
Danny Lennon: Hello and welcome to Sigma Nutrition Radio, the podcast that brings you evidence-based discussions with the world's leading researchers in fields related to nutrition and health. I am your host, Danny Lennon, and you are listening to Episode 155. And on today's podcast, I'm delighted to have Dr. Kimber Stanhope from the University of California at Davis on the show. Dr. Stanhope is a registered dietitian in addition to her PhD, and she's an associate research nutritional biologist in the Department of Molecular Biosciences at UC Davis and her work focus is on investigating the effects of sugar consumption on the development of metabolic disease, utilizing well-controlled diet intervention studies in human subjects. She has more than 20 years of nutrition research experience focused on contributing significantly to the clinical research base that we have and really helping inform how to I suppose optimize healthy nutrition and the diet that contributes towards disease within humans as well as trying to delineate the mechanisms that are involved. So, a really, really high-standing, renowned researcher in this area, probably has been more prolific than the vast majority of people I can even think of in the area of sugar consumption and human metabolism and health. And so that's why I wanted to try and get her on, to try and get a very evidence-based perspective of when we're talking about sugar consumption how it affects health and body composition, what exactly does the research tell us and what do we know, and how far to either extreme should this message go. And so hopefully we can get into some of the real nitty-gritty of sugar metabolism and then how that relates to human health.

The show notes to this episode are going to be over at SigmaNutrition.com/episode155, and if you've not done so already you can sign up there to receive the transcript to this episode and all other previous episodes, which will get delivered to your inbox completely for free, and also I'll be able to link up to some previous research papers published on this topic that Dr. Stanhope has provided.

For any of you regular listeners who haven't signed up to the Sigma Synopsis weekly email, then I'd like to remind you that if you're enjoying the content on this podcast you'll probably love the content posted to the email list. So every week you'll just get a short roundup email of the best written, audio and video content from the evidence-based nutrition and performance online community all in one place and you can click through to whatever ones take your fancy. That will be sent to you every week in a short little email you can just flick through. It doesn't take long and you can just pick out what you enjoy. And as well as that, now and again I'll send some content that's exclusive to the email list and maybe some insights on different things semi-regularly, and that'll be all to that Sigma Synopsis email group. So if you just go to SigmaNutrition.com, you'll see somewhere to sign up for the Sigma Synopsis and you just pop in your email and you'll be on that list. With that, let's get Dr. Stanhope on the line and get into this week's show.

Hey Kimber, welcome to the show. Thanks so much for coming on.

Kimber Stanhope: Thank you, Danny. It's nice to be here.

Danny Lennon: Yeah, I've been really looking forward to this conversation because you've obviously done a ton of work in this area of sugar consumption and how that affects various parameters of health. So before we get into some specific discussion around some of that research, maybe you can just give listeners an introduction to your work, where most of that has taken place, what you've been looking into, and just generally a bit about your academic background.

Kimber Stanhope: Happy to. I definitely started out with a bachelor's degree in nutrition, spent time in research right after graduating, and then became a registered dietitian so actually spent time in public health where I would do individual diet counseling as well as cholesterol, diabetes and weight management classes. Ended up back in research and as time…I got hooked. You couldn't get me to leave the lab. And it was around certainly

after I came back to research that we almost accidentally ended up being interested in sugar.

We really weren't looking at the effects of sugar on I cardiovascular disease or diabetes. What we were interested in, was it possible sugar increased body weight? And we were basing that on our mechanistic findings regarding the hormone everybody was talking about at the moment, and that was leptin.

Leptin was the hormone that they found that when they injected it into obese mice, and these were mice that had been studied for years and years and years as a model of obesity, they simply got fat for no reason at all except that they got fat. Even if you kept them from overeating, they still got fat. Well, when these mice were injected with leptin, the obesity disappeared, and we were very interested in leptin too. So we were doing mechanistic studies in Petri dishes with fat cells to try and determine the role…what made leptin go up or go down. Basically, what we had learned other labs had already reported was that leptin is a message that gets sent to the brain and it's released by fat cells, and when fat cells are perfectly happy and perfectly stocked with fat, they make a lot of leptin and they send this message to the brain saying, “Quit eating so much and burn more energy. We're fine down here.” But, in situations where there's not enough energy around for the fat cell, leptin goes way down and you get very, very hungry and you start overeating and you also conserve calories. Your energy expenditure goes down. Your metabolic rate decreases. And this was fascinating, and everybody thought leptin would be the next obesity drug and it would cure obesity.

Didn't happen that way at all. It definitely made the mice leaner because these were mice that were born without the gene that made leptin. But unfortunately, humans, we tend to have plenty of leptin around and, in fact, obese people tend to have even higher levels of leptin than a lean person. So leptin turned out not to be the problem, but that's why we started studying sugar and we found that the fructose component of sugar made leptin levels go down.

And so we thought, “Wow! Is this why everybody’s getting fat, they're eating too much sugar and their leptin levels are going down?” Our study, which basically started right around 1999, and then our definitive study that started in 2004, showed no, this wasn’t true. It was true that leptin went down when we gave human subjects fructose compared to human subjects who got the other monosaccharide, glucose, but they both still

gained the exact same amount of weight. So we didn't prove anything about the obesity crisis at that point in time, but what we did find was risk factors for both cardiovascular disease and diabetes went up only in the subjects that had the fructose sugar and they didn't change at all in the subjects that had the glucose sugar even though both groups of subjects gained the exact same amount of weight, and this was the data even before I published it, our group published it in 2009 in the Journal of Clinical Investigation. When I looked at that data, I kind of went, “Whoa, I'm going to be studying sugar for the rest of my life,” and that's exactly what happened.

Danny Lennon: Right.

Kimber Stanhope: So that was about 2005-2006. Ever since then, we have just kept writing grants to NIH. They all didn't get funded but enough of them have been funded that we have been working on sugar ever since. So our first major NIH study was the one with the glucose versus fructose and have those very interesting results, and that paper still is getting cited today because it's still just an amazing concept that two sugars, fructose and glucose—and the interesting thing about fructose and glucose is, chemically, they're absolutely identical. They both have six carbons, 12 hydrogens, six oxygens, but they're shaped differently and that's enough to make the difference that our bodies metabolize them very differently. However, even though those data were really important and interesting, they really didn't tell us much about the real diet because we don’t consume pure fructose or pure glucose as sugars. Instead, what we consume is sucrose and high-fructose corn syrup. Those are our added sugars in our food, and both of them contain both glucose and fructose.

So our next study was to compare one of the sugars, high-fructose corn syrup, to glucose and fructose. So where did it stem? Remember, with glucose we saw no negative health effects; with fructose, we saw very marked increases in risk factors. So how did the sugar we consume, high-fructose corn syrup, compare to the two? And that study was published in 2011 and, sure enough, basically high-fructose corn syrup, its effects were far more comparable to fructose. We saw the same increases in risk factors for high-fructose corn syrup that we did for fructose. And again, we did not see these negative health effects with the glucose-sweetened beverage.

That led us onto our next study, which we called the dose response study, and that was looking at consuming four different levels of high-fructose corn syrup in beverages. And so this was zero, 10%, 17-1/2% or 25% of

energy requirement as high-fructose corn syrup in a beverage. And just to give you kind of a perspective, the amount of sugar in the 10% beverage would be identical to one-and-a-half cans of Coke, so our intervention at the 10% level was the same as getting a half a can of Coke at breakfast, lunch and dinner. And this study, again, it was only two weeks long and our subjects were all young—the average age was 25—and about half the subjects were normal weight or leaner and half of them were overweight and even some of them were obese. This range of subjects, the effects were very, very consistent. We saw beautiful dose response effect. Basically, our graphs look like a little stairway with no negative effects in the subjects who received sweetened beverages that contained aspartame but no high-fructose corn syrup, then a little increase at the 10%, bigger increase at the 17-1/2, and then the highest increase at 25. Very significant dose response effects for risk factors for cardiovascular disease such as LDL cholesterol and a more scientific one would be called apolipoprotein B, postprandial triglycerides, and then uric acid, which for a long time we've known that uric acid causes gout but there's a lot of researchers now that are looking at uric acid a lot more closely because they think that's related to both diabetes, cardiovascular disease and hypertension. So this study basically showed that humans are quite sensitive to the amount of sugar in their food and we could differentiate very easily between the four doses even when we had subjects out in the real world eating their regular diet.

Danny Lennon: Yeah, super-interesting. There's obviously a number of things to dig into there that I want to go back on, Kimber, particularly with the dose response and how that relates to probably the typical diet people are going to be consuming. But even before that, if we take a step back and try and look and piece apart what's going on here when we see these differences in risk factors based on the types of sugar that you were seeing consumed, so when we're looking at either fructose or glucose in isolation or then, say, sucrose or high-fructose corn syrup, maybe for those who maybe aren't as familiar, can you maybe just give some brief overview on what those metabolic differences are in how those sugars are metabolized and then potentially why that actually matters or why we see differences when they're consumed?

Kimber Stanhope: I love to answer this question because I actually think the answer is very interesting and very important but also quite understandable to the general public, and if more people understood this answer they would be quicker to reduce their consumption of sugar. So we're going to start with glucose.

Remember, glucose is the sugar I said we really didn't see those negative effects. Now, we gave it as a beverage but in real life most of your glucose is consumed in the form of bread and rice and any other grains. It's broken down into these long chains of starch which are called complex carbohydrate. They get broken down into individual glucose molecules in your intestine, and then they leave your intestine via the portal vein, and that vein carries the blood directly to the liver. Basically, the liver is the first bus stop after the intestine and that gives the liver first rights to all the sugar we consume.

Now, the liver can pull that sugar in and use it for its own needs. It can turn it into energy. It can turn it into glycogen and replenish the liver glycogen stores. It can turn it into fat. It can do whatever it wants to do with it among four or five different pathways. But the decision as to whether the liver pulls the glucose in or leaves it alone, and if it leaves it alone then it just bypasses the liver and that glucose will get used by the brain, the fat cells, the muscle cells, the nerve cells, there is an enzyme that controls whether the liver leaves the glucose alone or pulls it into the liver and that enzyme is regulated by hepatic energy status. In simple terms, that means if the liver needs energy, in comes the glucose because that enzyme is turned on and it lets it come in. If the liver doesn’t need energy, the enzyme is turned off, the door is shut, and the glucose goes to the rest of the body.

Now, it's different for fructose. Fructose leaves the intestine just like glucose and comes straight to the liver, but there's a very different enzyme in charge of the fate of fructose. This enzyme is turned on all the time. It's not regulated by hepatic energy needs. That means whether the liver needs energy or whether it doesn’t need energy, in comes the fructose. Pretty much 90% of the fructose we consume ends up in the liver, and just think of how much fructose that is when you drink an entire Big Gulp. In comes that fructose and that liver, our livers do the best they can with all that substrate. It turns some of it into energy, some of it into glycogen, some of it into lactate, but it needs to do something with the overload. So it does exactly what our bodies whenever there's too much energy around, turns it into fat so it can be stored. So the fructose gets turned into fat and that increases the amount of fat in the liver. Well, the first thing that happens and we know this happens as quickly as six to eight hours later, that fat starts getting packaged into particles along with cholesterol and get sent out into the blood. So this increases our blood levels of triglyceride, which is a risk factor for cardiovascular disease.

But, over time, let's say you're drinking a Big Gulp every day, it doesn't all get sent out into the blood – some of it starts accumulating in the liver. That causes an increase in liver fat that's sometimes called fatty liver or nonalcoholic liver disease and that's a problem too, and we think one of the negative things related to having too much fat in your liver is that it makes your insulin stop working very well, and we call this insulin resistance. It means insulin is not doing its job, and that's a big problem because insulin has so many jobs and when insulin can't do it everything then escalates and gets worse. You end up with even more fat in the blood and you end up with even more sugar—fructose—being turned into fat. It's a vicious circle where everything gets worse. And as more fat ends up in the blood, what we end up with is some of that triglyceride ends up in the muscle cells and that causes insulin resistance in the muscle cells too. And therefore, you not only have liver insulin resistance, you have whole-body insulin resistance and that's definitely a risk factor for type 2 diabetes.

The other problem that's going on by a completely different pathway not related to the fact that fructose gets turned into fat but just because there's too much fructose around and it's going…this regulates a pathway that leads to increased levels of uric acid. So by two completely different pathways, we have two very negative effects instantly occurring with fructose consumption due to fructose overload and that's fat-making and increased uric acid levels, and both of those are related to metabolic… So did I make that clear?

Danny Lennon: Yeah, that's a really, really comprehensive breakdown. Thank you so much for that, Kimber. That was amazing. And I think that kind of gives us a clear picture of potentially why some of these findings are coming up in the research. Maybe one thing that people may be hearing now is obviously when you hear these results at first glance you start to see, "Okay, sugar is going to be surely some sort of problem particularly when there are high amounts of fructose around," but as with anything, it tends to be down to the dosage really making the poison, and I mean a lot of people will have seen people promote messages of having zero-sugar diets or sugar detoxes or completely avoiding any foods that they can think of with amounts of fructose in it, for example, where we know that probably to some degree there's going to be at least some leeway for minimal or smaller amounts of sugar in the diet and then depending on what sources we're looking at. So when it comes down to, first of all, sugar maybe in general, and then if we want to bridge into fructose just to kind of explain

that, what sort of dosages are we talking about when we're looking at when these detrimental impacts start to occur? So when we start to know that it's going to cause issues with insulin sensitivity or a lipid accumulation or the buildup of body fat storage, do we know what dosage is okay and when it starts to become excessive? Are there kind of numbers we can see or how should people think about how much is too much?

Kimber Stanhope: Excellent question, and there are two kinds of research evidence that gives us hints about the answers. Both of these types of research have their flaws. There's the kind of research I do where I actually intervene, change people's diet and look at the effects in the short term. My research gets criticized because, "Well, how do you know the effects you saw last?" or "How do you know those increases in cardiovascular disease risk factors get worse and worse and worse over time until they lead to cardiovascular disease?" Then, we have the other kind of research where we take thousands and thousands of people and we look at their diet and then we look at their change in disease status over time and we relate how their diet associates with their change in disease status. That data is important because these are people who are actually getting disease, and so we can look at the changes in their sugar consumption and can actually know, "Wow, they got the disease," but that data doesn't prove the sugar actually caused it. It only shows it was associated. But even so, I'm going to start with one of those kinds of studies.

This study was published in 2014 by the epidemiology experts from Harvard and they took over 10,000 subjects and looked at their sugar intake and then looked at how many people died of cardiovascular disease over the next 15 years, and almost 10% of them did. They then divided the groups, the entire population, into five levels based on their level of sugar consumption. The lowest level were those people that were consuming less than 10% of their daily calories as added sugar. So this is not just the sugar in a beverage but the sugar in the cookies and in the ketchup and in all the other hidden places there's sugar. So that group was 10%. Then, the next group was 10 to 13, and then 14 to 17, and then 17 to 20, and then it was the group that consumed more than 20% of their energy as sugar. They saw what I saw, a dose response effect. With each increasing level of sugar consumption, the number of deaths due to cardiovascular disease was increased in the group that 13 to 17% of their energy as added sugar, which is exactly the range where the average American consumes sugar. We consume about 15% of our calories as added sugar. There was 18% increased risk of death by cardiovascular disease in that group.

Now, that's our normal diet. That's what people are consuming and it's associated with an increased risk of cardiovascular disease of 18%, and when we're talking cardiovascular disease that's the number one killer in our country. The number one cause of death is cardiovascular disease. So 18% is a lot of deaths over a 15-year period.

Alright, the group that consumed over 20% of their calories as sugar, they had a doubling, an increase of 200% increased risk of cardiovascular disease. So it definitely looks like what the normal American is consuming is too much. That study was probably very helpful in getting our US dietary guideline experts to reduce our level of our previous guideline all the way up to these new guidelines which started in 2015, was 25%. Up to then, there was kind of a vague guideline saying, “Don’t exceed 25% of your calories as added sugar.” So at least from 2010 to 2015, the scientists, we should all feel pretty good because we have now gotten the guideline reduced to 10% of calories. How safe is 10% of the calories? Well, we are not going to be able to figure that out until we actually feed a group of subjects exactly 10% of the calories and check for increases in risk factors. Very hard study to do and, to the best of my knowledge, nobody’s done it. I've tried to get funding from NIH to do it and, unfortunately, the response I got from the NIH reviewers is, “Oh, everybody knows sugar’s bad for you except the industry. We really don’t need to keep spending all this money to prove it.” But you asked the question and I think it's an excellent question, “How low do we really need to go to be safe?” And I would like to know that answer, but I don't know where the money’s going to come from for us to be able to find out.

Danny Lennon: Right, yeah, completely agree. And I think one of the big points there that actually relates to something I was going to ask is obviously we can look at a lot of the observational research to kind of point us in the right direction and we see these associations with these excessive amounts of sugar consumption, and therefore death rates and then cardiovascular disease events for example, and so there's obviously a clear correlation there. But then, people can also state that, “Well, the people who are consuming the most sugar are also probably having terrible diet and lifestyle choices overall and that is probably feeding into it as well,” and we also know that perhaps one of the biggest issues with excessive sugar consumption is that in turn tends to drive excessive caloric consumption, which in turn drives excessive gaining of body fat levels, and we know that alone is going to affect health markers. So do we have any data looking particularly at do a lot of these detrimental effects we see on

health from high consumption of sugar, do they still persist even in the absence of, say, a caloric surplus and in the absence of gain of body fat? If someone's eating at a caloric level that's fine, does more sugar in the diet still have these same negative consequences on health?

Kimber Stanhope: Well, that's an excellent question, too, and I have three things to say to address it. The first is that in my study, that 10% group of ones who consumed the equivalent of one-half of soda at breakfast, lunch and dinner, in that group, even though they did not gain weight, we saw increases in their risk factors in only two weeks and that was postprandial triglycerides, LDL cholesterol and apolipoprotein B. That surprised me. I was hoping to see a stair-step effect but I did not expect to see that two weeks would be long enough to be able to pick up that difference in just, you know, just the addition of a half a can at breakfast, half a can at lunch, half a can at dinner. I wasn't expecting to see that. So to me, that's good evidence that we can see these effects without weight change.

Another study was done by a very close colleague of mine down at UCSF. He simply gave men energy-balanced diets. By that, I mean diets which they carefully monitored them and did not allow those subjects to gain weight. The diets either had fructose beverages or they had starch, you know, rice, bread. On the rice and bread diet, there was no change in risk factors. On the fructose diet, there were all the risk factors I talked about. There was increased fat-making of fructose, there was increased liver fat, there was increased triglyceride and cholesterol in the blood, and then there was decreased liver sensitivity or liver insulin resistance. All those things happened despite the lack of weight gain.

The third thing I have to tell you though is I'm doing this exact same study with high-fructose corn syrup. For weeks at a time, we are giving subjects all their food. They're either getting aspartame-sweetened beverages or high-fructose-corn-syrup-sweetened beverages and we're not allowing them to gain or lose weight, and we are looking at the changes in the fat-making, the changes in the liver fat, the changes in the blood risk factors, and the changes in insulin sensitivity. Unfortunately, I'm not going to have data to share with you for another two to three years, but that's our current NIH study that we are doing right now. Because what you said is an incredibly important question, "What are the effects of sugar that are not mediated by body weight?"

But I think it's also very important to put the two together. We already have data that suggests that sugar has effects, negative effects, that aren't

related to weight gain, but we have plenty of data that suggests sugar causes weight gain and we know weight gain has negative effects, so just how much worse is it when you put the two together?

Danny Lennon: Right, yeah. No, it's a really fascinating area particularly, like you say, when we know it's going to affect things like insulin sensitivity and then the role that that may play in excessive weight gain over time. And it's really great to hear that you've got research going on in that area because, like we've seen, it's probably an area and a gap in the literature that could really do with some comprehensive stuff and so that's really great to hear.

One thing I did want to do, Kimber, is to try and put some kind of caveats here for people listening who are maybe trying to think of some practical takeaways and they may be picking up that, "Okay, excessive amounts of fructose may be an issue or excessive amounts of sugar we know are obviously an issue," but then they list the kind of same thing where some of these…maybe sometimes people can extrapolate some of this stuff and come to maybe incorrect assumptions, and one big one I think that I often hear perpetuated online who maybe people haven't really looked at the full context of what's been discussed may start talking about, say, fruit consumption, and then telling people that they shouldn't be consuming certain types of fruits because of the fructose content, whereas we know a lot of this research is kind of done in the context of sugar-sweetened beverages. Could you maybe just give a bit of an overview for people what the kind of deal is when it comes to fructose that is contained in fruit and do they actually have a reason to fear it or be worried about that?

Kimber Stanhope: Another excellent question and one that I get asked a lot. There are at least four different reasons why the sugar in a cookie or a sugar-sweetened beverage is far more problematic than the sugar in your fruit. First of all, it all comes down to—we did this as a trial for fun in our lab. We have never had a human subject in one of our studies have any problem drinking 25% of their energy requirement as sugar-sweetened beverage. By that, I mean somebody that has a 2000-calorie-per-day energy requirement, we give them 500 calories worth of sugar-sweetened beverage. Nobody's ever had any trouble drinking that. But our group, we took lots of fruits. We took apples, oranges, bananas, pineapple, and we sliced it up and gave everybody in the group 25% of their energy as fruit and said, "Please eat it all in one day." Four people brought bunch of it back, said, "Sorry, I can't." Three people were able to eat it all but they said, "I won't do it again. It hurt." So that's one of the reasons why you probably don't need to be all that worried about the sugar in fruit. It takes a lot of fruit to get up

to the levels of sugar consumption that you can get with three sodas and two cookies and dessert every night.

Danny Lennon: Right.

Kimber Stanhope: You probably can't do that. The second thing is our sugar-sweetened beverage is absolutely devoid of anything of value with regard to vitamins, minerals, bioactives, flavonoids, and all the other good things a fruit has. Fruit has a tremendous number of positive bioactives that we know have benefits to our bodies. The epidemiology data almost 100% shows fruit consumption is related to protection against disease, whole fruit consumption. And of course, fruit has fiber, our sugar-sweetened beverage has none, but the thing about fiber is it has all sorts of beneficial effects on our microbiota and we're just starting to learn about those.

But the other good thing about fiber is it slows things down. There's nothing to slow the fructose overload when you drink a sugar-sweetened beverage. The speed with which that sugar can get into the liver is amazing. It's very fast. Within 30 minutes, undoubtedly that fructose is 90% in the liver, whereas with the sugar in fruit it would take far longer to get the fructose in that fruit available and in the liver. So that's another reason.

But there's another…this is lately new evidence…a new…people are starting to talk about and think about this a lot more, and that is the fact that our eating is controlled—let's call it mechanisms—by two different mechanisms. One mechanism is called homeostatic eating and that's the hunger that shows up when our energy levels are low. That's our bodies saying, “Eat! You need food in here because the energy’s low,” and all sorts of things happen when we eat to make that hunger signal go away and so we'll stop eating. That's homeostatic eating.

If everybody was paying 100% attention to their homeostatic signals, we'd all be ideal body weight, but unfortunately there's another eating system called hedonic eating. And hedonic eating is a completely different set of signals that relate to dopamine release in the brain and reward activation, and we have found that really make that reward activation signal go off is the high-fat and the high-sugar food. Basically, it's our highly palatable food system that we're surrounded with that people bring at every birthday party, at every celebration. It's by the check stands at every grocery store. Those foods that we absolutely can't get away from are the foods that are activating this hedonic drive. It has nothing to do with hunger. It's eating

because the reward, the taste, is wonderful, and you don't get that from a piece of fruit. You get it from the cookie. You get it from the chocolate. Some people get it from sugar-sweetened beverages, some people don't. It's really interesting. There's a lot of people…we don't understand why people are so addicted to sugar-sweetened beverages, but they say, "For me, I can't keep away from cheesecake." Everybody has a highly palatable food. It's never, almost never, going to be an apple or an orange that somebody just can't keep themselves from overeating.

Danny Lennon: Right. Yeah, completely agree.

Kimber Stanhope: Yeah.

Danny Lennon: It's really interesting. Go ahead.

Kimber Stanhope: Yeah. So I think I sort of covered all the reasons why you just don't need to start being worried about the fructose in your fruit. Yes, there's fructose in fruit but never more than 5% of that weight as fructose, and there are a lot of good things in the fruit.

Danny Lennon: Yeah, perfect answer. I think that helps a lot of people because, again, it's one of these issues that when it comes up it can cause confusion at first here unless they hear that proper context. So Kimber, this has been unbelievably informative and I'm sure there's tons of information people are going to take from this discussion. So before I come to the final question, can you let people know where they can maybe find more of your work online? Is there a bio they can go to? Are there papers on ResearchGate maybe that they can check out? Where can they find more of the work you've done or more information?

Kimber Stanhope: Well, actually, if they want to just start with the real easy basic stuff, going to YouTube there's quite a number of videos that are available that I've talked about issues related to the mechanism, related to fruit, related to nutrition labels and so forth, and there's even an entire one-hour lecture that goes on forever but a lot of people watched it that took place in Lake Tahoe. So they can see that, but my email is easily accessible under my name on Google. I send out PDFs of my papers all the time and, yes, I should set up a website where they're available with a click but I'm sorry, haven't gotten around to that. [Chuckles]

Danny Lennon: Perfect. I'll link up to all those videos in the show notes for people listening as well as where you can find Kimber's bio online as well, so click through that and do check that stuff out. And with that, Kimber, that

brings us to the final question that we always end the show on, and this can be to do with anything even outside of what we've discussed today and it's simply if you could advise people to do one thing each day that would have some positive impact on any aspect of their life, what would that one thing be?

Kimber Stanhope: It would still relate to the food and it would be let Mother Nature be your food processor, not the industry. Just pick those foods as close to nature as they grow.

Danny Lennon: Awesome. Thank you so much, Kimber, for your time today and even more so for the great information. It's going to help a ton of people and it's always amazing to hear this stuff from people that are involved in academia and actually doing this research and authoring these papers, so to come direct from you is awesome to hear this stuff. So thank you so much for the work you do and for your time today. It's been absolute pleasure.

Kimber Stanhope: Thank you, Danny. I enjoyed it.

Danny Lennon: So there we go. That was of course Dr. Kimber Stanhope from University of California at Davis. In the show notes, I'm going to link up to some of Dr. Stanhope's work. I'll include the full text, PDFs, of some of those research papers for those of you who are interested in looking at that as well as linking up to some of those YouTube videos and other pieces of content that Dr. Stanhope mentioned as well as her full bio and other places where you can contact here if you do have questions. You'll also get the chance to get the full transcript to this episode as well as all other previous episodes for absolutely free if you just go to SigmaNutrition.com/episode155. If you want to find me on social media, then you can either just search for Sigma Nutrition and Performance on Facebook or follow me on Instagram at my handle dannylennon_sigmanutrition.

And that brings this week's episode to a close. I really do hope you enjoyed the episode and if you did I'll be extremely grateful if you left a review for the show on iTunes or even if you just shared this episode around on social media or tagged people, whatever you can do to help spread the word of the podcast and help us continue to grow this and get more people interested in evidence-based information to try and counteract a lot of the nonsensical stuff that is probably floating around online. So if you can help us, that will obviously make a huge difference.

And for all of you who regularly listen, you are the ones that continue to drive the show forward and make it what it is and continue to get these experts on to be able to spread information from their research groups and what they're finding in the lab. So thank you so much for that, and just make sure that you've hit Subscribe on whatever app you are listening to this show on right now, whether that's iTunes, Stitcher, etc. just so you do continue to get updates of our future episodes.

And if you are listening to this on the week that the podcast episode was released, then I wish you a very happy Christmas and hope that you and yours have a wonderful holiday season and that you have a great time, and hopefully that it's something enjoyable for you, your family and your friends. And just want to say at this time, thank you so much for continuing to listen. It really does help so much to continue pushing the show forward and trying to put more into it because of the listeners that continue to listen to the show and really make it as big as it is. So thank you so much, guys. It means a lot.

That's it. Take care and I will be back very soon on the next episode and I hope to catch you then.