Welcome to the Huberman Lab Podcast where we discuss science and science-based tools for everyday life.
I'm Andrew Huberman and I'm a professor of neurobiology and ophthalmology at Stanford School of Medicine.
Today my guest is Dr. Peter Atia, his second time on the podcast.
Dr. Peter Atia is a medical doctor who did his training at Stanford School of Medicine, Johns Hopkins School of Medicine and the National Institutes of Health.
He is a world expert in all things related to health span, vitality and longevity.
In this episode we focused on many topics, focusing mainly however on health span and longevity and mental health.
Health span and longevity of course relate to how long one lives and Dr. Atia goes systematically through the seven major causes of death worldwide beginning with cardiovascular disease and cerebrovascular disease, also cancer, also accident related deaths, dementia, deaths of despair and in every case explains the three or four major levers that one can employ in order to offset that is to prevent those major causes of death.
What follows is an incredibly informative and actionable set of tools for anyone, male, female, young or old.
He explains the behavioral, nutritional, supplementation-based and prescription drug-based approaches
that one can use in order to extend health span and longevity.
Dr. Atia explains the key tests and markers that we should all pay attention to if our goal is to extend our health span and how to do so while maximizing our vitality.
This is something that not a lot of people think about when they think about health span and longevity but as Dr. Atia illustrates for us emotional health has everything to do with our physical health and vice versa and he shares quite openly about his own experiences in pursuing ways to improve emotional health and thereby health span, life span and vitality.
Dr. Atia is quite open about his own experiences exploring different practices to improve emotional health as ways not just to improve health span longevity and vitality but of course also to derive the most meaning and satisfaction from life.
Throughout today's discussion we also discussed Dr. Atia's newly released book which is entitled Outlive, The Science and Art of Longevity.
This is a phenomenal book, I've read it cover to cover now three times, I have extensive notes written throughout and the book of course focuses on longevity and health span and also has an extensive section on emotional health.
It gets quite detailed into Dr. Atia's personal experiences with emotional health and tools to improve emotional health that are very actionable for anybody to use.
I think the best way for me to summarize my feelings about the book would simply be to read the back jacket quote which I provided, so I read quote, finally there is a modern, thorough, clear and actionable manual for how to maximize our immediate and long-term health.
Firmly grounded in data and real life conditions, this is the most accurate and comprehensive health guide published to date.

Outlive is not just informative, it is important.
And indeed Outlive is an important book as is the discussion that Dr. Atia so graciously provided us in today's episode.
Outlive is released on March 28th, 2023 and is available for pre-order prior to that date.
You can find a link to where it's sold in the show note captions.
Before we begin, I'd like to emphasize that this podcast is separate from my teaching and research roles at Stanford.
It is however part of my desire and effort to bring zero cost to consumer information about science and science related tools to the general public.
In keeping with that theme, I'd like to thank the sponsors of today's podcast.
Our first sponsor is Eight Sleep.
Eight Sleep makes smart mattress covers with cooling, heating and sleep tracking capacity.
As I've talked about before on the Huberman Lab podcast, there is a critical relationship between sleep and body temperature.
That is, in order to fall asleep and stay deeply asleep, your body temperature needs to drop by about one to three degrees.
And in order to wake up in the morning and feel alert, your body temperature needs to increase by about one to three degrees.
The problem with most people's sleeping environment is that even if you make the room cool, the actual environment that you sleep on, that is your mattress and underneath your covers, is hard to regulate in terms of temperature.
With Eight Sleep, regulating the temperature of that sleeping environment becomes incredibly easy.
In fact, you can change the temperature of that environment across the night, making it a little bit cool at the beginning of the night, even cooler still a few hours into your sleep, which really helps getting into very deep sleep, and then warming it as you approach morning so that you wake up feeling most alert.
I've been sleeping on an Eight Sleep mattress cover for over a year now, and it has completely transformed my sleep.
If you'd like to try Eight Sleep, you can go to eightsleep.com slash Huberman to save up to $150 off their Pod 3 cover.
Eight Sleep currently ships in the USA, Canada, UK, select countries in the EU, and Australia.
Again, that's eightsleep.com slash Huberman.
Today's episode is also brought to us by Element.
Element is an electrolyte drink that has everything you need and nothing you don't.
That means plenty of salt, magnesium, and potassium, and no sugar.
The electrolytes, salt, magnesium, and potassium are critical for the function of all cells, in particular neurons or nerve cells.
And of course, proper hydration is critical for mental functioning and physical performance.
To ensure that I stay hydrated, I consume one packet of element in approximately 20 to 30 ounces of water every morning when I first wake up.
And I will also consume one element packet in about the same amount of water when I exercise or when I'm doing any kind of mental work, you know, preparing for a podcast, writing

grants, working on papers, and so forth.
I find that allows me to maintain my focus and physical performance at levels that I just simply can't otherwise.
If you'd like to try Element, you can go to DrinkElement, that's lmnt.com slash Huberman to claim a free Element sample pack with your purchase.
Again, that's DrinkElement, lmnt.com slash Huberman.
Today's episode is also brought to us by HVMN Ketone IQ.
HVMN Ketone IQ is a supplement that increases blood ketones.
I want to be clear that I am not following a ketogenic diet.
Most people fall into this category.
They are not following a ketogenic diet.
They are omnivores and they do eat carbohydrates.
So their standard fuel source for the brain and body is not ketones.
However, I found that by taking Ketone IQ, which we know increases blood ketones, I can achieve much better focus for longer periods of time for any kind of cognitive work and much greater energy levels for exercise, especially if I'm going into that exercise fasted and find myself a little bit hungry when I start that exercise.
This is no surprise.
We know that ketones are the brain and body's preferred fuel source, even if you're not following a ketogenic diet.
In other words, I and many other people are now starting to leverage endogenous ketones as a fuel source for the brain and body, and yet we are not following a ketogenic diet.
And of course, if you are following a ketogenic diet, Ketone IQ will further allow you to increase your blood ketones as a source of brain and body fuel.
If you'd like to try Ketone IQ, you can go to hvmn.com slash Huberman to save 20% off your order.
Again, that's hvmn.com slash Huberman.
The Huberman Lab podcast is now partnered with Momentus Supplements.
To find the supplements we discussed on the Huberman Lab podcast, you can go to livemomentus spelled O-U-S livemomentus.com slash Huberman, and I should just mention that the library of those supplements is constantly expanding.
Again, that's livemomentus.com slash Huberman.
And now for my discussion with Dr. Peter Atia.
Dr. Atia, Peter, welcome back.
Thanks, man.
Good to be back and sounding better this time.
Looking forward to talking about a number of important topics with you that you cover in your book.
But maybe we could start off by trying to set the frame for what people should be thinking about in terms of vitality and especially longevity.
So I mean, I think you have to be mindful of how you define these terms.
And I'm not going to suggest that the way I define them is the only way or necessarily the best way.

But I think from a clinical perspective, it's the way that makes the most sense to me having thought about this for the better part of a decade.
So it involves some bifurcation between lifespan and health span.
Lifespan is very easy for people to understand.
It is binary.
You are alive or you are not alive.
And clearly part of longevity is about how long you live.
Now I think for a lot of people, that tends to be where the discussion ends.
That tends to be the focus of it, right?
That longevity somehow implies living for 100 years, 120 years, something like that.
We talk a lot about maximum lifespan.
Even in laboratory experiments with mice, that's sort of one of the metrics that's discussed is what's the maximum lifespan of the animals.
But there's an equally, if not slightly, I think potentially more important part of longevity, which is health span.
But health span is squishier and I think it requires some definition.
Now the medical definition of health span is the period of time by which you are free from disability and disease.
I find that to be a not particularly helpful definition because by that definition, you and I have the same health span today that we did 30 years ago.
But I know you pretty well.
You know me pretty well.
30 years ago, we were twice the men we are now based on what we believe our health span is, right?
In terms of our cognitive function, our physical performance and things like that.
So I've clearly experienced the deterioration of my physical function as I'm sure you have going back to when you were a teenager, late teenager, early 20s.
And I think that needs to be captured somehow in health span.
So the way I think of health span really is along these three dimensions, physical, cognitive, and emotional.
I'm not necessarily suggesting that that's the only way to do it, but I do think that clinically it makes the most sense.
And so therefore, anything that really becomes a question of longevity has to address all of these issues.
Lifespan, physical health beyond that of just straight up disability and disease, cognitive health, independent of and separate from pathology such as dementia and emotional health, which of course is by far the most complicated of all of these because we have no biomarkers for it.
It's not like you can get a scan on somebody and determine the state of this, but nevertheless, it's important, right?
And it dramatically factors into quality of life.
So with all of that in mind, what are the major exit points for people along the lifespan route?

So start with the binary one, dead or alive, right?
I think most everyone who's healthy would like to be alive rather than dead.
So what are the typical ways that people exit from alive to dead and how can people stay on the free way of life, so to speak?
So this is, again, a great analysis.
We internally in our practice call this the death bar analysis.
And it's a surprisingly trivial analysis that I'm just surprised the death bars aren't plastered front and center on every doctor's office.
So if you simply just look at actuarial data, which are readily available through the CDC and do a little bit of data manipulation and analysis, you can pretty quickly realize what the horsemen of death are because there's largely speaking kind of four horsemen of death.
The first and most consequential in terms of the numbers is the diseases of atherosclerosis.
So that's cardiovascular disease being the lion's share of that, but also cerebrovascular disease.
So anything that has to do with atherosclerosis rises to the top.
Now that's true in the United States, but it's even more true outside of the United States.
It's even more true globally.
So in other words, when you look at the relative difference between the number one cause of death in the US and number two, which is cancer, the gap is actually smaller in the US than globally.
Globally, it's enormous.
There's only about 18 to 19 million people a year that are dying of atherosclerotic cardiovascular disease in the world, whereas number two is cancer at about 11 million.
How does the number change when you include cerebrovascular disease?
Yeah, it adds a bit to it.
Cerebrovascular disease, there's largely speaking, you can die sort of through embolic events, which are the majority of them.
Could you explain for people what embolic events are?
Yeah.
So taking a step back, what does the brain need more than anything?
It needs blood flow.
Anything that interrupts blood flow to the brain that results in ischemia is devastating, and it's devastating in a more readily apparent fashion than virtually any other organ.
So one way that that can happen is if a clot or disruption of blood flow occurs through obstruction of blood flow.
So that can occur through a clot.
So for example, a person has atrial fibrillation and a blood clot gets festering in the right atrium and they happen to have a hole in between the atria of their hearts called a patent frame in a valley, and a clot goes from right to left.
It can make its way up into the arterial circulation and happen that way where you include blood flow.

The much more common way it occurs is the same way it occurs in the heart, which is you have plaque buildup and that plaque becomes unstable, that plaque ruptures, and the rupture of that plaque results in an immediate attempt by the body to fix the problem.
But in doing so, it walls off the artery, meaning the blood flow distal to that point so that now blood is acutely being robbed of that.
However, there are other ways that people can have this problem, and so you have the whole hemorrhagic side of this.
So you can have blood vessels, small blood vessels in the brain that will rupture as a result of high blood pressure, for example.
So hypertension factors both into both sides of this equation, both in the heart and in the brain.
The majority of these are embolic, however.
So don't quote me on this exactly, but call it four, five to one strokes result from an embolic phenomenon as opposed to a hemorrhagic phenomenon, a bleeding phenomenon.
I don't want to take us too far off on a tangent, but as long as we're here talking about bleeds versus clots, what are some of the major risks for bleeds?
I mean, I know some people out there have genetic predispositions for being bleeders, as they're sometimes called, or clotters.
So things like factor five, Leiden mutations, which can be exacerbated in women, for instance, by taking certain oral contraceptives.
And there's a huge list.
If people are interested in them, they can look up what are the factors controlling bleeding and predisposed people to be in clotter.
But for the typical person out there who feels healthy, but might do well to know whether or not they are predisposed to be a bleeder or a clotter, what sorts of things rise to the top of that list and that people might want to check into?
Well, I mean, there might be sort of two different things going on in that question.
But I think if your question is, when we look at the subset of people who are at highest risk for hemorrhagic strokes, the far more germane question is not underlying coagulopathy.
The far more germane question really comes down to blood pressure.
Blood pressure would be the first, second, and third driver of that.
So hypertension is hands down the leading driver of hemorrhagic stroke phenomenon.
Okay.
So I'll just briefly interrupt and ask, since sometimes your recommendations deviate from the standards that one would find online or in the typical doctor's office, at what point do you get concerned?
Well, I actually find myself quite in line with the most recent available data on blood pressure.
And this has been, obviously, it's a topic that's of high concern to any doctor who's taking care of patients who even pays a fraction of attention to the available literature, which is that basically with each subsequent blood pressure trial, the data are becoming clearer and clearer that the more aggressively you manage blood pressure to be within the 120 over 80 range, the better.

So there's a recent study that even looked at going from what used to be considered acceptable, which was 130 to 135 over 80 to 85.
We used to basically say, that's kind of the first level of hypertension.
And we would say, well, do you really need to be better than that?
And the answer turns out to be yes, you do.
If you want to reduce heart attacks and strokes, it's better to be 120 over 80 than 135 over 85.
And this is a whole other rabbit hole that we don't need to go down, but it's a total obsession of mine, which is how do you measure a person's blood pressure?
I think this is potentially, I'd have to give it thought, but honestly, I could say top three underdiagnosed, fixable problems in the United States today and probably globally.
In other words, there are too many people walking around with high blood pressure who don't know it.
And I think part of the problem is, it's something that is mostly done in the doctor's office and the readings that you get in the doctor's office can be often misleading.
You've heard of this phenomenon of white coat hypertension.
So you go to the doctor.
Your blood pressure is virtually never measured correctly in the doctor's office.
That cuff they put on and that squeeze bulb, if you look at the rigor with which you need to measure a person's blood pressure, the right way to do it is the person has to be sitting like this for five minutes doing nothing.
Okay, folks.
What are the doctors now, you don't let them take your blood pressure sitting for five minutes.
And that doesn't include in the waiting room because if you get up and walk over it, right?
Okay, so make them stand there, right?
So you want to be sitting there like this.
A manual cuff is better than an automated cuff, but not enough people use manual blood pressure.
So a manual blood pressure means they put a cuff on you and they actually put a stethoscope on the brachial artery and they're using the human ear to listen, which believe it or not, you would think a machine is better, but it's not.
The machine can be misled by different sounds.
No, I don't want to suggest that automated cuffs are useless.
They're not.
But when an automated cuff gives you an answer that is potentially suspect, always back it up with a manual.
I'm pretty relentless about checking my blood pressure.
And so I'll do side to side manual versus automated every day.
And there's easily a 10 to 15 point difference between them.
Maybe this is a silly question, but can people check their own blood pressure?
Meaning manually?
Yeah, just could I get a cough in a bulb and learn how to do it?

Yeah, I think so.
I mean, I can do it, but honestly, I usually have my wife do it.
She's a nurse, but it's not rocket science.
Check blood pressure.
I guarantee you there's a great video on YouTube that explains the physiology of it.
And if you're willing to splurge on a good enough stethoscope and the cuff I have is really easy to use.
Like once you put it on, it's in a single thing, I'm squeezing the bulb and looking at the pressure gauge while I've got the stethoscope on my artery.
I mean, given the importance of blood pressure and this arteriosclerosis being at the top of the list of risks for dying, it seems to me it might be worth the expense.
What's a typical range of cost for the quality?
It's not an ordinance.
I feel like my blood pressure cuff is 40 bucks and the stethoscope is a couple hundred bucks if you're getting a good one.
And a good automated cuff, I have no affiliation with any of these companies.
I use two automated cuffs.
One's called withings and the other one's made by a company called OMRON and they're both decent.
But again, they tend to run high and I have yet to find a credible explanation from cardiologists as to why.
And that acknowledges that the manual one, when done correctly, is the answer.
But I've heard wonky answers about why automated ones are sometimes incorrect.
And again, it's just made me realize we're not checking blood pressure often enough on people.
We're overly relying on blood pressures in the doctor's office, which are not being done correctly.
So we basically have our patients do this relentlessly.
So how often, let's say someone buys this, because I think for $240, I realize that's prohibitive for some people, but given the cost of some of the other things that are discussed on this and many other podcasts.
First of all, I would just have people start with an automated cuff to begin with and just start with there.
We generally have people do it for two weeks.
We give our patients a little spreadsheet that automatically calculates averages and stuff like that, tells them what to record and where.
And we just say, look, for two weeks, we want to see two recordings a day.
And do a morning and an afternoon slash PM recording twice a day for two weeks.
And let us see those numbers and we'll scrutinize them further.
And if those numbers come in fine, let's revisit in a year.
Will a day ever come when a watch or a wristband can do this really well?
So I hope so.
And I'm investigating it.

I'm actually going to be trying one out in a couple of weeks with a company that I tried two years ago, two years ago when I tried it, I was not impressed.
So I kind of punted on it.
The company, which I guess I'll not share the name of the company just yet, but they claim that it's significantly better.
So I'm going to put it to the test again.
And it's basically a continuous monitor.
So it's a wrist device that about every 15 minutes throughout the course of the day will check your blood pressure.
To me, this would be, honestly, probably more important.
You know how much emphasis I place on CGM as a great thing to be able to test.
To use glucose monitoring.
Right.
I would argue this would be more important.
When the day comes that we can continuously assess people's blood pressure, it would be an integral part of a person's health checkup once a year is do two weeks of continuous blood pressure monitoring.
Right now to do that, which I've done as well is so cumbersome that it borders on absurd.
You actually have to wear a blood pressure cuff that is attached to a clumsy device that goes through the whole insufflation exercise every 15 minutes, including while you're sleeping.
You know, it provides some insight, but it's so disruptive that it's not what we really want.
What we, the dream would be like a patch that you could put, I don't know, over your chest that can somehow impute changes in blood flow or something like that and regulate, but we'll see, you know, between optical sensors and things like that.
I hope that we're getting closer to having something.
So I don't want to stroke.
I don't want to bleed in the brain.
I don't want to clot.
As long as we're at this number one on the list, archivular sclerosis being the number one killer, what are the major ways to prevent it?
Yeah.
So there's three big ones that stand out, you know, top and center.
And then there's kind of a fourth one that I think is the foundational piece.
So the three big ones we've talked about one blood pressure.
So if your blood pressure is 120 over 80 or better, that's important.
The second is not smoking.
So it turns out that smoking and blood pressure are both devastating for arteries, but for different reasons, right?
So smoking is devastating from a chemical perspective.
So it's completely irritating to the endothelium.
So the endothelium, as you know, is the single cell lining that is the innermost part of the arterial and arterial wall.

So this is a pretty special organ.
Again, it's a bit naive, but understandable that people just think of arteries as tubes.
They're much more complicated than that.
They have many layers to them, but this particular layer is unusually important.
It has an outsized importance because it is the one that's in contact with the luminal side, right, where the blood is flowing in the tube.
And anything that injures that has significant consequences.
So smoking is irritating to that in a chemical way.
And blood pressure is irritating to that in a mechanical way.
So those two things basically, that's the low hanging fruit in my world, right?
You just don't want to have those things causing irritation to the endothelium because that renders you now susceptible to the third factor, which is ApoB bearing lipoproteins.
I want to talk about ApoB in depth, but as long as don't smoke is the second recommendation on the list, can we better define smoking and what's being smoked?
So assume nicotine.
What about cannabis and what about vaping of nicotine and cannabis?
Because vaping has become so much more common.
It's a great question and it's sadly something we don't have a great answer for.
So I can certainly tell you that there's no reason to believe that smoking cannabis is somehow better than smoking cigarettes, but the dose seems to be significantly lower.
In other words, let's consider a person who smokes a pack a day for 20 years.
We call that a 20-pack-year smoker.
Someone who smokes two packs a day for 15 years is a 30-pack-year smoker.
That's a person who's dramatically increased their risk of many cancers, including lung cancer and also their risk of cardiovascular and cerebrovascular disease.
Again, I'm not a THC guy, so I can't necessarily speak for the habits of people that are smoking marijuana.
I can't imagine they're smoking that much.
Probably not.
Yeah.
So while on a joint-to-cigarette basis, they're probably equivalent in terms of harm.
I don't know.
Let's say a person smokes a joint a day.
That would be like smoking a cigarette a day.
That's a 20th of a pack.
Again, I don't want to say that there's no downside to that, but it's probably significantly less.
So I don't think the risk fully tracks.
I think the same is probably true for vaping.
I want to be clear.
I don't think vaping is a good idea.
The last time I looked at the data on this, it was surprisingly sparse, but to me, the only advantage I could see to vaping was if it was the only way a person would stop smoking.

So I looked at it as definitely the lesser of two evils, but by far the better scenario was not to do any of these things.
If nicotine is what you're after, there are better ways to get nicotine, for example, through lozenges and gum and things like that.
So you shouldn't be turning to those things to do it, but if gum is here and cigarettes are here, vaping was probably here.
But boy, I don't know.
For those listening, Peter spaced his hands far apart for gum and smoking and put vaping about a third of the way from gum toward smoking.
In other words, vaping isn't good for you, but it's not as bad as smoking.
That would be my, that would be my, I mean, do you have a, you've probably looked into this as well.
Yeah.
We did an episode on nicotine.
We did an episode on cannabis and the discussion around cannabis gets a little contentious for reasons that aren't important.
It's kind of funny.
People, the moment someone starts to confront cannabis as a potential health harm, people say it's not as nearly as bad as alcohol, which is a crazy argument, right?
Getting hit by a boss isn't nearly as bad as getting hit by a motorcycle in most cases, but sometimes, you know, so that's just kind of silly.
And clearly cannabis has medical applications, clearly.
And then it becomes an issue of the ratio of THC to CBD, pure CBD forms, actually being quite effective for the treatment of certain forms of epilepsy, so-called Charlotte's web, that's actually what it's called.
Very high THC containing cannabis clearly predisposes, especially young males to later on set psychosis.
Those data are starting to become clear, clear enough to me anyway, that people ought to be aware of them at least and maybe make decisions on the basis of those.
When it comes to the smoking versus vaping, it's just very, very apparent that the chemical constituents of the vape and what people are inhaling are terrible for people and are loaded with carcinogens and a bunch of other stuff, many of which cross the blood-brain barrier.
So that's what worries me the most.
Obviously, I'm not a clinician, but anytime I hear about small molecules, you know, these small inorganic molecules getting across the blood-brain barrier and then being maintained in neurons for many, many years, I worry because the experiment is ongoing mostly in young people.
So anyway, without going too far down that track, I think if people can avoid smoking and vaping, they should.
And as you mentioned, there are other delivery devices for nicotine and cannabis, tinctures and patches and gums and things that, edibles that if people choose to use those substances that can offset.
And sometimes people would benefit to imagine what the surface area of the lung is, right?

If you took the alveolar air sacs of the lungs and spread them out, you would easily cover a tennis court.
Remarkable.
So just think about anytime you inhale something, you are exposing, your body is so adept at absorbing it.
I mean, we have this unbelievable system for gas exchange that was designed for gas exchange and anytime you're putting something else in that wake, you're doing a really good job of getting it into your body.
So be mindful of what that is.
And that, look, that applies to pollution too.
I mean, the PM 2.5 data is pretty good.
I think once you, so particulates that are less than 2.5 microns are getting straight into the body, which is like a great argument for avoiding air pollution, right?
I mean, I always find it funny not to get off on this tangent, but to me, the most compelling arguments around cleaner energy have nothing to do with greenhouse gases.
They have to do with air pollution.
I promise you more people are dying from the particulate matters in air that result from burning coal than are ever going to die from the CO2 emissions that result from that.
And I would argue that's going to be two orders of magnitude.
It's not even in the same zip code.
Like sense during the fires, which seemed to follow me because when I was in Northern California, there were a bunch of fires and we were constantly looking, I mean, wake up in the morning, everything was covered with ash.
My dog was having trouble breathing.
I was having trouble breathing.
Everyone was suffering, but there are websites that one can go.
You can just look at air pollution.
And we tend to only do this during fires and then I'm, you know, when I'm in Southern California, there tend to be fires here, so, you know, it's correlation, not causation, but for sure I didn't set those fires, folks, but it's clear that it disrupts your breathing for a very long period of time, but it's the long tail of that that we're really talking about here, the very small particulate that we know firefighters, for instance, and certain industrial workers can end up with that stuff embedded in their brain tissue for extremely long periods.
It's just not good.
You make a really interesting point about the call for cleaner energy.
Can we run that one up to Washington or settle some of the debates about climate change just by getting straight to the health, bypass all the garbage that's, that's being spewed back and forth and just, and basically get to the issue at hand, right?
Yeah, just, just, just make it better for people to not die from the direct consequence.
I'd like to take a quick break and acknowledge one of our sponsors, Athletic Greens.
Athletic Greens now called AG1 is a vitamin mineral probiotic drink that covers all of your foundational nutritional needs.

I've been taking Athletic Greens since 2012, so I'm delighted that they're sponsoring the podcast.
The reason I started taking Athletic Greens and the reason I still take Athletic Greens once or usually twice a day is that it gets to be the probiotics that I need for gut health.
Our gut is very important.
It's populated by gut microbiota that communicate with the brain, the immune system, and basically all the biological systems of our body to strongly impact our immediate and long-term health and those probiotics and Athletic Greens are optimal and vital for microbiotic health.
In addition, Athletic Greens contains a number of adaptogens, vitamins, and minerals that make sure that all of my foundational nutritional needs are met and it tastes great.
If you'd like to try Athletic Greens, you can go to athleticgreens.com slash Huberman and they'll give you five free travel packs that make it really easy to mix up Athletic Greens while you're on the road and the car on the plane, et cetera.
And they'll give you a year's supply of vitamin D3K2.
Again, that's athleticgreens.com slash Huberman to get the five free travel packs and the year's supply of vitamin D3K2.
So trying to avoid such a difficult word to say, especially for a neuroscientist, arteriosclerosis.
Did I get right?
Well, it's athero, which is easier because, yeah.
Atherosclerosis.
Oh, there.
I've been making life more complicated for myself.
Typical of me.
So blood pressure, keeping it 120 over 80 or better.
Don't smoke.
Let's just throw in, don't vape.
Sure.
I'm going to just plant my flag on.
Just don't vape.
There are other ways to get those things in your system if you really want to get nicotine or cannabis into your system.
ApoB.
What's the story with ApoB?
Okay.
So to explain this, you have to tolerate a little bit of chemistry.
So everybody's heard of cholesterol, and I certainly devote quite a bit of time in the book to explaining this because it is so important.
And it's definitely one of those areas where I initially received a lot of pushback from the editor.
And there was a thought that, hey, this is a bit more technical than it needs to be.
But I think that sometimes you do need to resort to longer dissertations to dispel mythology.
So cholesterol is a lipid.
It is a molecule that the body synthesizes.

It is a molecule that is essential for life.
So if you cannot synthesize cholesterol, you can't live.
You'll die in utero.
So there are rare genetic conditions that prevent the successful synthesis of cholesterol, you know, embryos that have those mutations do not survive.
Okay.
So why do we need this stuff?
So we need this stuff primarily for two reasons.
First, it makes up a very important structural component of cell membranes.
So as you know, a cell is a sphere.
We look at them and think they're circles, but they're spheres and they're fluid, right?
They aren't just like little perfect, you know, big bowling balls or, you know, balloons.
They actually morph and shape and move in these pads.
And this is what allows cells to be next to each other and all sorts of things.
They also have channels across all of them.
And those channels are held in place by, among other things, cholesterol and phospholipids.
The second thing that makes cholesterol so important, it is the precursor to some of the most important hormones in our body.
So our sex hormones, testosterone, estrogen, progesterone, in addition to glucocorticoids, if you look at them, it's really funny, you know, people, if you're looking at, if you Google, like, give me the structure of these things, you're kind of like, wow, they're all basically the same.
They all look really similar and they're all pretty much just templates of cholesterol.
So understandably, when it's something that's that important, the body would leave nothing to chance.
We make all of our own cholesterol.
The cholesterol that you eat in food largely irrelevant.
It's a sterified cholesterol, so it means it has an ester side chain.
It's too bulky to absorb in the gut.
So most cholesterol that you eat in food just goes out your GI tract.
Okay, so we have this super important molecule that every cell in the body makes, but there's a bit of a problem.
There's actually two problems.
The first problem is not every cell can make as much as it needs all the time.
So you have this demand problem.
So for example, if you're sick, you're going to need to make far more glucocorticoids.
Your body's response is going to be to ramp up cortisol production, to mobilize fuel and do a whole bunch of other things, and certain cells like the adrenal glands are going to be called on to rise to a higher level of performance, and they're not going to be able to make enough cortisol.
So they're going to have to borrow or take cholesterol from other cells in the body.
In fact, one of the things we used to notice in the ICU, I never knew why it was happening.
I now know is the few times I would accidentally order the wrong set of labs on a patient in

the ICU and also order like a lipid test or something, you would always notice their cholesterol levels were dropping, serum cholesterol levels.
And I now realize why, because they were basically just funneling cholesterol to the adrenals to make more of the cortisol that they needed to combat whatever they were in the ICU for, which is usually the most severe form of stress the body is under.
So you have to be able to transport this stuff.
And then the second problem is, as you know, cholesterol being a lipid is not water soluble.
So the most dominant highway in the body is the circulatory system.
We can use the lymphatic system and things like that, but for the most part, we use our circulatory system as the highway to move stuff around.
And the highway is made up of water, plasma, which is the liquid component of your blood is water and therefore things that are water soluble move easily.
So glucose, sodium, electrolytes, all of those things are dissolvable in water and therefore they don't need a carrier, you just dissolve them in the water and they can go.
So that's why your liver can make glucose that your brain can easily get and there doesn't need to be a carrier or an intermediary or anything like that.
But unfortunately with cholesterol being a lipid, we can't do that.
Just as water and oil don't mix, cholesterol and plasma don't mix.
So the body had to come up with a trick and the trick was designing a vehicle that was water soluble on the outside and fat soluble on the inside, that you could bury the cholesterol inside along with triglycerides.
And on the outside, it was covered in protein, which is water soluble.
And that's the thing that moves around.
And that thing is called a lipoprotein.
And as its name suggests, it's part lipid, part protein, lipid on the inside, protein on the outside.
And those lipoproteins come largely in two different families.
So one family comes from a lineage called ApoB.
So the ApoB family, which is short for Apo Lipoprotein B100, is a family that is derived from the liver, and each of those lipoproteins has one and only one Apo Lipoprotein B100 on it.
We shorten it and just call it ApoB because we don't really worry about Apo Lipoprotein B48, which is attached to chylomicrons that are responsible for fat absorption in the gut.
They're very short-lived.
They don't really factor into atherosclerosis.
So we're going to just, for the purists out there, there's an ApoB48.
We're not going to talk about it.
So when I say ApoB, what I'm talking about is a protein that wraps around a subset of these lipoproteins.
There's another family of lipoproteins called ApoA, or Apo Lipoprotein A. This is a much more complicated family, and I'm not going to talk about it here because we would take an hour to just explain how the Apo Lipoprotein A family works.

I'll give the punchline is there are many Apo Lipoprotein A's. There's variable numbers of ApoA's on those proteins, and they are all part of a family called high-density Lipoproteins.
Back to the ApoB guys.
They are of the low-density Lipoprotein lineage.
So you've heard the term LDL and HDL.
What is it referring to?
It's basically referring to the relative concentrations of protein and lipids in the lipoproteins, and not surprisingly based on their names, the HDLs are higher density, more protein less lipid, the LDLs, low-density Lipoproteins, and VLDLs, very low-density Lipoproteins, and IDLs, intermediate-density Lipoproteins, are all lower density, which means more lipid to protein.
There are different sizes.
There's a whole bunch of other things going on.
The most important fact in all of this is that the ApoBs are athrogenic.
So what we're about to talk about next is perpetuated by Lipoproteins that have an ApoB on them.
So everything in the story right now is just about how do you get cholesterol around the body?
And these proteins that have lipid in the middle.
So let's just take ApoB, for example, many, many billions of them floating around in our body, even in the healthiest of people.
And they're being shuttled to tissues that need them, like the adrenals, muscle, heart, etc.
What sets the demand for these things?
So for instance, could somebody have relatively high LDL, maybe even higher than sort of high end of chart, or even above high end ApoB, but there's some sort of demand, metabolic demand, or they're weight training a lot, or they're running marathons, and so they need a lot of LDL.
The reason I ask this is because it's so easy for the uninformed person, which I include myself in that group, to just sit here, oh, LDL bad, cholesterol bad, ApoB bad, when in fact you very graciously spelled out the fact that these things actually perform a functional role in the healthy body.
So before we get into why they are, can be bad, why would you want a low density like ApoProtein, what is that doing for somebody, and is there any circumstance where the way people are exercising or thinking or not sleeping or sleeping too much, it's that a higher level actually reflects a healthy metabolic need?
We don't have any evidence of that to date.
All of the functions that I described can be done by the HDL.
So the high-density lipoproteins, the ApoAs can do all of it.
So ApoB and low-density lipoproteins are just, they're just the necessary, the weight, no, we don't understand why we have them, Andrew.
This is the part that's really interesting to me.
Most species do not even have ApoB, and as a result of that, most species are chemically

incapable of atherosclerosis.
So if someone could zero out their ApoB and their LDL, we assume they would function just fine.
We know they would because we have certain people who walk around with genetic mutations that render them that way.
Wow.
Furthermore, we also know that there's a bit of a myth out there that cholesterol, the cholesterol you measure in your blood is essential for brain health, for example.
It's an understandable thing, right?
You can speak to this very eloquently, the role of cholesterol in the brain.
Yeah, I wrote down when I was a postdoc at Stanford, so I always point out, I was born at Stanford, trained at Stanford, where is that pride diet, Stanford, hopefully a long time from now.
You'll tell me how long from there.
Well, we're going to do the Charlie Munger thing and make sure that you never go back to Stanford so that you can't die there.
There, exactly.
We cured already.
When I was a postdoc, I worked with a guy named Ben Beres, who I know you know probably as a different person then, for reasons that people can look up Ben's name.
Anyway, incredible scientist, but there was someone in his lab that discovered that cholesterol is a critical component of the synaptogenesis process, the formation of connections between neurons in the developing brain, and then that went on to lead to the discovery of things like thrombospondens being important for synaptogenesis, et cetera.
But cholesterol is central in the brain development mechanisms.
You want cholesterol around for brain development.
In fact, I think very low fat diets and very low cholesterol diets during early development can really impair brain development, as I understand.
Yeah.
It's not entirely clear why, but here's what we know.
When you're born, your serum cholesterol levels are very low.
So children, infants and children, have very low levels of cholesterol.
They would have, and I should explain one thing that's important.
Well, they're not myelinated yet, right?
I mean, they're sorry to interrupt, but myelin, of course, the sheathing around neuronal axons, which accelerates the propagation of nerve signals, and which is deficient in things like multiple sclerosis, is essentially fat made up of phospholipid and requires cholesterol for synthesis.
But young children are not very well myelinated.
I mean, the spinal cord is myelinated.
Spinal tracts are myelinated.
Right.
So this is what's interesting, right?

And I would all agree that cholesterol is more important to infants and children than to anybody else, right?
It would be the most important substrate for CNS development.
And yet, infants and children have virtually unmeasurable levels of cholesterol.
It really starts to take off in your teenage years, right?
So cholesterol, basically serum cholesterol levels rise, basically monotonically throughout life.
You can get a big bump at menopause, so it really goes up for them.
But what's interesting is how is it, how do we reconcile the fact that infants and children have really low levels of serum cholesterol, yet clearly undergo CNS maturation without any problems?
And it basically comes down to the following.
What you measure in the serum is but a fraction of the total body pool of cholesterol.
So we get a little bit of the light under the, you know, the, you know, the, you know, the street lamp under the drunk under the street lamp.
Just because we're looking there, we tend to think that that's what we're seeing.
But if you took the entire circulatory pool of cholesterol, it's about 10% of your total body cholesterol.
It's a tiny fraction of it.
So it's what we measure because that's all we have access to, but it really represents virtually none of it.
I do want to say something because you mentioned LDL.
I want to tie this back to the reader, right, or the listener rather.
ApoB refers to the lipoprotein, the singular lipoprotein wrapped around an LDL particle.
So if you happen to be lucky enough that your doctor measures an ApoB level, it's a blood test.
It says ApoB X number of milligrams per deciliter.
That's measuring the concentration of that protein.
It is a direct measurement of the concentration of LDL and VLDL particles.
When you have a blood test that says LDL, it usually doesn't say LDL.
It usually says LDLC or LDL cholesterol, because LDL is not a laboratory measurement.
LDL cholesterol is a laboratory measurement.
And it's just taking the total number of LDL particles, breaking them apart and measuring how much cholesterol is in them.
So LDLC measures the total concentration of cholesterol in the LDLs, ApoB measures the number of them.
And they're different, but one of them is far superior at predicting risk in its ApoB.
The number of particles is much more predictive of risk than the amount of cholesterol contained within them.
Fascinating, first time I've understood H-E-L-L-D-L and these lipoproteins in a way that makes sense.
So thank you.
I'm sure others feel the same way.

What ApoB level is your red flag cutoff, right?
I actually had my ApoB measured recently and I'm definitely above the high end.
We'll be discussing this over dinner on Saturday night.
And just to tie this back, I hope that's a steak dinner and that should be fine, given the fact that dietary cholesterol has no direct link to ApoB and LDL.
That's true, but dietary saturated fat does.
Okay.
So I'll...
Which is not to say we're not going to have a steak.
We will, but...
Not necessarily one of the fattier cuts, although probably will be for me.
So what's the high end that you...
High end flag.
At what point do you start saying, ah, we need to do something and then we'll talk about what people can do.
Yeah.
So this is a complicated question because it depends on so many factors.
The first factor it depends on is what is your objective?
And I do pose this question directly to a patient, right?
So I say, look, we've got this disease that's the number one cause of death.
Now you can die with it or you can die from it.
That those are your choices.
Statistically speaking, more people will die from it than anything else.
But if you live long enough, we will all die with it to some extent.
So if you're me and I come from a family history, as you know, I write about this in the book, where basically every man in my family except one has died of atherosclerosis and they have all done so very prematurely.
My dad's lost brothers in their 40s and 50s.
By some miracle, my dad is still alive at 86, but you know, I think that's in large part because he at least had the good sense to listen to doctors and take medication to lower his cholesterol and blood pressure.
If your objective is to not die from heart disease and only to die with it, then you want ApoB as low as possible.
Now how low you go depends on when you start because one way to think about this is it's an area under the curve problem.
The longer you wait to start doing something about this, the more aggressively you need to do something about it.
I think a better way to think about this, though, is to go back to what we talked about with smoking.
So would you agree that smoking is causally related to lung cancer?
Yes.
So just to be clear, Andrew, you do not think that it's just an association that smokers get more lung cancer?

No, I do not.
In other words, you believe that smoking causes lung cancer then?
Yes.
Okay.
I mean, there are a number of mechanistic steps in between.
I mean, if somebody was really wanting to drill into the logic, they could say, okay, it's not actually the smoking, it's a some disruption of the endothelial cell lining that you know.
The smoking triggers that, triggers that.
I assume so.
And I agree with you, by the way.
I think the data are very clear.
I'm very relieved to hear that.
But I'm going someplace very important here because if there's one topic that doesn't get enough attention in medicine, it's causality.
Causality is an obsession of mine.
Most of the day on some level, I sit around thinking about causality.
I think the hardest part about studying medicine with respect to human beings is how difficult it is to infer causality for most things that we do.
So if you believe that smoking is causally related to lung cancer, then smoking cessation reduces the probability of lung cancer.
That is a logical equivalency.
There can be no debate about that.
What if I said to you, Andrew, this is going to be our new philosophy around smoking cessation.
I'm going to anoint you the czar of smoking cessation.
So if people pick up smoking, no problem.
We're going to let them smoke.
But we're going to assess their risk for lung cancer using a model that predicts when their 10-year risk of lung cancer gets above a certain level, we're going to recommend that they stop smoking.
So we're going to look at their age, their sex, their family history, some biomarkers that might help us.
We're going to even do scans of their lungs.
And once we think they cross a threshold where their risk of lung cancer is high enough, let's just say it's 25%, boom, you make them stop.
You tell them it's time to stop.
Is that a logical approach to treating smoking and lung cancer or would it be better to say, given that we know cigarettes are causally related to this, how about you never start smoking and the minute you do, we pull the cigarette out of your mouth and explain to you that you're doing something that is causally related.
Of course, it would be the latter, not the former.
It would be idiotic to suggest that we endorse smoking until you cross a certain threshold.
Well, this now becomes the germane question.

There is no ambiguity that ApoB is causally related to atherosclerosis.
How can I tell you that?
I can tell you that looking at all of the clinical trial literature, all of the epidemiologic literature, and perhaps even most importantly, the Mendelian randomizations.
All of these things tell us.
Mendelian randomizations meaning genetic mutants, humans out there that make very little ApoB or excessive ApoB.
And very much, exactly.
So we have a whole grade.
So you can say if you make very little, you aren't going to die as quickly in your life as if you make too much.
That's right.
So Mendelian randomization is such an elegant tool where you basically let genes do the randomization.
And as you said, there is a gradation of LDL concentration or ApoB concentration that occurs from insanely low to insanely high.
And this is a wildly polygenic, polymorphic set of conditions.
And we can look at the outcomes of those people based on the random sorting of those genes.
And there's no ambiguity.
LDL is causally related, LDL cholesterol or ApoB, causally related to atherosclerosis.
Well, if that's true, and I haven't seen a credible argument that it's not, there are people who argue that it's not, by the way, but they just don't have credibility in their arguments, then you have to say that what we're doing in medicine today is very backwards.
Because what we're doing in medicine today is the following.
We're saying, I'm coming at this in a long way, but your question is so important that I want to answer it this way.
We're answering your question today as follows.
We're saying, Andrew, let's do a 10-year risk calculation of your risk of MACE.
MACE stands for Major Adverse Cardiac Event.
It is the metric we use in medicine.
So a major adverse cardiac event is a heart attack, stroke, or death basically resulting from these things.
And we have calculators that are pretty good at predicting your 10-year event risk.
They'll look at your cholesterol levels, your blood pressure, they'll ask if you smoke, they'll ask some family history questions, and they'll spit out a number.
Now we should do yours after the fact.
And I don't know if we did it for a person who's, you know, you're in your mid-40s, like it would probably spit out less than 5% risk for a major adverse cardiac event in the next 10 years.
In fact, the models don't even work if age is below 40.
So the first time I went to do one of these tests when I was in my mid-30s, I couldn't do it, like the algorithm breaks, that's sort of like, you know, just doesn't work.
So the implication there is if your MACE risk is less than 5%, the thinking is you do not

need to treat LDL or ApoB.
I argue that that makes absolutely no sense.
It's just as idiotic as the analogy I used around smoking.
If a risk is causal and it is modifiable, it should be modified regardless of the risk tail in duration.
So then the question becomes to what level?
And again, the earlier you start, the less aggressive you need to be, the less damage that's there already.
So for example, we do CT angiograms on our patients.
If the CT angiogram shows no evidence of calcification, no evidence of soft plaque, that means grossly
their coronary arteries are still normal.
Histologically, they're probably not because nobody probably makes it to our age with histologically perfect coronary arteries.
You know, we might be satisfied with a person's ApoB being at the 5th percentile of the population, which would be about 60 milligrams per deciliter.
But if we have any other factors, meaning we're starting later in life, or a person already has gross evidence of disease, calcification, soft plaque, family history is significant, any other risk factors are present, we'll treat ApoB to 30 to 40 milligrams per deciliter, which is probably the first percentile.
And if somebody's sitting up in the, say, low 130s, what kind of flag does that raise for you?
And I realize it's highly contextual age, et cetera.
No, no, it's a huge red flag.
Again, just because something is causal doesn't mean you're guaranteed to get it.
There are smokers who don't get lung cancer.
So there's going to be somebody listening to this who says, my grandmother's 95 years old, her cholesterol is sky high and she's alive and well.
And I will say, absolutely, there are a lot of people walking around that way, just as there are a lot of smokers walking around who don't get lung cancer, you can't impute these things on an individual basis.
You basically have to ask the question, how do I make the best judgment about an individual from heterogeneous population data and based on what are causal and non-causal inferences around risk?
So to me, if a person has very high ApoB and they do not want to be treated for it, then the best we would do is say, let's at least establish that there are no other risk factors present and let's at least do the most investigation we can around the existing damage.
And if that person has a perfect CT angiogram, I'm going to push less hard than if they have a devastating angiogram.
And by the way, devastating in my book is just any amount of calcification or soft plaque.
Anything that shows up grossly that you can see on a CT scan means that you've got a decade plus of really bad histology building up to it.

This issue of causality, I think now becomes very clear as to why that is so crucial.
And really appreciate the way you spelled that out.
So let's say somebody's ApoB is 80, 100, let's say 130, for example.
What sorts of things can they do to reduce that number?
Is this always going to be prescription medication?
And if so, what are the more common forms of prescription medication that work best?
What are their side effect profiles and so on?
So yeah, usually once you want to start getting down into the 30 to 60 range, you're going to require pharmacotherapy.
But usually we want to see how far we can get with nutrition.
So fixing insulin resistance in an insulin resistant person will bring this down.
So one of the hallmarks of insulin resistance is elevated triglycerides.
We haven't talked about triglycerides, but they warrant some attention because I mentioned it earlier, but one of the other things that the lipoproteins carry is triglycerides.
So you're carrying fat and cholesterol.
And if you recall, ApoB represents the number of particles.
So the purpose of them is to be carrying around mostly cholesterol, but if you have a high amount of triglyceride, you're basically using up cargo space on the ships.
And so you need more ships.
So if a person has elevated triglycerides, and I consider anything over 100 to be elevated, then the most laboratory tests would consider normal to be up to 150 milligrams per deciliter.
We would want to fix their insulin resistance, bring the trigs way down.
I would want to see trigs no more than two times the HDL cholesterol.
So if the HDL cholesterol is 60 milligrams per deciliter, I consider 120 to be through the roof high, and ideally we want trigs at or below HDL cholesterol.
Trigs being triglycerides.
So does that mean low in dietary fat?
No, actually it's most easily accomplished through carbohydrate restriction.
Triglycerides in some ways are kind of an integral of carbohydrate consumption.
Any energy restriction will get it for you, but it's most sensitive to restriction of even, even under eucloric conditions, carbohydrate restriction will lower triglycerides.
So again, energy restriction would be kind of first order of business, but within that carbohydrate restriction will probably get you there quicker.
So you know, you sort of take the low hanging fruit off the table.
And where does exercise come play a role?
Minimal role.
For improving insulin sensitivity.
No, no, no, I'm sorry.
For improving lipids in general.
Yeah.
For improving insulin sensitivity.
Absolutely, yeah.
Especially combinations of resistance training and cardiovascular exercise, correct, yeah.

So once it comes down to pharmacotherapy, you basically have several classes of drugs.
So the most obvious and the one that most people are aware of are called statins.
So statins work both directly and indirectly on the problem.
So directly they work by targeting an enzyme very high in the synthetic pathway of cholesterol production.
The enzyme is called HMG CoA reductase.
And I think it's the second committed step.
I might, I could be wrong on that.
It's, I don't think it's the first committed step, but you, that, that enzyme gets targeted kind of ubiquitously throughout the body.
And in response to that, the liver senses a reduction in the body's pool of cholesterol and the liver really tries to regulate this.
So the liver in response to that increases its expression of LDL receptors.
So the liver itself has LDL receptors on its surface.
And as the body's pool of cholesterol goes down, the liver senses this reduction and says, I want to bring more cholesterol in, more LDL receptors go up and more ApoB particles are coming out of circulation.
So that's really the dominant way that they work.
And in fact, that's kind of a dominant way that all of these drugs work.
So another class of drug is called azetamide.
It works by blocking, we could get as technical as you want on this.
It's called the nemenpixi one like one transporter in the enterocyte.
I like to explain this, I borrow this explanation from Tom Dayspring, but the enterocyte is a, is obviously the luminal gut side cell that is responsible for absorption of cholesterol.
Remember I said earlier, most of the cholesterol you eat, you don't absorb.
The reason you can't absorb it is an esterified cholesterol molecule cannot come in the nemenpixi one like one transporter.
It's too, it's physically too large, but the cholesterol that you synthesize, which once it makes its way back to the liver, gets secreted in bile down the intestine, that is unesterified and readily fits into that transporter.
So I kind of describe that guy as the ticket taker at the bar.
He lets everybody in as long as they fit through the door.
There's a checkpoint inside the bar that basically says, do we have too much cholesterol?
If so, spit it out and there's another door that acts more like the bouncer and he's called the ATP binding cassette G5G8 and he spits excess cholesterol out and if that system is working fine, everything is great, but in a lot of people that ATP binding cassette doesn't work very well and it can't properly regulate the total body pool of cholesterol.
So there's a drug called azetomib that simply blocks the ticket taker.
Are there side effects to statins and azetomib?
Azetomib has virtually no side effects.
You can think of it as a drug that's acting outside the body.
It's sort of acting on a turnstile door in your gut.
I have seen one patient get sort of loose stools from it that became enough of an issue

that we discontinued it.
I would say that when azetomib is combined with a statin, which is very commonly done, it's not unheard of, I can't give you a number, but it could be as high as 10% that you see an elevation in transaminases, which are enzymes that are made by the liver in response to some irritation.
So this is where I think it's unclear what the clinical significance of that is.
We tend to abort the strategy in the presence of elevated transaminases.
Even though the literature says you don't need to, our view is we have other options.
Why would we tolerate any inflammation if you don't need to?
Statins do have side effects.
So 5% of people genuinely and legitimately get a muscle soreness that can be debilitating.
It could feel like kind of the worst workout you've ever had.
The day after, imagine you hadn't lifted weights in six months and then you came over and I made you do the most brutal workout of your life.
You know how you would feel the next day?
That happens every time I come over.
I work out often, but every time I come over to your house, you put me through the most brutal workout I've ever been through.
I think you and Cam Haynes are the two people who've managed to put me through workouts that kept me sore for at least two weeks after each visit.
So that soreness, imagine you would have that persisting, 5% of people get that response from a statin and obviously that's just non, it's a non do.
There's a narrower subset of people that do get brain fog and do experience brain fog from statins.
And we don't really understand the why there.
We have some theories as to why, maybe they're getting too much of a reduction in central cholesterol synthesis.
Again, it's a subjective finding, but given that we have so many tools in the toolkit, we don't have to tolerate side effects with these drugs anymore.
There was a day when you had somebody who just had a heart attack and they're basically looking down the barrel of being on a statin for the rest of their life and there were like two of them and they had tons of side effects and it didn't matter.
Today, while there were probably nine statins out there, there were really only four that we even use and at least two of them have such a low side effect profile.
They're not as potent, but they have a, I mean, potent's the wrong word.
They don't have the same effect, but they're very potent because you're at least one of them you're taking at such a low dose that we've got lots of statin options.
The third side effect of statins, which again, not common, but can't be ignored is insulin resistance.
And this is one of the, I think one of the benefits of at least having periodic CGM tracking is we'll see this, you know, we had a patient who happened to be wearing CGM in general and then we started him on, you know, 10 milligrams of Resuva statin, which is probably the workhorse

statin right now.
It's a, that's a generic term for Crestor and he pings us like a couple of weeks later and he's like, man, my glucose is like 10 points up consistently from where it has normally been kind of hummed and hot.
We'd probably shoot it a few things.
After two months, we're like, let's just stop the Crestor and see if that fixes it and it immediately fixed it.
So there was, you know, we reintroduced the Crestor and it happened again.
So there was no doubt in my mind that, you know, or very low doubt in my mind that Crestor was responsible for that.
And again, you could say, well, maybe that's not that clinically significant, but I would argue why bother?
I have other choices.
So those are your two big ones.
The next one that is really the big one are PCSK9 inhibitors.
So, you know, gosh, coming up about 20 years ago, maybe a woman named Helen Hobbs made a discovery of a group of people that had a disease called familial hypercholesterolemia.
So FH or familial hypercholesterolemia is a very genetic heterogeneous condition.
Going back to that Mendelian randomization study, these are the people on the far end that show us how high lipid levels cause atherosclerosis.
So these people have very high cholesterol levels, typically north of 300 milligrams per deciliter.
Their LDL cholesterol alone is, by definition, at least 190 milligrams per deciliter.
Very high incidence of atherosclerosis in these people, along with other sort of injuries.
Like they have so much cholesterol, they accumulate it in their tendons, in their eyes, it's really devastating condition if not managed correctly.
And she discovered this mutation in a gene for PCSK9 that codes for a protein that degrades LDL receptors.
So these people had hyperfunctioning PCSK9 genes.
So their genes were just chomping down all the LDL receptors on the liver.
So these people weren't clearing LDL.
About five years later, another subset of the population were discovered that were the exact opposite.
These people had hypofunctioning PCSK9, they had virtually unmeasurable.
These people had LDL cholesterol levels of 10 to 20 milligrams per deciliter.
And not surprisingly, they had no heart disease.
So that led to the development of a couple of amazing drugs that are now used.
So I take one of these drugs.
I've been taking one of these drugs for, I don't know, I probably started in 2015.
So it's an injectable drug.
I take it every two weeks and it's called a PCSK9 inhibitor.
So the drug blocks the protein and therefore gives me more LDL receptors, yanks more apobiotic circulation.

Interesting.
When we were talking about side effects, I was thinking, are there any short-term benefits?
So I guess we could call this positive side effects.
But let's think of it more directly in line with the underlying biology.
Let's say my apob is high, mid-range to high, let's say 100, 80 to 100.
And I improve my insulin resistance through nutrition, but we decide it doesn't go down so much.
So we're going to continue to try and knock this number down and I take any number of different drugs to reduce it.
Do I immediately start to feel better?
So there's no...
You feel nothing.
Okay.
And I think that's an important point because of the causality issue that we were talking about earlier, because a lot of people are walking around out there feeling fine.
Their apoby might be a bit high.
They either know it or don't know it, but they think, well, I'm feeling fine.
And you gave a very rational argument earlier as to why, because of the causality involved, it makes far more sense to intervene.
Yeah.
We don't want to rely on feeling when it comes to atherosclerosis, just to put some perspective on this.
When I was in medical school, I think I even write about this in the book, we had a pathology lecture where the professor stands up there and he says, what is the most common presentation of a heart attack and, you know, as Keener first year med students, hands shoot straight up chest pain.
No, that's not the most common shoulder pain, aren't radiating down the left arm.
No, nausea, shortness of breath, we rattled this off for a few minutes and he goes death.
The single most common presentation for a myocardial infarction is death.
Now, I would say today, that was 25 years ago, today, it's probably not the most common because advanced cardiac life support is so much better, but it's still strikingly common.
So well, you could say that the best predictor of a heart attack is still a heart attack.
Well, I mean, not saying that the best underlying predictor, and actually this hits home when I was postdoc, I was living in San Francisco and I'll never forget this, taking my coffee and out on my porch in the morning.
This is right near the UCSF Parnassus campus, and this guy's walking down the street, he was probably about my age, and I said hello, and he said hello, he walked a few more steps and boom, he just hit the concrete and died right in front of me.
It took a minute or two to know that he was truly dead.
I'll never forget it because that's a non-surgeon, it's an event, right?
And I followed up on this because his family, you know, the whole thing, because they wanted to report and no cocaine in the system, no prior history of any kind of health issues.
But he was just strolling along and just boom, as if he'd been hit by a boss.

Yeah, so it's, I mean, again, this is just one of those things where we're going to, he's been a lot of time talking about things that feel good and feel bad when you change them, right?
Like if you take a person who's not sleeping well, but who thinks they're sleeping well, and you ask them for a leap of faith, which is, hey, give me a month to help you sleep really well.
Yeah, you're going to feel better.
You might not know it now because you don't know how bad you're sleeping now.
You've become acclimated to this, but this is not one of those domains.
You know, exercise, nutrition, sleep, all those things.
When you do those things better, you feel better, but you know, I don't want to overpromise on this.
You're not going to feel better in the moment when you fix your lipids, but you'll feel better when you don't have a heart attack.
So by all this logic, everybody should get their APOB measured.
How early in life should people do that, starting in their 20s, in their 30s?
Certainly if you have a family history that is of any concern, like if I could live my life over again, knowing, if I knew everything then that I know today, yeah, I would have had mine measured in my 20s.
I didn't get my APOB measured for the first time probably until I was in my 40s because that's, well, yeah, maybe late 30s, early 40s, right?
I had my first calcium scan when I was 35 and I had to beg Boro Steele to get it done because everyone was like, why does a 35 year old want to do this?
But I, something, I just felt something was wrong given my family history and I'm glad I did.
I'm glad I did that because I learned something that, that completely changed the direction of my life.
Okay.
I know my APOB numbers and that I might be that guy who's up in the, you know, above a hundred.
So I'm going to get this treated.
That's a promise to myself.
I'd like to just take a brief moment and thank one of our podcast sponsors, which is Inside Tracker.
Inside Tracker is a personalized nutrition platform that analyzes data from your blood and DNA to help you better understand your body and help you reach your health goals.
I've long been a believer in getting regular blood work done for the simple reason that blood work is the only way that you can monitor the markers such as hormone markers, lipids, metabolic factors, et cetera, the impact your immediate and long-term health.
One major challenge with blood work, however, is that most of the time it does not come back with any information about what to do in order to move the values for hormones, metabolic factors, lipids, et cetera, into the ranges that you want.
With Inside Tracker, changing those values becomes very straightforward because it has

a personalized dashboard that you can use to address the nutrition based behavior based supplement based approaches that you can use in order to move those values into the ranges that are optimal for you, your vitality and your longevity.
Inside Tracker now includes a measurement of apolipoprotein B, so-called APOB in their ultimate plan.
APOB is a key marker of cardiovascular health, and therefore there's extreme value to knowing your APOB levels.
If you'd like to try Inside Tracker, you can go to insidetracker.com slash huberman to get 20% off any of Inside Tracker's plans.
Again, that's insidetracker.com slash huberman to get 20% off.
We covered the three major risk factors, which were blood pressure, keeping that in check, don't smoke, and APOB.
We've now talked about the things to adjust APOB levels.
We did not really talk about things to adjust blood pressure.
I'm assuming exercise sits as one of the foremost-
Exercise nutrition.
Yeah, weight management is a huge one here.
You take a person whose blood, and this is one of those things where we don't immediately jump on the pharmacotherapy train with blood pressure, because here there are side effects sometimes, and you do have to worry about overshooting.
You don't really have to worry about overshooting a person's lipids.
We do back off if we overshoot, but it doesn't cause a symptom.
There's not a short-term immediate risk from doing that.
If you overshoot somebody's blood pressure medication, you trade one problem for another problem.
They become lightheaded when they get up to pee at night.
They fall and bang their head.
That's a devastated consequence, totally unacceptable.
Our goal is to see how much we can lower blood pressure without medication before we turn to medication, and let's be clear, the meds today are so much better than they used to be.
Again, there was a day when the side effects of these medicines were miserable.
That's simply not the case today.
I mean, ACE inhibitors, angiotensin receptor blockers, I mean, these things are very well-tolerated, especially the ARBs, so again, almost anybody can be on these things, but if we could get a person to lose 10 pounds and exercise every day, we see great effects with zone 2 stuff, right?
So kind of the low-intensity cardio.
What's in your recommendation there?
I know you talk about this in the book, but I've thrown out numbers about 150 to 180 minutes per week.
You go a bit higher.
Yeah, we go 180 to 250, 240.

Yeah, I'd like to see three to four hours a week of zone 2, so that's an important piece, and sleep is an important piece.
So get the sleep right, get the exercise right.
If you're overnourished, let's correct that problem.
And if all of that doesn't work, and by the way, that works a lot of the time, that works most of the time, if that doesn't work, then we've got pharmacotherapy.
There is still a true phenomenon of essential hypertension, which is in individuals for whom all the fixable stuff has been fixed and they still have high blood pressure.
We still have to medicate those folks.
By the way, there's something that I want to mention here that doesn't get much attention, but it's so important, which is the effective high blood pressure on the kidney and also the brain itself.
We've talked about the brain, we've talked about the heart, but the kidney doesn't get enough attention.
The kidney is a remarkable organ, and I think if you're really in this game of trying to live longer, if you think, hey, maybe we'll live 80, 85 years, but if we start doing all of these other things and really optimizing our behaviors, that could be 95.
Well, you have to start thinking about the capacity of the kidney.
Once the glomerular filtration rate falls below a certain level, you have to be very careful with how you live your life.
Unfortunately, this is one of those things that is another mistake that's made in modern medicine, which is we don't pay enough attention to how to measure kidney function correctly.
We rely very heavily on something called creatinine, as opposed to looking at another biomarker called Cystatin C, which is far more accurate.
We also tolerate too low of a kidney function for a person's age.
We might look at someone who's 50, whose kidney function is at 65% and say, you're totally fine because it's true that at 65%, there is no problem.
You're not thinking, well, if this person has to live another 40 years and this continues to go down, they're going to potentially be staring down the barrel of needing dialysis the last five years of their life.
Again, you want to die with compromised kidney function, but never from compromised kidney function.
In fact, the hazard ratio of all-cause mortality associated with compromised kidney function is even greater than that of heart disease.
Once you cross that threshold, I mean, lights out, once you are needing dialysis, your risk of death is higher than that of someone with high blood pressure smoking, even someone who has cancer.
You have a higher risk of death, having end-stage renal disease than you do having cancer.
The kidney is so sensitive to blood pressure, this is a tiny organ that on every pump of your heart is getting 20% to 25% of your blood.
Just imagine how sensitive and susceptible it is to elevated blood pressure.
We've covered quite a few corners of avoiding the major killer, arthrosclerosis.
Let's talk about cancer.

Nobody wants cancer.
Everybody seems to know somebody who has had or has died of cancer and probably no surprise given that it's number two on the list.
What are the numbers and what can people do to offset cancer?
Of course, there are a huge number of different types of cancer.
Inside of this conversation, I just want to earmark that might be good to have a conversation about alcohol, which we didn't talk about in the last discussion.
If alcohol is involved or is a risk factor rather for cardiovascular disease or cerebrovascular disease, that would probably be the time to mention it.
This has been looked at in a number of ways.
If you look at top-line epidemiology and you've heard of these things called the French paradox, which is, oh, come on, they eat all of this fatty stuff and drink all this wine and they have a slightly lower risk of cardiovascular disease, you just have to throw that stuff out the window because there's so many confounders there that it's useless epidemiology.
If you really look at the data clearly, and there was actually a really elegant analysis that included some genetic studies that came out in JAMA about a year ago, it's actually pretty clear that there is no dose of ethanol that is healthy.
There's no J-curve.
It used to be this literature that said there's a J-curve associated with ethanol, meaning at total abstinence, there's a slightly higher risk of death than if you're drinking one drink a day, and then if you go beyond one drink a day, the rate of death starts to climb.
The problem with that analysis, so there's just been a lot of consternation around that, but the problem with those analyses are multiple, but the most important of these are that the abstainers have a reason for abstaining typically, and those reasons can't be extracted statistically from these analyses.
I've written many blog posts about this, if people are really interested, they can go and talk about that.
I also do talk about this a little bit in the book, by the way, but the short answer is there is no dose of ethanol that is healthy.
I would argue that it's not a straight line of risk, but it probably goes, I think from zero to one, there's probably no measurable harm for most people.
One per day or one per week?
Probably one per day, up to one per day, it's probably very difficult to discern the harm, but I'm going to put a caveat on that that I'll come back to, and then I think the risk starts to climb pretty steeply after that, and I think it climbs non-linearly after that.
That is my reading of the literature.
So then, how do you decide if you're going to have up to one drink a day, and by the way, that's not the same as seven a week, because that doesn't mean seven in a day.
Which we know is really detrimental.
Especially for the brain, but also the cascades that result from disrupted sleep, not just for that one night, but multiple nights.
The literature I've seen on alcohol, again, this is an emerging literature because what you're describing is exactly right, but people are now, some more conservative folks are

starting to place it at two drinks per week total, beyond which you start running into the issues.
Especially for women in terms of breast cancer risk, which is something maybe we can circle back on.
Yeah.
I mean, look, my view is, if you can not drink at all, you're better off not drinking at all.
And people always say to me, well, Peter, what's your view on this?
And my view is, I do drink.
I'll go weeks at a time without having a drink.
I haven't had a drink, you know, I've had one drink since I saw you last a couple weeks ago because I've been sick.
So I'm thinking, well, gosh, like the deck is stacked against me right now.
Why would I do anything to stack in more?
But my philosophy, which is half tongue in cheek, but is true is like, I just don't drink bad alcohol.
You know, I sort of, my wife saw me do this the other day, we opened up a bottle of wine and it was a very expensive bottle of wine and I took a sip and I was like, yeah, I just dumped my glass.
I was like, I don't know, just doesn't taste right to me.
And it tasted fine to her.
So I don't think it was that the wine had spoiled.
It was just, I didn't like the taste of it enough to justify drinking it.
I was like, I don't feel like drinking it.
Yeah.
I've fortunately, there were times in life, you know, certainly college and portions of graduate school when I drank, but I've never really enjoyed the, the taste or experience of alcohol.
So I, all the alcohol in the plan could disappear.
I wouldn't even notice, but I'll have one every once in a while.
I'm sort of of that, of that mindset, but great to hear that zero is better than any because I think everyone agrees on that.
So it doesn't appear that alcohol can be directly linked to cardiovascular disease and cerebrovascular
disease, although these indirect effects through insulin, altering insulin sensitivity.
I think, I think the, the impact of sleep on cardiovascular and cerebrovascular disease is profound.
And I do think that the impact of ethanol on sleep is underappreciated.
And here I think we should do a little nod to Matt Walker, the great Matt Walker, because you know, 10 years ago, if we, if someone had a conversation about sleep and how critical it is and how not getting enough quality sleep is dangerous, people would have just kind of shake their heads and say, what's the evidence for that?
I think Matt really deserves most of the credit for alerting people to these issues around not getting enough sleep.

It's just remarkable what's happened in the last decade.
Thanks to Matt.
And, and while we're on that topic, we, you know, we have the other next horsemen of death, the neurodegenerative diseases.
I think those were also heavily impacted, especially on the dementia side, by ethanol.
So again, I want to be careful when I say this stuff, right?
I don't believe in fear-mongering.
Okay.
You know, I just said a moment ago, I'll say it again.
I drink alcohol and I'm going to continue to drink alcohol.
But I think that one has to make the trade-offs, which is like, if I really do love the taste of certain Spanish wines, I really do love the taste of certain tequilas, certain mezcals, and I really do love the, the, the taste of certain weird esoteric Belgian beers.
And it really does give me pleasure to consume those things in the same way it gives me pleasure to consume certain foods that are quite vapid, right?
You know, there's no upside in consuming a brownie that my kid just made, except for the fact that my kid just made it and it's fun to eat the brownie with them, right?
You know, we come back to this thing about like longevity is also about health span.
And part of health span is quality of life.
And you know, I write about this in the book that I think there was a day when my approach to this was purely an engineering approach, which was we are going to optimize every molecule of my being for this.
And if you, if you, if you go so far down that rabbit hole that the quality of your life deteriorates, what's the point?
So that's why I think for somebody like you who says like, you could take all the alcohol off the face of you that I wouldn't even notice, then that's a great reason not to bother drinking.
I wouldn't put myself at the opposite end of that spectrum, but I'm probably further to the spectrum, you know, where yeah, if you told me I could never drink alcohol again, I would be fine with it, but I'd be giving something up that I enjoy.
But at the same time, I know if I have two drinks with dinner, my sleep sucks.
And therefore that's, that's just a threshold I rarely, rarely cross.
I certainly have my vices of alcohol just doesn't happen to be one of them.
What about cancer?
Again, nobody wants cancer.
We've all known people have died of cancer or have had cancer.
What can be done to reduce one's risk of cancer?
Well, you asked earlier about the numbers, so let's throw some numbers out there, right?
So globally, we're talking about 11, 12 million deaths per year, about half the number of ASCVD, still a staggering number.
At the individual level, put it this way, somewhere between one and three and one and four chance, anyone listening to this or watching this is going to get cancer in their lifetime.
But what's the probability they will die from that half of that, about a one in six chance of dying.

Okay.
So is it true that every male gets prostate cancer, most in other words, every man will die with prostate cancer and some will die from it.
You and I have prostate cancer right now.
Thank you for informing.
Yes.
Hopefully we will not die of it.
We should not die of it.
Breast cancer, colon cancer are cancers that no one should ever die from because they're so easy to screen for.
They are so easy to treat when they are in their infancy that it's totally unacceptable that people are dying from this.
There are other cancers for which I can't really say that breast cancer, much more complicated.
Pancreatic cancer, much more complicated.
Gleel, Bustomy, Multiforme, much more complicated.
So there, you know, as you said a second ago, cancer is not a disease.
It is a category of diseases.
It's not just that each organ is different and breast differs from pancreatic.
It's that within breast cancer, ERPR positive, HER2-NEW positive, is a totally different disease from the triple negative breast cancers.
Those with BRCA mutations or non-BRCA mutations.
Well, even putting that aside, just looking at the hormone profile of the individual breast cancers, they're totally different diseases.
So it's not just that breast cancer is different from prostate cancer.
It's that all breast cancers are quite different.
Maybe I should frame the question a little differently than given the vast number of different types of cancers and categories within those.
Your question is still a fair one.
I just wanted to throw that caveat out there.
So now to your question.
Okay.
So what do we know?
It turns out that we can very comfortably speak to several things.
One is the role that genes play.
So maybe I'll just spend one second on gene 101 thing for the viewer.
We want to differentiate between what are called germline mutations and somatic mutations.
So your germline and my germline are set.
When we were born, our germline mutations, any mutations we have in germline genes are inherited from our parents.
They're non-negotiable.
They're non-negotiable.
You got those things.
So question one is how much of cancer results from those types of genetic mutations?

And the answer is very little, less than 5%.
You mentioned one a moment ago, BRCA.
Okay.
So mutations in BRCA are germline mutations.
A woman will get a BRCA mutation from one of her parents and we will often have a sense of that just from the family history, you know, when mom and sister and aunt and grandmother had breast cancer, you've got a breast cancer gene.
Now, it might be BRCA.
It might be another gene that's not BRCA, but there's no ambiguity.
And we test for these genes mostly just for insurance purposes, frankly, but there's no ambiguity that that was a germline transmission of a gene that is driving cancer.
But 95% plus percent of cancers are not arising from germline mutations.
They are arising from somatic mutations or acquired mutations.
So the question then becomes, what is driving somatic mutation?
And the two clearest indications of drivers of somatic mutation are smoking and obesity.
Smoking we've talked about.
Let's put that aside for a moment.
I'm so surprised about obesity.
I don't know why I'm surprised, but I've never heard this.
I'm probably just naive to the literature.
Yeah.
So obesity is now the second most prevalent environmental driver of cancer.
Now I will argue, and I think I argue this in the book, hopefully pretty convincingly.
I don't think it's obesity per se.
I think obesity is just a masquerading proxy.
What is obesity?
Obesity simply is defined by body mass index.
Well, first of all, I don't think I'm obese, but I'm way overweight on BMI.
You probably are too.
So let's just acknowledge.
I'm clinically diagnosable as obese.
Are you?
Oh, no.
Well, clinically.
That would be BMI over 30.
Sorry.
I don't think you're probably there.
No.
But if I measure my weight by height.
Yeah.
My BMI is probably 27 or 28.
Okay.
It's been a little while since I've checked.

I can, I only know body fat percentages and things like that.
So basically like BMI is a far from perfect proxy, but at the population level, it's what we use.
I wish we would get off it, by the way.
I think it's really crap.
Because it doesn't take into account lean versus non-lean tissue.
I think we could get better data if we looked at waist to height ratio.
That's a way better metric.
So this is just a quick test for everybody.
And I'm going to argue your BMI is less relevant to me than your eye color.
But if your waist circumference is more than 50% of your height, you should be concerned.
Okay.
Well, then I'm okay.
Yeah.
You're fine by that metric, right?
That's important.
So if you're six feet tall, your waist better be under 36 inches.
And if it's over, I would argue that's the definition of obesity, not your BMI being over 30.
So back to this issue, because we're using such a crude measurement, it basically is catching a whole bunch of stuff.
But the question is, what's driving it?
And I think if you really look at the physiology of cancer, I don't think it's obesity.
I think it's two things that come with obesity.
Insulin resistance, which is two thirds to three quarters of obese individuals are insulin resistant and inflammation.
And I think those two things with the inflammation and the immune dysfunction, with the insulin resistance and the hyper, basically tonic growth stimulus that's coming, that's what's driving cancer.
So again, is it because a person is storing extra fat, you know, and their love handles that that's driving the risk of cancer?
No, that's, those are just two things that are coming along for the ride.
So beyond those two things, and along with certain, we, there are also certain environmental toxins.
We absolutely know we're doing this, right?
So we understand that people who, you know, have exposure to asbestos have a much higher risk of certain types of lung cancers and things like that.
But for the most part, those are our big risks.
Beyond that, we talk about alcohol in certain cases, absolutely.
Alcohol is a carcinogen.
The dose part still isn't clear to me, I don't know, is one drink a day moving the needle much on cancer risk per se?
It's not clear.

And it might depend on those genetic predispositions.
Yes.
So, so yeah, if step one is don't get cancer, you have no control over your genes.
You have control over smoking.
You have control over insulin sensitivity.
I wish I could sit here and tell you that there is a proven anti-cancer diet, or that if you do X amount of exercise per week, you're going to not get cancer.
We just don't have a fraction of the control over cancer that we have with cardiovascular disease.
We don't understand the disease well enough, so we don't understand kind of the initiation process and the propagation process, and we have to rely much more on screening.
Are there good whole body screens for cancer?
In other words, can I walk into a tube or a cylinder, rather, and get screened for the presence of tumors, any and everywhere in the body, outside the brain, because the brain is a little harder to get to, right?
Maybe or not, the brain is actually pretty easy to screen for.
Because it's so fatty and floating in water.
Well, and also the head, when you put the head into an MRI scanner, there's no movement.
It's the least motion artifact is in the brain.
So when you use something called diffusion weighted imaging with background subtraction in an MRI, a technology that was actually pioneered in the brain for stroke identification, it's also really good at looking for tumors as well.
So let me make the argument for why screening matters, because this is again kind of an area where I go far down a rabbit hole in a way that I think traditional medicine would argue against.
So my argument for screening is an argument at the individual level, and it goes as follows.
To my knowledge, there is not a single example of a cancer that is more effectively treated when the burden of cancer cells in the body is higher than when it is lower.
So the two examples I think I talk about in the book are colon cancer and breast cancer.
So when you take an individual with stage four colon cancer, that means that the cancer has left the colon and is now outside of the colon.
So it's usually in the liver at a minimum, potentially in the lungs or in the brain.
That person's five-year survival is very low, their ten-year survival is zero.
We will treat them with a very aggressive regimen of multiple drugs.
And again, you'll get a five-year survival of maybe 10 to 20 percent, and by ten years nobody's alive.
If you take a person with stage three colon cancer, so the colon cancer is big, and it's even in the lymph nodes around the colon, but at least grossly you can't see those cells in the liver.
Microscopically, of course, we know they're there because if you don't treat those patients, they still die of colon cancer.
But you whack them with the same chemo regimen that you were going to give the metastatic patients, 80 percent of those people are alive in five years.

So night and day difference in survival.
What's the difference?
When the person with metastatic cancer, you're treating a person with hundreds of billions of cells in the adjuvant setting, which is what we call it, we call it adjuvant when you treat people who have only microscopic disease.
You're treating billions of cells.
The same is true with breast cancer, so we have the clinical trial data to put them side by side.
So rule number one is don't get cancer.
Rule number two is catch cancer as early as possible if you're going to get it, which brings us to your question of how do you screen for it?
We basically screen, the first line of screening is imaging, is a sort of visualization.
So you have cancers that occur outside the body that you can look at directly.
So skin cancer, you can look directly at the skin, esophageal, gastric, colon cancer, those are outside the body, right?
Mouth to anus embryologically is outside the body.
So you can put a scope in and you can look directly at the cancer.
But for all other cancers that are inside the body, yeah, you have to rely on some sort of imaging modality, although now we're starting to look at things, things called liquid biopsies.
So blood tests that are looking for cell-free DNA and the cell-free DNA gives us a sense of based on the epigenetic signature of what you're looking at, hey, is there a cancer in the body?
And if so, what tissue is it potentially coming from based on these epigenetic signatures?
So the problem with relying on any one modality is a problem of sensitivity and specificity optimization.
Now with MRI scanners, which are in some ways the best way to do this because they don't have radiation, so you don't want to be incurring damage as you do this.
The irony of doing a whole-body CT scan, screen for cancer, is your whole-body CT scan would be close to 30 to 50 millisieverts of radiation, staggering some of radiation.
So does that mean that people should, sorry to pull you off this, but I was going to ask about this anyway, avoiding going through the whole-body scanner at the airport?
Noise.
So low.
So low.
Yeah.
You know, going through a whole-body scanner at the airport or even getting a DEXA scan, I mean, these are trivial amounts of radiation.
What about flying?
You know, you hear that pilots get more cancer.
If you're a pilot who's flying over the North Pole back and forth and back and forth, you're probably getting, you know, five to 10 millisieverts a year.
The NRC suggests that nobody should get more than 50 millisieverts a year.

So you and I both travel a fair amount, but typical travel for the busy person, let's say two round-trip flights of more than two hours per month and an international trip every three months.
Still less than a millisievert a year.
Living at sea level, one millisievert a year.
Living at a mile elevation.
If you lived in Denver, you're at two millisieverts a year, basically.
I have to ask, standing in front of the microwave.
I'm just, yeah, well, we've got friends.
They ask.
With or without testes on the counter.
That's an inside joke that, unfortunately, unfortunately deserves no description.
And Peter's not referring to me.
But people worry about other sources of radiation.
So it doesn't sound like the microwave is a concern.
What are the other major sources of radiation?
I mean, outside of sort of nuclear stuff where things go, yeah, if you live near a plant or there's been a, yeah, there's been a, it's mostly, it's mostly at the hands of medical professionals, right?
It's the CT scanner and the PET scanner are hands down the biggest source of radiation.
What about the X-rays of the dentist when they're very scurry behind the wall, they're very low, relatively speaking.
Fluoroscopy is very high.
They tend to try to cover up all of you that.
So for example, if they were doing a fluoroscopic study of your kidney because you had a stone, or if you were getting an injection into, you know, if they were doing a fluoroscopic guided injection of one of your discs in your neck, that would be a locally pretty high dose, but they're going to cover the hell out of you elsewhere.
And again, if you get one of these things, it's not the end of the world.
But boy, I wouldn't want to be getting one a month.
And back to the point about screening, you know, a chest, abdomen, pelvis, CT scan is probably, I mean, look, there's probably a scanner out there now that's moving fast enough that it's much lower, but I'll give you an example.
Okay.
Remember how I talked about, we do CT angiograms on all of our patients for coronary artery disease and off the shelf scanner for this is 20 millisieverts of radiation.
Okay.
So calibrate, calibrate me because 40% of your annual alignment.
Oh, wow.
So the medical practitioners really are the major culprits here.
That's right.
So what we say is, and I think most doctors are now realizing this is, no, no, it behooves you to pay a little bit more to go to a really good place that can do that scan for two millisieverts,

meaning they have a much faster CT scanner, much better software, and they're better engineers.
So they have better engineering that they can do on the scanner to get that done.
So if someone listening to this, here's my take.
Do not get a CT scan or any imaging study without asking how much radiation am I seeing?
And if a person can't tell you how many millisieverts of radiation you're being exposed to, then just say, I'm going to wait a minute until somebody can tell me that.
I realize.
And keep in mind, if 50 is the most you should ever be exposed to in a year, there better be a damn good reason why I'm going to get 25 in a day.
Now there are some people who have to do this.
If you're a cancer patient and they're scanning you as a part of your treatment, I mean, you have to pick and choose between those two opportunities.
So I don't want to, I don't also don't want to create some fear mongering where, oh my god, if you hit 50 in a year, your hose, no, it's just, I wouldn't want to hit 50 a year every year for my whole life.
And I certainly wouldn't want to be hitting hundreds a year for any period of time.
And I think we're just trying to raise awareness and also calibrate people to what the sources are and so they can make good choices, not to place them into his chronic state of fear or even an acute state of fear.
So for that reason, we prefer MRI scanners because there is no radiation.
I realize this might sound like a specialized circumstance, but I'll just start off with my own, which is when I was a graduate student, I worked with fixatives, so paraformaldehyde, excuse me, glutaraldehyde, we know that these are mutagens, they mutate cells, not good.
Now you do some molecular biology in the lab, you use DNA intercalating dye, those little bands and gels, the reason they label is because they get between the DNA, not good, to get into your own DNA.
And that's a very specialized circumstance, I also injected tridiate radioactive proline into the animals and things of that sort, again, very specialized.
And yet, most people, I think, will be exposed to pesticides, they'll put stuff on their lawn or they'll have paint thinners and things of that sort.
Is there any sense of what the average, if one can, average risk is incurred in terms of carcinogens, just through this interaction with weed killers, paint thinner, detergents around the house that we now know, there's some major lawsuits that have been successful against the manufacturers of these things, and what is the real cancer risk created by having those kinds of solvents and pesticides and things around?
I don't think I know, truthfully.
I think it's very complicated to calculate such things when their ubiquity is so high.
So one argument is, look, it's kind of baked into the baseline prevalence of cancer today, because these things are so ubiquitous.
In California, for whatever reason, it seems that there's an asbestos warning on pretty much every building, if you look carefully enough, except maybe the one's built in the last five years.
I don't think I've ever worked in a building where the elevator was updated in terms of

the inspection.
It was almost like 10 years back, you always see it while you're in the elevator, no one seems to worry about those, or where there was not an asbestos warning or a lead warning.
It seems like it's just kind of everywhere, and they're noting it in these little flags.
I don't walk around worried about it, I don't lose sleep over it, but it sounds like a real risk or else they wouldn't bother, right?
Clearly they're just trying to cover their legal masks.
Yeah, it might be more CYA than anything at this point.
I mean, I don't know how much of a risk asbestos poses when it's not being agitated.
In other words, I don't know that the asbestos in the ceiling four layers up is really a problem, but if they had to come in here and rip this ceiling apart, I don't want to be in here either.
It was like post 9-11, a lot of the workers selling the World Trade Center pits, because that's what was left, sadly, were developed cancers, probably from exposure and those kinds of things.
Well, I mean, I would argue it's also just the unbelievable amount of pollution, micro pollution that was in the air following those things.
I mean, that's devastating stuff.
So yeah, those are fortunately the outlier events that are dramatic.
But again, my focus is basically, look, I could hermetically seal myself somewhere in the world maybe, and maybe that would reduce my risk by 1%.
But I'm going to focus my energy on what I control, because that's really hard for me to control.
I like focusing my energy on things I can control.
What I can control is the timing and frequency of my screening.
I can't control my genes anymore.
They are what they are.
I got whatever predisposing cancer genes I'm going to get.
I might be lucky in this regard in that I seem to get all these horrible heart disease genes and maybe not as much.
But you can also argue, there are cancer-bad genes in me that we don't really know about because everybody was dying of heart disease so young.
But boy, am I going to control the screening thing?
What source of genetic screening do you recommend to your patients?
Because there are a lot of them.
There's 23 of me.
There's whole genome sequencing in place available now in a variety of formats.
This is actually one of the questions our research team is working on as we speak.
We're trying to decide.
So we do genetic screening for certain things, like ApoE is a gene we want to know in everybody.
For its role in neurodegenerative disease.
Correct.
Specifically in Alzheimer's disease.

We are selectively using cancer screening in some patients.
But in our practice, it's less important because we're generally so aggressive anyway that it turns out to be a little bit moot.
We don't learn a lot in the genetic screening that's changing our screening practices because we're so thorough in our family history and we're so aggressive in everybody regardless of family history.
But I think there's a place for these things, for example, if you're looking for reimbursement on certain tests, give an example.
So colon cancer historically was not covered by, colonoscopy screening for colon cancer was not covered until you were 50.
That's been bumped to 45.
We still think everybody should be screened no later than 40.
No.
I haven't had one.
So I suppose I should.
Yeah.
I mean, look, I'm 50 and I've had three already.
So again, why, because colon cancer is not just the third leading cause of cancer death.
It's 100% preventable.
Why?
Because every colon cancer comes from a polyp and every polyp can be seen on a colonoscopy.
So there's simply no reason to not know that.
And that has to be weighed against the cost of the colonoscopy, both the financial cost and the risks, which are very low, but not zero.
You know, there's a risk that comes from electrolyte abnormalities and hypertension from the bowel prep.
There's a risk from the sedation.
And there's obviously a risk of, you know, bleeding or perforation that comes from the colonoscopy itself.
Again, in a generally healthy person, those risks are so low that they're almost difficult to quantify as evidenced by a recent New England Journal of Medicine paper that was a very anti-colonoscopy
paper, which I won't get into because it's probably a little bit of a tangent.
But what's interesting is, despite being a very anti-colonoscopy paper, this paper does a better job demonstrating the safety of colonoscopy than anything else.
It just was a oddly designed experiment.
So the biggest challenge with aggressive screening posture is the specificity problem, which is when you stack more and more modalities around these things, you're going to start finding things that aren't cancer.
So MRI has a very high sensitivity.
In English, that just means if a cancer is present, an MRI is very likely to see it.
But it has a very low specificity, which means in English, it will see a bunch of things and think they are cancer when they are not.

And it's most troubled by glandular tissue.
So glandular tissue is the Achilles heel of MRI.
And therefore, when you use, as we do, whole body MRI for cancer screening, we tell our patients going in, it's like a 25% chance we're going to find something that is not cancer, but will require us to do further investigation.
If you're not cool with that, which is totally fine, we probably shouldn't do this.
And again, most people are okay with that, but it helps to set that expectation going in, that you're going to probably be chasing your tail, looking at some stupid thyroid nodule that is absolutely nothing.
I mean, I can't tell you how many useless thyroid nodules we've had to get ultrasounds on that prove to be absolutely nothing.
And you have to follow them for a couple of years to make sure they're nothing.
What is the typical cost of a whole body MRI?
And so for people who are not your patients, how would they go about getting those?
Because I think most people's general practitioner is not going to script that out for them.
Correct.
I don't know the short answer because I don't know how many different places are doing it.
I can tell you that we use a couple of different facilities, and I should disclose that I'm a founder of one of them, but we use a scanner that probably, we send our patients to anywhere they want to go, but within a certain company that we like, that's not a company I have an affiliation with, and I believe they're charging about $2,500.
Since you don't have an affiliation, can you mention that?
Because, for instance, you are not my physician, sadly, for me, and luckily for you.
But I'd love to get a whole body MRI.
So what is this company?
So the company that makes the MRI that we're using right now is called Prenuvo.
I interviewed the chief technology officer and the head radiologist of that company on one of my podcasts.
It's a super interesting technology based out of Vancouver, and for a long time, that was the only scanner in the world.
So I had my first scan back in 2015.
I went up to Vancouver to get it done, probably had my first two up there.
They've now opened locations all over the country, so they've got one in the Bay Area, they've probably got one here in LA, I know they have one in Dallas, so they've got them all over the place.
And then the company that I'm affiliated with is a different type of company that does all sorts of diagnostics, but among them is, we have a Prenuvo scanner in that company.
That company's called Biograph, and that's in the Bay Area.
Biograph.
Biograph, yeah.
Spelled as one word.
That's very helpful in terms of understanding the general risk and ways to offset cancer, to the extent that one can, and certainly what the consideration should be.

Number three on the list of ways to die, we should just title this ways to die, or we should title this how not to die too early.
Neurodegenerative disease.
This is an area I'm somewhat familiar with, not because of my own experience, thankfully, but because of my relationship to the neuroscience community.
And last time I checked, I was told that everyone experiences some age-related cognitive decline, so we all get less proficient at focus, memory, complex, context-dependent tasks, switching, all that stuff as we get older, but it's the slope of that line that really can be controlled to some extent, and that Alzheimer's dementia represents just a steep acceleration, downward acceleration of all of that.
That was what I was told, I'm guessing, that even though I reside in this, not kind of, but I reside in that community, that some of that is being revised, especially with respect to the underlying causes of Alzheimer's, because there's a lot of controversy, even scandal around this whole APP, ApoB, amyloid, plaque, tangle, stuff, which is the stuff of textbooks, grammatical students, and neuroscience students.
What is the story with neurodegenerative disease, Alzheimer's in particular?
How can we offset it?
And perhaps, as importantly, how can we all slow our own cognitive decline, irrespective of whether or not we get what is called Alzheimer's dementia?
So Alzheimer's disease is both the most prevalent form of dementia, and the most prevalent neurodegenerative
disease, so it occupies that unique spot.
We're talking about roughly 6 million people in the United States have Alzheimer's disease.
That's one in, let's see, I mean, about 2% of the total population, but that doesn't include those with mild cognitive impairment, or pre-dimension, or other forms of dementia.
And of course, the right metric is not what percent of the population, which of course includes children, things like that, it's, you know, so the function of age is age, the major risk factor for getting Alzheimer's.
We say with glaucoma disease, I'm much more familiar with because my lab worked on it for many years.
The biggest risk factor for getting glaucoma is age.
Yeah.
The greatest risk factor for cardiovascular disease is age.
The greatest risk factor for cancer is age.
We tend to not spend a lot of time talking about that because it's not a modifiable risk.
So you know, we tend to focus on modifiable risk factors.
So what else can we tell you just to give you kind of lay of the land?
So the second most prevalent neurodegenerative disease would probably be Lewy body dementia followed by Parkinson's disease, although the rate of growth of Parkinson's disease is the highest.
So anything, we'd probably be most, you know, we, those three diseases we want to really be paying a lot of attention to.
As you know, there are a lot of other neurodegenerative diseases.

Every one of these things is devastating, like multiple sclerosis, ALS, Huntington disease.
These are awful, awful diseases.
There are also other kinds of dementia, vascular dementia is not Alzheimer's dementia, but it is, it produces comparable symptoms.
Each of these things, by the way, are slightly different.
Lewy body is a dementia.
It's a dementing disease, but it also has a movement component.
So it sort of sits on a spectrum that's sort of, you know, I mean, loosely halfway between Alzheimer's disease and Parkinson's disease.
We talked obviously about age being the number one risk factor, kind of not that interesting because can't do anything about it.
So the real goal is as we age, what are we doing to reduce risk?
Well, let's start with an important gene, the gene that everybody's heard of certainly came up a lot on the limitless special where Chris Hemsworth was, you know, made the decision to reveal something that none of us expected when we started that whole series, which was that he ended up being homozygous for the ApoE4 isoform.
So maybe folks understand we have two copies of every gene.
So for gene X, you have copy that you got from your mom and copy that you got from your dad and the ApoE gene is kind of a unique gene and that it really, it has three different isoforms that are all considered normal.
None of them are mutations.
So you have the E2 isoform, the E3 isoform and the E4 isoform.
The E4 isoform is the OG isoform.
That's the one that we have historically had as far back as we can go.
We actually think the E4 isoform offered a lot of advantages back in the day.
It's a bit of a pro-inflammatory isoform and it certainly offered protection against infections, especially parasitic infections in the CNS, which would have been a really important thing to select for 200,000 years ago.
Kind of parasites get into the CNS.
I mean, you have blood, brain, barrier, thick skull.
I mean, I'm not calling, I'm not telling you, you have thick skull, but I mean, it just seems like parasites and other tissues would be an issue because what we're talking about here is brain disease.
Yeah.
Anyway, but it also could have protected them.
It probably offered some protection outside of the brain as well.
Anyway, the E3 isoform I think showed up, I think 50,000 years ago and the E2 isoform showed up very recently, about 10,000 years ago.
Now, today, we realize that there's a clear stratification of risk when it comes to Alzheimer's disease that tracks with those isoforms.
So, because you have two copies, you basically have six combinations of how you can combine those genes.
You could be 2, 2, 2, 3, 2, 4, 3, 3, 3, 4, 4, 4.

The prevalence of them is basically as follows.
3, 3 is now the most common.
3 is the most common.
So, double 3 is 55-ish percent of the population.
The next most common is the 3, 4, which is about 25 percent of the population.
And then after that, most things are kind of a rounding error.
So, 2, 3s and 2, 4s would be the next most common.
4, 4s are very rare and 2, 2s are the rarest of them all.
2, 2s are less than 1%.
4, 4s are about 1% to 2%.
Very important point here is that the E4 genes are not deterministic.
So, they're highly associated with the risk, but they're not deterministic.
There are at least three deterministic genes in Alzheimer's disease.
One is called PSEN1, another one is called PSEN2, and another one is called APP.
Those genes collectively make up about 1% of cases of people with Alzheimer's disease.
So, they're fortunately very rare genes, but sadly they are deterministic, meaning if you have those genes, you do get Alzheimer's disease.
And what's perhaps most devastating about those genes is how early the onset is of the disease.
These are people that are usually getting Alzheimer's disease in their 50s.
So, we do have a patient in our practice, actually she's spoken about this very openly, whose mom had one of these genes, and she got Alzheimer's disease in her early 50s.
I think she might have made it into her 60s before she died, but absolutely devastating consequences here.
Why do people with Alzheimer's die?
Because I know about the hippocampal degeneration, hippocampus of course being an area of the brain important for learning and memory, but is there brainstem degeneration?
Do they lose breathing centers or cardiovascular control?
Really what happens is it's sort of failure to thrive, aspiration, things like that.
So, it's usually they just stop eating, or they can't control secretions, they aspirate, they get an ammonia, or they really lose the ability to even sense pain in their body, and therefore they'll get an ulcer and they don't realize it and it'll become cellulitic and they'll develop a horrible infection and response to it.
I see.
So, it's a body vulnerability.
The reason I ask is, every once in a while a news report will come out based on a legitimate case study where they'll do a scan on some person and discover that they're missing literally half their cerebral cortex, like huge chunks of brain and they're functioning relatively normally.
And so, here we're talking about a neurodegenerative disease of relatively, it's widespread, but there are a few hot spots of course in the brain that degenerate more profoundly than others and the people dying.
So, that makes sense, it extends to lack of peripheral awareness or control and then some

acute injury or infection, got it.
You mentioned earlier some of the controversy, right?
So, what are we talking about here?
Well, it's, and I do write about this at length in the chapter on Alzheimer's disease because I think this is a very important point, right?
Which is the index case for Alzheimer's disease, there's always an index case, right?
There's the quote-unquote patient zero.
The index case was a woman who, you know, 100 years later we realized had an APP mutation.
I mean, these are APP or PSEN1, but she had one of these deterministic genes that led to a very early onset of disease, which by the way, without which we may not have come up with the diagnosis because had she just got Alzheimer's disease in her 70s, it would have just been referred to as senility, which is, you know, was not interesting enough to pay attention to.
But I think it probably set the field on the path towards an overemphasis on amyloid beta.
And it's not really clear how important amyloid is, which is not to say it's not important.
It is important.
And there's no ambiguity that amyloid is responsible for the changes that we see in the brain.
But it's not crystal clear because there are lots of autopsies that are done on people that are completely healthy and have died with no cognitive impairment, and they're chock full of amyloid.
So what we don't fully understand is exactly what does removing amyloid do.
The other thing that complicates the story is there has been no shortage of drugs that target amyloid that have seemed unsuccessful.
And just to clarify, when you say amyloid, you mean people have died with their brains examined in autopsy and see that there are tons of so-called amyloid plaques, different than arterial plaques, of course, but within the brain.
So the two hallmarks of Alzheimer's histopathologically would be plaques and tangles.
And even that now is, of course, coming under question.
But that's what we teach every neuroscience graduate student.
It's what we teach every undergraduate.
It's also what we teach every medical student, and not just at Stanford, but everywhere.
So I have heard that the link between APP and whether or not one develops or genes for it relate to APP, and whether or not it's cleaved at one site or another is just what you were describing and risk for Alzheimer's is a cleavage question, right?
So people with the APP mutation, I think, have one extra cleavage site.
They result in one extra cleavage of amyloid, and then it misfolds.
And the misfolding is what the plaque is that's being created.
That also then predisposes them to the neurofibrillary tangles.
And again...
But all this is under question now, right?
Well...
I mean, this is what I was told.
And when I look, it sounds like there were some papers early in the chain of discovery

in the research in Alzheimer's that were either wrong because they were falsified, intentionally falsified.
There was an intentionally falsified paper on one particular amyloid variant.
And that clearly set the field back a decade, because a lot of people went down that rabbit hole based on deliberately falsified data.
Then what happened to that guy?
I don't know why I assume it was the guy, but what happened to that guy?
Yeah, that's a good question.
I think I wrote one piece about it when it happened.
I actually reached out to the person who broke the story, because I wanted to have them on my podcast, and I forget why he didn't do it.
I forget why he wouldn't commit to it or something like that.
I thought it was a little odd, because I thought this would be a great way to talk about this.
I do not know what came of that scandal.
In other words, I haven't paid attention to it for probably nine months.
So I don't know...
Obviously, the paper has probably been recalled, but I don't know what disciplinary action was taken.
The field is...
I don't know.
I don't want to speak like I'm in the field, because I'm not.
So I want to be careful what I say.
But I think the field is probably in a bit of a crisis, because there have been so many bets placed on anti-amyloid therapies, and amyloid biomarkers, and amyloid everything, and we just haven't seen efficacy.
So contrast that with cardiovascular disease, where you have this ApoB biomarker, you understand the pathophysiology of how it works, you have drugs that target it, so you have a biomarker, so you give somebody a drug that lowers ApoB, you can measure ApoB.
That's a really important and obvious thing to be able to do, and then you have clinical outcomes, which is, oh, when you take a bunch of people in primary prevention, it takes this long before you see an effect.
In secondary prevention, it only takes this long to see an effect, different risk stratifications, all these different things.
We don't have any of that for Alzheimer's disease.
So we do use...
There are now serum-amyloid biomarkers that we use, and we do track these in our highest risk patients, but only because we believe, and I don't know if we're right, by the way, that lower is better, and therefore, if we make these changes to you, and your serum-amyloid levels come down, that that tells us something about what's happening in your brain that's favorable.
But, I mean, I would hate to represent that we are practicing nearly the level of precision medicine there that we are in cardiovascular medicine.
When it comes to Alzheimer's disease, maybe take a step back.

When it comes to brain health, I think there are a handful of things that seem unequivocally true, and there's a lot of stuff that is signal-to-noise ratio that's really low.
So the unequivocally true things for brain health are sleep matters.
Another unequivocally true thing for brain health is that lower LDL cholesterol and ApoB is better than higher.
Another thing that is unequivocally true is not having type 2 diabetes matters, so having really great...
Yeah, but...
The insulin-sensitive...
The insulin-sensitive matters, sleeping adequately matters, having lower lipids matters.
Those three things are clear, and the fourth one that is unequivocally clear is exercise matters.
And a specific form of exercise?
Very...
So I tried to answer this question on a recent AMA that I did, because the answer is more is always better, but if you...
If I tried to have one of our analysts look at it through the lens of, if you could only exercise three hours a week, what would be the highest use case, and our interpretation of the literature was if you could only spend three hours a week exercising, you'd be best off doing one hour of low-intensity cardio, one hour of strength, and one hour of interval training.
So if someone said, like, I only want the minimum effective dose, you're going to get a pretty good bang for your buck doing that, but I would argue if your brain really matters to you, do more.
No one hour of interval training is no joke.
No, because you're going to spread that out over probably at least two workouts, yeah.
But those four things are basically the only thing where there's no ambiguity about the benefit.
What about head hits?
Don't hit your head.
Seems almost assuredly true in a susceptible individual for sure.
So I put that, yeah, maybe we could include that as well.
Well, I just mentioned, you know, one of the things I've been learning recently is I know you boxed for a number of years when you were younger, I boxed a little bit, hit my head a number of times, skateboarding.
But you know, we think about sports injuries as the major cause of head injuries, but then I've got colleagues at Stanford that say car accidents, bike accidents, I've got so many colleagues and children of colleagues growing up in and around campus that were hit by cars on Woodside Road or, you know, I mean, or small objects surrounded by, you know, three was a car weight, 3,000 pounds or something like that.
You know, it's unbelievable that the number of head injuries and then construction sites because those ridiculous little hard hats, which don't protect against anything, except I don't know, maybe a windblown hair that they basically predispose the whole situation

predispose people to head injuries, very common on construction sites and then say nothing of military, etc.
So I think that I was told that the best thing to do if you get a head injury is to knock it another one.
In other words, if you can stop doing the activity that leads to more head injury.
Yeah.
The other thing that I think is emerging and I hope it is studied rigorously is the use of hyperbaric oxygen immediately following a TBI, Traumatic Bering Injury, reached out to Dom Degustino a little while ago to kind of, because he knows a lot about this lit to say, Hey, is there anything out there that's really kind of turnkey convincing?
And he said, not yet.
They're still doing it.
Right.
So I would do this.
If I, if I was in a car accident tomorrow and sustained a concussion, and by the way, I'm not a proponent of hyperbaric oxygen.
So I, you know, we have an internal white paper that we wrote inside quite recently where I examined when I say I examined, you know, the analyst team examined and I pushed back and reviewed, um, and I, I came away very kind of bearish on hyperbaric oxygen.
I don't think, I don't think it's harmful, but I think all of the claims are nonsense.
You know, telomere extension is totally irrelevant.
And if you actually look at the studies, they're the worst done studies I've ever seen in my life.
I'm sure you've seen some of these where it's like, you put these people in a hyperbaric chamber and then watch them do cognitive tasks act after, and they're so much better.
Well, the fine print is they don't even have placebo groups here.
Like, can you imagine doing a study without a placebo group or your placebo group doesn't go into a sham chamber?
Yeah.
I mean, one of the big problems of the proliferation of all these pay to play journals, meaning journals that will basically publish a paper with minimal or poor peer review, um, because they charge in order to publish, um, and then offer free access, you know, free access sounds great, but when it's pay to play type journals, there's been a huge proliferation of papers, most of which you find on Twitter, um, in which the study design is, is beyond that.
Like, like a ninth grader who woke up late for school and was parting all weekend to design a better study than most of these studies.
Yeah.
And there's some excellent studies out there as well, of course, but presumably out of, and eventually on hyperbaric chamber too.
So I'm not picking on hyperbaric chamber per se, but the, the proliferation of truly terrible science that's published in peer review journals is, is just overwhelming.
Yeah.
It's insane.

And all of that is to say, I think there are places where hyperbaric oxygen makes sense clearly in wound healing.
It does.
It's, it's a miracle treatment for wound healing.
And I would absolutely use hyperbaric oxygen if I suffered a concussion.
Um, but you know, beyond that, I think it's pretty, pretty tough to make the case.
Where do people go for that?
I mean, they're clinics.
Yeah.
They're clinics you basically go to and protocols have to be very precise.
I mean, you're, this isn't something that cowboy at home, you know, no, no, no, no, you have to go into a real chamber.
Um, I think the TBI protocol that's most commonly used is God, I want to say it's pretty intense.
It's like five 60 minute sessions a week at two atmospheres.
Oh boy.
Like it's not, it's no joke.
Um, so from a cost and time perspective, it's enormous and, and the time and cost are reasons why I think when I see people doing hyperbaric oxygen, just because they think it's going to help them live longer, I'm like, dude, you know what you could do with five hours a week, plus the commuting time that you put into that, like it's put that into exercise and I promise you you'll get a bigger benefit than you're getting out of hyperbaric oxygen.
Um, but there's a lot of other stuff that I just think is maybe helpful.
There's tons of supplements that I think about when it comes to brain health, you know, what about Theracumin?
What about magnesium with L3 and eight, the transporter?
What about methylated vitamins that lower home assisting?
What about EPA and DHA?
And we've gone through all of the literature on that stuff.
And many of these things we still are recommending through a kind of basically like the potential benefits outweigh the potential costs.
But the evidence is really unimpressive for most of those other interventions.
So when you think about the big four or big five, if you include not getting head injury, everything else is probably a rounding error compared to those big ones.
Maybe just for sake of, um, thoroughness, we can just list off those four again, exercise, exercise, sleep, insulin sensitivity, um, and lipid management.
Well, along the lines of head injuries, we should probably move to the next category of, um, uh, how not to die as to avoid accidental death, uh, how common is accidental death and what are these accidental deaths because we are separating this out from automotive death.
So is this people, um, falling while hiking self selfies gone bad?
Um, you know, what are we talking about here?
I'm not chuckling because I like it.

It's just, I mean, it seems like there's a near infinite, uh, ways that ways to, um, to die accidentally.
Uh, and one user, I think there's two ways to kind of look at this, um, uh, and, and so here I kind of merge two categories, um, so I would call it that are, that overlap in the way that they're characterized by the CDC, but I would sort of, we'll, we'll, we'll talk about them separately and bring them together.
So if you talk about true accidental deaths, automotive, uh, and falls and overdoses are the, are the three, that's basically what it comes down to.
So you know, when our death bar analysis, we kind of list all this stuff out.
In fact, I think that's actually one of the figures in the book is I have the accidental death, uh, figure that we've put together where we've adjusted by population and you'll see a couple of things.
If you look at it in absolute terms, it's basically a pretty constant.
So regardless of what decade of life you're in, once you're above, you know, 20 accidental deaths are a pretty sizable number of, of deaths.
Now car accidents seem to be pretty constant throughout life, little more common if you're under 60 than over 60, but they never go away.
I was told that, um, in teenage and boys and, and, uh, boys in their, in their early 20s, alcohol induced, uh, automotive fatalities, but placed them at this, at an, an astronomical risk.
Is that just not true?
It's not true anymore compared to overdoses because young people now, um, aren't getting their driver's licenses.
I've also heard that.
Yeah.
Well, I think it's also because we're seeing such an uptick in the deaths that come from fentanyl.
Got it.
So fentanyl related deaths have basically squashed all other deaths below 65 on the accidental front.
Really?
Oh, it, it's not even close because of the number of different substances that fentanyl is being woven into winding its way into everything.
All right.
So all counterfeit drugs, all illicit drugs, and look, most of the time you're not getting a lethal dose.
So it's, you know, it's, it's, but, but you're getting lethal doses so often now that, um, well, you know, I did a little analysis, actually the other day when I had looked at how our deaths of despair increasing over the last five years.
So what did I define as a death of despair?
Suicide.
All related death or overdose, accidental overdose.
So that we differentiate that from suicide where suicide is obviously deliberate and

accidental is not.
So if you just look at those three things, so accidental overdoses, suicides and alcohol use or alcohol related death, um, not including driving, by the way, this is like cirrhosis of the liver that comes from that number is going up at almost 20% per year since 2019.
So the, I couldn't get 2022 numbers yet.
So at the time of the time I did this analysis, which was last week, um, the 2021 numbers was about 210,000 Americans, goodness up from 180,000 in 2020 up from like 150,000 in 2019.
So is this, um, and that is driven almost entirely by fentanyl use.
So I'm trying to, um, get a sense of how this would happen.
A while back there was an article in the New York times that some photographs of people that, um, died of fentanyl overdose and said they, they went out to buy cocaine and I thought to myself, this is a really kind of odd, socio biological phenomenon, right?
Because I mean, here they're, they're not demonizing these cocaine user.
I mean, they went out to buy cocaine, right?
This is not a, um, I know cocaine has one narrow clinical use as a prescription drug, but in general when people buy cocaine, they're, they're quote unquote partying with it or using it to work longer hours or something like that.
Um, so the whole nature of the article was a bit strange to me, but it clearly pointed the fact that people are using cocaine.
Okay.
That's no surprise, but people are going out and buying cocaine.
They're presumably buying Valium.
They're presumably buying.
This is where it's really killing kids.
I mean, this is online.
This is in person.
I mean, the reason I'm so, so baffled by this is let, let me, uh, re contextualize what I, what I've said so far about this question.
I was surprised that the times would write a paper about the tragedy of cocaine users dying of fentanyl and I think they did it to highlight this fentanyl problem, um, because people have been using cocaine for a long time.
And typically those are not the members of the population that we're really focused on since the mid eighties, the so-called cocaine and crack epidemic.
So basically it tells me that people, like you said, illicit drugs, so cocaine, but also, you know, what other sorts of drugs are buying the majority of people are dying from fentanyl poisoning.
And I had a guy on my podcast recently named Anthony Hippolito.
And if anybody's interested in this topic, they really need to go listen to that.
So watch the YouTube version of this and your podcasts are excellent.
So people, if you're interested in this, and I think everyone should be interested in this.
If you have a child or know somebody who has a child, you just got to get this podcast into their hands because it's the most important public service announcement I'll probably

ever do in terms of saving more lives potentially.
Where the majority of this is making its way into the, into the accidental poisonings is through illicit counterfeit pills.
So it's when kids are out there buying, you know, oxy, they want oxy, well, they can't if they can't get real oxy, right, because they're not going to go to a doctor and get real oxy.
So they're going to buy it through, you know, Snapchat, right, they're going to buy it through some drug dealer that they're finding on social media.
They're buying sleeping pills.
They're buying all sorts of counterfeit stuff like Adderall.
Any of these things are being laced with fentanyl.
Adderall.
Absolutely.
Wow.
I assume the fentanyl.
And again, the reasons are it's insanely cheap to use synthetic fentanyl.
And secondly, and again, but the effects of fentanyl or nothing like that are all.
So cocaine doesn't make sense for that reason.
It doesn't make sense either.
Yep.
And yet it's still showing up in cocaine.
Again, I don't think that's the dominant place it's showing up.
I would, I would guess that the dominant place it's showing up is in counterfeit opioids.
So any opioid, barbiturate, any sedative, let me tell you what I'm telling my daughter, right?
Because this is to me, it's a frontline problem.
I have a 14 year old daughter.
I'm like, listen, I don't care which friend of yours it is.
I don't care how much she's amazing.
If she tells you to try this sleeping pill, because she took it the night before and it was really helpful, or this will help you study better, or this will help you do anything.
I'm like, just come to us.
We got a better pill for you, right?
Like in other words, you can't trust anything because you don't know where she got it.
She has the best of intentions, I'm sure, when she's given it to you.
And by the way, she probably took it the night before and was just fine.
But the people who are making these pills are not exactly up to GMP standards.
So you just have no idea which pill is getting what dose of fentanyl.
One thing that Anthony Hippolito told me that I simply couldn't believe, I had to ask him six times, was that some of these pills have like one milligram of fentanyl in them.
Now I made the point on the podcast that a hundred milligrams of fentanyl for most people is a hit like they've like, I've had fentanyl before, I've been in the hospital and I've had fentanyl, a hundred milligrams is like, wow, that is such a trip.

Why are people dying from one milligram intake?
Prespiratory inhibition.
You can't breathe.
That shuts the brainstem off.
Well, I don't think we can highlight this enough.
You know, adults are dying, kids are dying.
I met someone just earlier this week who told me her 35 year old son died of an accidental fentanyl overdose and he wasn't, at least by her description, a drug addict or anything of that sort.
Yeah, this is, we're talking about a different game now, right?
So it's like, these are kids that have anxiety.
These are kids that are, you know, are sort of addressing another issue with these pills.
And that's why I think this whole concept of deaths of despair is a really important one.
But back to your question, what do accidental deaths primarily amount to for the aging population?
Again, it is so clear that it is fall related.
This is where once you hit 60, 65, the risk of a fall that results either immediately in death.
You know, you hit your head and die going back to like cerebral hemorrhage or it is the straw that basically leads you down the path to death within the next 12 months is astonishingly high.
That it's sort of hard to wrap your head around.
But if you're over 65 and you fall and break your femur or hip, so you either crack the femoral neck or the femur itself, your 12 month mortality, the probability you will be dead in 12 months after that break, if you're 65 or older, depending on the study is about 15 to 30%.
Wow.
Wow.
So, in terms of offsetting the probability of falls, you talked a little bit about this before, but you and I have talked a little bit about this before, but maybe we could go a little bit deeper.
People's ability to jump and land seems to be highly correlated with one's ability to not fall or at least fall and control the fall in a way that leads to no or less severe injury.
Andy Galpin talked about this on your past, he talked about it on my podcast.
What is the hallmark of aging on the muscle?
It is atrophy of the type two muscle fiber.
That's the hallmark.
Fast twitch.
Fast twitch muscle fiber.
So, if you want to understand what looks different in 50 year old Peter versus 18 year old Peter, it's not my type one fibers.

It's my type two fibers.
It's my fast twitch fibers.
It's my explosive fibers.
When I was 18 years old, I could vertical jump over 30 inches.
Today, I'm lucky if I can vertical jump 24 inches and when I'm 60, boy, it's like my goal is to be able to vertical jump 20 inches when I'm 60 and I don't know if I'm going to be able to do it.
I've seen some videos of some 80 year old sprinters that are pretty impressive and certainly 80 year old gymnasts that are impressive.
I've not seen very many videos of 80 year olds dunking basketballs, for instance, who are not taller than six feet, five inches.
So when we lose, so again, if you just think about size, strength, speed, we lose speed first.
We lose speed, then strength, and the last thing you lose is size.
So again, size is agnostic to fiber, right?
You could have big type one fibers and still have lots of size.
They're not going to be that strong and they're certainly not going to be fast.
So what I mean, we could go through, we could spend hours on this particular topic, but I think the most important thing that people need to understand is you cannot age well if you are not doing the type of training that is there to strengthen and delay or minimize the hypertrophy of your type two fibers.
So everything matters, right?
You have to be doing your zone two.
You have to be doing all of these other things.
But some component of your training needs to be stressing the type two fibers.
You have to be doing strength training that taxes those fibers.
You have to be doing reactivity training.
You have to be doing explosive training.
And ideally, some training that involves jumping and landing.
Well, jumping is a very big part of it.
And landing is a very big part of another one of what I kind of think of as my four pillars of strength training.
So one of the pillars of strength training is eccentric strength, which is breaks.
So you know, you're going to hurt yourself 10 times more likely, I'm making that number up by the way.
I don't know if it's 10 times, but experientially, it seems to be you are 10 times more likely to hurt yourself stepping off something than stepping onto something, right?
Walking down versus stepping up, because when you step up onto something, you are concentrically controlling the muscle.
When you step down, you have to apply the brakes.
And that's where most people falter.
Much harder to walk downhill than uphill.
Uphill is taxing your cardiovascular system, but if you slow down enough, you're fine.

But a lot of people don't have the ability to slow themselves down when they're walking downhill.
And so when an older person steps off a curb and can't fully stop themselves, and that results in a fall.
So you know, I like doing things like a broad jump, broad jumps, a fun little test set I like to do every once in a while.
I always want to make sure I can broad jump six feet.
That's kind of my arbitrary number that I've chosen.
And the reason is on the takeoff, that's a very explosive movement.
But the landing is just as important.
If I can't stick that landing, it means I don't have the brakes.
So those are kind of some of the tests I want to be able to do to make sure that I'm utilizing that system.
Because I do think, you know, look, I've watched my mom, my mom fell probably been about four months ago, just fell in a typical way that people fall.
By the way, it could have happened to anybody.
It's not like, you know, my mom walks around and moves around just fine.
But in this particular day, she just tripped on a uneven stone and fell and landed and broke her hand.
And she's really lucky she didn't break her hip.
And I told her that because my mom was probably in her mid-70s.
And I said, look, you know, if that was your femur, I'd give you a 30% chance of dying in the next year.
I mean, it's just an, those are such difficult to recover from injuries.
Because first of all, you're dealing with the immobility of, you know, the hospitalization and immobility that follows that.
And the amount of muscle loss that occurs could easily be, you know, four or five pounds of lean tissue lost that for most people at age becomes almost impossible to get back.
And that says nothing about sort of the acute causes of death, like a fat embolism that results from a broken femur, a blood clot from laying in bed.
Those things are also catastrophic.
But what happens is a lot of these patients just never get back to the same level of mobility.
And you know, now I think in many ways we're kind of pivoting from what kills you to what ruins your quality of life.
And we've spent so much time talking about what kills you, but I think you might as well be dead in some ways if you can't do the things you want to do.
And if playing with your grandkids or gardening or playing golf or going for a walk with your spouse or think of any of the things that we all do today and take for granted, if you can't do those things, I don't know, you sort of lose the reason to be around.
And oftentimes the inability to do those things is associated with pain, which is psychologically and obviously physiologically so distressing.
You mentioned the four pillars of health, maybe just list those off for people.

Well, the four pillars of longevity through physical.
Oh, yeah, sort of the exercise pieces of them.
Yes.
So strength, stability, aerobic efficiency and aerobic peak output.
I guess aerobic peak would be so VO2 max and zone two.
In my analogy, your zone two is how wide the base of your pyramid is and your VO2 max is how tall the peak of the pyramid is.
So the best pyramid has a wide base and a high peak.
So you could have a reasonably wide base and a shallow peak.
If you just did zone two training, you're going to get a reasonable peak, but it's not going to be that high.
You have to do some of that specific training.
If you just focus on high intensity, you might drive up that VO2 max, but you're actually going to have a relatively wide, a narrow aerobic base.
So you think about just maximizing the area of that triangle, widest, tallest.
Stability and strength.
Stability of course encompasses everything we're talking about in terms of reactivity.
I dedicate a chapter in the book to this concept because it is so foreign to most people.
And for understandable reasons, it's not sexy, it's the hardest one to train, it's the hardest one to understand, but it's so important because it's the thing that I think differentiates people who age well and people who don't age well.
And I should perhaps throw in there, please correct me if I'm wrong, but also most of the machines that are in typical commercial gyms that allow people who are not very experienced to start doing some resistance training, don't really tap into the stability factor terribly much.
So while there's value to leg extensions and leg curls and chest presses and shoulder presses done with machines, certainly for a number of reasons and can often be safer than free weights, especially for people who are approaching it later time or new to the whole thing, they don't really lend themselves to real life stability, walking down, as you mentioned, walking downstairs in the absence of a handrail or movements in kind of odd planes, having to step aside to avoid a bicycle at an angle as opposed to just moving linearly.
Yeah.
And by the way, a lot of things that don't involve machines still don't give you that, right?
If you're doing a deadlift, you have to be stable to lift a heavy weight like you would a deadlift without hurting yourself.
That requires an unbelievable capacity to harness intra-abdominal pressure and to be connected.
If you're going to lift 500 pounds off the ground, you're stable, but that still doesn't prepare you for what you just described.
So stability is multifaceted and it involves doing a lot of things.
Today, for example, I finished my, today was a cardio zone two day, so I did my cardio zone two and, you know, had it extra 10 minutes before I needed to kind of get moving.

And so all I did was step ups for 10 minutes.
I just did single leg, very slow step up and insanely slow step downs off a box in a gym.
So two second up, four second down, two second up, four second down with, you know, and I would do them with ipsilateral loads, contralateral loads, all sorts of different things.
And you know, basically that's just a stability game for me.
It's like I'm building that concentric strength in, in a movement where it's easy to cheat, but can I do it without cheating?
It's terrific.
And it's terrific that you cover all of that in the book, in addition to these other topics.
So several times during our conversation today, you alluded to quality of life.
And one of my favorite segments in your book, indeed, the segment in your book that I believe could be its own entire book of tremendous value is the section on emotional health.
If you could just share with us a bit of what inspired you to include that section was this, for instance, based on communication with your patients, to what extent it was based on your own life experience.
And then maybe we can drill a little bit deeper into what's contained in those chapters and what really constitutes emotional health.
Well, I mean, I think that that chapter of the book, which is a pretty long chapter, it's the final chapter as well, is certainly different from all of the others in that there is no, there's no confusion about expertise, right?
I think in the other chapters, I at least try to come across as having some knowledge on the subject matter.
And I'm writing them most often as, you know, quote unquote, the doctor, right?
Whereas I think that last chapter is much more about an experiential side of my knowledge acquisition and therefore really it comes across more as a patient.
And I think you're right.
I think that that's a chapter that initially was resisted by all other parties involved in the book.
So my co-author, my editor, everybody else sort of felt like this is interesting, but it's a, it's a separate topic.
If you want to write about this, you should write another book about it, but it doesn't really belong in this book.
I disagreed for two reasons and ultimately, I guess my opinion prevailed.
The first is I didn't want to write another book.
So it just that, you know, not including this in this book to then write about it in another book was not something I was interested in doing.
But I think more importantly, I do think that this book is about much more than how long you live.
And while we have talked about and we'll talk about in the book, that is, you know, how cognitive and physical health are just as germane to quality of life as they are to length of life.
This other piece of emotional health, you know, it's potentially the most important of them all.

It's also the hardest to define, but without it, none of this other stuff matters, right?
So there's, you know, infinite lifespan, if, if, if, if you're miserable means nothing.
Maybe worse.
It's, that would be a curse, right?
You could argue, how could you punish somebody the most?
Allow them to live forever and be miserable.
Is there a, yeah, there's a Greek God, Tithonus, yeah.
Tithonus.
Yeah.
He was granted immortality.
It's a bit different.
He was granted immortality, but without a health span, basically.
So he aged forever, dreadful, yeah.
And this would be dreadful too, right?
And I feel like, why did I need to write about this?
Well, I think that, you know, this is probably my greatest struggle, I think, you know, way at the outset of the podcast, you asked me kind of like, what are the obstacles to longevity?
And that got us down a path of some very black and white things.
But when I look at a patient, I create a dashboard and the dashboard is, what are all the things that are a threat to every component of your longevity, both lifespan and health span?
We talked about a bunch of those things.
So how, what is, what is your risk for atherosclerosis and what are we doing about it?
What is your risk for cancer?
What are we doing about it?
What is your risk for neurodegeneration?
What are we doing about it?
What is your risk for accidental death?
What are we doing about it?
What is your risk for physical decline?
What are we doing about it?
And one of those things is, what is your risk of emotional health or poor emotional health and what are we doing about it?
So when I do that exercise for me, which I do, right, I mean, I can, I have that spreadsheet laid out for me and I know where my factors line up.
And interestingly, despite my family history being horrible for atherosclerosis, it's like sixth on my list because, I mean, basically I intervened early, I have a clear understanding of the pathophysiology and I'm doing everything to the maximum.
So I'm actually very confident I will die with and not from atherosclerosis.
But the top thing on my list is actually emotional health.
It's the one that is the hardest for me to manage.
And it's the easiest to get out of balance and it creates the most pain in my life.
So that's a long answer to why I felt this needed to be in here.
Well, in the book, you go into very honest detail about some of your journeys through

and challenges with emotional health and paths to overcoming those.
Maybe we'll get into those a bit.
But before we do, how should we define emotional health?
This to me seems like one of the most difficult areas to calibrate oneself, like even just measuring emotion is tricky, language is the dissection tool for psychologists, psychiatrists and indeed for all of us, how are you doing today, great or I'm miserable or I'm depressed.
I mean, it means such different things to different people, obviously suicide being the far end of we presume misery.
There are instances of manic suicide, but depressive misery.
But setting that aside, how should we evaluate, think about and communicate emotional health to ourselves and to the relevant people that could potentially help us.
Yeah, well, you're right.
It's very difficult and so much of what goes into this book is about things that are much easier to quantify.
I could sit here and talk for days about all the ways we quantify from the histologic to the gross of each of these diseases, genetically, all of these other things.
With emotional health, it's far more vague and I don't even attempt to come up with a definition.
I can tell you things that make up components of it.
Connectivity with others just seems to be an inescapable part of this, so the ability to maintain healthy relationships and attachments to other people, by the way, these are no particular order, having a sense of purpose, being able to regulate your emotions, experiencing fulfillment, experiencing satisfaction.
All of these things matter and I think that for many of us, if we're taking an honest appraisal of ourselves, we'll notice that we have deficits in these areas.
Being present, by the way, that's something that may have been less of an issue 100 years ago than it is today.
Certainly for me, being present is very difficult.
It's not my default state.
I don't know that it's the default state for most people truthfully, but I'm very often predisposed with thoughts about the future, occasionally thoughts about the past, but it's much more often thoughts about the future and planning and thinking about what I need to do and what do I want to do next and never really being satisfied with anything that's happening in the moment.
I have to work hard to overcome those things.
I'm sure you can appreciate this, but when you are present, you generally are in a much better frame of mind.
Yeah.
There's an interesting study.
I think it was initially published by Dan Gilbert's lab, one of these long-term happiness studies that was published in Science Magazine that pinged people for their level of happiness, unhappiness, presence, or lack of presence multiple times throughout the day.
This was in the early years of smartphones.

This is around 2010, 2011.
The technology wasn't as good as it is now, but it was good enough to do this in a very large number of people.
I forget how many, but it's certainly more than 10,000.
That number is, I'm stating it intentionally low.
What they found was regardless of whether or not people were doing something they enjoyed or not, boring to them or not, the degree of presence to what they were doing was a stronger predictor of their happiness in that moment and overall than was anything else and also a fairly rare feature for most people.
It seems like it's something that we do need to work at perhaps nowadays as you point out more than we perhaps do in our ancestral past.
I'm a little bit surprised that you say that you find it hard to be present because you strike me as somebody that is not just willing, but has a strong, almost reflex toward drilling and observing the contour or something and then really drilling into it and really getting to the guts of most everything that interests you.
You strike me as somebody who's very present, and I guess that maybe this gets back to this vagraness.
It's usually exclusive.
For example, I'll notice that sometimes if I'm playing with my kids, especially my boys because they're younger and playing with them is really being in their world.
If I'm with my daughter, we can be doing things that are mutually, we'll do things together that I would probably do by myself or she would do by herself, but with my boys, it's generally doing something I wouldn't otherwise be doing.
If I'm paying attention to it, I'm constantly amazed at how after five minutes of searching through a bin for just the right Lego piece that we want to do to build this one little thing, like my mind will start thinking about something else, like, oh my God, like I got to go, ugh, I didn't email that dude back and I got to do this and I got to do this and I got to do this and I got to do this and I just get into the, I got to do, I got to do, I got to do.
It's like, dude, you've only been here for five minutes.
Why don't you just find the Lego piece that you need to finish building that thing over there that is this beautiful moment that you're not going to have many of, right?
There's a very finite number of these moments you're going to have.
So you want to savor every one of them.
So again, I don't think I'm alone in that.
I think a lot of parents, for example, can relate to that.
And that's literally just one of many different things.
And by the way, I wouldn't have said that that was my greatest challenge either, but it's something that requires, I think, deliberate attention.
What you're alluding to is a challenge with holding a single time perception or perception of time.
One of the most remarkable things to me about the human brain is our ability to be present or think about the past or the future or the present and the future and we can occupy

different time bins.
And in a recent non-recorded conversation of ours, you showed me something that I've seen before, but for some reason this time it had a profound impact on me, which is that you have a chart of the number of weeks that you're going to live and you mark them off one week at a time.
We were talking about this in the context of major life decisions.
And it illustrates the fact that we need such a chart that we can't really move through our day being present to the beauty of working on a Lego with our kid while also paying attention to the fact that, wow, this is week number, whatever, 600 in the X number of weeks of one's life.
So that ability to contract and dilate our time perception is marvelous, but it's also a double-edged sword because it takes us out of what's meaningful in the moment.
One has to wonder, then, whether or not our challenge is in being present.
I guess the psychoanalyst or psychiatrist, maybe we'd ask Paul Conti, who you know and I know and respect greatly, whether or not this is some subconscious refusal of our own mortality or something, right, that if we were to really contemplate our mortality on a regular basis, not just when we're marking off the weeks of the poster, we wouldn't be able to be present because it's kind of overwhelming, right?
I don't know.
I feel like the literature says that people who spend more time contemplating their own mortality are actually more at peace, kind of a little bit of the exposure therapy idea.
And so I'm not sure it's an unhealthy thing to be aware of your mortality.
I suspect it's helpful in as much as you accept it, right?
And you feel like you have some agency over parts of it, right?
Like I don't think I have nearly enough agency over the length of my life.
I think I've got five to 10 years of wiggle room that I can extract.
If I do all of the things that I've written about in that book, I bet I can stretch my life out 10 to 15 years at the maximum, call it 10 over what would have happened if I didn't do those things.
Maybe it's more, but it depends on what we're comparing it to, right?
From being reasonable to maybe being a little bit hyper-functioning, maybe it's 10 years.
But where I know I have a much greater agency is on quality.
And for me, now a big part of that is in terms of quality of relationships.
I think that's a big thing.
And I think for most people, that's what I hope this chapter does, is it sort of allows more people to kind of take an appraisal of that and ask that question, which is, before it's too late, am I living my life more for my resume virtues or for my eulogy virtues to borrow from David Brooks's work, The Road to Character, which I talk about as being kind of one of the many aha moments that I had during this journey.
Yeah.
And there again, thank you.
You recommended The Road to Character.
To me, I do an annual solo wilderness trip, and I listen to it during the drive to that

trip and on that trip, and it's a, I would just say it's a truly important book for everyone to listen to.
Yeah.
It's really quite impressive.
What are the things that you do on a regular, let's say on a daily basis to try and enforce, forgive the word, but enforce emotional well-being and health in terms of relationships?
Because as you point out, it's not reflexive for everybody, and that doesn't make them bad people.
I think it does have to do with this challenge in balancing expectations of work and other things.
And for some people, a more inherent selfishness, and for some people, they aren't selfish enough.
I know plenty of people are running around trying to serve everybody, and then their health is crashing or their mental health is crashing.
So it can cut any which way or always.
What sorts of practices do you incorporate or just even thoughts within your own mind?
Do you use charts and lists?
I mean, you're very regimented about your workouts, building grip strength, eccentric training, zone two, et cetera.
Why wouldn't we also script out the things to pay attention to each morning and day as a list of to-dos?
Well, I have done those things, right?
So certainly, and I write about it in the book, I've gone away a couple of times, right?
So in 2017, I spent two weeks at a facility in Kentucky.
In 2020, I spent three weeks at a facility in Arizona.
And on the back end of that facility, three years ago when I got out, I had a very clear list of daily things I needed to do.
And so at that point, for about six months, following, getting out of that stint of rehab, I mean, I was, I mean, got the list of behaviors I was doing every single day.
I mean, twice a day standing in front of the mirror, reading my list of affirmations, writing in my journal every single day, I had therapy every single day.
I mean, all of that stuff was highly regimented.
You know, today I would say there's no one single behavior that is quote unquote mandated as part of my recovery.
But perhaps the most important thing that does come up every day is being mindful of and acting on as quickly as possible every time I do something damaging to a relationship.
So I would say that like, if you compare formula one, one of my, my favorites work by far, if you compare formula one, 40 years ago to formula one today, the difference is not in the number of accidents that takes place.
The difference is in the fatality of those accidents.
There are just as many, if not more accidents in formula one today.
The difference is nobody dies in those accidents.
The cars are so much safer.

They're engineered first for safety, second for performance used to be the reverse.
And that's why there was a day when every second or third weekend, a driver was killed.
It's catastrophic to imagine what took place between the mid sixties and about the mid eighties and formula one.
And similarly, I would say that the frequency with which I have an interaction with a person who matters to me that is not the best interaction it could be is only slightly less than what it was five years ago.
The difference is the severity of that is much lower and more importantly and most importantly, the length of time between when I screw up and when I make amends is infinitely shorter.
Right.
So from being, I would never make amends to if I'm a dick to my wife, I usually am trying to rectify it within a few minutes or at most a couple of hours.
And so it's really, you know, one thing I learned throughout this journey was if you hold yourself up to this goal of I have to be perfect, I have to be the perfect dad, I have to be the perfect husband, I have to be the perfect friend, you're going to set yourself up for failure because, you know, you're just not going to be perfect.
But if instead you can say, what I'm going to be perfect about is repairing damage when I cause it.
That's what matters.
You know, the other day, I yelled at my son for something, it was a while ago actually because it was before I lost my voice.
So, you know, I don't know, he was just doing something and he was wrong.
You know, like it was like, he did something, I told him 150 times not to do and I yelled at him and punished him like, you know, but I was way too harsh, like, because basically I basically, the first 27 times he did it, I didn't respond.
And then when I finally did, it's like I blew a gasket, right?
But what I realized is, yeah, you could say, well, maybe it hurts a child to do that, but I think it hurts them way less if you can immediately go and repair and say, hey, buddy.
Daddy was a little harsh in that.
I'm sorry.
I didn't mean to yell at you like that, but what you did is wrong and you're not going to get to go out and play right now as a result of it, but I love you very much and I want us to do better.
I want you to do better and not doing this thing and I want to do better and not yelling at you when you do this thing.
So it's not rocket science, right?
But I just think I used to live my life in a way where all I did was break shit and never fix it.
So you're living in a house where everything is broken, whereas now I still break things, but now I clean up the mess and oh, like all of a sudden the house is better.
What is your process for when there's a need for repair, but you feel that it wasn't you, it was somebody else's error or potential error.
So you very humbly express how you go about repairing your errors.

But what about situations where I loved one, a co-worker, you feel screwed up or wronged you, right?
As many people do, we all do from time to time, feel this way.
Do you approach them and try and repair the situation because there's a little bit less or far less control than the situation you described?
And by the way, the situation you describe everything is a perfect one because I think we all screw up.
And so the second question is sort of the answer to the first, which is if everyone did what you were doing, the world will be truly a far better place, but not everyone's doing what you're doing.
So if you feel wronged, assuming that wrong wasn't sociopathically motivated, what is your process for going about repairing a relationship fracture like that?
Again, this assumes that this is a relationship that matters, right?
So in every interaction, you're only really able to optimize around one thing and you have to decide, is this one thing that I'm optimizing around the relationship or is it the outcome?
There are other things to optimize around, but you understand that those are different, right?
Maybe you could elaborate on that a little bit.
I think I get it, but flesh that out a bit.
If I'm at the market and if I'm trying to buy a new car and I'm sitting there talking to the car salesman, that's a relationship.
That's an interaction.
Now, I want to buy this car for as little as possible and he wants to sell the car for as much as possible.
Well, in that interaction, my relationship with him means nothing.
Just assume I don't know this guy and he's not like my best friend.
I'm optimizing everything around the outcome.
So everything I do in negotiating and in interacting with him personally is based on getting the best outcome for me.
It's very selfish, right?
Nothing wrong with that, by the way.
Well, he's doing the same.
Absolutely.
Exactly.
But now, for example, pretend that you are the car salesman and you're one of my closest friends and it's your dealership, like it's your money.
You can't sell this thing to me at a loss.
I don't want you to do that because I want you to be able to make money.
And similarly, like you care about me and you don't want me to overpay for this.
So now we're negotiating and we're both trying to optimize for an outcome, but our relationship also matters.
It's a very different negotiation at that point.

And so I think I always try to ask myself this question when I'm having some interpersonal conflict, which is what am I optimizing for?
So if I'm having a quarrel with my wife, I have to remind myself that the objective or outcome is not necessarily the top priority.
Being right all the time, which is my default state, it's just to be a bull in a china shop.
It's to be authoritarian instead of authoritative.
And that doesn't work if the relationship matters.
So to answer your question, the first thing I'm going to ask myself if I feel slighted is what is the nature of the relationship?
Is it even worth trying to do something about this?
And presumably you're asking the question because the lens is yes.
This is someone who you care about more than in just a transactional way.
Usually what I've realized is I can't try to approach the situation without fully understanding myself and that takes a while.
So generally, and this is where I still one to two times a week, I'm still working with a therapist.
I have to kind of try to figure it out on my own and then usually bounce it off a therapist and say, well, I think this is why I'm upset about this.
I think that when this person did this or said this, I felt this.
First of all, am I am I correcting what I felt?
Because remember, sometimes you might, at least for me, this was the case.
I would just feel anger in response to every interaction.
But what I didn't realize was that anger was really just another emotion that was superimposed on top of hurt or superimposed on top of fear or superimposed on top of shame or superimposed on top of something else.
But I didn't know how to articulate any of those other emotions.
So the only thing I could really articulate was anger.
So if anger is the only thing I know and anger is the only response I see, it's not very helpful.
It's not very insightful.
So that's that's a big part of it is being able to deconstruct what I'm feeling.
Oh, what I really feel is loss or what I really feel is abandonment right now.
And that sometimes takes a while to figure out, at least for me, like I'm still, you know, I'm only a few years into this journey and maybe other people figured these things out when they were in their 20s.
And so they're veterans, they can do this more, more naturally.
But that's step one, if I don't really understand what's going on, I can't even begin to try to approach this person to say, this is how I feel.
This is, you know, how do you feel?
And what are we optimizing for in this interaction?
I certainly know you are not alone in this, this sense of it's a process and it takes a lot of time and and on a case by case basis can take a lot of time to figure out exactly what one is feeling.

And I think it really goes back to the the coarseness of language as a way to sort one's feelings.
It was actually your other, because we mentioned Paul Conti, who is one of your Stanford Medical School classmates, but another previous guest on this podcast, who was also one of your medical school classmates, Dr. Carl Dysaroff, right, psychiatrist and bioengineer of phenomenal stature and doing amazing things in the world who said, you know, most of the time we have no idea how other people feel, even though we think we do.
And most of the time we don't even know how we feel.
I mean, our ability to really know what we're really feeling is terrible.
And yet we recognize the the broad the broad bins.
I'm pissed off, I'm super happy, I'm relaxed, I'm tired, I mean, just think about how coarse that that language is for that for all the nuance and all the underlying things conscious and subconscious that could be driving an emotional state, it's really, it's really quite unbelievable.
Yeah.
Beyond the valence that would, you know, positive versus negative, that was about the extent of my emotional language until, you know, somewhat recently.
Well, it strikes me you've come a very long way, maybe you could share with us a little bit about what you learned on these, what you called retreats or, I mean, in the book chapter you describe deliberately going off to a treatment center, multiple treatment centers over time to really drill into this process of understanding oneself better and how one's current state of emotional processing and emotional stability are influencing relationships and the key importance of that.
Was there any kind of overriding theme for you, for instance, could you trace back to specific events or themes of childhood that made a lot of it make sense or is it far more nuanced than that?
Well, you know, the first thing I would say is I wish I could tell you that this was a very deliberate and wonderful choice that I just decided I'm going to go on a little, you know, self healing journey, but unfortunately that was not the case in both cases in 2017 and 2020, I was as close to having no choice in the matter as one can have.
So both of these experiences represented total rock bottom moments in my life.
So these would have been the two lowest points in my life for different reasons, but they were nevertheless the two absolute low points in my life.
And I would say, you know, in the first instance, I guess I could have chosen not to go, but I would have lost everything that mattered in my life at that point.
And I had, you know, our good friend Paul Conti basically telling me that I needed to do this, that I really needed to do this.
And in the second situation, though completely different circumstances, you might think how can one person in just a span of three years find themselves in a situation where they almost without having any choice in the matter have to go away to a place where you're basically blocked up without a phone for, you know, three weeks and you're doing 12 to 13 hours of therapy a day.
So nothing about this was something I wanted to do, nothing about this was pleasant.

I would describe these as the most difficult things I've ever done in my life bar none.
And I've done some difficult things in my life, but they've always been physically difficult.
I love doing physically difficult things.
But this was emotionally the equivalent of for me, you know, climbing K2 and swimming the English Channel in the same month, you know, something that just I could you couldn't fathom.
So so with that said, yes, I learned a lot and I learned that people like me can be overly analytical and that that hyper analytical nature can lead you astray when you think that your intellect is giving you a fact based explanation for a set of circumstances and you rationalize them away.
Well, this happened to me when I was a kid, but, you know, like, I get it and it's not really a problem.
And as a result of that, you know, it's it's these are actually some positive things that came out of that experience and and and I think the real aha moment in my journey, which occurred on a day that I remember very well was the day I finally dropped that I dropped that that rationalization and I allowed myself to experience what a child would experience in that moment and then understood what the implications are for a child going through these things.
And I think that was that was really the first time in my life I ever accepted emotionally something that I had intellectually always said, you know, it doesn't really matter.
I mean, it's just, you know, that's just life and those things happen and lots of worse things happen to lots of people and that's OK.
And I think it it's not that once I emotionally accepted this, I became a victim.
It wasn't at all.
It just finally allowed me to realize, oh, I can let that go now.
Like I don't have to.
I don't have to.
I don't have to be a slave to the adaptations that came from that I can I can I can surrender that's beautiful and and inspiring to me.
I think that yeah, there's this incredible ability that the human brain has to script a story and to compare to other people's circumstances and as you said, you know, rationalize what are essentially emotional traumas or physical traumas from the perspective of the adult.
But if I know one thing for sure and make it very clear, I'm not a clinician, but is that the brain doesn't discard of any circuitry.
We repurpose the same circuitry we used as children as adults.
And so the ability to go back to that and to and to parse it.
But as you as you point out, not from a from an intellectual standpoint standpoint, but from an emotional standpoint seems to be the really hard work.
Do you do that on a regular basis?
No, not not at all.
It's been done a handful of times.
It's been exhausting.
It's very difficult.

It's it's it's I don't know if this is the right word.
I would almost describe it as emotionally violent.
And it's it's it's not something I need to revisit often truthfully.
I think that yeah, it's it's it's been done a finite number of times.
And I think I've captured so much so much value from it that there are lots of other things I continue to do.
I mean, I, you know, I use a system called dialectical behavioral therapy that is a regular part of the therapy that I do, but I don't have to go back to my childhood.
I don't have to go back to uncovering and re exploring a lot of that stuff.
I I've I've I've learned the lessons and now it's really about practicing the skills.
I know I know what I want now.
And I know, you know, you talk about plasticity, I'll share one example, which I know I wrote about in the book, but just for for folks listening that you'll appreciate.
So I, you know, just one of the one of the hallmarks of my existence has always been, you know, just an insane amount of anger and rage.
It's been there as long as I've known.
So I don't have a conscious memory of not having rage, right?
So earliest memories of life when I'm five years old, I have rage like you can't believe.
And it's it's a problem all my life.
So as a teenager, if I go more than two weeks without punching a hole in the wall of our house, it's a miracle.
I mean, I am so good at drywall.
You can't believe how good I am for all the stuff I have to repair around our house.
Like I'm breaking windows.
I'm breaking.
It just doesn't like I just and so in a way, and of course, I rationalized how much boxing saved my life because I had this amazing outlet for my rage, right?
If you I got to basically exercise six hours a day, I'm hitting punching bags and people all day long, and it's just a beautiful outlet that keeps me out of jail.
And a big part of that rage was inward, right?
So it's it's not rocket science to understand that a person who has that much hatred for everyone has an enormous amount for themselves.
And so one of the things I didn't realize was happening was what my inner monologue was because as you can appreciate, your inner monologue is so frequent and ubiquitous and present that it's easy to almost forget that it's there.
I mean, that's the that's the that's the sort of dangerous part about it, right, is kind of the, you know, the David Foster Wallace, this is water thing, the fish are swimming through water.
The water is everywhere.
They don't even realize they're in water.
You don't unrealize.
You don't realize the subconscious stream of thoughts that constantly flow.
But eventually, I became aware of just what that self talk was.

And it is it was no longer the case.
It was the angriest, the most violent self talk you can imagine.
I mean, it was like, there is no mistake that I could make that was anything other than my perfect, perfect standard that didn't result in what I would call my inner Bobby night going ballistic.
So it just didn't matter.
Like it sounds silly under it didn't matter if I didn't perfectly cook a steak.
If I didn't perfectly nail something I was doing.
If I didn't do anything that was perfect at what I described as match grade perfect.
I mean, I would want to beat myself to a pulp and I would scream at myself.
I mean, it just it's it's again, it's hard to describe and I hope that most people listening to this don't understand what that feels like.
Well it became very clear that that had to change because when you are when you are that when you hate yourself that much by definition, you are going to be an insufferable prick to everybody else because you're just that's going to spill into how you interact with the world.
So I was working with a therapist who was one of the people who was sending me to this place in Arizona.
And basically it became clear that they proposed that I could shed this trait if I was willing to do a certain amount of work and I was like there's no chance like I'm 47 years old.
This is the only way I've ever interacted with myself.
How in the world could this be undone it would take another 40 years to undo this and they're like no no here's this exercise you're going to do.
So the exercise was every single time I did something where I would have that self talk I would have to immediately stop myself and pretend that it wasn't me that just did that but it was one of my closest friends and instead I would audibly speak to that person there's nobody else there but speak to that person as though they are the one that made the mistake and I was to record that on my phone.
So I'm out there shooting my bow and arrow and I don't get a bullseye instead of screaming at myself I have to say oh imagine it's my buddy JR who just missed that shot what would I say to him pick up the phone or you know pull out the phone and say of course something different and of course what I would say in that situation was much kinder I mean infinitely kinder it's like from saying it to my closest friend I'm gonna say it in a very kind way and I had to take a copy of that audio and text it to my therapist.
Oh wow.
Yeah.
Talk about vulnerable.
I was all on board this practice until you mentioned that at which point and I trust my therapist to a very deep level but I thought wow that's a mountain.
Well this poor person got a lot of text messages a lot of audio files but here's the part that just blows my mind it only took I don't know I can't remember exactly I have to go back to look at my journals it took about four months to get rid of Bobby Knight like you know again that we had kind of a mental model for what this looked like which was Bobby Knight

was the chairman of the board he sat in the board room and nobody else got to talk and for those that don't know Bobby Knight had a terrible temper yeah yeah the worst right this is the guy that was throwing chairs across the basketball court level 11 out of 10 and all of a sudden like we got to the point where Bobby Knight is not even in the board room anymore in fact I as I say this today like I don't really remember what he sounded like it's it's amazing to me and and I've had some really amazing opportunities to bring him back like it's not like I'm making fewer mistakes right it's not like I'm better today than I was three years ago at all the things that I do I'm not I'm actually probably worse in many regards but the difference is you know I can communicate with myself I think I can say this I think I can say lovingly right and and maybe not as lovingly as some people can I still think I'm probably maybe just a little higher standard with myself than maybe I need to be at times but but I'm just not beating myself up like I used to and I think by extension I'm beating other people up a lot less well I don't know the extent to which your internal narrative reflects the narrative that others have about you but first of all I want to thank you for sharing what you just shared I think as a practical step it it first of all it's one I've never heard of before but certainly represents this incredible phenomenon of neuroplasticity because four months sounds like a bit of time and yet you were 47 years old that's 47 years of accumulated just absolutely berating self-talk is what it sounds like so it's something that people can can think about for their own for their own purposes and their own challenges also you know I've read the book twice now and love it as I put in my endorsement of it I think it's not just informative but it's indeed important because it centers on so many of the key actionable items related to vital health span and lifespan vitality longevity whatever people want to call these things that are essential but also this the section on emotional health is was absolutely profound for me it inspired a huge number of changes and the book as a whole represents a very important contribution to everybody there are numerous points and I would say every chapter is applicable to everybody and there are very few books out there like that so I want to thank you for that and especially for including the section on emotional health and especially for sharing what you did today because I think it doesn't just take a bit of vulnerability but a ton of vulnerability and humility to be able to share what you just shared and my only request or wish is that you also hopefully internalize it the tremendous gift that you're giving everybody through coming on podcasts like this doing your own podcasts writing the book you know I look out on the landscape of front facing public facing health out there and you sit not alone but in a unique stance as the medical doctor that I do believe that people trust the very most because of the fact that you have that intense rigor you're just I wouldn't even say your desire your absolute obsession with measurement and precision many of the things that a moment ago you were pointing to as potentially you know hazards for your emotional life but that serve all of us the general public so preciously and so with it just incalculable value so I hope that internalizes as well maybe it'll even weave into yourself top maybe I need to send you a script every day but in all seriousness I also want to thank you for taking the time today and even though it's a personal thing I really want to thank you for your being an amazing colleague to me in the podcast space in the in the health and medicine space whatever that is and also

just an incredible friend you've been a tremendous source of support and guidance in every one of the domains that we talked about today and many more and again I just want to say that this emotional health component I agree with you I think it's it's not just vital I think it's it's the the most vital of all of them so you've just made numerous important contributions and I'm just want to thank you for sharing that you clearly put everything you have into everything you do thank you Peter Andrew thank you I really appreciate you making the time for us to sit down and talk in a long form way which I enjoy and yeah it's it's an it's an honor and it means a lot to me that you have read it twice and that you've appreciated it and praised praise that as you have thank you thank you once again for joining me for today's discussion with Dr. Peter Atia I hope you learned as much and enjoy the conversation as much as I did please also check out Dr. Atia's new book which is releasing on March 28th 2023 entitled Outlive the science and art of longevity if you're learning from and or enjoying this podcast please subscribe to our YouTube channel that's a terrific zero cost way to support us in addition please subscribe to the podcast on both Apple and Spotify and on both Apple and Spotify you can also leave us up to a five star review if you have questions for us or comments about the podcast or topics you'd like me to cover or guests that you'd like me to include on the Huberman Lab podcast please put those in the comment section on YouTube I do read all the comments in addition please check out the sponsors mentioned at the beginning and throughout today's episode that's the best way to support this podcast on various episodes of the Huberman Lab podcast we discuss supplements while supplements aren't necessary for everybody many people derive tremendous benefit from them for things like improving sleep supporting hormones improving focus and so on the Huberman Lab podcast is proud to have partnered with momentous supplements if you'd like to see the supplements discussed on the Huberman Lab podcast you can go to live momentous spelled O US live momentous dot com slash Huberman if you're not already following me on social media it's Huberman Lab on Instagram Twitter Facebook and LinkedIn and at all of those places I cover science and science based tools some of which overlap with the contents of the Huberman Lab podcast but much of which is distinct from the content covered on the Huberman Lab podcast again it's Huberman Lab on all social media platforms if you haven't already subscribed to the Huberman Lab podcast neural network newsletter it's a monthly newsletter that includes free tool kits things like toolkit for sleep how to enhance the quality and duration of your sleep toolkit for focus toolkit for neuroplasticity toolkit for deliberate cold exposure heat exposure and summaries of podcast episodes all of those tool kits can be found by going to Huberman Lab dot com go to the menu scroll down to newsletter and simply give us your email we do not share your email with anybody and again the newsletters and tool kits are completely zero cost and you will also find some PDF examples of previous tool kits again that's Huberman Lab dot com thank you once again for joining me for today's discussion with Dr. Peter Atia and last but certainly not least thank you for your interest in science.