David
Hey, Ryan. So good to see you.

Ryan
Yeah. Great to be back, David. Thanks for having me.

David
Where are you today?

Ryan
Oh, so I'm in Lexington, Kentucky. This is where our lab is located.

David
I got to ask why. Lexington, Kentucky.

Ryan
Is sort of where I was born and raised. Was it a small town in Kentucky where I was born? I came here for my undergrad in medical school and then started a couple companies here, so it's just been a great place for us. It's always a place I love coming home to.

David
Nice. My wife's from that part of the world. We're down there a few times a year.

Ryan
Good time of year. The derby's about to happen. Horse racing is in full swing. And, you know, some bourbon being, you know, released. So it's a good time to be here.

David
Nice. So, Ryan, let's just jump into it. You know, we last spoke a couple years ago about two diagnostic tests, and I know there have been a lot of changes in the test environment. Talk to me a little bit about what you're doing right now.

Ryan
Yeah, absolutely. So I think that, you know, this quest to measure biological age is still obviously our end goal. Right. And so that's what we're always oriented towards. And our growth there has been really impressive, not just our personal sort of company

growth but also the science of measuring biological age. And so we've come a long way and just a few years in creation of new algorithms and how we interpret.

Ryan
I think that if I had to summarize maybe some of the biggest changes, probably since the last time we spoke, I would say that most of the focus has been on explainable algorithms, sort of. Now, instead of just saying you're ten years older or ten years younger and really recommending the same recommendations for every single person. We have ways to make personalized recommendations and see what this heterogeneity in aging is sort of being caused by.

Ryan
We all might have different causes of aging. And now we can sort of isolate and provide more specific recommendations. I would say that's one of the big movements. The other secondary big movement is just focusing on increased precision in factoring out all of the previous errors or problems with the clocks. And then lastly, I think we're starting to get into another new area of the clocks where we are more able to separate disease and aging.

Ryan
So getting a much more purified aging signal and even getting to some causal effects of these clocks, which I think is really exciting. So those are really the four big areas. I think that we've seen some improvements since last time we spoke.

David
When you use the word causal, you're meaning causing like what is causing me to seem to age faster or slower.

Ryan
More so than that, actually, I think with some of the newer clocks that are explainable, we're able to say, hey, this feature is maybe more abnormal and causing an overall higher biological age. But I think that we're also getting into some clocks that have used a process called Mendelian randomization to say these changes in the genome or causal versus correlative.

Ryan
And that is an exciting breakthrough, again, very much at its infancy. But we're starting to see some of those tools developed. Wow.

David

Okay. So that's sort of like through me. So causal versus correlative. That's always the huge thing in so much of like health and wellness is like what's actually doing it. You're able to discriminate that at what level.

Ryan
Yeah. So so at an individual CPG level is sort of what's happening. The best known example of this comes from a lab at Harvard, the clerkship lab. They released some what they call these causal aging clocks and the output of these causal agent clocks, actually, three, it's a it's sort of, adaptive age. What are adaptive changes, maybe to stressors of aging.

Ryan
What are some protective. And then what are the actual causal points of aging. And so those clocks are new. They still I think have room to be improved. But it was the first of its kind, sort of a big breakthrough in using this process of Mendelian randomization to sort of ask the question of, you know, what is that just correlated to these events as we get older versus what is actually precipitating these events as we get older?

David
How are you verifying this?

Ryan
Yeah. So so it's a good question. And I think that it's one that's pervasive among all biological age measurements. Right. At the end of the day, how do we know these work? This is always something I'd like to talk about because it is again is a, I would say, weakness of the industry. Hundreds of clocks out there and not all of them have the same, I would say degree of validation, but validation is absolutely required.

Ryan
And so when we were talking about, you know, first generation early clocks, we were able to validate them by saying how close today to what they're predicting, which is chronological age. So we could say, you know, how far off are we? But whenever we're really trying to capture the biological process, it's really hard to define what the true standard is there.

Ryan
Right? Some people might say the absence of disease. Other people might say total lifespan. You know, others can have a whole host of different true targets. And so generally, I think the rule that we adapt here in the industry is adapted is that we're using and comparing these things by how predictive they are of all disease outcomes.

And that's because we know that aging is the biggest risk factor for almost every chronic disease and death.

Ryan
And so if we can predict outcomes better than previous clocks, we know we're making progress. and so in order to do that, you have to use a biobank which has taken samples from many, many years ago. And then you can test those samples from the time they were collected and then see if you can predict outcomes of those individuals.

Ryan
And so you need big biobank systems in order to effectively prove that. But really, the clocks with the best hazard ratios that are most predictive disease are the ones we recommend using.

David
Okay. So say I take a test, what am I going to learn.

Ryan
A lot and a growing amount. And I think that there is probably some importance right now in separating the testing and all the things that it can do from the age tests and what we can do there. and so our reporting right now does way more than just aging. We can tell you again how likely you are to lose weight with caloric restriction.

Ryan
We can tell you how much you smoke and drink across the lifetime. We give you even performance markers like VO2 Max and and grip strength immune cell subset.

David
If you're getting this from a blood test.

Ryan
Oh, absolutely. Yeah. Yeah. We trained algorithms. Yeah. To do this. Yeah. And one of the definitely one of the big updates that's I think coming in the testing is all the, the other things we can do with this data. So there's a lot that we can do and a lot that you can find out. But in the case of aging, traditionally these age reports have been doing a couple things.

Ryan

They've been telling you your overall age, right. In some cases, they might tell you telomere length. In some cases they might tell you your rate of aging, which we use that you need and pace algorithm. And so so we can give you those things. But the newer things that we've been able to do now are really focused around two algorithms.

Ryan
One we created with Harvard, which is called our Omega age. This clock is able to to actually estimate certain proteins, clinical values and metabolites to sort of say which of these is driving your aging process a little bit more. And so we can actually give now individual resolution on maybe what things you might want to fix in order to improve that.

Ryan
the the other one is one we also are deploying just next week with Yale called Symphony Age. And this is an algorithm that gives you your overall age, but it also breaks it down into individualized system organ age resolution. So we can actually say, you know, your lung system is five years younger versus your your musk in skeletal, which has a two year acceleration.

Ryan
And so now we can start to pinpoint specifically organ systems that that may be, I would say driving aging in a heterogeneous way. And it all comes down to the fact that we all live very different lifestyles. We know that these lifestyles and everything that we do within them are affecting our aging process. And some people might be aging differently.

Ryan
Now we can actually apply an individual resolution to say what's most important for you.

David
So this is very interesting. Now, the last night I got in late because I flew, so I slept like less than I would like to. It versus like in a couple of days I'll be very rested. And what's the variance in this depending on the day.

Ryan
Yeah. It's it's a great question and to be honest, one that I don't have a complete answer to. But one thing I will say is that the answers to that are dependent on the algorithm, because every algorithm is going to be slightly different because each are trained to a different outcome. So for instance, you know, the pace of aging has inclusion of lipids, right?

Ryan
So you're able a apob ratios for probably maybe affect that and maybe not affect as many of the other clocks. And so how they're trained is certainly important to answer that question. Every clock is different. But generally that brings us to the other side of how I would validate these clocks, which is their precision. How good are they at detecting the same result multiple times.

Ryan
So we test the exact same sample. What is the variance? The good news is that we see less than 0.5% variance. If we test the same sample, you know, across multiple batches across multiple time points. So what that tells us is that our technical validity is very, very good. So if we're seeing change, we're seeing change in a biological basis.

Ryan
Right. Which is really important. The question is how much do we sort of oscillate on a biological basis. And that's a hard question to answer. And every clock is different. Actually, a study just two weeks ago came out showing that circadian rhythms might even affect your biological age with sort of peak biological age being later in the evening.

Ryan
So if you're testing, maybe later in the evening, that might be, sort of a problematic confounder. And so the biological variation in oscillation is still a question we need to answer. But right now the technical variation is still very, very good.

David
Wow. Okay. This is super interesting. Based on this. But just talk me through like what sort of results I could look at. If we're talking about organ systems or specific things like what are the actionable things I'm going to be able to do once I get to this test?

Ryan
Yeah, certainly. So so let's just take Omega to start and then we'll maybe get into, you know, some of the system organ aging things. And so to take Omega Omega is an algorithm. Again we created with you. It is when we validated against every other clock the most predictive of death and disease. we are 92% accurate at predicting death within a five year time period.

Ryan

So that's super accurate. Again, I know that five years is a lot of time, but it's still better than than anything that's been published to date that I could find, at least in terms of death prediction or lifespan prediction. And so whenever you take that test, what it comes back with or what we call these epigenetic biomarker proxies, and I'm sorry to introduce a new term, but this is how we predict clinical value.

Ryan
So we can actually predict what your HPA when see is your fasting glucose, your creatinine, your your C-reactive protein. In some cases, these predictors actually even better than the biomarkers that we started with. So our predictor of CRP is more predictive of every health care outcome than even regular CRP. If it's more precise. And so we just think we have a better biomarker of overall inflammation.

Ryan
But that's the individual resolution that you're getting. So you can actually get that back and say, where do I rank among the true diagnostic cohort for my glucose? Is it higher or lower than average? Is my fattening higher. You know. And we start to see some patterns here. So for instance, you know, one pattern that is readily visible is in metabolites.

Ryan
We have some metabolites that we quantify that that can be indicative of things like high blood pressure. So I can sort of now after reading a lot of these reports say you're vanilla. Acetic acid is elevated. Your hydroxy phenols your glutamine is elevated. You probably have high systolic blood pressure and say that might be a process which is driving aging, because we know that those metabolites are correlated definitively to high blood pressure and cardiovascular risk.

Ryan
And so we can start to make really personalized recommendations. This can also get very complicated, very, very quickly, especially because, you know, we we actually quantify 36 individual variables within that omic age, about about ten proteins, about 13 metabolites, and then the remainder being proteins. And the algorithm has selected these things is important, not us. So a lot of these things most people have never heard of, or they might have 1 or 2 publications on, on PubMed.

Ryan
So it's very, you know, avant garde and cutting edge, but we're starting to see connections that can definitely inform us about how you're aging. So let's just say that you have poor metabolic control or maybe an unhealthy diet. We might see that in things that have higher, you know, some things like our fasting glucose measurements or our HPO and CS, or we might see that, you know, other metabolites, which lead us

to maybe suggest things like in Acetylcysteine or, you know, some of those things to help with some of the components we see within processed foods.

Ryan
And so it can get very, very complicated, very, very quickly. On omega age. But we're getting such resolution. It's really, really exciting. And to sort of say these are all different ways we can all age. And this is how we make personalized direct improvements.

David
I have so many questions. So say I'm a practitioner and I've just run a patient's blood okay. Once a year or something like this. How would I use your information in combination with the blood work?

Ryan
Yeah, it's a good question. If you have the blood work, definitely still stay with the blood work because what we're creating is not directly compatible. You know, we're measuring a signal of a particular thing. So for instance, I'll take CRP as a good example. right. Because our CRP is much better. We're not directly measuring CRP. We're measuring the genetic, I would say expression that's associated with CRP levels.

Ryan
So when we're doing that we have different hazard ratios for disease. For instance, our CRP for instance, can our brain phenotypic outcomes 6.4 times better than regular CRP. So it's a great brain outcome biomarker. we also still have better hazard ratios for the other things like diabetes and cardiovascular disease. Things are also markers of CRP, but it's completely different.

Ryan
You know how it changes. We also call our as a little bit more like an HBA one C of inflammation because it's not as immediate really reactive. It might not change as quickly. For instance, it might be sort of a couple week average of that particular thing. So we want to make sure we mentioned first and foremost this is a different biomarker.

Ryan
You know fundamentally a different biomarker. But we are still seeing that whenever we're comparing these associations they have the same associations to disease. So we look at our HBO and see we see that that's, you know, highly related to type two diabetes risk. Right. We see, you know, creatinine highly related to chronic kidney disease. Right. We see, you know, all these these same associations.

Ryan
So we're picking up similar biological signals. But they're different. So we always want to put it into context in the way the context we're using right now is within aging itself. Because all of these things are used in sort of an integrated predictor to predict time until death. and so if you're above average or, you know, let's just say we want something's low.

Ryan
If those are high, we want to take action. And if some things are high which we want low, we also want to take action because we know that as those are improved, you're improving your omega age. And then that algorithm is incredibly predictive of death.

David
Let's go back to actions I take. If my current knee level shows a higher predictor of kidney disease, what is the recommendation that is sent to me as an action? I should take?

Ryan
We try and stay pretty broad so we're not giving direct medical advice, but we have what we call sort of these epigenetic biomarker cheat sheets. And these these epigenetic biomarker genes is actually directly say what has our known associations if each biomarker and then what are classical recommendations to change that particular biomarker. We can go through these for every single one.

Ryan
But in the terms of creatinine, right. In terms of improving kidney health, we really recommend a few things, right. We recommend increased hydration, right. To make sure that your your, you know, have enough kidneys to filter. We recommend, you know, avoiding any types of medications. Right. That might be never toxic or sort of looking into that we, you know, recommend trying to reduce blood pressure.

Ryan
And the best ways to do that are obviously through improved exercise and other dietary changes. And so those are the types of recommendations we give is investigate this or make these broad general lifestyle changes that have been known to be healthy for that particular outcome.

David
And where do you see these tests going?

Ryan
Yeah, yeah, I think at one point in time we'll be able to consolidate a large proportion, maybe 60 or 70% of all clinical biomarkers with just this one test. and, and I think that that's very, very exciting. Again, we have to prove out that each of these individual biomarkers is effective for for what the classical has been used for.

Ryan
But I think that's certainly where we're going. I think that in aging in particular, the more resolution we get, the better and the more again, we can say that resolution is correlative or causative and make specific recommendations for better. So so just like over the last few years, we've now seen improve precision. We've seen, you know, removing confounding factors which might affect the results.

Ryan
We've seen now resolution of the why you're aging. I think we're still going to go more in that direction for biological age to really identify causes. And then to say, this is what you need to focus on for optimized lifespan or health span.

David
So I have a question. This is this might be crazy, but so what I'm understanding here is that if I have my regular bloodwork done, I'll just make something up so my LDL is high or something. So that would lead to someone saying, okay, you probably need to bring this down because you're going to have these Z problems.

David
If I understand your test, what you're saying is it doesn't matter. The absolute level of the LDL, it's the epigenetic effect of the LDL. So some people could have, you know, higher LDL with with very low impact. Did I get that right?

Ryan
Yes and no. So so I think that that that we're certainly measuring the epigenetic signal of that particular event. We're not necessarily always saying that the epigenetic signal is more predictive than other biomarker. And so in the case, there's another published study out there from a, from the I'll pretty much the only other person who's creating these surrogates who DNA methylation.

Ryan
His name is Ricardo Mariani. He's from the UK and he's worked on things like GDF 15 or Probnp. These biomarkers, he can predict through DNA methylation. And he's

saying, do they work as well in other categories? In some cases the answer is yes. Other cases no. And so so for instance, you know, our triglyceride predictor is pretty awful.

Ryan
It's not really accurate. And it shows different trends. Our triglycerides to predictions tend to go down across the population while regulatory tech goes right in the go up. So we might not be getting the best signal of that process. Epigenetics. And so it might not be a good biomarker or one that's that predictive event. But with that being said, we can start to pick and choose, I think, based on what is the most predictive of outcome, creating new biomarkers with new data.

Ryan
But but sort of starting in jump starting these new predictors based on things that we already know are important, like the measures that are used in clinical settings.

David
Okay. So now because you've done a lot of these and at a population level, and so you've seen what certain groups of people have been doing to change their biological age. So if I I'm just going to like give you a few examples. And you can if you want, you can just say, oh, we see positive or negative.

David
Let's talk about min or GnRH, this sort of stuff.

Ryan
Yeah. So within a minute in our at a population level we don't see much change quite frankly. So we haven't done an interventional trial on just in a man or in R. It's hard to say directly, but if we do like a cross-sectional analysis of people who are taking those products within our large cohort now, we generally don't see a huge difference.

Ryan
So I would say that it's been a little bit disappointing, but I think that some of the additional data in those areas are also showing that, you know, there are differential responses and even things like it might be negative in some cases. There was a a recent study to show that, you know, some of the metabolites can accumulate in the kidneys, which can cause a negative kidney related outcomes.

Ryan

There was another study that showed the metabolites could increase even cardiovascular risk as well. And so I think that we might not have enough resolution because of our data sets to pick up on any clear, specific change.

David
All right. Another one sort of in that category like resveratrol.

Ryan
Yeah. Resveratrol again we don't have any direct studies but cross-sectional. And we are certainly not seeing an impact. I think that we probably wouldn't expect too with some of the other data coming out there. I'm not too hopeful for that one. But we are about to release a trial that's been led by Yale and it should be released, I would say, in the next eight weeks or so as a preprint, which will actually combine every longitudinal epigenetic study in an analysis of biological age.

Ryan
and so we're really excited to, to release that, to actually start to answer some questions that will go over things like, you know, B12 and folic acid supplementation, umbilical cord plasma, exercise, diet, radiotherapy, metformin, Covid, syphilitic treatments, the Mediterranean diets, dementia and several others. And so we're really excited to release that as well.

David
When you say longitudinal study, how many people are we talking.

Ryan
Across the multiple different studies? You know, tens of thousands is what we'd be getting at each study individually might be pretty low powered. You know, we published one on that and even quercetin, which only had 21 patients. But even then we were able to find some really unique things in some of those data sets. And so this will be, you know, we'll include hyperbaric oxygen, we'll include supplements, we'll include gene therapies even in this analysis.

Ryan
So we're really excited to to put this all together in one comprehensive analysis and sort of show what's working and what doesn't.

David
At a population level up again, not personalized.

Ryan
Well, these will actually be direct interventional studies. so we don't have to do cross-sectional. We're doing, hey, how have these individual patients change over time?

David
I see okay, I'm going to ask you some more here. Everybody's favorite rapamycin. What are you seeing there.

Ryan
If you take it long enough positive effects. And we're seeing here we actually do have some direct interventional data. And so we're seeing that that over the course of six months probably not a lot of change. but if you're someone who's been taking it for several years, then we're seeing more of an effect and benefit and so I would say it's positive but slow to act.

Ryan
Interestingly enough, we we're now comparing some of our initial data sets at different dosages of rapamycin two. And it's it's too early to speculate or make a recommendation on dose. But what we are seeing is that between 6 and 10mg we're getting remarkably different epigenetic profile differences.

David
Good or bad.

Ryan
We don't know. It's hard to interpret. I would say at the moment the I would just say that, you know, one of the metrics we look at in these interventional trials or what gene regions change and even what immune cell subsets are changing, especially with rapamycin. That's something people worry about as it can be immunosuppressive. And I can say that that the immune differences have you seen it at ten and six milligrams is very different, as well as just what methylation regions are changing in a significant fashion.

Ryan
And so it's certainly a question that we hope to to dig into a little bit further. But the really long term large scale data are going to take a little bit while longer to collect. And so we might I think, in this Yale study include that rapamycin data preliminarily, but definitely differential responses in women. Excellent. Generally especially, you know, after menopause.

Ryan
There have been several studies also to show that the testosterone to estradiol ratio generally improves neurodegeneration clocks like remains. So I think that we're getting certainly some positive outcomes there that are pretty definitive.

David
Anything else that's really moving the needle that that I don't know about?

Ryan
Yeah, I think we're certainly getting ready. Data in the umbilical plasma the young plasma. We're seeing some benefits. But I would say minor benefits with plasma exchange, you know, sort of filtering out that plasma. You know, I think that we are seeing, again, a lot of intuitive things, right? Things that time and time again always pop up, you know, exercise it particularly, you know, mixtures of strength and endurance related exercise or improvements, vitamin D, you know, DHEA.

Ryan
interestingly enough, in one of our analysis of that, the Multi-omics data that we created with Harvard, we're seeing even some supplements, like earthing in some of those mitochondrial supplements, which have been postulated to have some big effects on aging, also coming out at the top of our list. So Orchid Dining is probably one of those newer ones that I think is super exciting, fascinating.

David
And this large study that you mentioned, when is that coming out and in what form?

Ryan
So it'll be a pre-print, probably released sometime in the next eight weeks with collaborators at Yale who have been taking the lead on that. We've contributed quite a bit of our longitudinal data sets in a variety of interventions, and so that should be sort of right around the corner.

David
And for someone who's interested in taking these tests, you know, we mentioned about what the biological variability is. So I take a test and I've been given, you know, a bunch of recommendations to improve myself in some way. What's the time interval. The one test again to see like did this really work.

Ryan

Yeah. We still generally recommend no more frequently than six months for most algorithms. There are few algorithms. So we're in since we did, I think probably since the last time we spoke with Stanford that was on Netflix. Vegan versus omnivore, sort of dietary study in twins, we were able to see notable, noticeable and significant differences in just eight weeks in that particular cohort.

Ryan
So so we were able to see, I think, resolution, even with small cohorts and small intervals, but generally on an individualized basis, we do recommend sort of taking that a little bit longer, just so that we can pick up on true biological aging signals and not be, I would say, is confronted with any confounding information that might directly result.

Ryan
So six months is generally, I would say, the frequency we would recommend as the shortest interval. But some people do it even just once per year.

David
What really excites you?

Ryan
Yeah, a lot, I think. I think that biological aging is in certainly always going to be one of our major, major focuses because we fundamentally believe it underlies the development of every chronic disease as the biggest risk factor. But I also think that as a platform, DNA methylation really excites me because we can start to do things like I mentioned, these epigenetic biomarker proxies where we can start to quantify your omega three levels, your sperm reading levels, all these other different things within an integrated data set and test.

Ryan
And so I'm really excited about the ability to start to report on multiple different pieces of biology, to really find connections that we haven't seen before, to maybe answer the bigger question of, you know, what is the most likely process to intervene on to improve biological age? And so I think that that we're starting to get to an area where we can answer those questions a little bit more rapidly, that we can then directly put into clinical translation, right, so we can directly start to say, this is how we can improve our lives now versus, you know, trying to to make changes in the future.

Ryan
But I also think that DNA methylation beyond this is really exciting because with one test, we can start to predict multiple different disease outcomes independently of

aging. So sort of like polygenic risk scores have been used in the genetic space. These methylation risk scores have been shown to be really, really exciting. And that's the other thing that we'll be publishing with some of our Harvard collaborators toward the end of the summer.

Ryan
our methylation risk scores for different diseases. So in addition to biological age, in this test, we can also monitor the risk of progression of all those things. And unlike genetics, you know, you might have taken a genetic testing that you have an AP 3 or 4 variant. And then that might increase your risk of certain outcomes. We can actually change these methylation factors.

Ryan
And so it sort of empowers people a little bit more. Certainly the resolution to find unique patterns of aging excites me, but also that we can start to tackle both disease and aging in the same test with methylation risk. Words that are more actionable and changeable than the polygenic risk scores we're used to.

David
Do you see some point where, for instance, I would take my standard blood based biomarkers? Some, like functional info, like what's my actual VO2 max? Some of these sort of things put them together with the DNA methylation test, which would be a massive data set, and then there would be some sort of output from that. Is that what people are thinking about?

Ryan
100%? I think that I always talk about this with reference of the Human Genome Project is a good example, right. The Human Genome Project, I think everyone expected to solve so many issues for us. And, and I think that, you know, if you Google that now, some of the top news articles would be the failure of the Human Genome Project, why it didn't work out.

Ryan
And I think one of the big reasons for that is because it's only a piece of the puzzle. And just like epigenetics, it's the same. It's only a piece of a very large puzzle. And so we consider that the whole puzzle board to be what we call these multi-omics, right? All the different things that we can measure in the body, going from, you know, genomics to epigenetics, from epigenetics, transcriptomics, which are those mRNAs, those, you know, mRNAs going to peptide and protein creation, and then those create the infrastructure of the body, which then have metabolite and processes like hormones or, you know, other factors.

Ryan
And then ultimately the clinical biomarkers and the phenotypes, do they have disease? How do we perform right that that integrative sort of assessment of omics. Once we have all the data we can make the best models and decisions. so integrating all those data sets is something that we've tried to do. That's how we created our omegas. That's why it's called Omega.

Ryan
We measured all of those different parts of the process. And I think that if you can start to integrate them into clinical models, that's great. The only problem is, while that might give you the best answer, it also might be quite expensive. you know, proteomics and metabolomics are done by plasma as well. So sometimes harder to collect, sometimes harder to transport.

Ryan
And so for what we're trying to do with DNA methylation is to learn lessons about gene expression profiling from the other omics, but to integrate it into one functional model at a low price test, which can be done anywhere on a simple blood spot that has really good stability. So I think that that I think you're absolutely right.

Ryan
The best models, the best actionability will come from integrating multiple data sets. But in the sake of I think, time, convenience and price, we're trying to consolidate all into just epigenome methylation. We think it's uniquely suited to do that.

David
Brilliant. Ryan, if somebody wants to take a test, what do they do?

Ryan
Yeah. So so this testing is available to anyone who wants to order it. They can go to our website at Tru diagnostic.com to order. And I think we're hopefully going to see some some additional improvements, which particularly in additional improvements with those epigenetic biomarker proxies. so we can sort of report on things like functional nutrition and the methylation risk course for disease, which can also report on disease risk.

Ryan
when we do that though, we will have to go through a physician because we're reporting directly on disease. So you can order directly from us if you really want to

quantify your age or if you have a physician, you can encourage them to order to get sort of a robust panel of all the things we can do with this amazing.

David
Okay, super. Thank you so much for the work that you're doing. I think this is fascinating. My North Star on all of this is being able to take all of this data together, and then I just get told, like, stop eating yogurt in the morning, it's bad for you or whatever.

Ryan
I doesn't like.

David
I, I want that really granular. Yeah. Our mutual friend Brian has put together a blueprint, but, you know, he's spending millions of dollars every year to test himself on this sort of stuff. And for us normal people to just have, like, the algorithm. Like what? What do I do? Yeah, that would be great.

Ryan
Yeah, exactly. And I know that, you know, sometimes this can all sound really esoteric, right? It can all sound too complicated and sometimes so much so complicated that we can't actually apply it. And I think that that's one of the things we're hoping to change with this increased resolution, where we can say, target your heart rate or target your inflammation, and then give you really clear steps on how to do that because it's the leading risk factor for you as an individual.

Ryan
And so again, that process has just happened over the last year, really. Both of those symphony genomics papers are coming out. So I hope that the next time we talk, maybe in another year from now, I'll be able to give you those recommendations and say, hey, for information age, these are the five things or proliferates. These are the five things.

Ryan
I'm really make those. And I think we're still a little ways off from those direct, you know, clear and concise recommendations. But it's not far away.

David
Amazing. Ryan, thank you so much. I know you're a busy guy. You're doing a lot of amazing work and it's always so inspiring to speak with you. I really appreciate it.

Ryan
Yeah, thanks for having me again, David, I appreciate it.

David
Take care now.