copied all 55 of them into a document that I now have and I will schedule with Kiran sometime soon to do all the questions. No presentation, no more teaching. We're just going to come on and I am going to bombard-

Kiran Krishnan:

I love it.

Michael Roesselin:

... bombard Kiran with questions, so thank you [inaudible 01:04:48]-

Kiran Krishnan:

In the meantime, if you go to SIV's Instagram page, Isabelle and team have been putting up a lot of great information, so some of those questions may get answered just from looking through their Instagram feed and looking at all the information that they've posted, but certainly one of my favorite things to do is answer questions with Michael. We'll definitely prioritize the scheduling of this over the next couple of weeks and come back on and do that.

Michael Roesselin:

Yeah, yeah, yeah. We'll make it happen. I'm traveling to the U.S. It's Thanksgiving, but we'll figure out a way to make it happen. You were just in Costa Rica. I saw your-

Kiran Krishnan:

Mm-hmm.

Michael Roesselin:

... Instagram story, it looked lovely-

Kiran Krishnan:

Oh, so nice.

Michael Roesselin:

... so yeah, that's a good... I've only been there once, but it was a nice time, so-

Kiran Krishnan:

Yeah.

Michael Roesselin:

... cool. One question that I will ask because people are saying like, "The SIV thing is small. You're using pictures of faces. You're talking about skin all over the body." There's a lot of questions about scalp. There's a lot of questions about other parts of the body.

Kiran Krishnan:

Yeah.

Michael Roesselin:

You guys, the intention is not just to make the one facial serum, right? Like you guys-

Kiran Krishnan:

Yeah.

Michael Roesselin:

... are working on more stuff?

Kiran Krishnan:

On more, yeah. Think about it this way for now. The way the tube is designed right now, it's designed for typical face use and the way you typically use it is you put one drop on each side of the face and then you rub it in all throughout, including the neck and chin area. You can do the same thing on the scalp as well. You can either directly apply it on the scalp and rub it in all over the place, or you can put it on your hands and rub it in as well. Now, one tube, if you use it every day on the face will typically last about a month. That means that you could use it actually in other parts of the body. One of the ways I use it, I actually not only use it on my face, I use it everywhere because I love to keep control of streptococcus overgrowth and all of that from all of the travel I do.

Actually, when I put my lotion on my hand, I put a couple of drops of the SIV Serum on there and I mix it into my body lotion and then I apply it on myself, so I get the SIV all over the place, If I end up with a spot somewhere, if I get a dry spot or somewhere on the body, I'll apply it directly on that spot and rub it in, but generally I apply one or two drops in the lotion that I then apply throughout the rest of my body. If you're doing both your body and your face, that one tube will probably last more like two and a half weeks, but if you're doing it just on your face or your scalp, it'll last you about a month. I use it quite generously. We'll make a bigger version of it for the full body. That'll be next, but right now you can adapt it that way.

Michael Roesselin:

Okay, that makes a lot of sense. Good. All right, I've got a giant document filled with questions and I will track you down and we'll get you back on here. I will fastball lob questions at you for an hour very soon.

Kiran Krishnan:

I love it.

Michael Roesselin:

The presentation was great. I took three full pages of notes and it was like the old days when I was learning all of that stuff for the first time-

Kiran Krishnan:

I love it.

Michael Roesselin:

... and it's really interesting because this isn't being talked about at all. I've-

Kiran Krishnan:

Right.

Michael Roesselin:

... I've been in functional medicine circles now for almost 15 years and I've never heard half of that before, and nobody really pays attention to it. There were some questions I'm interested to see your response on for some of the more conventional things that people do to their skin and what that does to the microbiome, so-

Kiran Krishnan:

Yeah.

Michael Roesselin:

... we'll get into that next time. Everybody, give Kiran a shout in the chat if you enjoyed the... Whoa, our lights just flashed. There's a storm outside, so if I disappear, my power went out, but let Kiran know what you thought of the presentation and we will be back on as soon as we can.

Kiran Krishnan:

Thank you, everybody. I really appreciate-

Michael Roesselin:

[inaudible 01:08:54].

Kiran Krishnan:

... your attention. We're very excited to pioneer this space and really drive into it like we did with the gut and leaky gut and so on. You guys are at the beginning stages, again back with Rebel Health Tribe. We started in Microbiome Labs early, early on with Michael and Rebel Health Tribe, so you guys are the innovators in our space and we're excited to be able to do this with you. For those of you that end up getting it, please report in to us how it's going, how you're doing and all that because that feedback that we got from MegaSpore early on was really critical for us to understand how the product works and even make adjustments if needed and direct the clinical research. Really would love your feedback on it. We trust your feedback and your opinion, so thanks again for this opportunity.

Michael Roesselin:

Thank you, and everybody, we will see you guys soon.

Kiran Krishnan:

Cheers. Bye.