Hypertrophy Past and Present (59) Hypertrophy is muscle-fibre specific

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SUMMARY

In this episode of "Hypertrophy Past and Present," the hosts discuss a classic workout plan from Alan Steven, a former Mr. America from the 1940s. They emphasize the effectiveness of sticking to the same exercises for extended periods, challenging the modern notion that constant variation is necessary for progress. The conversation also delves into the physiological aspects of hypertrophy, particularly focusing on muscle fiber-specific growth and the implications for training programs.

- *Alan Steven's workout plan consisted of 10 exercises performed three times a week for four years without variation, leading to significant strength gains.*
- *The hosts argue against the common belief that exercise variation is essential for progress, highlighting that consistent training can yield substantial results.*
- *They emphasize the importance of muscle fiber-specific hypertrophy, suggesting that understanding this can lead to more effective training programs.*
- *The conversation touches on the role of fatigue mechanisms in training, explaining how they can impact muscle activation and growth.*
- *The hosts advocate for a structured approach to training that includes multiple exercises for each muscle group, performed frequently to maximize hypertrophy.*
- *They highlight the need for creativity in programming while maintaining the foundational principles of effective training.*
- *The episode concludes with a call for listeners to engage with the material and consider how it applies to their own training practices.*

Welcome back. Thank you for joining us for another episode of Hypertrophy Past and Present. Chris, how are you doing today? >> I'm doing okay. I'm not I haven't got very good Wi-Fi at the moment. So if I drop out, I will come back on again as soon as I've as soon as I can. But you might notice the listener might notice occasional kind of hiccups as I appear and disappear. it's basically just cuz we're having a big storm or we've had a big storm and the and it knocked the Wi-Fi out. So, other than that, I'm doing well.

>> I will do my best editing job. I I must have trauma from this because you paused for like half a second before you answered how you were and immediately I was like, "Oh no, he's gone. We've we've just started and you're gone." But no, you're still here. I'm I'm being a little bit pessimistic. So, welcome. Great. We What are we talking about today? We are talking about You know what? Before we even launch into the topic for today, the the workout for today, I think this is a really interesting workout. We're talking about Alan Steven. We've talked about Alan Steven before. not for a long time. I think only one of his plans in the past, he was a former Mr.

America. We're talking 1940s, 1950s. One thing I really like about this workout plan, and this is a me thing, okay, so it's what's really irked me lately is this idea going around online that you you cannot do the same exercises. you need to keep changing exercises and obviously we've kind of like pushed that conversation a little bit further you know I guess into the general kind of domain and people now understand okay I don't need to change an exercise every 4 weeks but there still seems to be this idea that I still need to change it at some point I still can't do it for you know 6 months or I can't do it for a year like it maybe I can do it for more than one phase but I can't do it for two or I can't do it for four can't do it for six and I came across this workout from Alan even and I just laughed to myself cuz Allan says this is the workout I did for the first four years that I was training. I didn't change anything. I didn't change a single exercise. I kept it exactly like this for 4 years and he got really strong like he did I mean obviously bodybuilder as well. He looked amazing but just to give you guys a little bit of idea as to how he continued to progress over that time he started lifting his overhead. He was doing nothing like 30 kilos or something like that. And he progressed to the point of doing over 130 kilos for military press.

And he kept this exercise. He did not change it. He didn't, you know, use variations to work on sticking points or anything like that. He didn't get sore joints and sore connected tissues and whatever else. He just did the same exercises three times per week like everyone else back then and got really strong and really muscular. So this I love this plan. This is classic classic silver era. This is what good bodybuilding plans look like. So let's jump in. And Alan Steven, we have talked about him in the past. The former workout we talked about of his was really interesting. It was kind of like a really early version of classes. So if you do want to go back and find that episode, it was a it's a pretty fun way of training. But this was a 1940s plan, late 1940s. So we're, you know, talking kind of, I would say, relatively early silver era and it's just classic silver era plan we've got here. So three times per week, we have 10 exercises if we include the pullover, which actually we should include the pullover because he didn't do it after the squat like they generally did. So he was actually training the pullover. Usually they would do the pullover after the squat and they do it light and you know it probably wasn't really much of an exercise to be honest, but here he actually was training the pullover. So he started with a barbell military press. He then went into his barbell pullover and then barbell curl. So supernated curl and then prone press. So that'll be a bench, a barbell bench, and then a straight arm fly. So dumbbell fly, a squat, which you know what? I'm going to pause and make a comment here. When you guys see people talk about in the silver era, this gets really confusing.

because often they'll call them laterals and they'll call them like lying laterals, flat laterals, bent over laterals, or bent arm laterals, straight arm laterals, and sometimes I mean flies, sometimes they mean lateral raises. It gets really tricky. So what I try to do is find photos of that person doing that exercise. So here he was in fact doing a dumbbell fly. Then he went into the squat, went into a deadlift, dips, barbell bend over row, and then concluded with a barbell reverse curl.

10 exercises. I think there's a a fair bit here that's quite interesting. So, I'm interested what jumps out to you, and then I'm going to mention a couple of things I think are interesting here. >> So, just kind of setting the framework for this. This is 10 exercises. and he's doing these as a full body routine three times per week. >> That would help. I should say how often and how many sets. He was doing three times per week and he was doing three sets of each. and I don't believe he he he went up to 10 repetitions. I don't believe he specified the lower end because obviously they tended to do double progression method. I don't know if he was going six to

10 or if he was doing 8 to 10. I'm not sure on what the low end was there, but he went up to 10.

So there's there's things I like about this program. I mean, I like the I like the framework that and I like the context. So I I'm also a fan of just kind of finding a program that works and sticking with it for a long period of time. my experiences have generally been that that is far and away the best way to get strong in specific lifts. I did it for my power lifts, which are not particularly impressive at all by any means. And I did it for my dip and my kind of weighted chin-up, which are a little bit more interesting. you know, longtime listeners will know I got my weight to pull up to a little over body weight. So, I think I was weighing in at about 72 and a half kilos. I was kind of doing weight to pull-ups with 75. my weight to dip time was about 82 and a half. so you know, not amazing by today's standards, but kind of 15 years ago, you know, that was not too bad. and I literally just trained exactly the same kind of protocol. I was just doing, you know, sort of, I don't know, maybe two or three sets of maybe three to five reps two or three times a week. And I just literally did the same thing for a couple of years. And I did, as everyone knows, Heburn for my powerlifting stuff.

And I just did that literally for two years. So I think that you know Alan Steven here has really nailed how to you know get strong over long periods of time and also you know add muscle mass along the way. So definitely supporting this this kind of context of it >> just the exercises. I think that ultimately I I'm just going to comment a little bit on what I see as being different from a lot of the other kind of programs we've looked at.

>> There's more chest work in here than I think I've seen in most other silver era programs. So, we've got chest fly. >> So, that's a lot. And if he's doing three sets of each, that is a lot. It really is a lot. So, I would guess that the bench press probably had a slightly narrower grip if he's making that work. That would be my guess. the >> one thing I know about Allan is he actually did change not like from workout to workout but in later workouts he did various grips. So I believe he actually would do three sets and he would do one set like quite wide, one set a little bit like halfway, one set narrow.

>> Okay. >> Yeah. I don't think he did that here. He did that with dumbbells. Obvious well I say obviously he did that later on with dumbbells but I'm not sure what he was doing in in this workout. >> Cool. and then obviously we've got a deadlift in here, which we don't always get. So, we don't always have any kind of hamstrings stimulus at all in a lot of the silver era stuff. so those are the things I think are jumping out at me as being immediately quite different. in terms of back work, I think we're still limited, which is a general feature of silver era programs. No, bent over row. Not a fan of the exercise by any means. I think it's hugely unstable and doesn't really target what people want it to target.

but we've got a pullover in here that's actually functioning as a pullover, which might give you a little bit of that stimulus. So, you know, it's not it's not quite as bad as a lot of other routines, but you know, the rest of it, it's really good. It's really solid. I mean, it's interesting to see two different curl variations that are actually targeting different stuff. which is nice. You know, >> you made a comment before we started recording about how, you know, we're going to find out one day that all these guys were just doing pull-ups and they have never told us.

>> And one thing that is actually funny on that comment, I tell you this. >> Well, so Allan, I've seen Allan say to

quote that the best exercise he did for developing his back was a behind the neck chin-up. >> And yet it's not in the past. >> Nobody ever lists them. Yeah. I I genuinely think that there's there's something weird going on that means that like they were all kind of only ever writing down the exercises they did with barbells or something. It's like >> if it's body weight exercise it doesn't count it just doesn't get listed and I think that's probably there's probably something in there, you know. but anyway, that's that's kind of the observations I got. I I I like it as a program. I like the fact that he's doing three times a week you know, one three sets. I like the fact it's a full body routine. I mean all of the the big boxes have been ticked from my point of view and it's a solid set of exercises. I mean he's going to make a lot of progress on that over a long period of time which clearly he did as you as you pointed out. So what are you seeing there?

>> So I find it interesting that this is in some ways is kind of like a beginner and intermediate plan and I guess almost early advanced. So he's, you know, obviously stuck with this plan for four years and got to the to the point that many people would never even get to in terms of strength and physique wise. So really he's used this one plan for beginner, intermediate, and maybe early advanced. And even as a beginner plan, we have, for example, you touched on it, a supernated curl and a reverse curl, you know, so we've got and you know, if you look at chest, we've got, I guess, the straight arm flies kind of doing similar things to the bench, but you know, if we're getting some chest involvement then the pullover as well, like he's kind of got these exercises selected for, you know, different regional purposes for different muscles.

And I think that that's an interesting observation in and of itself because obviously there's this idea that while beginners, you start with fewer exercises. And I think there's merit to that. I don't think a beginner, not that they shouldn't do more exercises, but obviously there's a steeper learning curve. So I understand why someone would start start with fewer exercises. But I find it interesting that as a, you know, technically speaking beginner plan to at least begin with. He had the bend over row, but he still had reverse curls. He still had supernated curls. So this idea that, well, you just need, you know, a row and you don't need to do any other arm work. It's like, well, no, not only did he have a row and arm work, he had two specific elbow flexor exercises that were very different. I think that's quite interesting. And obviously the chest work as well. you know, even the dips, like he's got the military press. A lot of people were using that as a primary triceps exercise back in the silver era. He's also got the dips in there. So, he's really got by and large kind of two exercises for most of the muscles that he's focusing on. you know, obviously hams are neglected, calves are neglected, so lower body, you know, a little bit less. But for the upper body, he's he's kind of generally doing two exercises per muscle.

Yeah. Know, that's that's a really good point. I mean, the fact that he did start it with, you know, such low starting weights and then take it all the way through to, you know, something that would be superhuman, I think, is is pretty cool. it's a really good demonstration. you know, and yet we've got people running around who will never get close to those kind of numbers, you know, screaming that they need to periodize and, you know, have all kinds of weird body part splits and, you know, kind of train with >> various different methods and training techniques and and he's just there, you know, banging away in the background just doing the same thing all the time >> and overtaking everybody. I mean, it's it's such a such a great story.

I also think it's interesting where he's placed these curls. So, he's got his his pullover and then the curl and then

he's got the bend over row and then the curl or he's got the bench and then the fly. And it's kind of like this this idea we've seen repeated elsewhere and Dorian obviously did it as well where you kind of have that more, you know, whether it's a single joint or whether it's a whatever an exercise that's using less muscle mass than the exercise before it.

He kind of has that happening a little bit here as well. >> Yes, that's true. No, I didn't see I didn't spot that. That's a good point. >> I think this is a nice plan. Obviously, exercise selection, priorities are different. You know, hamstrings, cars. Okay, there's stuff here that's being neglected. Yes, you want more back worker there. You want your pull-up, but all in all, I think this is a pretty interesting and exciting plan for someone who, you know, pick pick the 10 exercises that are going to do what you want them to do and just nail them. just doing for for years, you know.

>> Yeah. And I think ultimately you've been making this point on social media recently, which is that an exercise doesn't stop working, you know, just because you're not making very good progress on it. It's still doing exactly the same thing that it was doing previously. You know, you're still activating the same muscle fibers and you're still applying mechanical tension to them. And I think so Rob and I have talked about this on the high performance physiology podcast which is the podcast where I talk about athletic related strength training stuff which is that I think that people fundamentally don't go back to the adaptions enough.

They don't think about what's actually stimulating the adaption. They just kind of think, you know, oh well I'm I'm getting stale or they make up and that that's a madeup word. I mean that that literally doesn't mean anything in in physological terms. They go I'm getting stale. I'm not I'm not making progress. I need to do something different. I'm like, >> if what you were doing previously was working, it's still working now. It's just that it's working incredibly slowly. And yeah, you might want to think about adding an extra exercise in if you feel like there's growth that you're not getting that you could be getting. But that analysis takes place based on looking at the gaps in your program. It doesn't start from the I mean if you've covered all your bases then you probably are just fundamentally hitting a plateau and you've just got to take it very very slowly from this point onwards.

>> you know that's just how it is. The stimulus doesn't magically go away just because you feel like you're stale or you feel like you're not making very much progress or you feel like something isn't working. It's like well no nothing's changed you know. So yeah, analyze your program and go, okay, actually I probably should have an extra elbow flexor exercise or I probably should have an extra back exercise or I probably should have an extra shoulder exercise. Fine, not a problem. Maybe this is the time in your training career where you want to add that in. But, you know, if your program doesn't have those gaps, then you're just going to need to carry on doing what you're doing. It's just going to take more time. And I think that's that's just really well illustrated by this program that you brought us today, >> which I wanted to use this plan because you want to talk about muscle fibers, which sounds like a pretty basic thing to talk about on a muscle physiology podcast, but there we go. That's where we're going to go today. And I just thought this is a perfect opportunity to talk about this idea that this guy was hitting the exact same muscle fibers with the exact same exercises, doing the exact same thing for four years, and it still worked. Nothing here stopped working.

Yeah. So, the reason I want to talk about mus is because it's a central component of the stimulating reps model that what you'll find is that most critics sort of ignore it and pretend that they haven't heard it. So they'll end up arguing it's like a straw man version of the model because they don't want to talk about muscle fibers because as soon as they talk about muscle fibers any alternative explanation that they've been kind of constructing just falls over completely. so ultimately what you'll find is that at the moment there's been a movement away from I mean you'll still find people in the fitness industry saying silly things like you know metabolic stress causes hypertrophy or inflammation causes hypertrophy or you know cell swelling causes hypertrophy. You'll still find those people and they're they're kind of dinosaurs really. I mean they're from a forgotten age that isn't really that relevant to today's conversations but they do exist. they're still out there, you know, and they they're just kind of a little bit out of touch with what what the rest of us are talking about. But in terms of the people who are actually in the loop, what you'll find is that they all kind of sit there on their podcasts and they'll go, "Oh, yeah, you know, hypertrophy is just motor equipment to mechanical tension." And then they'll stop.

And the the problem is that people interviewing them never take the next step and go, when you say mechanical tension, where is this mechanical tension? What's producing the mechanical tension? what specific structure is producing the tension and and what is it signaling to happen and what you'll get even if you did ask those questions you'll get a very handwavy word salad answer >> I was going to say to be gracious I have heard some interviewers ask those questions >> but the answers tend to be >> a serious answer >> yeah a little bit like laughter >> and basically they will say oh no no it's just motivation so let me explain why we need to talk about muscle fibers we need to talk animals fibers producing and experiencing mechanical tension and then responding by growing individually. We need to have that conversation because otherwise what happens is that you kind of let these guys get away with it. They kind of go oh it's just motion mechanical tension and they just then handwave themselves onto the next thing that they want to talk about. But the reality is that you can't explain the fundamental findings of the last kind of 20 years of hypertrophy science if you don't talk about single fiber mechanical tension. And the reason for that is because the literature currently shows that if I do three sets of you know 10 rep max or three sets of 25 rep max to failure couple of times a week then I should end up after a couple of months of training. I'm not saying you get the same result after a year of training because I do think they'll be slightly different. But after a couple of months training, you're going to end up broadly in the same kind of place.

You're going to have the same muscle growth from doing, you know, three sets of 10 or even three sets of sort of six to eight versus, you know, kind of three sets of 20 to 25. Now, the problem with that is that the light load training group with 20 to 25 rep maxes is never producing high levels of muscle force. Not at any point. Never. there there's no high levels of muscle force ever being produced. Yes, both groups are producing high levels of motin recruitment. So yes, you can kind of get to the end of a set to failure and you'll be producing relatively high levels of recruitment in both cases.

Okay, I do think that light load's a little bit kind of less on the top end than a heavier load. but you know, it doesn't really matter for this context. For a couple of months training, you're not going to notice the difference. So the point is that you don't get high levels of muscle force in that scenario. You get high levels of recruitment in both scenarios, but you don't get high levels of muscle force. So basically the question is, okay, so you you're talking about mechanical tension, but you can't have high levels of whole muscle force, high levels of whole

muscle tension. And the problem is, well, there's several problems here, but one of the problems here is that the wider fitness industry doesn't understand that effort doesn't equal tension. So they will get to the end of their light load strength training set and they will be kind of, you know, really pushing to get that last rep and they've done a absolute grinder. They've taken 5 seconds to get to the top of the rep. You know, the eyeballs are kind of really bulging out their heads and they're going, "Oh, wow.

Now I've got so much tension in the muscle." I'm like, "No, not really. You you're curling a kind of, you know, 12 kilo dumbbell. You know, you're doing a light load set. There's no there's no real muscle tension there at all. What you're feeling is the sensation of innovation which is produced by the brain in response to central motor command which is creating motor unit recruitment. So what you're feeling is effort. So yeah go ahead say you're producing really high effort because you are congratulations you know great that's good but you're not actually creating creating high levels of muscle tension. Mechanical tension at a muscular level is still pretty low. So when we say high mechanical tension, so you say, you know, hypertrophy is all about motin current plus high mechanical tension.

>> You're talking nonsense. If you're leading people to believe that that is whole muscle tension that is doing it, you don't have high levels of muscle tension. You got massive differences. And this is why you've got you've still got researchers running around trying to find additional mechanisms for muscle growth. They keep going, "Oh, well look, you know, light loads are producing, you know, low muscle tension. Therefore, we've got to have another mechanism. We need cell swelling. We need metabolites.

We need to find some reason in order to justify how light load training produces the same hypertrophy as heavy load training. And they can't do it. And the reason they can't do it is because they're not thinking about single fiber tension. They keep thinking about whole muscle tension. And this ultimately is why we're having this conversation today about single muscle fibers is to try and bring a little bit more light to this issue and get people to understand that the sing that the mechanical tension that we're talking about is single muscle fiber tension. It's got nothing to do with the entire muscle and how much force that the overall muscle produces.

Now before I kind of pause for a moment, I just want to say one final thing on this overall kind of framework. We know that it's not solely high motin recruitment that causes hypertrophy. Now this is really really important because some people will hear what I'm saying and go okay well if it's not if mechanical tension is high in the heavy load group as a at a muscle level and you know whole muscle force is high is low in the light load group. So we got a high force in the high muscle force in the heavy load group and a low muscle force in the light leg group but mechanical but motin recruitment is high in both of those situations. Is it just motin recruitment? Can I just activate the muscle fibers at the top end of the motion pool? I've achieved high recruitment. I've activated all these muscle fibers. And does that now mean that just simply activating the fibers is sufficient to make them grow? No, it's not. How do I know that? We know that because light load movement like power training high velocity fast movement training also causes incredibly high levels of motion probably higher than like those load strength training to failure you don't get any hypertrophy from that so we know and I actually I just as like a way to kind of fix that in people's minds when I first proposed this idea on Twitter as it was then I don't know sort of 10, 11, 12 years ago. I I I I kind of said to people, it's not just recruitment. And some people were

pushing back on me going, "Okay, well, no, I think it is just recruitment. Just activate the fibers that you want them to grow at the top end of the routine pool and they'll grow." And I'm like, "No, it's not because, you know, of these fast movement studies that show you hit high recruitment, but you don't actually get any muscle growth." And I said to them, do do you genu genuinely think that it's just the amount of time that you're spending at a higher level of recruitment that is deter the determining value? Because they were like, oh, well, you know, if you're just doing a vertical jump, a max of vertical jump, then you only hit recruitment a high level for a very short period of time. So, you need a high duration of time at high recruitment. I'm okay.

Okay. Well, jump up and down all day. >> Seriously, do like enough time under. you get about a second worth of of time under attention with a vertical jump. So do a you know count out the number of seconds that you're going to do for a single set to failure. I don't know maybe 12 to 15 seconds. Do do 12 to 15 max effort vertical jumps and tell me before you do the study tell me you think that's going to cause the same amount of hypertrophy as doing a set of squats because I'm all ears. If people genuinely think that's going to happen then I'm all ears. But it's not. But that's the kind of thing that you would have to do. We actually have some animal model studies showing that you know that isn't what happens. We got some really nice animal model studies showing that you compare the same level of activation, same you know kind of duration of time under activation, different velocities which is what I'm going to talk about next. therefore different mechanical tension levels at the fiber level and you get totally different outputs. But that's the reason you know that overall kind of context is the reason I want to talk about single muscle fiber tension today. Does that all make sense?

>> It does. At some point we need to define some terms cuz people will use motor units and they will use muscle fibers and that I think sometimes is used intentionally to confuse people on this topic. >> Sure. I mean basically and if sort of people who work in this area are going to kind of hate me when I say this but this is the easiest way to think about it. basically your motor unit is the motor neuron plus a bunch of muscle fibers stuck on the end. So essentially you recruit switch on a motor unit and signal goes down the motor neuron and it activates all the muscle fibers that are you know kind of connected to that motor neuron. So essentially you've got a group of muscle fibers and that is your motor unit.

when people say, you know, oh, hypertrophy is motor unit specific, what they mean is that the muscle fibers attached to that motor unit are all going to get the same kind of stimulus. That's not 100% true, but let's say it is because it's more or less true. You're getting the kind of same stimulus when that motor unit is switched on. So, you kind of work your way up the motorunit pool as you produce higher levels of central motor command in response to higher levels of of effort. And basically as you switch on those motor units every single fiber attached to that motor neuron you know activates. And so yeah I mean ultimately what I'm going to defend today is that you know hypertro is muscle fiber specific for the mechanical tension reasons but also for the motion reasons.

if somebody wants to argue that hypertro is multi-unit specific. They're not really saying anything different. They're just saying talking about groups of muscle fibers instead of single muscle fibers. Arguably, if you had muscle fibers in different parts of a muscle that were exposed to different shortening velocities or different length tension relationships, you might get different hypertrophy across those fibers despite them being in the

same motor unit. But I'll kind of clarify all of that as we go through with the jumping example you gave. So obviously you've mentioned how it's not just the duration of those fibers being active because you could just jump all day or you could jump for the same amount of time as it would take you to do a set of to failure of of a leg press or whatever. Yeah. But there will be some and I know you haven't gone into mechanical tension much yet but when you do there will be some mechanical tension that will be happening in those occurring in those fibers of that are active in a vertical jump. Is there no amount of vertical jumping where fiber is active that could accumulate enough mechanical tension to have any significant hypertrophy stimulus?

So I think it would depend on the body weight of the person. but you know right at the very very beginning of the movement you might get a tiny blip of mechanical tension. But the thing with concentrics is they very very quickly start to you know get to the point where the crossbridge cycling is is losing the the level of tension that you would need to generate hypert stimulus. But you know we can get into that as we go through.

>> Sure. So what I'd like to do is just walk through why both sarcimeogenesis and transverse fiber hypertry are muscle fiber specific phenomena and I want to do it in that order not because it's kind of well ultimately because it's a lot easier based on the literature to see that sarcomogenesis is fiber specific than it is to see that transverse fiber hypertrophy is specific. I think you can make the case very very strongly for both. But the sarogenesis case is so unbelievably slam dunk that anybody arguing the contrary really has a like a massively uphill struggle to defend their position. So it's useful to kind of start with cycleenesis because actually you kind of having shown that sarcimeogenesis is definitely fiber specific. kind of like well okay then our default sort of assumption for transverse fiber hypertrophy is that it will also be fibers specific because you've just talked about how adding you know myaphbrals on the end of a muscle fiber in the form of a new sarcom is fiber specific why would we assume that transverse fiber hypertrophy is not so I think it's useful to do it this way around even though you know kind of smoenesis is less interesting from a kind of overall fitness industry point of view so when we say smoenesis we're talking as I say adding smears on the end of a muscle fiber and typically speaking smear genenesis will happen in static stretching situations it'll happen in eccentric training situations and to a lesser degree it'll happen in stretch position exercise sort of in the context of strength training as well so that's kind of you know where the where the adaptions are happening now a couple of years ago people were kind of screaming that there's no human studies showing think genesis happens. We've now got those. So, they've stopped screaming about that. I guess they'll go and scream about something else that I've said, you know, and I'm sure that'll never really stop, but you know, that's kind of where we are. So, starting at the beginning, sois is one of those adaptions that has been shown to vary depending on which region of the muscle you're looking at. So, one of my favorite researchers did a Butterfield did a study called the magnitude of muscle strain does not influence serial cycle number adaptions following eccentric exercise. Now, when you read that you kind of go, "Oh, that sounds a bit confusing." But the reality is what the title is alluding to is that it's muscle fiber elongation, not muscle elongation. So, the magnitude of muscle strain does not influence serial sum number reductions. You could kind of see that the the title really wanted to be the magnitude of muscle fiber strain does and the magnitude of muscle muscle strain doesn't influence but you know obviously they probably got a word kind of limit. So ultimately what it shows is that if you look at an individual muscle and how it elongates during an eccentric contraction different regions of the muscle elongate to lesser and greater degrees just according to how the architecture of the muscle is arranged. Most people have a mental image of a muscle as being like a fuser form kind of biceps muscle. You know, they don't really think about how penation works and how it changes

the way in which different muscle fibers and different they don't even think the muscles are compartmentalized which they are. So muscles can have very very different structures and I mean like glute medius is a great example. It's like really clearly delineated sections which are doing very very different things from each other. you know, PEX probably is a better example for most bodybuilders and what they're interested in. But if you look and this study is really cool because what it shows is that the amount of elongation of the individual regions, the fibers in those regions was what determined whether or not you were going to get any sarogenesis in those areas. So you only really got addition of scommers in the areas where muscle fibers were elongating a lot. the whole muscle was obviously elongating but you weren't getting the same sulcom edition in every single fiber of the muscle. You're only getting it in the specific region that displayed large single fiber elongations. And I think for me I mean already I feel like I've built an almost insurmountable case for anybody to argue against. I mean I think this is a colossally important study that nobody really talks about but very very cool demonstration of how similaris is multifiber specific. Is this making sense so far?

>> Yeah. Yep. >> Cool. the next kind of point in the argument I would say really is the entire limb distraction literature. So like most of the psychomeis literature is built on limb distraction which is kind of basically a surgical procedure where you put a little metal cage around a limb and you lengthen the bone. was you know kind of developed for you know kind of helping people who have limb length discrepancies of large magnitudes. ultimately now I think they use it for cosmetic purposes but you know people who want to get taller but you know it is a kind of surgical model that we use in animals often in rabbits and you can kind of elongate a limb and what you'll find is that the muscle is being elongated according to how much you kind of elongate the cage and what you can do is you can see that the amount of the amount of muscle lengthening doesn't actually determine whether or not you get sarcime addition. What matters is whether or not the sarcime length inside the muscle fibers themselves has actually exceeded a certain threshold. If you lengthen the muscle but you don't pull the sarcomas past the threshold length, nothing happens. But when you lengthen the muscle a little bit more and you push past that kind of threshold cycle, suddenly you start getting the adaptions.

If you stop lengthening the muscle and you just leave it at the new long length, nothing happens. You just have added all your sizes in series lengths have gone back to their original level because you've added a whole load of extra sime in and the sam now just sit there doing nothing. Now technically you your muscle at the end of the experiment your muscle could be like 20% longer than it was at the beginning. So the whole muscle is now really elongated but it's not doing anything. It's just sitting there and you're like okay well if whole muscle length like strain is the determining factor then why is the muscle not adding sime in series because it's got nothing to do with the muscle.

It's the sarcime length that matters. So really the distraction literature kind of goes this one step further than the Butterfield study because it goes okay it's not just fiber lengthening it's actually sarcime lengthening that matters and s lengthening is really kind of the driver because it's what determines how much passive tension you're able to create inside that single fiber. Does that make sense? >> Yeah. Yeah. So the so you couldn't actually just assess how much a single fiber is being lengthened and determine whether that's going to cause sarcenesis or not. No, you'd have to actually look at whether or not the threshold length of the individual sarcimees within those and that's where the length tension relationship comes from. And just to kind of be clear, a lot of a lot of people when they talk about length tension relationships, they think that muscles have length tension relationships and

they don't. Sarcom have length tension relationships.

Sarcom inside muscle fibers have length tension relationships. nothing else. You can argue that a muscle might display a kind of force length profile. Okay, fine. But strictly speaking, the actual mechanistic length tension relationship comes from the individual sum. >> And so we're talking inside the muscle fiber, you got these bundles of myaphibr sections within those. So we're talking like heaps of sarcomies within a single single >> the sime the sarime basically is like a kind of contractile unit if you like it's the smallest contractile unit. So ultimately it's going to have a specific length and if you pack more sarcom in series into the mus fiber then each sime then moves to a smaller length. So you can have a muscle fiber that's really quite long and yet if it's got thousands of sares all in series with each other each sar can be quite short and you'd have a relatively short kind of actual resting length. So what you can find is that in some muscles if you got just really really low to circumin series all the way along the muscle fibers inside that muscle the muscle can lengthen quite a lot and actually you don't leave the plateau region as we would call it of the length tension relationship and people are going wow you know this muscle kind of is elongating a long way it must have this you know really pronounced length tension relationship like well no it doesn't because the length tension relationship ultimately comes from the sarcom behavior not from the muscle behavior and that's kind of the really important thing when it comes who comes to sac.

>> Mhm. Yep. >> So in terms of kind of keeping this going, I've mentioned basically the Butterfield study which has regional sarogenesis which shows that you're not talking about whole muscle strain. I talked about distraction which shows that it's a sarcom phenomenon and even within muscle fibers you got to be aware that that can cause things to vary. and as I said at the end of that section, if you look at how if you look at how the if you look at the reason why that sum is important, it's because the sarcom is what determines the passive mechanical tension that's being produced and experienced by the muscle fiber. Now I get a lot of push back or I've had a lot of push back on this in the past.

People have said, "No, no, no, no, no. There's all kinds of other stuff inside muscles that's producing passive tension." I'm like, "Yes and no. There is stuff inside u muscles that produces passive mechanic tension. Yes, we've got like collagen in there. But the reality is that the titan inside a sarcime is what is producing most of it and it's what's producing the relationship. Okay, so let me kind of just clarify what I mean by those two things. it's one thing to argue the relative amounts of passive mechanical tension that you can generate inside a muscle from well not generate but experience as a result of stretching a muscle. And it's another thing entirely to argue the shape of the relationship that we're looking at. And the shape of the relationship is really really clearly caused by Titan. So if you I've got some really nice kind of studies on this, but basically if you look at the single fiber data, you can see that the exact same shapes of passive tension versus sarcime length.

If you graph the amount of force that sad produces as you stretch it and you kind of graph force on the kind of y- axis and then sat length on the x-axis. So what you should see then is just like a a kind of curve going exponentially upwards as you go you know to longer longer circumatics you should find that force goes exponentially upwards. If you can visualize that essentially you'll find that the three curves if you graph whole muscle passive tension intact muscle fiber tension passive tension so when I say intact I mean with the

surrounding collagen endomsium and then if you skin that and take the endomsium off so you've got no collagen at all so the important thing is in these three scenarios whole muscle you've got all the collagen you could possibly cram into a muscle. So it's like everywhere.

It's like not just ends, you've got parramysium, you've got, you know, epimesium, you've got all kinds of fascia, you've got everything going on. You've even got kind of, you know, stuff in between musifiers that perhaps maybe shouldn't be there from fibrosis. So you got all kinds of stuff crammed into that muscle. then you look at intact mus, you've got the endomissium. That's kind of all you've really got at that point. That's actually the one that most people tend to focus on when they argue with me. They go, "Oh, no, no, no. I think that, you know, passive mechanical tension comes from endomsium." Okay, fine. And then they'll argue okay what about you know kind of skinning the muscle fiber and just removing all the all of the endmissium as well. So you kind of you can look at these different scenarios and you can go you know each of them have progressively less and less if you go from whole muscle down to kind of endsium covered muscle fiber intact muscle fiber down to skin muscle fiber. You're basically kind of reducing the amount of collagen you've got from one section to another. And you will find that passive tension does decrease as you kind of strip out all the collagen. But the shape of the curve is identical. It's absolutely 100% identical. So what that tells you is that the underlying shape of the length tense relationship is actually coming from the Titan forces and it's the driver of the actual relationship that we see across the entire kind of muscle versus muscle fiber spectrum down sus.

It's all coming from passive tension created by Titan. It's not coming from other stuff like collagen. that collagen. Yeah, it's giving you a little bit of extra resistance and it's minuscule really compared to little tightens, but it's not changing the shape. The actual generator of kind of mechanical tension from a passive point of view when you're stretching a fiber, you're stretching a whole muscle is coming from Titan. So like I feel this is important because it kind of takes things a step further. It goes you know the reason why the distraction literature shows you that there's a threshold sare length that you have to reach is because Titan is producing the passive tension that is then stimulating simogenesis which the adaption that we're talking about which is the addition of sar on single muscle fibers.

>> Mhm. So, you know, okay, fine. You know, not necessarily something that is, you know, as important as the previous kind of section of the argument, but I feel like it it kind of does help to clarify what what is actually happening when it comes to single fiber passive tension stimulating the addition of sarcom on the end of that fiber. And if you kind of just to kind of round the argument off, I don't talk about my tube literature very much. I think that you can get yourself in a pickle with myotube literature the same way that you can get yourself in a pickle with mtor signaling literature that we talked about last week. If you don't really really know what your limitations are, you can end up in situations where you're making speculations about what's happening in vivo based on what's happening in my tubes and they're really not comparable.

I think that the lactate ideas of when people have done research into lactate and speculated that it might directly stimulate hypertrophy as a way to keep the metabolic stress hypothesis alive. I think they get into trouble with the myotube literature because they don't really take into account the limitations of what they're doing. But that's probably the best example of that going wrong. But you can look at myot tubes being subjected to

mechanical loading in different directions. you can look at them being u subject to longitudinal stretching which is basically kind of what would be experienced by passive kind of mechanical tension in the kind of static stretching or eccentric training kind of situations or you can get transverse loading. So that would be kind of reminiscent of crossbridge formations that we'll talk about later.

and you know what's really cool is that if you stretch my tubes longitudinally they increase in length. whereas you kind of don't do that and you stretch them radially, you stretch them transversely, they they do the opposite. They increase in in in diameter. So, you know, I think that's just like a really useful way to kind of again just confirm. I I wouldn't I wouldn't build a an entire model around that if that was my starting point.

but you know it's useful just to kind of add on the end of a a number of other observations and say oh yeah and also the myotube literature kind of does pretty much the same thing. you know so again an interesting kind of add-on but really for me the region specificity and the distraction literature are where it is most important and that is then supported by the fact that the actual length tense relationship shape comes from Titan which is kind of the thing that stimulates hypertrophy sorry not hypertrophy the thing that stimulates sarogenesis and just to be clear we know it doesn't stimulate hypertrophy because if you look at mouse models which have modified Titan to make it stiffer by just reducing its length. It stimulates cycogenesis, doesn't stimulate hypertrophy.

>> Can you just clarify those two points? So, what does it mean to simulate cycogenesis? Because we're still adding parts to a muscle fiber. How is that different to hypertrophy? >> So, the way that the word hypertrophy is used in the fitness industry is wrong. It's used to refer to an increase in muscle mass, which is not true. the word hypertrophy refers to an increase in the diameter or cross-sectional area of a single muscle fiber. you can increase muscle mass or even whole muscle cross-sectional area by adding sarccom in series which is sarcenesis. So you can make a muscle fiber longer and it will either bulge out like sort of the entire fiber will curve away from the bone if the sire lengths stay the same. If so lengths shorten so that each sum is now resting at short length, what you'll find is that the actual muscle fiber now increases radially without adding myophibrals in parallel because what you've done if you think about it, you've just squeezed a tube kind of to a to a sort of shorter length. It's got to bulge out sideways.

So that kind of causes all kinds of problems in measurement situations. But yeah, basically smear genenis is adding contractile material on the end of a fiber. hypertrophy is adding contractile material inside the existing sums that are already there and just making those kind of larger you know outwards which is radially. >> Yeah. Yeah. But like you said both can just an increase in CSA. >> Oh muscle CSA absolutely. I mean ultimately single I think that psycho modesis can make single muscle fibers appear larger in terms of CSA as well if you don't normalize the length tension relationships when you measure them.

that's the problem. if you if you don't if you if you take two muscle fibers and you keep them the same whole muscle fiber length as in when I say that that's not in physiological terms that's actually in numerical terms. So if you got a like a a 5 cm muscle fiber and you keep it 5 cmters and you add sarccom in series and you keep them both 5 cmters, what you'll find is that the sarcime in series will force the muscle fiber to bunch up and it'll end up getting bigger and that's just just kind of geometry. That's not any other kind of complicated kind of

physiological explanation. That's just geometry. The problem is a lot of physiologists tend to when they refer to muscle fiber length they tend to assume that you've normalized the sarccom for their you know kind of standard lengths. So you you you're just kind of measuring you know the the new muscle fiber and you've you've normalized it. so actually you'll end up with a longer muscle fiber by adding more cycle in series.

>> Okay. I think that's an important point because it is I don't know if you hear this much but it is an argument against how all of this works and against passive tension and against sarcamogenesis is if it was sarcomogenesis we wouldn't be seeing increases in CSA because it's not hypertrophy. So those two things kind of get equated for some reason. So it's helpful that you >> I mean the the Hornberger lab did a really good analysis showing geometrically that if you add sarcime in series then you will actually automatically increase whole muscle cross-sectional area. I mean you know I mean anybody who wants to go up and argue against those guys is welcome to do that but you know just be aware that they're pretty much at the top of their game. I don't I don't think they're you know kind of messing around. So geometrically you would kind of already get to that conclusion if you just thought about it but they've gone actually done a proper analysis to show that it does actually happen. So yeah psychogenosis will increase whole muscle mass it definitely also increases whole muscle cross-sectional area. Hypertrophy is not an increase in whole muscle cross-sectional area or an increase in whole muscle mass even though we use it like that unconventionally incorrectly.

technically fiber hypertrophy is what we should be talking about which is the increasing I mean ultimately just to be clear you can get muscle mass increases and cross-section area increases from hyperlasia in a whole bunch of animal models and probably a little bit in humans as well and that's definitely not hypertrophy. So again you know we need to just be if people want to have the arguments at these levels of detail they probably need to adopt the correct terminology. I'm I'm sympathetic to people who just want to call, you know, like, oh yeah, if you want to hypertrophy your glutes and you need to do these exercises. I'm not going to argue with those people. I'm not going to say, oh, no, you should be using that. No, I'm not going to argue with them. That's that's silly. That's that's just being picky for the sake of making, you know, kind of a point. what I'm saying is that if you want to argue about somogenesis, you know, and hypertrophy and those kind of things, then you need to be adopting the terminology. I think at this point I've been guilty of not doing that in the past, but I think really at the point we are in the fitness industry now where people have a pretty good base understanding although there's a lot of kind of weirdnesses around where people are kind of arguing about things that don't exist because they've heard second and third and fourth and fifth hand versions of things that you know other people like me have created then you kind of you end up in a bit of a weird situation. Yeah, I think really if you want to have this conversation, you need to be separating out fiber hypertrophy from psychogenesis and understand geometrically that both of those can cause an increase in whole muscle cross-sectional area.

>> Cool. So that is really all I wanted to say about somogenesis. I think that's a I think that is a really strong case for arguing that somogenesis is happening on a single muscle fiber level. I think you know mechanistically and the data that we've got make it pretty much undeniable that that is that is what is going on. and so really the next thing that I would then go on to talk about is then say well okay is muscle fiber hypertrophy a single fiber phenomenon.

now this is really important because ultimately what we're saying then is that if you if you say that so I think there are two starting hypotheses. You either hypothesize and when I say hypothesis I literally mean hypothesis. I'm not talking about like models or anything like that. I'm literally just saying hypothesis. I think you either start with the hypothesis that hypertrophy is muscle fiber specific and when I'm say that what I mean is the same way that sarcomogenesis is fiber specific which is to say the hypertrophy happens both because the fiber produces tension and then experiences that and then tells that single fiber to grow. That's the fiber specific model. The other hypothesis you can start from I think is that you've got a whole muscle phenomenon whereby you say okay there is some kind of tension being detected at the whole muscle level like at the tendon and that then I I've actually had conversations with people who've argued that there is there are receptor I mean there are tension receptors at the tendon but that's beside the point.

they argue that those kind of tension receptors at the tendon could then back talk to the muscle fibers and tell them to grow as a result of the whole muscle tension. Now this this hypothesis I think dies immediately when you look at the like load strength training to failure literature that's what I said when I introduced this entire conversation I said if you start by the hypothesis that you think whole muscle tension is somehow triggering muscle growth then you basically have to stand in front of you know the amazing work done by a whole bunch of kind of dedicated hypert researchers for the last 20 years and say well I think all your work is nonsense which obviously is not really true so You can't say that light load strength training failure literature is you know >> is valid and then at the same time you can say oh well you know I think it's whole muscle tension that's causing hypertrophy of individual fibers. You just can't do that. So I think that's that's really the kind of the starting point of of of where this entire conversation kind of comes from. But if I kind of go through the same points that I went through with somogenis with somogenesis I started out by saying look we got this butterfield study that shows regional kind of somogenesis happening as a result of regional fiber strains. Can you do that with single fiber hypertrophy? Well not really but you can get relatively close. So what you what we can't do is look at obviously single fiber forces in vivo.

You can't really do that. What you can look at is single fiber activations. And the reason that's not actually as bad as it sounds is that we know that the primary driver of single fiber force from a crossbridge point of view is the force velocity relationship. So essentially when a muscle fiber is shortening at a slow speed, you are going to get a relatively high single fiber force. If it's shorting at a faster speed, you're gonna get a really slow a really low level of force.

>> Now, other people kind of go, "Oh, no, no, we we want to talk about force you know, kind of length relationships, length relations. We want to talk about rate coding and that kind of stuff." Like, okay, cool. But those factors are really tiny compared with force velocity. They they do make a little difference, but honestly, if you just look at the magnitude of the force velocity relationship, it obliterates everything else. So if you're moving slowly, you're going to find that the fiber is producing high force.

recoding really doesn't change that much in the in the context of the movement velocities that you're going to get in strength training. If you go from a normal strength training repetition, even a max effort repetition at a you know unfatigue in an unfatigued state, so you're doing a 10-minute max and you move the weight as fast as

possible, you're still not moving that very quickly. you know, you compare that with your last repetition, there is a difference, but it's not like massive.

If you compare that heavy strength training repetition with like a fast movement like a throw, the velocities are just utterly completely and utterly different. So you know there's there's a huge range of possibilities that you can get on the force velocity spectrum that are bigger than that we see even in the context of strength train. So basically we've got a situation where force velocity is determining the is the major determinant of crossbridge mechanical tension and crossbridge mechanical tension as I say what you can see is that activation of the fiber and if you assume that all fibers are shortening roughly at the same kind of speed depending on the rep that you're doing you're going to find that activation whether the fiber is activated or not and then can therefore produce cross bridges is the primary determinant of whether those fibers are going to experience mechanic potential they need in order to grow. And we do have relatively good data mapping regional activation to regional growth after strength training. it's not done with EMG, it's done with MRI scans, but it is actually still measuring the activation in specific regions of a muscle and showing that it does associate with long-term growth after training with exactly the same exercise. So you can broadly see that the psychomeogenesis conversation that we started with where they got the really good data from the butter pill study is mapped across into strength training not as accurately because you can't measure the fiber tension in the way that you can measure fiber strains but you can see regional activation tracking and doing the same thing as long-term hypertrophy.

>> Okay. I'm I'm trying to think, you know, there's there's a lot going on here and hopefully people are keeping up, but you know, so far it's I'm I'm trying to play devil's advocate and it's hard to it's hard to actually argue, well, how could it be anything other than >> at a fiber level? >> No, I know. >> We know that's where mechanical tension is occurring. We know where that's where, you know, stretch is occurring.

It's like I don't even know how to argue the opposite at this point. I I think that that really what's happening as I said at the very beginning what's happening is that people who are are arguing with the stimulating reps model as I've presented it physiologically what they do is they pick a couple of things highlights that they can argue with like maybe they'll argue with the fact that I've said in the past I think it's you know kind of five reps the last five reps to fail it's probably the stimul and they'll pick that and they'll just say well that's the stimulating model no it's not that's just the kind of the output number that I think is most likely to reflect what's going on.

The stimulating model is a physiological model that says u muscles will grow when you hit high levels of recruitment so that lots of fibers are activated and those fibers are each experiencing high levels of tension because the muscle as a whole is shortening slowly. If you get those two things to work at the same time then you'll end up with hypert stimulus across the whole muscle. That's the stimulating model. How do you get there? Well, you get there because metabolite related fatigue largely just slows muscle fibers down and that's largely the primary fatigue mechanism and like those train train to fail. So you don't really get reductions in mechanical tension at the fiber level that you would expect to get as a result of other fatigue mechanisms. So ultimately that's how the stimulating model explains the light load strength trained failure literature versus heavy load strength training literature producing the same muscle growth over time. everybody else just waves their hands

in the air. They just go oh well it's motive mechanical tension. like okay now take the next step and tell me where the tension is coming from because I guarantee if you do that you will then actually have to start thinking and you'll end up in this physiological model I've created you won't actually be able to explain it in any other way if you reject it outright and you say well no I think it's whole muscle tension then you are absolutely in big trouble because now you're like those strength training literature just smacks you in the face and says no you're wrong >> so I think what they've got themselves into is a a bit of a a dead end because they kind of they don't want to come out of the dead end and say actually no Chris is because they lose massive amount of face to do that, but they can't extract themselves in any other way. So, they're just going to sit there and pretend that they've got arguments and they don't they don't have any arguments. They're just kind of you know, waving their hands around and just saying tension and and recruitment without actually telling you what is going on because they can't.

Anyway, I don't want to rant about other people because it's not really the purpose. I want to walk through how this stuff actually works. So as I said you you can track the same kind of arguments through from so genesis as you can with the the kind of the hypertrophy stuff fiber hypertrophy stuff. interesting we've got a couple of important other blocks in this in this kind of argument. One of which is that if you are looking at muscle fiber growth in developmental situations or you know kind of maybe even in in myotubes if you stop acting meiosin across British from actually forming then you end up not h having myofaphibbrition. So if you're kind of looking at a growth situation as I say either you're growing myotubes or you're looking at you know kind of sort of in vitro muscle fibers in some other way you're trying to make the the kind of the structures grow then you can activate them that's not too difficult to do u if you block actin kind of formations even though the fibers are activated you don't get the myular edition I think that's a really good kind of sense check on the fact that the crossbridge formations is the thing that we're looking for to stimulate my edition. A lot of people kind of go, "Well, no, it's just activation." We've had this conversation back in the beginning of our episode today. It's not just activation. It can show that by looking at the high velocity work, but if you really needed a like granular sense check that it is crossbridge formations that matter, just look at the literature blocking those and you'll find that even activation won't do the hypert stimulus. You need the crossbridge formations to be able to get that next step. So I think that's a good stand to take.

>> So you made a point a while ago. You said you if you're using say a 10 rep max load or or heavier there's going to be immediately there's going to be some reduction in velocity, right? Like compared to very very light loads. Yeah. But that's going to the I guess reduction velocity is going to increase obviously the heavier it goes and then we're going to see more mechanical tension at a fiber level the heavier and therefore slower that that contractions occurring. Now, if you were to use say an 8 to 10 max load, so there's not very slow slow shortening velocity, but there's some reduction in shortening velocity and you were to perform that with a max effort. So, you were actually trying to perform that velocity as quick as possible. So, there was as much mod recruitment as possible. Could you potentially get some some stimulus in those higher threshold mode units, but it may just be very small amounts in that situation?

>> Yeah, I mean it's the same comment that we that I made when I talked about the vertical jump. Yeah, right at the very beginning. You might get a little bit right there at the very beginning. yeah, I mean ultimately the physiological model, the stimulating rest model is is kind of it's it's a map of the territory, but the map isn't the

territory. Yeah. You know, so the stimulating direct model is designed to give you a framework that physiologically largely explains well, it does explain what's going on, but yeah, you will find situations where it's like, oh well, because you have to accelerate, yeah, you will have that little bit of slow movement velocity right at the beginning of the repetition where you've got that little bit of you know, kind of tension which does go above the threshold necessary to trigger the hypertrophy. Yeah, you will.

physiology is messy. The framework we're working with is messy. This map that I've got is the simplest kind of like explanation that works for you know as much of the thing as possible without completely you know getting to a comp a level of complication that that is just makes it unusable. So, you know, I think yeah, you could get that kind of trailing edge and you know, I've been through the kind of reps to failure the reps in reserve literature before and you can find little studies here and there that kind of make it look like maybe you've got kind of a bit of hypert happening at eight reps in reserve or seven reps in reserve occasionally. And it's like, well, yeah, that's probably what that is. does that mean you're now going to change your entire training model to train seven or eight reps in reserve? No. you know, the the the model still track. It's just, you know, people are just looking for little tiny bits of areas where they can criticize me because >> Yeah, totally. No, I think where there's potential something that's interesting in this I think is when we look at obviously atrophy and obviously we see that occurs very quickly you know over the course of a week we don't really know how small amount of stimulus is needed to prevent that atrophy right like as soon as there's sort of any hypertrophy stimulus I guess we almost buy ourselves this sort of protection window so I just wonder are the opportunities with max effort work if we do have activation even with small amounts of mechanical tension at a fiber level. Is there potential for that to actually sort of stave off some of this atrophy?

>> Yeah. So, I mean, basically that would be kind of like saying, well, you know, if I've been doing heavy squats you know, through the offse, can I can I get away with jump squats in season? I mean, that would be the athletic way of looking at it. >> Yeah. >> And I think that's a really interesting point. I've always been interested in that as a concept. I kind of suspect that I I kind of suspect that the answer will displease both me and and anybody who's arguing against me really.

I think it'll end up being a bit of a lukewarm kind of answer. I I don't think it will be fully protective. I I think that you would probably end up having to do quite a lot of jump squats to make that work. and I think you'd probably end up with a confounding effect of smearogenesis happening from the landing phases being super maximally eentrics. >> So I think it would be quite difficult to do in that content only variation >> to make that work leg press type thing. I don't know but it's something that I have thought about in the past and I think it's it's a totally valid point. So, if you kind of can get to a, you know, maybe sort of training three times a week with some power training type activities, you might be able to get a kind of enough of a stimulus to keep at bay.

>> Obviously, you'd end up doing a bunch more reps than you would have done if you'd just done, you know, kind of single serve squats twice a week. >> Yeah, totally. Yeah. so in terms of keeping this kind of single fiber hypertrophy model going as I said you know starting with the idea that you know hypertrophy does appear to be regional the same way it's talking about genesis is regional we do have kind of a sense check showing that you do need to have crossbridge activation crossbridge formation sorry not just activation in order to create

hypertrophy and really what it comes down to though the the kind of the slam dunk in this in this sequence is the study that came Now around about the time that I was pulling the model together called increased hypertrophic response with increased mechanical load in skeletal muscles receiving identical activity patterns 2015 16 study.

basically it's a rodent study where they did identical activations with electrical stimulus maxim kind of titanic contractions of muscles with under anesthesia and they as I say matched the total activation but one group was experiencing isometric contractions and the other was experiencing high velocity contractions. Now, I've talked on the athletic kind of strength training podcast about how calcium ion accumulation is what determines fiber type shifting and I've explained that comes from the calcium ions that are stimulated by the activation. So, ultimately it's really cool that this study showed that both groups experiencing the same activation durations experienced the same fiber type shifts but totally different hypertrophy. only the slow movement velocities caused hyperty. So what you can see is that this is one of those amazing studies that shows you you've got a velocity dependent response despite activations being absolutely identical and fiber type shifting being absolutely identical. A lot of people tend to assume that fiber type shifts kind of just come along for the ride with hypertrophy. It's not true.

they're absolutely 100% separate adaptions started by completely separate stimuli. So for me that really is kind of the slam dunk. And you know if you then go back to the PSI stuff on sort of moss of fiber kind of single fiber force in force velocity kind of experiments you can see that the actual crossbridge formations are changing number of attached cross bridges are changing according to velocity. So the faster the the movement velocity, the faster the muscle fiber has to shorten. And what you find is that the number of cross bridges that remain attached any one moment time decreases because the detachment rate increases. So basically the fiber the sorry the cross bridges are coming off earlier than they would do if you're moving more slowly. And that really just kind of explains the link between the force velocity relationship and the actual mechanical tension that the individual mus experienced. And I've seen so many people, not so many I suppose really because I tend to just watch when I when I get sent them by Rob, but Rob Rob sends me videos to the time and I've sort of kind of clicked on them and I just navigate away because I can see what the problem is straight away, but so many people like want to argue about the force velocity relationship is not just about, you know, crossbridge formations. Well, it it it pretty much is. there's a few other bits and pieces going on if you want to kind of look at whole kind of multi- joint exercises. But when you're looking at like sort of muscles really it just comes down to crossbridge formations. That's that's really what matters. So it's really cool. We've got the PSSE literature explaining mechanistically you know how that velocity is affecting the mechanical tension produced by single mus. So we can see that as the velocity tapers down towards the end of the set, as we get slower and so we get the higher recruitment because of the effort increasing, we get the velocity loss as a result to metabolite accumulation and we end up producing higher muscle fiber forces through more cross bridges attaching at any one moment in time.

And in case anyone missed, you did say it there. So if you are intentionally slowing down the velocity, well then that's going to be reduction in effort. So there's going to be fewer motor units that are going to be recruited in that. So you can't just be like, oh kind of time and attention, guys, isn't it? I mean, they're still around today. I mean, I don't I think sometimes what happens is, you know, people who don't really know very much about

how the physiology works behind strength training, they kind of find something that somebody was talking about years ago and they go, "Oh yeah, this this idea is great. I should reintroduce it and maybe people will follow me on the internet." I'm like, okay, if the only thing you're interested in is people following you on the internet, great. But honestly, there's literally no physiological reason why you would assume that slowing down a repetition is going to do anything if you know how central motor command is related to effort. You're just dropping motion recruitment levels, as you just said, is not going to get you where you want to go.

U but I don't really want to focus too much on recruitment because I think today is about why single muscle fiber tension both from a passive point of view and active point of view in form of crossbridge formations is important to consider in the context of the stimulating model. We've got to talk about how tension is fiber specific because that's the only way that the kind of the people who are arguing against the model will understand or be encouraged to understand is probably a better way of putting it. Be encouraged to understand that they are not being what's the right word? They are not they are not clearly delineating their position in opposition to the stimulating model.

They are just bleeting complaints and not actually being specific about what those complaints are. You know, so when they complain about five repetitions or when they complain about how it's, you know, oh, it's just tension and recruitment, you know, there's nothing more complicated than that. Well, no, it is quite a lot more complicated than that because you have to defend which hypothesis you want to go with. Do you want to say that it's whole muscle force, in which case you are going to have an uphill struggle with the light load strength training to to failure literature, or are you actually going to go down the single fiber route, in which case you've got to do what I've just done in this podcast and explain why you think that's happening and how it's happening and what that then means for muscle. because one of the things that we got enraveled in one of the arguments we got unraveled in probably the biggest argument I think we've been enraled in was the neuromechanical matching argument where basically we've been pointing out that because hypertrophy is muscle fiber specific you actually are going to end up needing a couple of exercises to maximize muscle growth and you're going to need to know which exercises targeting which regions and the best way to do that is neuromechanical matching. now a lot of people argued against neuromchang matching as a thing which is just silly cuz it's pretty much the only model that exists. And a lot of other people have argued against the idea that different exercises are indeed targeting different muscle fibers which again you know is pretty silly but ultimately different regions of a muscle are going to have different muscle fibers in them.

And you're going to find that because hypertrophy is muscle fiber specific which what I've been talking about today. you are actually going to be training different fibers and just kind of saying oh well no I'm training the muscle. No you're not. You're training muscle fibers. That's why I keep saying to people you're never training a muscle. You're always training muscle fibers. The fact that one muscle fiber in a muscle is being trained and and another muscle fiber if it's not being activated and loaded doesn't know.

>> It's got no idea that you're doing that. And that's really coming back to what we've talked about today. is why it's so important. >> Yeah. I mean, ultimately, the only reason this is still an argument is because people want to defend splits. They want to defend not doing full body ultimately. And, you know, becomes the argument, well,

there's a crossover stimulus. You know, you're doing you're doing your back work and you're doing a pullover and you're getting some chest work in there, chest activation, >> some some chest activation, some chest work, some that that word some is doing so much work in that, it's like, oh, you're getting some. Okay. Now, what does sum mean? What does sum mean? And people think, oh, it means that okay, the fibers are experiencing a smaller stim. It's impossible. The magnitude of stimulus is going to be determined by the force velocity relationship if the fiber is activated in the first place.

>> And that's the problem because people go, "Oh, well, it's just a smaller stimulus, so they can kind of be kept going." No, you're not thinking properly. >> Yep. >> If the vib is not activated, it might as well be in a completely different part of the body. Yeah, it's like they might as well be saying, "Oh, I'm doing I'm doing a chest day. Therefore, my calves are getting some stimulus because I'm standing on the ground." I'm like, "What are you talking about?"

>> Yeah. >> You know. >> Yeah. Yeah. There's no actual logic to that argument because, you know, obviously you look at something like the chest in that example and you got shoulder flexion and extension. It's like, well, half of the chest is doing one thing, the other half is doing the other. can't possibly argue if I just do one of those joint actions the whole chest is somehow going to you know not experience atrophy or it's all going to experience hypertrophy like there's just it just doesn't make any sense but that becomes when people accept ultimately oh okay it has to be things have to be happening at a fiber level or you know there has to be whatever atrophy occurring in a week or you know based on the maintenance literature or you know whatever people will pick up one point and be like Okay, I have to concede that point, but now I need to still find a way to make all this other stuff work.

And that's why we then end up seeing people argue, well, actually maybe every exercise is causing maximum activation in that muscle, right? And you know, I'm seeing people now argue variation is just enjoyment. >> They they have to yeah, they have to work backwards from what their their desired answer is to to then find justifications. So they start arguing silly things like I mean the whole we've I've done we've done a whole podcast on how motin recruitment is not maximal in strength training exercises and I don't want to repeat it here but you know there's a really really good episode on this but it it's not an argument it is you cannot claim that you've got 100% motin crewman at the end of a strength training set in the muscle that you are using. It's absolutely 100% impossible to make that claim. If you literally just look at the voluntary activation data and then work backwards from that, deducting all the reasons why dynamic strength training in a gym is going to involve lower recruitment than the tests in dynamometers. you know dynamometry gives you kind of numbers of 80 to 95%.

which is you know showing a clear deficit to vary activation clear you know you haven't got full recruitment if you then work backwards and go okay well those are done in very stable situations with single muscles in single joint situations if I work backwards from that and I've got you know less stable because I'm I'm not strapped to a chair I've got and I'm laughing because there are actually some science based lifters who pretty much do strap themselves to a chair at this point but you know you kind work backwards from that and say, "Okay, well, I'm not strapped to a chair. I'm using more than one arm or leg at a time. I'm doing kind of, you know, multi- joint

rather than single joint." And you work backwards from that and deduct the fact that you're going to lose recruitment for every one of those things that you do. You're ending up in a situation where you're a lot less you've got a lot less activation at max effort than you would have, you know, kind of and certainly nowhere close to 100%. But as you say, people go, "Oh, well, you know, I can't make my way of training sound justified unless I you know, remove this particular observation. Therefore, I'm just going to argue against observation." And because people just kind of want to follow that person, they just believe it. It's wild.

>> So, I don't know if you got anything else you want to say about the single fiber stuff, but if you're finished with that, maybe what we can conclude with is a bit more kind of rapid fire. what's the application or the the actual practicality part of if hypertrophy is fiber specific what are some things that means so you know obviously we've touched on a few so far but if we could just run through some of those >> well the biggest one really is I well leaving aside the fact that it explains how the literature works which nobody else can do you know ultimately if you say that hyper >> which is an important one so ultimately you're Well, it's the reason I built the model. I built the model to explain how the light load strength training literature works. You know, I mean, ultimately, we've got this really important. I mean, I was kind I was kind of, you know, thinking about all this stuff at the same time as researchers were doing studies showing that three sets to failure with eight, you know, eight to 10 reps or 20 to 25 reps produced the same hypertrophy. And people were running around, you know, waving their hands in the air, you know, screaming that there had to be something, you know, kind of metabolic stress going on because, whole muscle forces were different. And they're right. Whole muscle forces are different. And I was like, well, hang on a minute. Is hypertrophy a whole muscle phenomenon or is it a fiber specific phenomenon?

>> And that was it. That was the start of the whole model. And like 10 years later, people still can't bring themselves to say it because as soon as they say it, they're going to have to go down the same route that I went down. and they're going to end up in the same place as me. and they don't want to do that now because they've got ego demands in the in the equation because they didn't figure it out. Therefore, it can't be right.

>> so but yeah, ultimately the the big reason to kind of think about it is because it explains that literature and nothing else really can do that while maintaining the observations that everybody is making which is that the only things that matter to hypertrophy are recruitment and tension. Okay. Well, then where is the tension? Is it at the whole muscle or is it the single fiber? Because as soon as you say single fiber, you're going to end up having the same set of thought processes that I've just gone through today and you end up with a stimulating reps model. so yeah, I think that's really the big the big deal and everything else just kind of follows from there. So if you think that fiber is if you think that hypertrophy is fiber specific then the big two things that matter then are firstly neuromechanical matching is going to tell you or whatever other motor control model you want to cook up but you know new mechanical matching is the only one that has any kind of validity. It's the only one that's really been tested.

so and I know that people are still bashing it but there's like nine or 10 studies in humans all showing that it's validated. So I don't really know why people are so exercised about it but you know so neon matching as I say it's going to tell you which exercises training which muscle regions but the point of muscle fiber specific

hypertrophy is that we do have to train individual muscle fibers and that means that you do need to have a number of exercises in your program if you want to capture the entirety of muscle growth u that you've got available to you.

If you don't want to do that, if you're just quite happy with a lower level of total growth and you just want to, you know, bang away on four or five exercises, that's absolutely fine. It's just that you've set a different goal. >> But if you want to reach the peak of bodybuilding, natural bodybuilding, sorry, to be clear, then you're probably going to need at least sort of 12 exercises, probably more than that. you know, I think that's the important that's probably the really big practical application. And I think that's one of those things that gets a lot of people a bit aggravated.

>> Yeah. >> Carrying on from that, you can then immediately go well okay so given that we know that from the maintenance literature atrophy happens relatively quickly, you know, within a few days of leaving the hypertrophy stimulus period, then you are also going to need to do those exercises with a relatively high frequency. And that's the one that I think is really sticking in people's kind of is really causing problems for people at the moment. I think that's the one that they're rejecting. No, no, no, no. I want to I want to do, you know, kind of upper lower kind of sort of four total workouts a week. Two two upper, two lower, and I want to do different exercises in each workout. I'm like, well, good luck because you're going to need to do three or four kind of sets just to stay pretty much static if you do that. because you basically train the same muscle fibers, you know, only only kind of once a week at least at the top end of the motiv because different muscle fibers are going to get used in different exercises unless you're literally doing this sort of a machine exercise and a free weight variation that's basically the same. I mean, okay, fine. I mean, that's that's not what I'm talking about. But if you're doing like a white, >> then that brings you back to the first point where you need more exercises if your goal is to maximize the growth in a given muscle.

>> Yeah. So, it's not really going to work out that well. But basically, some people are going, "No, no, no. I want to do, you know, I want to do my pull-ups on that day and I want to do my kind of rows on the other day. And I'm like, well, you know, I don't want to do one one of each, one set of each on both days. I'm like, well, then you're going to find that you're kind of pretty much just spinning your wheels the whole time then. so >> you've turned them into the same exercise because anything unique about that exercise, you've you're you're losing by the time you do that exercise again.

>> I wouldn't phrase it I know why I know why you said that. I wouldn't phrase it quite like that. because I think you're overselling it. yeah, I would I would say you've you've not turned them into the same exercise. You've turned them into a much worse version of one of the exercises. >> The lowest common denominator almost between >> the lowest common denominator. Yeah. Is what's what's getting trained. Nothing else is getting trained. So, if you pick two exercises that are have some pretty good overlap, you know, I don't know, maybe you've got a bench press that's not quite fully transverse plane. It's got a little bit of kind of you're kind of moving towards the sit plane as most people do because it's more comfortable, you know, and then you do a fly on the other day. So, you're doing a bench press which is kind of somewhere between the sagal plane and the transverse plane and you're doing a a kind of a fully transverse plane fly.

you're gonna have basically a kind of somewhere in between exercise is going to be what you end up with. The lowest common denominator between those two. It's probably not the end of the world. >> In contrast, if you were doing a wide grip pull-up and a narrow grip row, you'd be pretty stuffed because those two are training completely different parts of the back. So, I think it really depends on which kind of level of variation you're talking about.

>> Yeah. Yeah. And so as as I say, I think practically the first thing that you kind of come across is exercise selection is really important. The second thing you come across is that frequency of exercise selection is also really important. You can't just kind of stick the exercises anywhere you like during the week. You actually need to be doing them a little bit more frequently. And that really starts to create issues for people who want to make really creative looking programs. and this comes back to the Alan Steven issue really, which is that a really good program is pretty much going to look really, really boring.

and a lot of what gets sold as really good programming today is basically just entertainment, which you know is paralleling what you I mean, I've made this comment in a previous podcast and I got absolutely hammered for it. I said most of what people are doing today when they talk about educating themselves. They're just basically entertaining themselves watching people argue about stuff on, you know, kind of Tik Tok or Instagram. They're not really learning how things work. Like, you want to learn how things work, read the FAQ. you don't, you just kind of want to entertain yourself kind of arguing about something that you don't really understand yet because you haven't done the work to understand it. And I got absolutely hammered for that and I'll probably hammer it saying it again. But it's basically the same thing that you're seeing in programming. People want a really entertaining kind of exciting looking program that's changing all the time. And it's like Alan Steven absolutely you know destroys your kind of progress with his four years of doing the same thing three times a week.

>> Yeah. I was gonna like that's just the I think that's the challenge that we've got with to I mean obviously human nature doesn't change but I think social media tends to facilitate some of the worst tendencies that we have in wanting novelty and excitement and instead of actually just sitting down and grinding through the work that needs to get done. But again you know if if that upsets people then you know just kind of you know treat me as old man yells at cloud and and ignore that bit.

you know >> well with with the programming I have thought to myself before if no one ever sold programming like if that just if we just lived in a world where that didn't happen how would these programs look different >> and I just think they would look >> exactly and what would happen is you would get like one exercise that would change here or there maybe someone would add in an exercise like it would just be this slow evolution of a singular program we just we would not end up with these silly you know four-week phases where you're doing completely different things every time. Yeah. I mean, like even to the point where I actually had this thought yesterday in my home gym, I've got all these unique pieces of equipment. I've got all, you know, 30 different specialty bars and all this stuff and I don't use 95% of it. And I think to myself, you you wouldn't there's no need to use this stuff unless you're going to find the best bar for the whatever exercise and you're going to keep that exercise in forever or more or less forever. There's no value to all this variety. the variety is there to find the best of that thing and keep it.

You don't change it. And you know, obviously I had a different mindset when I set up my home gym because I've, you know, got all this other stuff. But, you know, you look at these, you go back to that first point you brought up where it's like, hey, well, if you want to maximally develop an a muscle, you'll need at least a couple different exercises for that muscle. And then the second point you made was, well, you need to be doing them relatively regularly. And you can't just drop them out. Like, those things are in for good now. And you go back to Allan's plan.

Yeah. Okay. Two exercises for his high priority muscles and he did them three times a week for years. That's what it ends up looking like. >> Exactly. Exactly. you know I think anyway that's I don't I don't want to kind of get too ratty. I'm as I say I'm going to get hammered for this anyway. So you know we are where we are. the the last thing I want to kind of probably add as as a big rock item that kind of follows from the observation that hypertrophy is muscle fiber specific is that now you can look at fatigue mechanisms and predict exactly how they're going to cause problems for you in your strength training programs.

>> Yeah. Because now you can go okay so what do your fatigue mechanisms actually do? Well you've got kind of four main categories of fatigue mechanisms. got two CNS mechanisms and you've got two groups of peripheral mechanisms CNS you've got superpinal and spinal and basically what they do is they gradually reduce access to the highest threshold motor units and work their way down the Henman size principle. So you're basically just going to switch off units at the top and work your way downwards and that is going to then obviously stop those muscle fibers from experiencing any mechanical tension because they won't be activated and you won't get those fibers experiencing hypertrophy. Now people who've listened to that explanation and have listened to my work before are going to be going well yeah obviously. I'm like, well, yeah, but it's not obvious for people who are sitting on the opposite side of the fence going, well, we think whole muscle tension is what's generating muscle growth. Because they're going to be like, oh, well, you know, CNFT just reduces recruitment a little bit. That's no big deal. You still got high muscle tension.

>> And do you see how that works? They they don't understand that you're now getting selective loss of stimulus for specific muscle fibers and that means they're now going to basically have no growth. So, imagine you're in a situation where and a lot of people are in this situation unknowingly. Imagine you're in a situation where you've done you're doing a training program that's got a little bit too much volume and you've got a little bit of accumulated background muscle damage and therefore an inflammatory response and therefore some superpinal CFT and that's just sitting there in the background. Okay. Now, if that's happening, you're basically just reduced access to the top end of the motorunit pool. Every time you now go in the gym, there's no way you can now activate those fibers. Now, you've just got full-on atrophy happening of those fibers at the top end of the motorunit pool. People think that it's all just about a matter of degree. I'm like, oh, I'm just getting a little bit less stimulus, so the whole muscle is not going to get quite the same challenge.

And they use words that are not physiological because they're trying to be vague about what's happening because they don't know. like well no it's not what's happening what's happening is you actually fundamentally lost access to being able to train those fibers now I mean they might as well now be like immobilized this or in space or something there there's no stimulus being they're just going to shrink back to baseline levels >> so

straight away you've got to be aware that CNS is is going to have these very very kind of pronounced effects if you let it kind of stop you from ever training those vibes because you're in a state of accumulative fatigue peripheral fatigue. Then you've kind of again got a whole bunch of interesting stuff going on. You've got calcium related stuff which reduces tension. So we want to try and avoid that. Well again that kind of then starts to change the way we think about doing strength training programs. We don't want to end up with calcium because it reduces tension which reduces the stimulus. So we can now go okay well what types of training going to produce calcium accumulation. Well, it you know, eccentrics, stretch positions, light loads, and you can start to see how those things will then have negative implications for you. Not just from, I tend to talk about that stuff from a post-workout point of view, but that actually is going to create problems within the workout. So, you can kind of go, well, okay, so I don't want to do a stretch position exercise before I do a contracted position one then.

>> But everyone goes, oh, no, no, no, no. You need the you need the stretch. That's where all the stimulus is coming from. Like aside from the fact that what you just said is nonsense, >> it actually isn't a good idea because the contract position you can actually recover from because it doesn't have that calcium. You can do that stretch position exercise afterwards if that's really what you want to do. >> so I think you know it starts to then give you a framework for understanding how fatigue mechanisms are working in the context of your strength training workout. You can now program much much more effectively because you think about the fiber and what it's exposed to.

people thinking in this is why I think hypertroy researchers have never got their heads around how fatigue works in the context of hypert because they can't think in terms of fibers they keep thinking about oh it's the muscle it's the muscle it's muscle the muscle is experiencing the fatigue mechanisms they're like well how does that affect them well I don't know because if I go and look at the fatigue literature they're all talking about single fibers and I don't want to do that I want to think about the whole muscle so I can't really make the fatigue literature fit with what I'm talking about well I man because I think about hypertrophy as a single fiber phenomenon. So now I can use the fatigue literature. This is why I use the fatigue literature and very other very well it's not the only reason it's cuz it actually took me like two or three years to work through it all. And that's just a really tedious process that I wouldn't wish on my worst enemy.

But you know it is a really kind of easy thing to then do if you've got a hypertrophy specific sorry a musc hypertrophy model you can use the pod literature very very easily once you've been through that horrible process of reading it. which I've simplified by giving people the FAQ which nobody wants to read. then ultimately you can kind of now apply that literature very very easily because you've got this very kind of clear framework about how it is impacting on either recruitment as in number of fibers that are activated or potential on a single fiber level.

So, I think >> someone should create an app. >> You know those like there'd be heaps of apps like this and it'll be like read the Bible in a year app and it gives you like passages each day of the year. >> Read the FAQ in a year. Someone who's more apps savvy created put it online and tag us. >> No, please don't. But anyway, so yeah, basically I think those are the big rocks. It's nechanical match. Okay, don't get hung up on the terminology of

new mechanical matching. If you think you've got a better motor control model that determines which muscle fibers are activated in which situations, use that.

But it doesn't matter. The point is muscle fiber, >> there's something to determine which fibers are being used. >> Yeah, exactly. Whatever model you want to use, I think your mechanical matching has the best data behind it. whatever motor control model you want to use, it means that different fibers are being activated in different situations. and therefore you are going to have different regional muscle growth depending on which exercise you do. That means that you're going to need some different exercises. frequency is also going to get affected because of that. So you are now going to need to train those exercises multiple times per week. and then obviously as I say fatigue is going to guide you towards more sensible rep set kind of exercise type selection variables because you're now going to understand how the fatigue mechanisms are negatively affecting the hypertrophy stimulus that you're getting and a very granular way. So I think those are probably the big rocks.

>> Yeah. Yeah. And what's cool is we've come at it from the physiological perspective, right? And then retrospectively we can look back and be like, "Oh, cool. This is how the silver guys made that work. This is what it looks like. >> This is how every single human being on the planet trained before anabolic steroid introduced." Yeah, exactly. Which is a really good sense check. >> And they did it the other way. they've intuited it and they've, you know, just through trial and error found out what worked and they maybe tried to put some language to it and tried to put some physiology to it, but obviously they didn't have that level of understanding and knowledge that we have now. And you know, the fact that we've been able to meet at the same location be like, well, actually what they're doing it did make sense. It does make sense. and and you know the the model even if you if you started by ignoring all this silver era stuff and you just went through the physiology and you went through the model and you tried to write out a program that made sense it's going to end up looking almost identical to what these guys were doing >> more or less. I mean I think I think that there's some sort of science-based lifters that are doing interesting stuff. I've been on I've been on a kind of bit of a rant today, so I might as well just carry on and rant a little bit more. And some of this there's going to be a little bit of a positive rant here and a bit of a negative rant.

so one of the very very few things that gives me a little bit of hope for the human race is when I see people who have taken the model that I've built and then created some really cool practical applications off the back of that. Now, I've talked in the past about how you've done that with your training programs. And I think, you know, they're probably the best training programs out there at the moment, you know, for the the general population who who want to well, I mean, right the way up, I mean, I would say right the way up to competitive bodybuilding really, but you know, for everybody from general population who want to kind of add kind of muscle mass at a recreational level right the way through to competitive bodybuilding. I think you know that's what's cool about the group that you're running and I'm kind of ask you to talk bit about that in a moment but >> what I kind of I also see though is that there are science-based lifters who are doing you know really experimental stuff. So there's people out there who are looking for maximum stability.

They're looking for the ability to train muscles in all kinds of different kind of ranges of motion and and directions and trying to look for ways to create a you know an incredibly comprehensive program. and I think

those guys are doing some really really creative stuff and that that I think is really really brilliant. And I I guess what this little kind of mini observation is about is to say I wish that more people would use my model in that way.

So what I mean by that is that I wish more people would use the model and just kind of start from it and build on it practically. Yeah. What happens what 99.9% of people do and I've I've said this before and I hope people don't think that I'm bitter about this because I'm not. It's just this is just how human beings are. they they steal most of it and then they argue viciferously about why I'm wrong about one 2 3 4% of it that they don't like >> and that achieves nothing. It achieves absolutely nothing because their opinions won't be there in a few years time. 5 10 20 years time they won't be there. The only thing that will be there is a stimulating model. that's that's just how it is because the model is the only thing that is actually you know new and interesting. All the observations that people are making when they go oh no no no I don't agree with Chris about this and I don't agree with Chris about that doesn't matter. Your opinion will be gone in a few years time. Nobody will care. you know but what people will care about is you know do we actually have any practical programming that's built on the back of that? You know, that's why I think, you know, your programs are awesome because they do actually probably produce the most valid but simultaneously practical training program that can be done in a variety of different context even you know in a garage style gym. I think that's really important. but similarly, you know, like other people who are out there who have gone to the absolute extreme of doing like 60 exercises in a workout and they're all kind of, you know, incredibly creative exercises and they're strapped into all kinds of weird positions in I think that's amazing. I think that's really really cool because they're actually starting from a physiological model and they've gone to the absolute extreme and gone, what would this look like if I maximized absolutely everything? I'm like, "This is amazing that you actually you've actually taken my model and done something creative with it and that's just fantastic, >> you know." whereas people who are kind of going, "Oh, well, no, I'm I'm just going to kind of, you know, steal all the Christmas stuff and then argue about 5% of it." I'm like, you know, how do you even look at yourself in the mirror? I mean, like, what are you doing with your life? But anyway, that's just kind of a little bit ranty. I I accept and I apologize to anybody.

>> Well, I actually think I I do think that that's where the the most value that people can can have or produce or give occurs anyway because, you know, I sort of again I don't want to add to the rant too much, but I do look at the industry a lot and I think that there's often there's not a lot of creativity and not a lot of of yeah, I guess creation and sort of, you know, lifegiving that's occurring and there's there's a lot of diminishing and trying to tear people down and that stuff doesn't tend to actually move anything forward. And I think what can move forwards is and you know I've seen this happen a lot and you know sometimes through applications I've made where I take a model and you know might try to use that in certain ways in in programming contexts and then that stuff actually gets adopted and then people realize oh that's a a creative way of using the model or that's a creative you know way of programming that actually makes sense physiologically and then some of those bits you know some of them might be small but they become widely adopted you know and I see people now in you know, science-based lifting kind of world and it's kind of just accepted or this is the way we program this or this is, you know, something that we just all do it this way. it's just become accepted as this is the best way of programming for this thing or, you know, this muscle or whatever. And I look at some of that stuff, I'm like, well, 5 years ago that wasn't accepted. You know, 5 years ago, even even when we were having conversations around your model, that wasn't the way that we were generally programming these things. And now people just

accept, oh, this is the best way of programming XY Z. And so I actually think that's where there's a lot of utility is is go look at the model, think about it laterally, think about it creatively, experiment with some different things in the gym, and that's the stuff that can catch on. You know, that's the stuff that can actually influence people. Hey, this is a better way of programming. is a better way of doing this rather than you know like you're saying hey let's just kick this one particular you know part of the model or this one particular argument or no neuromechanical matching is not the thing that determines you know which muscle fibers are going to be active it's like okay we can we can have some of those academic conversations that's fine but the average person coach or influencer educator who's trying to kind of help people in the industry that's not the stuff that's going to help them by no means is that going to do Yeah. And I mean, as I said earlier, new mechanical matching isn't even part of the stimulating model. It's just, you know, the prevailing motor control model that we've got. So, it doesn't really matter. The point is that whatever motor control model you use, you're still going to be ending up using the stimulator reps model to explain how it works in the context of a strength training program. I just think that your mechanical matching makes more sense than anything else. so yeah but you know let's kind of come back to the to the group because we don't talk about this enough.

>> Yeah. So obviously we've we've launched our first phase of the hypertrophy past and present train heroic group. So that is a group where you can sign up you can be part of a community part of other people following the same program. It's going to be silver well it is a silver era based program. This first phase is doing a Regge Park variation program. Not his sort of classic 5x5, but more of his sort of defining type programming. And it's an opportunity for you to get a good home gym based program, one that you can make some alterations based off your own equipment. You've got access to me in there. You can ask me questions. You can send me form check videos. you know, I'm I'm there to help guide kind of what is going to help you be successful in that.

And then every probably eight weeks or so, we're going to shift to a a slightly different program. Obviously, we're going to keep the the bones the same. So, a lot of the exercise selection is going to stay very similar. you know, based on what we've talked about today, we're going to be programming in a way that makes sense physiologically where we know, hey, hypertrophy is happening at muscle fiber level. We're not going to just throw random exercises at you, but we're going to shift it. So, it is going to be I guess inspired by a different silver era lifter or a different silver era program. And you know that it didn't really come up today in the conversation, but I was going to mention when you said programming is kind of boring, like the the the best programming for natural lifters ends up looking boring. I think in some ways it does, and in some ways there's actually opportunity for there to be a lot of creativity. And I think Alan Steven actually did that well because he kept these exercises the same, but you look at some of his other programs and he's doing clusters, you know, he's doing, you know, 10, 15, 20 sets of of single repetitions or doubled repetitions. And there can be some cool stuff like that where, hey, if we keep exercises the same, we can actually use some really creative other methods that does add variety, does add variation, but it actually keeps all the underlying principles the same.

Yeah, absolutely. I mean, I think that was a great choice that Alan Steven planned today. It was a a really inspired pick. I'm pleased that you you dug that one out. >> So, that was one of our longest episodes. I think hopefully some of you are still here at the end. And I mean, I guess we kind of wanted to cover this because there's so much that actually rests on this particular topic. You know, if you you mentioned, you know, you can either look at it as hypertrophy is happening at a muscle fiber level or it's a whole muscle level. And depending

on where you start with that one question, it will take you down completely different paths. And unless we actually acknowledge what it is we're talking about there and how much that influences everything else, we just end up talking past each other. So hopefully that >> the acknowledgement is the point. The acknowledgement is the point. I think what I'm seeing is that people are just skating past it. You know, they're saying, "Oh, you know, hypertroy is just, you know, motin to mechanical tension and they're skating past it or they're handwaving past it." I'm like, "No, no, no, no. You need to really tell me exactly what you mean by that statement because it matters a lot whether you're saying that you think it's whole muscle force or single fiber force." If you're saying it's single fiber force then we need to go down the route of actually understanding where are you justifying that or how are you justifying that and which is what I've done today and secondly then what does it mean which is what we've done when we've gone through the practical implications and I genuinely think that if people would just kind of pause and think about this issue a little bit more they'd probably end up in the same place that I am which is the stimulating match model.

Well, thank you guys if you made it this far. I know this was a relatively dense episode and maybe maybe next week we'll do a a slightly easier, more accessible episode. Who knows? Maybe it'll be even more dense. I don't know. But thank you if you made it this far and I hope you'll join us again next week for a new topic.