Danny Lennon: Hello and welcome to Sigma Nutrition Radio, the podcast that brings you evidence-based discussions with the world's leading researchers and practitioners in fields related to nutrition and performance. I am your host, Danny Lennon, and you are listening to Episode 143.

Before we get into the interview, especially after all the questions and inquiries I received off the back of last week's episode which was related to some of the topics that come up within our coaching process her at Sigma, I just want to outline a couple of things just to cover off typical questions that were coming in. So we offer an extremely comprehensive weekly coaching service where you'll work intensively one-on-one with one of the Sigma Nutrition and Performance coaches. You have the option of nutrition-only coaching if you have your own training sorted or you have someone else doing your programming, or you have the option to combine the nutrition coaching with training programming, so whether that's S&C if you're an athlete, whether just to get you a bit stronger or to get in shape, we can cover off your training and exercise, or then we have specifically powerlifting programming as well if you are a lifter. So if you're interested in learning more about any of that and seeing our application process and just who we work with because we're quite specific about who we do work with, if you just go to SigmaNutrition.com and just look for Online Coaching tab in the menu, and then you can find

the details there about who we work with and how to apply if that's of interest.

So getting on to today's episode, I'm just absolutely delighted to be joined by a really truly brilliant academic and one of the smartest guys you will ever listen to in relation to muscle physiology, so Dr. Keith Baar from University of California at Davis. Keith has been really one of the true leaders in muscle physiology research for a number of years and has been involved in a number of different fascinating research areas and has really been at the forefront of a lot of that work. And one of the most novel areas that he's been working on recently has been examining the role of connective tissue and tendon stiffness in both performance and injury risk as well as the nutritional and training protocols that could potentially modify tendon stiffness to, again, potentially improve that performance, decrease skeletomuscular injury, and then recover from ligament or tendon rupture or bone and cartilage damage. It really is quite amazing when you look at some of the stuff. So I'm going to get Keith on in just a minute to discuss some of this fascinating work. I think you're really, really going to enjoy it because he's really one of the smartest guys you will ever listen to in this area.

The show notes are going to be at SigmaNutrition.com/episode143, and if you haven't done so already you can sign up there to receive transcripts to this episode as well as all previous episodes, which will be delivered to your inbox as a PDF, like I said, completely for free. So let's get into this week's episode with Dr. Keith Baar.

Hey Keith, welcome to the show. Thanks so much for joining us today.

Keith Baar: You're very welcome. It's good to be here.

Danny Lennon: I think there's so much to get through and there's a lot of the research that I've come across that's come out of your lab that has been particularly fascinating, so I want to dig into some of the details later on in the show. But maybe just as a way of introduction to everyone listening who maybe hasn't come across you before, can you maybe fill people in on your academic background, some of the work that's currently been going on in your lab, and really the main objectives of the work that you and your colleagues are doing?

Keith Baar: Sure. So I'm a professor at the University of California Davis. I study basically the molecular and cellular responses of musculoskeletal tissues to exercise training, nutrition and aging. And so what we've done in the

past with my PhD work, I looked at the effect of resistance exercise on specific molecular markers that are related to muscle growth, and so as part of that work I identified that mTOR complex 1 was activated in response to resistance exercise and that deactivation was proportional to the load across the muscle. And so that was a starting point. I then went to Washington University in St. Louis and did a post doc with the sensei of exercise, Mr. John Holloszy, and there I identified or I described for the first time that PGC-1 alpha was upregulated by endurance exercise. And specifically, what I was looking at there was how…it wasn't the full-length or the native PGC-1 alpha that was upregulated by exercise so much as it was that there was a novel isoform of PGC-1 alpha that's now known as either PGC-1 alpha 2 or 3, depending on who you ask. It was upregulated specifically in response to exercise.

And then as I've developed over the years, what I've done is I spent five years in Scotland developing a lot of the models to understand how different types of exercise affect musculoskeletal tissues, and along the way there what we have been doing is we've been looking to understand how the noncontractile part of muscle works. So these are the proteins that are involved in force transfer. So the force transfer is the process by which you take the force that's being produced by the actinomyosin and you transfer that force out of the individual sarcomeres to the connective tissue overlying the muscle all the way down to the tendon that is going to allow for movement to occur. We started this in part because a colleague of mine, when I went down to talk with the UK cycling team, they described how they were able to increase, significantly increase, strength without changing muscle mass, and that kind of went against a lot of what the paradigm in muscle biology is, which is that force is proportional to cross-sectional area.

And what we've been discovering in the last few years is that there are a number of different situations where we can either decrease force or we can increase force and have that happen in spite of the fact that muscle cross-sectional area or the fiber cross-sectional area isn't changing. And so what we're realizing is the ability to transfer the force is almost as important as the size of the fiber itself. So whereas a lot of my early career was to determine skeletal muscle hypertrophy in the mechanisms underlying that, what we know now is that even if you're not hypertrophy you can still have a really significant effect on your strength gains that you can get from your training. And in fact, from some of the work that we've done with the USA track and field team and the throwers on that team, I

would say that the best predictors of your performance as an athlete—and this is true across a number of different sports when I've gone through the literature—it's not your absolute strength, it's the rate at which you can develop force, so your rate of force development. So the faster you can develop force, the faster or the better you're going to perform. And whereas historically we've thought that the rate of force development was strictly down to skill acquisition or learning or neurological adaptations, what we have been showing is that we can actually reproduce a lot of these things by looking at the force transfer apparatus in the skeletal muscle.

Danny Lennon: That's super-interesting stuff because we've actually chatted on the podcast in recent times about some of the applications of that potentially in the sport of powerlifting where, like you said, especially because it's a weight-class-based sport and we're trying to look to see who can have the most strength for that given weight class, typically the general maybe logical way to think would be, "Well, for someone to just be the best lifter they can, over time they just add more muscle hypertrophy for that given weight class and then they can go then and express that as more strength," whereas we're starting to see competing ideas and like you say some things pointing towards that actually you don't necessarily need to see the increase in cross-sectional area of a muscle fiber, you can actually get increase in strength. Is that something you're starting to see people maybe…or where can we see the applications of that in something, say, like powerlifting and how should people think of the potential to get stronger without necessarily always having to chase hypertrophy?

Keith Baar: Yeah, it's definitely something you can see in a variety of different sports. So we have been applying this first and foremost as a health-based thing to try and prevent a lot of musculoskeletal injuries in classically areas where you get repetitive stress injury or you get fatigue injury, in fact. So that's been long distance runners, rugby players, football players, all of these things where there's huge repetitive load and the musculoskeletal injury is the number one cause of time away from sport. So what you see a lot of times in those situations if you're looking at the English Premier League, over 65% of all injuries are musculoskeletal injuries that are noncontact injuries that are related to this health of the musculoskeletal tissue. We have only recently begun looking at the performance-based aspects of this to try and say, "Can we get in with specific things and specific training modalities that are designed entirely to improve the functionality of the force transfer apparatus? And, yes, things like powerlifting, things that are

weight-class-based, things like cycling, you definitely can see that altering training to go away from some of the muscle hypertrophy aspects and go towards some of the force transfer aspects is definitely a way that we're going to see more and more of in the future.

Danny Lennon: Yeah. And you mentioned something that's particularly interesting and actually related to some of the stuff that I wanted to get to when you talked about the high rates of injury we see in certain sports related to the musculature, but really like you've outlined it's not necessarily just because of that, it's also dependent on, say, the connective tissue that's there as well. And I know this kind of centers around a lot of the really fascinating work that you've been doing looking at connective tissues and the stiffness of connective tissues and potential ways to enhance that, but maybe before we get into that specifically, just to set the scene for people listening and to try and lay out exactly why is it important from a practical perspective to consider something like the stiffness of connective tissue in an athlete.

Keith Baar: So it's a great question. So the number one thing is that most people have this idea physically from Huxley's classic work in the sliding filament theory that the way muscle contracts is that the sarcomere shortens and then the next sarcomere in line shortens, and then that continues to happen all the way along the muscle until you get to a myotendinous junction and then all of the force from all of those individual sarcomeres contracting is what causes the whole muscle to shorten and to pull on the tendon. But there's beautiful work out of John Faulkner's laboratory where they show quite elegantly using a little yolk. And so what it is is just a little plastic circle that they sew onto the outside of a muscle and all they do, because they work with the plastic and reconstructive surgeons there, they sew it into the outermost connective tissue layer of the muscle, and then what they do is they measure force either at the ring or they measure force at the tendon. And so if the Huxley model of longitudinal force transfer, which means that one sarcomere shortens, the next sarcomere shortens, and the whole muscle shortens, if that's how we're getting energy to the tendon, then when we measure force at the outside ring we should see almost no force. And in fact, when you stimulate the muscle and you measure force at the ring versus the force at the tendon, what you see is that the force of the ring is about 80% of the force of the tendon, and what that means is that at least 80% of the force produced by the shortening of the sarcomeres is directly translated or passed laterally. So it's transferred laterally out the sides of the muscle as soon as that force is created, and what that does is

that protects individual muscle fibers from injury. The other thing that it does is the better you are at transferring the force laterally using the connective tissues, using dystrophin, using all of these force transfer proteins, the better you are at doing that the more your muscle is going to contract as a single unit, the more each individual fiber is going to be protected. The stiffer those structures that hold the fibers together are, the faster and the better you're going to be able to transfer force.

Now, when we look at the tendon, we look at a very different structure. So what we've got is we've got two force-generating or force transfer components that we're looking at when we talk about the musculoskeletal or a muscle-tendon-bone system. We've got the extracellular matrix within the muscle, and then we've got the extracellular matrix within the tendon. So what you can do is you can do exercise that's going to increase the stiffness of the extracellular matrix of the muscle, but it could also at the same time decrease the stiffness of the connective tissue within the tendon, and the reverse is true and all of the different combinations are true. So what you're looking to do is as you train or as you do other things that are going to improve muscle function is you're looking for the connective tissue within your muscle to become stiffer and better able to transmit force and you're looking for your tendon to also become somewhat stiffer and to be better able to transmit force. If you're only training with fast movements, and fast movements are specifically important for the tendon stiffness, the faster your movement is the more your tendon is going to become stiffer over time with training; the slower your movement is the less stiff your tendon is going to be over time. We know that because everybody who has ever had Achilles tendon problems, what they're told to do is they're told to do slow lengthening contractions. Those slow lengthening contractions increase the shear within the collagen and the tendon and that decreases the overall stiffness of the tendon. If you only do fast movements and you continue to increase, increase, increase the stiffness of the tendon and you're not doing anything to really strengthen your muscle, what’s going to happen is your tendon is going to become stiffer than your muscle is strong, and what that means is when you hit the ground at full speed, instead of what should happen which is the tendon lengthens a little bit and acts as a shock absorber for your muscle, if your tendon is stiffer than your muscle is strong, then what happens is the tendon is so stiff that it doesn’t extend upon impact and instead of your tendon extending your muscle has to extend even though it is fully contracting. So it's maximally activating and it's trying to shorten, but because the tendon is so stiff it now has to lengthen abruptly, and that's

where severe muscle injuries occur. So a lot of the hamstring injuries that we see in high-level sport are, when people are accelerating or at high speed, their foot hits the ground way out in front of them, their tendon of that hamstring is stiffer than the hamstring muscle is strong, and instead of the tendon slightly giving a little bit to provide a little shock absorption, now that lengthening component has to go to the muscle. The muscle's not strong enough to stay contracted. It abruptly lengthens and that's when we get muscle pulls. So this whole system working together, the connective tissue, the extracellular matrix within the muscle and the extracellular matrix that is in the tendon, those things have to work optimally to support muscle function in any of the muscles that we use but especially the muscles that we use when we're doing high-intensity contractions.

Danny Lennon: Right. So we then essentially have, when we're considering the stiffness of the tendon, for example, really whether someone ideally would have that to be stiffer or less stiff is all context-dependent on that particular situation in relation to the strength in the muscle. Would that be correct?

Keith Baar: Yes, to a large degree the stiffness that you can get your tendons to is going to be dependent on the strength of the muscle. Really, we know from work by Andy Jones a long time ago that the stiffer you are, the higher your passive stiffness. That means the less flexible you are, the more stiffness you have within your muscle tendon units, the better efficiency you're going to show when you're running, so the less energy you use to run at a high speed. So the longer your tendons are, the stiffer they are, the less energy you have to use to run on a flat surface. We know that if you increase the stiffness within your muscle and tendon units that you're going to increase the efficiency of your cycling and all of these other sports that we follow, that we participate in. So a lot of what we're trying to do as we try and improve performance is we're trying to increase the stiffness through the system.

Now, the caveat is if you do that and you're only increasing stiffness upon stiffness, what you're going to find is that when you go back and you go to do your performance again to play your sport, now that's when you pick up injuries. It's most commonly seen in the American football in the rookies who are going to participate in the combine. So all they're going to do in the combine is they're going to do bench press, they're going to do short sprints, they're going to do agility drills, they're going to do long jump, high jump, all of these types of things that are all based on stiffness and power. And so what they do is they spend months training only to be at their most powerful, their most stiff, and then immediately after that

they're going to go back out and they're going to…or after they get drafted they're going to go and they're going to go to a mini-camp and they actually have to play their sport again. But if they've only trained to increase power and stiffness and all of these other things, what you find almost every year is that people are pulling up with hamstring problems, with ACL problems, with all of these other musculoskeletal injuries as a result of the specific type of training that they were doing over that time.

Danny Lennon: Yeah, that's super-fascinating. So essentially we have this case where, like you say, because the athlete is training almost exclusively at high speeds and using rapid changes in direction similar to the way you would prepare for the combine, because of that we're getting this massive increase in stiffness, and then when they go back to a typical training that they'll be doing outside of that combine prep they're essentially not able to…or their system is set up in a way that that stiffness is too much and then leads to injury. I think that's really, really fascinating. And you mentioned earlier collagen. So where are we talking about the role of collagen in all this? Is that what the stiffness of the tendon is dependent upon, just how much collagen is there, or what other factors at that level apply?

Keith Baar: So a tendon stiffness is determined largely by the amount in the cross-linking of the collagen. So there are the two things. There's how much collagen is there and there is how cross-linked the collagen is. And the cross-linking of the collagen is mostly done through enzymatic processes in your body. So there are specific enzymes which cross-link collagen, and when you do any type of exercise those enzymes go up, the activity of those enzymes go up and the expression of those specific enzymes go up. So enzymes like lysyl oxidase, for example, which its specific role is to cross-link lysine residues within collagen, as you increase the amount of lysyl oxidase you have and you increase the activity of that protein, what you get is you get an increase in stiffness, and it's especially important that it's not only how much collagen you have but it's how many of these cross-links you have.

The other way that you get cross-links are through sugars. So sugars can form cross-links within collagen matrix like your tendons, and this is one of the reasons why diabetics who have a high circulating sugar content have a greater risk of musculoskeletal injuries and they have a greater risk of other types of injuries, because what happens here is instead of an enzyme putting a specific cross-link into the collagen, what happens in diabetics is that the sugars can be cross-linked across collagen molecules and that's going to stiffen the collagen up as well as the enzymatic

reactions, but because you don't have specific control over where those cross-links occur they can provide a cross-link that is more difficult to turn over and results in a more permanent change to the collagen structure. It's one of the reasons why skin quality and other qualities of collagen-containing tissues are relatively poor in diabetics. So if you have athletes who are diabetic, you have to also be concerned with how that's affecting the stiffness of their tendons and other structures within their body, because what you would find is that they do have a higher incidence of tendon and ligament rupture due to just overall changes in the stiffness within the structures.

Danny Lennon: Yeah, that's super-interesting and it's actually a really good segue into discussing some of these nutritional factors that we can potentially modify to change collagen production and then therefore tendon stiffness, for example. And I know this is one of the areas which you've been at the forefront of looking at and because there's almost no other research at least that I'm aware of really outside of your group that has looked at it in quite this detail, so you can you maybe just talk about some of the things that you've been trying to evaluate in terms of nutritional interventions that maybe play a role here and what you've started to see or what type of picture is starting to emerge?

Keith Baar: Yeah. So we started looking at the tendon probably about almost 10 years ago and the way that we started doing this is, because we've talked about one way that you increase stiffness is by training using high-speed movements, and paradoxically the other way that you increase stiffness within the tendon is through inactivity. And so when we first started looking at the tendon, we were trying to figure out how it worked and how you could get and connect muscle which is really compliant, is really jello-like, to something as stiff as a bone which is almost steel-like in its stiffness. And so what we've discovered in a paper with my colleague Ellen Arruda is that when we looked at a tendon across its length, it was less stiff near the muscle and more stiff near the bone. But the interesting thing that we also did in that experiment is we then inactivated the muscle, and so we then looked five weeks later and what we had done is lost completely the compliant region near the muscle, right? So what that told us was that low levels of activity are needed to keep the tendon moving to keep this regional difference in mechanics, and that's really important, again, for injury rate because we talked about the athletes who go from the combine to practice, what we haven't talked about was the fact that this year especially you see this huge increase in Achilles tendon ruptures in

football players, and that's because they've gone from having no activity to having full activity.

So we started by looking at that regional variability, but tendons are very, very difficult to analyze and there's very little known about them. So what we thought would be a really good way to get a better understanding of what's happening within the tendon and to get really a lot more control over what we could actually measure is we started to actually make our own tendons. And so we did this just in vitro in a culture situation. We made three-dimensional tendons that are basically they're made from human cells, they're made from human ACL remnants. So when people rupture their ACLs here in Northern California and they go to our sports medicines doctors at the University of California Davis, what we do is that sports medicine doctor will cut out the ACL and they'll collect that and we'll bring that back to laboratory and isolate the cells from it. And then what we can do is we can put those cells into a gel, then over about a week's time period the cells will form what is for all intents and purposes an embryonic ligament or an embryonic tendon. And so we can then start to figure out what makes a tendon stronger just by adding things into our little dish.

And so when we first started doing this, the things that were obvious to us were, "Okay, let's start with amino acids that are enriched in collagen and we start with vitamin C because we know that vitamin C is important in collagen synthesis and extrusion from the cells." And sure enough, what we found is that if we added amino acids that were enriched in collagen like glycine, which is every third amino acid in collagen, or proline, which is one of the most common amino acids in collagen, what we found is we could increase the strength of our engineered ligaments especially in the presence of ascorbic acid or vitamin C. So we took that to mean that if we started feeding people things that were enriched in these amino acids that we'd be able to improve the functionality of the tendons and ligaments. And so when I went to look around to see what kind of foods were enriched in proline and glycine and all of these amino acids that we wanted for tendons, what I found was the most obvious thing of all, which was that these amino acids are enriched in things like gelatins and things like bone broths, because bone broth is essentially the creation of a gelatin as well where you're boiling bones, you're extracting the collagen, the collagen is getting broken down, and then if you let that broth sit in the fridge it'll form a jello just like you would from a gelatin.

And so what we started considering was the idea that gelatins or bone broths would be really beneficial as a way to improve connective tissue function, improve return to play or accelerate people's return to play after an injury. And so we started doing some tests together with some colleagues at the Australian Institute of Sport. Our first report is hopefully going to be out here in the next few weeks where what we've done is we've done a series of studies that we've looked at increasing doses of gelatin and we're taking it, just we're doing it in a randomized clinical trial where we do a crossover design so every one of the subjects comes in for three trials. In one trial they get a placebo that is a granular material just like the gelatin, tastes a little bit wonky just like the gelatin, and then the other two groups, one group has 5 grams of gelatin and the other group gets 15 grams of gelatin. And then they drink that, and then we measure amino acid enrichment over the next three hours and what you find is that the amino acids are going to peak within the blood at about an hour after the consumption of the supplement. We then take a large amount of blood at one hour and we put it into our engineered ligament model just to see whether the blood from a person taken before they've taken the gelatin or after they've taken the gelatin, can we see any difference in the function of the ligaments that we get from these? And sure enough, what we see is that if you make the ligament and you feed it media that contains the serum of the people after they've had the gelatin supplement, you get more collagen in a step-wise response, whereas at the 5 grams of gelatin you get more collagen than the placebo and at the 15 you get more collagen than you do at either the 5 or the placebo group. And then what we look at is we look at the mechanics and sure enough, the mechanics are improved. They are stronger. They're stiffer than they were without the supplement.

And then what we did with our randomized clinical trial is we then had the individuals jump-rope for six minutes, and we jump-rope for six minutes and only six minutes because the connective tissue cells in your bones, in your tendons, in your cartilage, they respond very quickly to exercise and then they stop responding to exercise, and what we'd found previously is that most of the tendon cells stop responding to exercise after about five minutes. And so they jump-rope for six minutes and then they take a six-hour rest, they then drink whatever supplement they're supposed to drink again, and then an hour later they jump-rope and they keep doing this three times a day for three days. And what we find is that the anybody who jumps rope, those people who jump-rope show a doubling in their collagen synthesis and most of this collagen is synthesized from the bone,

from the impact of the jump-rope. And because when we look at the marker that we look at in the blood, because you've got such a big bone mass, that marker really indicates the bone adaptation.

When we add the gelatin, the low dose of gelatin wasn't enough to get a significant effect of the supplement, but when we increased the gelatin content in the supplement to 15 grams of gelatin, what we see is a further doubling of collagen synthesis. And so what this is showing us is that if we want to improve the collagen response to an exercise bout, we can easily do that by adding gelatin as a supplement, and this is really important. The first study, this one, is just mostly…it can be looked at as a bone recovery protocol. So if you've got an athlete who breaks a bone – a bone in the foot, a bone in the leg, bone in the back, what you can do is you can have them take the gelatin and then do five minutes of exercise—and this doesn't need to be weighted exercise. You can be in an AlterG. You can be in something that's just going to allow a little bit of impact but enough impact to target those nutrients to where you want it to go. So if you have significant break-in in your lower leg or in your back, you can get into a supported AlterG or something that's going to allow you to take the weight off of it and only do, say, 5 to 10% of your body weight on that, five minutes—you can be walking, you can be jumping, it doesn't matter—and then you repeat that every six hours because it takes about six hours for the cells to return their responsiveness, and then by having that gelatin load before the exercise what you're doing is you're targeting your nutrients just to those specific areas that need to recover and you can really decrease the time it takes you to recover from an injury.

Danny Lennon: Mm-hmm. That's just so fascinating, that whole story that's emerging from that research, and just some of those results are pretty significant. So you mentioned there then when we're looking at the practical application potential for this obviously with things like bone injury or even maybe a tendon or a ligament rupture, something like that, there's obviously the potential for an application there. Do you think there's the same potential when someone's potentially looking for, say, a preventative measure or to stop something from happening or to minimizing future risk of, say, soft tissue injury, or for the moment do you think we're really looking at a rehab strategy?

Keith Baar: We definitely use it as one of the biggest things that we do to prevent injury. So what we do is we're trying to roll this out into baseball players, into…we do it all the time with our long distance runners. What we do is we say, "Okay, these are athletes and baseball players who have these

CCL ruptures where large numbers of young boys are going to be getting [00:35:52] surgery before they're done high school because they're overloading this ligament. There are huge numbers of runners who get stress fractures or who get all of these other basically mechanical-fatigue-related injuries. What we do in this situation is we just do exactly what we do for the rehab except we're not doing it as frequently. So if I know that I have an athlete who's going to be practicing in the afternoon, I have them get up, I feed them gelatin, and then I'm going to have them do an exercise bout that's going to load the area that we're most concerned with. So if I have somebody who's a pitcher and they're going to go to practice in the afternoon, in the morning I have them wake up. They take their gelatin. They're going to go into and they're going to do a series of exercises in a race path or in some sort of resistance so they can load the tendon that we're trying to, or the ligament in this case, that we're trying to access. We're going to do a series of exercises they could throw for three or four minutes as well, so that all you're doing the activities that are going to be the things that are going to provide the mechanical damage, the fatigue-based damage, so that you load the exact ligaments that we're trying to target the nutrients to. For our long distance runners, they do five to six minutes of jump rope because if you have a history of tibial stress fractures or hip stress fractures or Achilles problems or plantar fasciitis, all of those structures are going to be loaded by the jump rope. They're going to get just enough of a stimulus in that six minutes to have a response. The cells are going to get a response. And then by giving them the supplement an hour beforehand, what we're doing is we're getting them to do their exercise when the nutrients are at their peak.

The reason we do this is because unlike muscle where I can give you a supplement in the next hour to two hours afterwards because muscle has a huge blood flow, so if I give you a supplement after you've done exercise that blood flow is still going to be really, really high for a long period of time, in tendons and in ligaments and in other structures like cartilage, what these tissues are is they're far more like a sponge than like a muscle in the sense of how they're perfused. So a tendon is a virtually avascular tissue. It has almost no blood supply. So the way that it gets nutrients is when just like a sponge, if you squeeze out a sponge and then you put it back into an environment where there's a liquid, it'll expand again and it'll suck nutrients in from the environment. That's what's going to happen in your cartilage, your ligaments and your tendons. Every time that you impact the ground or load the tendon, a lot of water is squeezed out, and then as it recovers that water is sucked back into the matrix and with it

comes any nutrient that's in the blood and in the extracellular fluid that you have at the moment. So if we want to get something into our cartilage, into our tendon, into our ligament, we want to get it in beforehand. Bone and muscle, they have really good vascular supply so that they get good blood supply. If you have an injury, and a lot of the bone injuries occur in places where you have poor blood supply, again, you're going to want to be bathing that area with the nutrients when you're doing your impact, okay?

So a lot of what we're trying to do is we're trying to, yes, it's preventative because we can target things before they get an injury, it can also help in recovery, but what we're trying to do is we're trying to use our activity as a way to target where we're sending our supplement. If you do all-over-body work, if you're somebody who uses shoulders and knees and all of these things, then maybe we're going to have to modulate how much of the nutrients you get beforehand. But right now, if you're mostly a leg-based athlete or mostly an arm-based athlete, we can target the nutrients really well and that we can prevent a lot of injuries.

Danny Lennon: Yeah, that's just super-impressive. And I think just from a practical perspective just to get it right, when you're talking about those doses you're using, one, is it still that 15-gram dose that you use in the research study that you're using, and two, do you use vitamin C along with that or can that be taken in any time, say, via the diet as well?

Keith Baar: So what we tended to do is we used squash. In this case, we used orange squash because it has vitamin C, about 50 milligrams of vitamin C in it in a single dose, which is what we gave to the subjects. So the vitamin C isn't something that we're trying to get at a very high level. We're just trying to get a permissive level of vitamin C and it seems like any low level of vitamin C is sufficient. You can get it from the diet. So when I do the gelatin for…I have a 9-year-old who is athletic, so I give her…I make it up into jello, I make it up actually into gummy bears and all these different shapes, and most of the time I'm making it up with a juice that has vitamin C in it, and it just has to be about 50 milligrams per, you know, within the serving. So like I said, it doesn't have to be very much. So if you're taking it in in the diet, if you're just making up a pre-exercise concoction that's going to be your pre-workout drink, if you've got something in it like if you've got caffeine in it or if you've got other things in it, if you've got beetroot because you're trying to get a really high-quality workout, all of those things are fine. Having a little bit of vitamin C in there is not going to cause too much of a problem.

As far as the done right now, the dose that we did in the 15 grams—so all we've tested so far in humans is 5 and 15 grams. The 5 grams has been effective as far as anecdotal work with people just using it before we have had our study results looking at the 15 and the 5. So we're not certain as to exactly how much of a dose we need to give, whether it's going to scale with body weight. So do we need to say, "Okay, so if you're a bigger person you need to take in more, if you're a smaller person you can take in less?" We don't know those things. Those are still things that we're in the process of studying.

Danny Lennon: Super-interesting. I just think there's so much to this that's coming out and it's really fascinating like I've said and really interesting to see where this develops with future research. Before we get to the final question of the show, Keith, maybe can you just let people know if they can track you down on social media or Twitter, that type of thing, or if there's a ResearchGate profile that they can find more of your research on?

Keith Baar: Yeah. So you can follow me on Twitter at @musclescience. So I started very early, so I have a really good handle. So it's just @musclescience, all one word. And then, yeah, I do have a ResearchGate profile just as Keith Baar—it's with two A's—and so you'd see most of the work that I've done there. You can also look on the Gatorade Sports Science Institute website because I've done a number of different things for coaches and athletes on this and other topics, so there are some simple reviews there as well that people can use.

Danny Lennon: Excellent. And for everyone listening, I will of course link up to all of those things in the show notes to this episode so you can go and check all of that out. And Keith, that brings us to the final question I always end the show and this can be to do with anything outside of what we've discussed today and it is quite a broad generic question, so apologies if it catches you off-guard, but it's simply, if you could advise people to do one thing each day that would have some positive benefit on any aspect of their life, what would that one thing be?

Keith Baar: Yeah, so the easiest thing to say here is to exercise and it doesn't need to be high-end training, it just needs to be training. So that's the easiest thing. I would also say the most important thing is to learn something new, and the third thing I would say is to have a really high-quality dark chocolate that's epicatechin-rich because I think that that's also something that's going to provide things. Especially as you start getting older, it's going to

actually provide things that are going to help you be able to perform longer in your life.

Danny Lennon: Awesome. Keith, I just want to say thank you so much for all the amazing information today that you've put out. As I've said in a number of times, I just find this whole area of research quite fascinating and it's really interesting to see how it's going to develop and it's exciting to see just the practical applications that are available with it right now for athletes. So thank you for your information today. Thank you for your time and for the continued great work that you're doing within science.

Keith Baar: You're welcome. It's a pleasure.

Danny Lennon: So there we go. That was our episode for today. Remember, the show notes are going to be at SigmaNutrition.com/episode143 where not only can you get a transcript but you'll also get links to all of Dr. Baar's research, to find more in different areas that has published on.

For anyone, again, interested in the comprehensive personalized online coaching for performance nutrition that we provide as well as strength and conditioning and powerlifting, then check out the details at Sigma Nutrition and Performance by going to SigmaNutrition.com and just clicking on the Online Coaching tab, which will be in the menu. I'll also put a link to it in the show notes if you want to click directly through and check out some of that information.

And that brings this episode to a close. I really hope you enjoyed the episode in getting into some of the details around this physiology in the same way that I did. If you did enjoy it, then as always I'm extremely grateful if you share the show on social media or if you tell someone about the show or if you leave a review on iTunes, for example. And so with that, I will talk to you all next week in our next episode, and until then, enjoy.