The New Science of Becoming Ageless with Andrew Steele

Andrew Steele: [00:00:00]you start to see aging as the cause of this huge amount of human suffering. The reason I call it in the book, the greatest humanitarian challenge of our time, which could sound slightly, counter-intuitive a bit of a weird thing to say about this inevitable process, , is because it's responsible for, by far the majority of the death and the suffering in the modern world.

Diana Hill You're listening to Dr. Andrew Steele on psychologist off the clock

Yael Schonbrun: We are four clinical psychologists here to bring you cutting edge and science-based ideas from psychology to help you flourish in your relationships, work and health.
Debbie Sorensen: I'm Dr. Debbie Sorenson, practicing in mile high Denver, Colorado, and coauthor of act daily journal.
I'm Dr. Diana Hill coauthor with Debbie on act daily journal and practicing in seaside, Santa Barbara, California.
Yael Schonbrun: From coast to coast, am doctor Yael Schonbrun. a Boston based clinical psychologist and assistant professor at Brown university.
Jill Stoddard: And from sunny San Diego, I'm Dr. Jill Stoddard author of be mighty and the big book of act metaphors
we hope you take what you learned here to build a rich and meaningful life
Thank you for listening to psychologists off the [00:01:00] clock.
Diana Hill: If you listen to this podcast, you probably know by now that we are partnered with Praxis continuing education, and there's a reason why it's because Praxis really can help you your client's lives by learning how to effectively promote lasting change with evidence-based approaches DBT, compassion, focused therapy.
we love Praxis so much, especially because. Our very own Debbie Sorensen is going to be doing a workshop through Praxis. Tell us about it, Debbie.
Debbie Sorensen: Yes. I'm doing a webinar on acceptance commitment therapy for burnout. This is for therapists who are working with clients who are burnt out. And of course, as therapists, we are also occasionally may experience our own burnout. So hopefully it will be helpful for that too.
It starts August 25th and it's on Wednesday afternoons just for a few Wednesdays in a row. So you can check it out on the Praxis website and learn more. I hope you can join me if [00:02:00] you're a therapist, be great to have you there. And for all of the live online courses that Praxis offers, you can go to our website OFFTHECLOCKPSYCH.COM and get a discount code.

Jill Stoddard: Hey listeners. It's Jill here. As you know, in addition to being a POTC co-host, I'm also an author and part of being an author is having a platform or an online presence. So if you like the types of interviews I do, and you want to hear more from me on act imposterism, anxiety, and more. I'd love it if you would help me out by signing up for my monthly newsletter. And by following me on social media. Just go to jillstoddard.comand scroll to the bottom of any page to sign up for the newsletter and click the social media buttons in the upper right-hand corner. Thanks for your support.

Diana Hill So today on the show, we have Dr. Andrew Steele. Who's going to be talking about the science of aging. And I like to bring folks like this onto the show every once in a while, because I always like [00:03:00] to skirt the edges of psychology. I think sometimes we live in a sort of a silo, and it's always interesting to learn about how different fields are navigating.
Uh, Ideas and concepts that will influence our mental health and our psychology. And I have Debbie here. I'm curious what your thoughts were after listening to the episode with Dr. Steele.
Debbie Sorenson: You know, I really, it was a really interesting episode. I feel like there was a lot of. Um, things I hadn't thought about before and some new ideas related to what's going on with AGN that I had never considered, but you know what I really appreciated most about him. Very smart guy and very, um, reasonable and science-based in his approach.
And I have to say Diana, I was a little bit unsure about it when I was listening to it, to be able to introduce the episode. I was, I have a little bit of skepticism around this topic because what I've found over the years is that, you know, there are some, yeah. There's [00:04:00] some fear that people have about aging. And I think there's some age-ism behind that. I studied Gero psych a bit in grad school and learned a lot about age-ism and how we have perceptions around aging. But I think because of that, people are afraid of it and are a little bit prone to latch onto some, I would say. Maybe pseudoscience, like people spend a lot of money if the claim is that, you know, it will, you know, anti anti-aging products and that kind of thing.
And so, you know, again, I went into it a little bit skeptical, but what I found there was a lot of really interesting science here. And he kind of debunked some things, or at least says, you know, the research really doesn't support this and when you talk to him about some of his, you know, what he puts into practice and the behavioral strategies, I liked the simplicity of it and the research backing. Um, it's funny. I, I found some articles over the years where people, this is just an [00:05:00] example. Um, you know, a woman lives to be 105, what's her secret. And then she, it turns out, you know, she ate bacon every day for breakfast. This is something I really read in an article. Uh, in the newspaper and it's like, oh, well that must be the secret to longevity. We should all go out and eat bacon every day. It's just, I think people are so desperate for the fountain of youth sometimes that, um, can lead to some, you know, some unhelpful beliefs around.

Diana Hill Yeah, it's interesting. I guess my perception around aging has shifted so much over the years after just. Getting interested in this topic and learning and reading more about it. Just as a side interest that for me, it's been, he talks about this. It's, it's less about living longer and more about just wanting to.
Have health spans, so wanting to do what we can, as soon as we understand the science behind what our roads, our bodies over time, do what we [00:06:00] can to take care of our bodies, to the best that we can. But also. Not at the cost or consequence of living a full life. And I think that that's the other component of it, which is sure we could have like these perfectionistic tendencies around movement or eating or, uh, whatever, never go outside.
So your skin is never exposed to the sun, but then what is the consequence when you, you know, you miss out on your kid's baseball game? So I think that there's this balance between like, between it all and I'm, I guess I'm always sort of. A little bit open-minded in the science end of things. I believe in evidence-based approaches. And then I also am always thinking, well, there's some stuff that hasn't been discovered yet. And, um, there's also some time time-tested wisdom, you know, my grandma always. You know, she never made, she never put artificial stuff in it and things because she just thought, Ooh, probably not a good idea to, if it can last on yourself for, for hundreds [00:07:00] of years, maybe you don't want to eat it.
And you know, it turns out maybe some of those preservatives aren't so great for you. So I think there's a balance in it all I guess, is what I'm trying.
Debbie Sorenson: Yeah, I really loved that idea that you were just talking about of looking at more at health span instead of lifespan, right. This isn't just living. For the sake of living longer, it's more like being able to stay healthy and take care of yourself, to do the things that you want to be doing. I love that
Diana Hill So enjoy Dr. Steele. He's still likable. He talks really fast, but, uh that's because he has a lot to say his brain is going probably even 10 times faster than he's talking.

Diana Hill: Here we are with Andrew Steele, who is the author of ageless, and after obtaining his PhD in physics from the university of Oxford, he decided that aging was the most important scientific challenge of our time and switch fields to computational biology, which actually I had to.
Look up what computational [00:08:00] biology is. And now that I know what it is, I have a deeper understanding of your approach and how you use modeling and statistics to understand aging. But Dr. Steel has worked at the Francis Crick Institute using machine learning to decode our DNA and predict heart attacks using patient's medical records.
And he's now a full-time science writer and a fantastic science writer. So I'm really excited to share his work with you today, uh, based in London. So welcome Dr. Steel. It's great to have you on and talking today all about aging and actually how to grow old without maybe growing older.
Andrew Steele: Thank you very much. Thanks for that very kind introduction.

Diana Hill: I think that a good place to start is just with the term aging and maybe what some some folks initial responses to the term aging may be. And I know just in talking with friends and colleagues about aging, I've heard different angles. One is sort of, Oh, I want to know how to, anti-aging more for cosmetic purposes.

[00:09:00] Uh, others take the approach of, we just all need to accept that we're all aging and we're all going to die and we really need to accept impermanence. And then others may say things like, uh, anti-aging is for the super wealthy or the Silicon Valley, you know, multi-billionaires that want to just live forever, but you take a different approach to aging and, and maybe you can share why you think that aging and, and working on the science of aging is one of the most important humanitarian, , things that we can do of our times.

Andrew Steele: Yeah. Yeah. Wow. There's a lot to unpack in that opening question there. Um, I think it really does depend on how you approach aging. I think a lot of us just think of aging as this inevitable process, this, you know, it's just a side effect of being alive. We grow old, we see our pets girl, our farm animals grow old.

It just seems like this universal process that afflicts all living things. But actually, you know, when you approach it as a biologist, then you start to see aging as the cause of this huge amount of human suffering. The reason I call it in the book, the greatest humanitarian challenge of our time, which could sound slightly, counter-intuitive a bit of a weird thing to say about [00:10:00] this inevitable process, um, is because it's responsible for, by far the majority of the death and the suffering in the modern world.

So if you think about all the major things that kill us, things like cancer, things like heart disease, things like dementia, And these are all essentially caused by the aging process. The single biggest, what doctors called a risk factor. The thing that predisposes you to getting one of these diseases is simply how long ago you were born.

And that means, as I say, sort of biologically speaking, they're essentially caused by aging and the statistical way. I like to get a handle on this for us. The simplest definition of aging is to say, it's the amount of time it takes for your mortality risk to double. So let's unpack what that means. That means that if you've got a particular risk of death, then if you're a human it's seven or eight years later, your risk of death will have doubled.

So I'm in my thirties, I've got a risk of death somewhere around one and a thousand per year. But if that carries on doubling and doubling and doubling, by the time I'm 65, I'll have about a 1% chance of dying that year. It still isn't that bad. But then if you're lucky, you're lucky enough to make it to 80, your chance of death.

That is about 5%. If you're lucky enough to make it into your nineties, then your odds of death in any given year are greater than one in [00:11:00] six, that sort of life and death at the role of a dice. So, you know, what this really shows us is that this is incredibly powerful process. It's radically changing.

What's going on inside our bodies. It's predisposing us to all kinds of different diseases and. Although I talk about death. That's where it's the easiest way to statistically get a handle on it. Actually, you know, all these different ways that aging can kill you. You know, things like heart disease, things that cancer, particularly things like dementia.
Um, they, they take an awfully long time to take your life and they can have a huge toll on your quality of life. All they do so they can take away your independence. They can basically remove the ability for you to get around the house, to play with your grandkids, do whatever it is you want to be doing.
So there's this huge, um, Sort of collection symptoms of aging, these, these various diseases as the frailty, the forgetfulness things, we might not necessarily give a specific diagnosis in your doctor's office, but nonetheless affect your quality of life dramatically. And what that all means is that, you know, if so, let's take a global perspective now of the 150,000 people who die on planet earth every single day, over a hundred thousand of them.
So more than two thirds die because of aging. And what that really means is that [00:12:00] we've got huge, huge numbers of people suffering for years, maybe even decades of this variety of diseases until one eventually becomes severe enough to take their life. So viewed as a biologist, that's sort of the pessimistic side, but the exciting side.
And I really try and get across in the book is having gotten to this point where aging is responsible for so much of the death and suffering in the world. Then, you know, maybe if we can do something about aging, we can do something about all of that suffering too.
Diana Hill: I love how you boil it down to these hallmarks are these core processes that are involved in aging because right now medicine is so built around curing diseases and there's 1,000,001 diseases out there. So we just set in all these different directions of cure, you know? Cancer for this and cure macular degeneration and cure, you know, this kind of disease, but really when you boil it down to some of the core hallmarks of aging, then you can start to have a really big impact on a whole lot of things.
And there's actually a parallel there with psychology because the field of psychology and the science of psychology is doing something very similar where we're moving [00:13:00] out of diagnoses into more of a process-based model of what are the core processes involved in being a psychologically well, and it really does boil down to just a few like six core processes.
So I'd love to unpack with you some of those hallmarks, those you write in the book, 10 key things that contribute to aging. And then we can look at what the newer science is saying about how we can target them. But what are some of the hallmarks of aging? What are the things that cause us to age.
Andrew Steele: so I said that was a, the definition I just gave us the statistical definition of aging. If you want to sort of drill down into some of the biological nitty-gritty, that's where you get to these hallmarks. And there are a variety of cellular processes, molecular processes that the kinds of changes that happen

on every scale inside your body that eventually give rise to these various diseases, give rise to the frailty, give rise to it.
Eventually you dying of old age, and this can start at the very, very small scale. So you can have things like damage to your DNA. That's obviously the instruction manual that's inside every single one of [00:14:00] yourselves. And if you get mistakes, basically typos in that instruction manual, then as you get older, those typos can accumulate.
They can cause your cells to misbehave in certain ways. And that's one of the processes that can cause us to age. Then there's the other stuff inside yourselves as a little proteins that are, um, that they to be built from your DNA, the DNA is the instruction manual. The proteins are the machines that go around doing stuff in your cells that are structural elements in our body that hold our bodies together.
Um, they can be degraded with age and that can be another cause of aging. They are the things inside ourselves are made up of proteins and things like the mitochondria, which is commonly referred to as the powerhouse of the cell, these tiny little. Uh, they're called organelles that generate all of the energy that our cells use for this whole variety of different tasks.
Then, uh, you know, dysfunctioning all of those things that DNA going on in the mitochondria going on, whatever it is, can cause misbehavior in the cells themselves. And a really great example of that is senescent cells that cells that basically become aged they've, um, either divided too many times, or they've got too much damage in their DNA and that can cause them to stop dividing and basically put themselves into a state of arrest that like, hold it right here.
Um, possibly cause you're at risk of becoming a cancer cell. [00:15:00] And so that means they can accumulate our bodies as we get older. And then all of these things can combine together to massive hallmarks that essentially incorporate whole systems in the body. And a really great example of this is the dysfunction of our immune systems as we get older and nowhere has that been clearer in the last 12 or 14 months then with the coronavirus pandemic.
Cause we've really seen how people who are in their eighties. Well, statistically, they're literally hundreds of times more likely than, uh, someone who's in their thirties to die if they get infected with COVID. And one of the big reasons for that is that their immune system is just weaker for a variety of different reasons.
There's all these different processes. These sort of sub hallmarks create this massive hallmark, which is immune dysfunction. And that has a variety of other side effects as well as reducing, uh, defenses against infection. So that's sort of the biological way of looking at it. And as you say, I broke it down into these 10 categories.
I based it on a 2013 scientific paper that actually had nine hallmarks of aging, but I shifted things around a bit, partly for narrative reasons. And partly because some new sciences emerged, the one I added that isn't in that 2013 paper at all is about the microbiomes of the change in the bacteria and fungi and various microorganisms.

And I had guts [00:16:00] that we're starting to understand it has an influence on the aging brain, but what, although, you know, I, you know, do, it's talking about giving you quite a long answer there and it's quite a meaty chapter. Yeah. Book what's really amazing about this is exactly, as you say, you know, there are 10 of these hallmarks, um, that, that, that they are collections of process.
They're not individual things, but they're so, so much fewer in number and it's such a. Uh, a simpler way of looking at things. Then if you look at the disease, it's seen that there are hundreds of kinds of cancer. There are loads of different ways. You can get dementia or cognitive decline or things that can go wrong with your brain.
If things happen in your art, using of heart disease, you can have a stroke. You have just so many different ways your body can go wrong. And I think the, the, the, the paradigm change that we need to make in medicine is that we need to move away from trying to treat these end points, these diseases that come after all the hallmarks and hopefully into being a bit earlier and stop people from getting it, getting ill in the first place.
Diana Hill: I really want to get to some of the, uh, interventions that are cutting edge around that, and that are probably going to be showing up fairly soon, according to your research. But first I want to rewind a little [00:17:00] bit and talk about. Why we, why our bodies would even do this in the first place. So you, you offer a quote that's actually often used in psychology as well, which is nothing in biology makes sense.
Except in the light of evolution and a psychology has co-opted that a bit of very few things in psychology also makes sense, except in the light of evolution, but maybe you can talk a little bit about how you tie aging to evolutionary theory. And why would we evolve to grow old? Maybe even this concept of evolutionary neglect that you mentioned in the book.
Andrew Steele: Yeah. And the, the sort of a classic thing that the other very famous quote about evolution is it's the survival of the fittest. That's the one that's really entered the popular consciousness. And so you look around and think, you know, what is possibly fitness optimizing about growing old about this process of progressive degeneration and the way you've got to look at it as you know, what does evolution really care about evolution?
I'm sorry to say, you know, if you're a man that cares about your sperm, if you're a woman that cares about the babies you can produce. And basically [00:18:00] beyond that evolution, doesn't much care for you as an individual. And the reason is that if you can pass on your genes to the next generation, the genes, which are the sort of units of evolution can carry on into the indefinite future.
And if you imagine a gene that makes an organism more likely to reproduce for whatever reason, by whatever means, and that gene is going to become more frequent in the population and it's going to continue to expand. So why might that mean that something like aging could evolves and it's obviously bad for the individual that's carrying, you know, that combination of genes that makes it age.
When you got to think about, um, the fact that you might die of something else or the strange fact is that the reason that we die of aging is effectively because

other things can kill us. So let's think about, do you use the easiest way to imagine this is, imagine you're a little tiny animal, like a mouse, and that means you're an animal that lives in a very hazardous, natural environment.
You've got a, you know, you've got predators, you've got cats with a sharp eyes and claws that are out to get you. Um, you can dive infectious disease. Uh, you can even just dive exposure. You're so small that if it gets really cold, you can basically freeze to death. So there are loads and loads of different ways that mice in the wild can die.
And that means they tend to have a pretty short lifespan in the wild, their [00:19:00] lifestyle is probably months, maybe a year if they get lucky. And so what that means is that mice tend to gear up and reproduce very quickly. They have a big litters, they can have babies once a month or so, which is obviously much quicker than humans can manage it.
And the reason is that they're going to die of something. And if you, if you were to wait. Um, you know, 15 months or two years before you were able to have kids, then you'd probably statistically be dead before you were able to pass on your genes. And that obviously wouldn't be a very good deal for evolution.
So evolution does, is it prioritizes reproduction over absolutely everything else. And if you're an animal that lives in a dangerous environment yeah. It gives you up to reproduce as quickly as possible, even if that comes at the cost of degeneration in later life. So you can imagine, you know, on a, on a very simplified level, evolution is willing to throw your body together and sort of slapdash way, as long as it gets you to reproductive fitness, as quickly as you can pump out those kids.
And then you can basically die as far as evolution is concerned. Cause you've, you know, you've, you've served your purpose. And what that means is that we have this a variety of. Molecular detritus in our genome, which, um, you know, doesn't conspire to kill you in your first 20, 30, 40, 50, even 60 years of life.
But if you do live long enough, [00:20:00] then you know, effectively are irrelevant to the gene. Portland's evolutions happy to let these mistakes happen. And that's why ultimately, you know, we do succumb to cancer. We do succumb to heart disease at the surprisingly synchronized time in our lives, because that's the point we've got old enough that we've, you know, we've done our reproduction. We passed on our genes. Evolution just doesn't care about us anymore. Unfortunately.
Diana Hill: and that's why some of those larger animals that are harder to kill maybe live longer. You mentioned things like the whale that lives or the tortoise, that's the cover of your book that, that maybe it takes them a little bit longer to reproduce. So there they get to stick around
Andrew Steele: Yeah, so I mean, size size, and aging is one of the most famous correlations in evolutionary biology, which is that, you know, the bigger you are, the longer you tend to live. There's probably a little bit of, um, sort of circularity in that statement because you also need longer to grow to the size of a whale. You can't become a whale, a full-sized blue whale in six months. However, as you say, right, that, you know, if you're a whale you're in the ocean, you haven't

got any natural predators. Cause you're absolutely enormous. Then that is one really, really clear way that you can evade other forms of death and [00:21:00] thereby evolve to grow older.
Um, tosses are another great example. They've got this hard shell and actually the reason the tort is on the cover of my book. I think they've, they've got an even more exciting property they've they? They, they are something called negligibly senescent. And what that means, that's that senescent. There's just the scientific word for old.
And so what that means is they are that they are negligibly old, they don't age. So no matter how old or taught us it is his risk of death stays constant. So it appears that, you know, for whatever reason, evolution moving in, its mysterious ways, this decided the optimal way to pass on a tortoise's genes is to have it not deteriorate with age.
And what's most exciting about that is that, you know, tortoises don't have an increased risk of death. They don't get frail, they stay reproductively active even until very late in their lives. And this means that aging isn't a law of biology. Although, as I sort of mentioned at the top, we age our animals age, almost all mammals age, there are this handful of creatures tortoises.
There are some salamanders, some fish aren't, there's even one mammal called the naked mole rat, which are negligibly senescent. They don't appear to grow older as they grow old. So yeah, it's, it's, it's, it's [00:22:00] really fascinating. The sort of diversity of aging across the tree of life. It really shows us that this isn't necessarily something that we're forced to do by some law of biology.
And so maybe, you know, with human ingenuity, we can work humans way out of that too.

Diana Hill Hi folks. If you're listening to this episode, you're probably interested in health to one degree or another psychological health and physical health and exciting news. I'm going to be taking a group of folks to know Saara Costa Rica in April, 2022.
We're going to be staying at blue spirit, which is a beautiful retreat center there, and we're going to be exploring psychological flexibility. I'm going to have a yoga teacher with me, so we'll be doing some movement and then just a lot of rest and re restoration on the beach in Costa Rica in one of the blue zones of the world. So join me. You can find out more at Dr. Diana hill.com.

Diana Hill: So I want to go back to that word senescent, because it shows up when we're talking about senescent cells and in sort of pop, uh, science talk, people use the [00:23:00] word zombie cells. I think that's sort of the, I often think that scientists are really bored in their lab doing this bench work hour after hour. And they come up with these fun names, like zombie cells and, um, you know, sort of find little tricks of the trade, but with senescent cells, what is happening there and how is it contributing to our aging? And then maybe how are you using comparative biology and, and techniques to start to target those cells?

Andrew Steele: so that's, the sellers are probably, I think the, the, the most fascinating example of, of a hallmark of aging, I think actually, because, well, firstly, because they're quite easy to explain, which is always nice. And secondly, because the treatments for them are very, very close on the horizon. It's actually by developing some of those treatments that we've really started to understand just how widespread their role is.
So let's rewind what is a senescent cell? Well, as I mentioned in my long answer about the hallmarks senescent cells, they're basically aged cells. They're cells that have either been around in your body a very long time and therefore accumulated some DNA damage. And, uh, that you know, that therefore that is sort of damaged.
They're not in a fit state to go on. They could [00:24:00] just have divided a lot of times. So we know that if you have a cell in a dish, it's actually how senescent cells are first discovered. If you know that divide and divide and divide. Uh, the first human cells, this was done with some cells called fibroblasts.
They can get to about 50 cell divisions and then they just stop. And actually they get really weird under the microscope, even to it in a non-expert microscopists like me, they, they, they exhibit what's called the fried egg phenotype by some scientists. That's another name. They probably came up with our board in the lab.
They basically like a fried egg spotted on the dish. And so they look vastly different to a regular cell. And when these cells stopped dividing, we think this is an anti-cancer mechanism. So the idea is that, you know, say, say you have accumulates and DNA damage. The way that cancer works is that you get damage to the DNA of a cell and that damage, it can turn off genes that cause the cell to stop growing and it can turn on genes that encourage the cell to grow. And it shouldn't. And some combination of mutations can therefore allow that cell to divide an infinite number of times effectively so that cell can divide and divide and divide it, continue to assumer that tumor can metastasize. It can spread around your body and that's how cancer ultimately kills you.
So our bodies are really, really keen that we don't [00:25:00] get cancer because that's obviously a huge threat to all multicellular organisms. And so what we do is, you know, if a cell does divide an awful lot of times, if a cell does have suspicious looking damage to its DNA, our body basically slabs up, slammed on the brakes.
And this is actually a great example of evolution of aging we were talking about because on the, you know, when you're young, this is a great move, cause it means the cell stops dividing. Actually your immune system comes and gobbles. Those cells up, they're taken out of circulation. Everything's fine. But as you get older, you get more DNA damage just because your cells have been around longer, more of your cells have divided more times.
That means there's another incentive for them to become senescent. Your immune system we've already talked about, becomes less vigilant it's, uh, you know, aging itself basically. And in fact, ironically, because some of its cells can

start to become senescent. And that means it gets less efficient at picking up these senescent cells.
And that means they can accumulate in our bodies. And so suddenly, you know, what was beneficial in youth is detrimental. In old age, these senescent cells are pumping out this toxic cocktail of molecules. It turns out they're not just sitting there, you know, not dividing nice and benign. They're pumping out all these different molecules, basically saying to the immune system, Hey, I'm over here, come and clear me [00:26:00] up, but actually because your immune system is getting less effective at that.
And because these senescent cells are accumulating more and more rapidly, uh, these molecules are just being pumped out constantly and they cause this background effect called chronic inflammation. And that's, uh, basically, you know, it's, it's overreaction of the immune system, the immune system's in this constant state of paranoia, because of things like senescent cells, increasing the inflammation inside your body.
And what we now know is that that can cause all kinds of different diseases. Uh, it can cause heart disease. It can cause dementia, ironically, it can even cause cells to turn cancerous. So it's got this, certainly it's this bizarre sort of a sting in the tail so that it was created as a counter defense mechanism actually turns out to cause cancer when it becomes too severe.
And so, because it can accelerate this whole range of age related diseases, this is actually classic hallmark. And what you'd hope is that by targeting one of these hallmarks of aging, by targeting the accumulation of senescent cells, you can hopefully prevent multiple of these diseases at the same time.
And actually that's exactly what we can do. There was an experiment done in 2015, uh, using mice. And what they did was they gave them some drugs that would selectively kill these senescent cells [00:27:00] while leaving the rest of the cells in the mouse, his body unharmed. And what they found is that by clearing out those cells, they effectively made the mice biologically younger.
So they gave these drugs to mice. That was I 24 months old, which is about 70 years in sort of human terms. Obviously, as I said earlier, mice had a much shorter life span than we do. And what they found was they lived a couple of months longer. So it may be a few years in human terms, but they weren't just staggering on an old age having, you know, having had some disease prevented and therefore living longer, but still frail, still, you know, suffering from all the other consequences of being old.
They got this heart disease, they got this cancer, they got fewer cataracts that sort of on the disease side, they were also less frail. They, um, they, they, they can run further and faster and a little mousy treadmill. There are some fascinating sort of psychological results for as close as you can get to psychological results with a mouse, which is that they seem to be more curious. So if you put a young mouse in a maze or in any kind of novel environment, they will tend to explore. That'd be like, Oh, you know, where am I, what's going on? Can I find some food, et cetera, et cetera. Whereas if you put an older mouse

into a similar environment, they're often a bit less exploratory, they're a bit more anxious.
Um, they're less willing to get about. And they [00:28:00] found that by giving these analytics to old mice, they could rejuvenate some of that curiosity. And frankly, the mice just look great. They even have better for and better skin. So, you know, compared to the sort of ragged wrinkly, gray-haired blotchy furred, mice, it didn't get the treatment.
These are the mice just look great. And so what this shows us is that by targeting these senescent cells, you can have this phenomenal global effect on all the different things that happen to us as we get older. And hopefully the idea is that we can then transfer those treatments into humans as well.
Well, actually what's most exciting is there are currently 20 or 30 companies trying to turn these analytics from an idea in the lab so we can actually have in the clinic, this is something that's happening right now. So, um, that's, that's definitely why I'm very, very excited about these things because now we understood how global our effect can be.
I'm really looking forward to it. You know, seeing if these results can translate into people.
Diana Hill: Yeah, well, I'm sure a lot of the listeners that are hearing you talk and Andrew, we talked about how quickly Andrew speaks. So we all, you want to listen at 0.75 to be able to download the amount of content [00:29:00] that you just offered in
Andrew Steele: Absolutely. No
Diana Hill: Um, go for it. Uh, and, and also his book is incredibly detailed and you kind of need to go through it with the final.
Tooth comb, but you can also think about the big picture of what you're saying, which is basically as we age, our body does all sorts of things because it doesn't really prioritize our aging. It prioritizes our reproduction. And we now have some technology that can change our, the process of aging and in doing so it can lead to massive, massive impact on our health span, which ultimately my interest is not as much about like living forever.
It's just that when I'm older, I want to be able to be as active as I am now. I'm I love to hike. I love to run. I love to garden. I see how, uh, as our bodies decline, it really, they kind of can impact our quality of life. And I really love my kids and I want to be around and healthy for them as long as [00:30:00] possible.
And one of the things that, uh, sort of it in my, in my interest, in, in aging is also, I did some stuff in my twenties to my body.
Andrew Steele: didn't we all
Diana Hill: Now I'm in my forties. I want to do it differently. I want to like take care of, and hopefully I'm do some of the damage that I've done. And I imagine a lot of our listeners can relate to that. So, okay. So we have both behavioral stuff that I want to look at. Like, what can we do right now? Behaviourally with, before these medicines come out and then there's also the, the, um, the interventions and the medicines, and some of them just in going to the intervention part of senescent cells, you, you talk about some of the ones we've heard.

So rapid myosin Metformin, uh, which seemed to be especially Metformin. There may some be some promising, um, uh, results with that. But there was one that I hadn't heard and it relates back to sperm, which is sperma Dean, which sounded kind of, it's just kind of fun to talk about and learn about. So what is permitting and how could [00:31:00] that be helpful for us with these, with these nasty senescent cells?

Andrew Steele: Yeah. So there are loads of these naturally occurring compounds. Basically, I try to dig into, to understand, um, you know, whether they could potentially intervene in various parts of the aging process. A rapamycin was something that was discovered in a bacterium on Easter Island, actually. So that's the Island of the massive stone heads that you might have seen.

Um, um, nature's just full of these things. These biological compounds that have incredible specificity, they can do very precise things. And some of them do seem to have some effect on the aging process. So if you give rapamycin to old mice, it actually seems to slow down their aging and allow them to live longer in good health Metformin is another really exciting one that you mentioned.

Uh, this is a diabetes drug it's incredibly, uh, well prescribed. I think there are literally tens of millions of prescriptions for this written in the U S every year. And in the UK, we've been using it since the 1950s. So that's, and that we've got an incredibly good safety record for actually the original source of Metformin or the drugs, the drugs that preceded it, rather than the sort of molecules that chemists optimized intimate.

It was a particular kind of flower. Um, and what they found [00:32:00] is as well as being a diabetes drug, it seems that diabetics are on Metformin, uh, live longer than non-diabetics who aren't taking Metformin. At least that's what it appears from the data. And that's very suspicious because diabetics, they tend to be less healthy than people who aren't taking Metformin.

They tend to be more overweight. And yet, nonetheless, there's a slight signal that, uh, the diabetics who are taking the drug live longer than them, the non-diabetics, there are some hints it's got anticancer properties. And so the idea is we're going to try a trial giving Metformin to people who are basically quote, unquote, healthy, what we would currently call healthy people who are in their sixties or so.

And hopefully what we'll find is that they get fewer of these age related diseases. They die later than the people who are in the control arm, who just getting a placebo sugar pill effectively. And finally, you mentioned sperma Dean and this is one of the most fascinating ones, just because that's at this, this as the name suggests was first discovered in sperm or other in semen.

So, uh, the microscopist who was first, uh, actually the guy who invented the microscope, a Dutch guy, and I'm going to get his name wrong. Now it's Anthony Vaughn, even who it costs, something like that. And what he found was that as he was, uh, he, he started examining all kinds of things under the microscope. [00:33:00] And I guess being a guy, one thing he tried was looking at his own semen. And as the seaman, uh, basically dried out these little crystals formed as

he called them. Sperma Dean. We now understand that it's a particular kind of a particular molecule. And if you give it to 'em. There's there's some reasonably good evidence.
There's not compelling, uh, like human data at the moment. I don't think, but there's some evidence that it seems to increase a process called autophagy, which is a cellular recycling process. And therefore, again, this could be another one of these compounds that either, you know, maybe we will be taking spermatids or maybe we'll be taking something related to it.
Maybe it'll just give us, uh, you know, some inspiration as a, sort of another Avenue for research, but there are just so many different compounds all throughout the natural world that it's going to be well worth rifling through our drug makers back catalogs, to see if any other things can slow down the whole aging process.
And we can just, uh, you know, take advantage of those for ourselves.
Diana Hill: and how I understood it when I was reading your book about slowing down, the aging process is more kind of like how we can like dribble a ball down the field to get to the point of making the goal of when we actually come up with, um, these big interventions, like the dribbling of the ball is the [00:34:00] behaviors that we can engage in.
Now that will support autophagy that supports anti-aging so that as the, um, the. The science catches up and the things the clinical trials are, um, now available to the public, we can stay around long enough to have access to them. Is that accurate in terms of like how to, what we can do now to get to the point where we can actually use some of these interventions.
Andrew Steele: how I look at it. I've, I've found that writing a book on aging is the single best way to make you really pay attention to your own health. And the first reason for that is obviously that you sort of, you know, you begin to understand a bit more about the biology behind a lot of these bits of health advice.
But secondly, this stuff really is, uh, you know, it's on the horizon. It's it's well, within the lifetimes of most people who are alive today. So Metformin, I mentioned, uh, the clinical trial for that actually should already have started. I think it was delayed due to COVID, but we're going to potentially have results for that.
In the next three to five years, these analytics, the first trial was started in 2018. What you're going to find is probably the first, um, th th the first successful [00:35:00] cytolytic drugs. If there are, if, you know, if they work in humans are going to be for specific diseases. So we know there are certain diseases where senescent cells are known to be a problem.
Things like lung fibrosis, things like arthritis, And if the drugs prove effective at treating those conditions, and most importantly, if they prove safe, they don't have severe side effects. Then we could imagine, you know, giving those things preventatively rather than giving them to people who are already sick.
We could give them, you know, say you're 50 or say you're 60, just to clear out some of those senescent cells and hopefully, you know, slow down your aging,

stop you from getting it in the first place. And, you know, while that might be five or 10 years away, that's still not miles and miles into the indefinite future.
So the really exciting stuff about, you know, following health advice, things like eating well, things like, you know, exercising, the stuff that I talk about in the health advice chapter of the book, um, is, you know, the longer that you live in good health, the more chance you have of being alive to benefit from the sort of latest and greatest, and anti-aging treatments that come online from a pharmaceutical point of view.
And so I think that, yeah, that's the, that, that's certainly a huge motivating factor for me to try and keep healthy, to make sure that, you know, when it's the first or second or third generation of some analytics on, there are [00:36:00] some other of the more speculative STEM cell or gene therapies that I talk about a bit later in the book, you know, when those come online, I want to still be going and I want to be healthy enough to have something.
So it's definitely a huge motivator for me.
Diana Hill: Well, I have to confess, that the strategies that you give are pretty, um, Basic strategies for us to, to, to do that we can implement today. And that the ones that I've been trying or more of the out there kind of strategies, which I think you do bunk a little bit. I actually was , I was reading your book, I went into my cupboard and just pulled out all the supplements that I have purchased over the years.
And I'm not using most of them at this point, but I, I could cover my whole counter with, you know, everything from the, you know, the basic ones like vitamin D and vitamin C to more unusual ones. Like NMN , we interviewed Dr. Sinclair on the show and, uh, you know, he has some ideas around what we should be doing, and then I've done, I've done kind of like the unusual staff
, The message that I got from your book is that really it's much more simple than we think it's as simple as brushing our [00:37:00] teeth.
Andrew Steele: So some of it is surprisingly basic and I found that things like exercise, things like not smoking, uh, eating well, you know, making sure you get a balanced diet and not too much of it. These are all things that everybody knows, but I found that understanding the biology behind them and how it is that they help you, uh, you know, H how they help you get healthy and how they literally slow down the aging process.
They make you live longer, but not just longer, they also make you live healthier. Now I just found hugely motivated, even though they can sound like quite, you know, regular bits of health advice in terms of the more out there stuff. I, I really tried to confine myself to what we have really solid clinical evidence for.
I think the vitamins is a really great example because there've been these huge studies. The thing, if, you know, if you're a scientist, if you're a doctor, the absolute best gold standard evidence or something is what's called a systematic review, this is where scientists don't just look at a single study.
They don't just look at a thousand people and, you know, and, and so on. And so on. They look at all the studies that had ever been done on a particular topic. And they'll go into the literature. They'll dig through all the studies, they'll find out, you

know, which ones are trustworthy, which ones aren't, they'll pull their effects together and do the biggest statistical analysis you can possibly dream of.
It's an [00:38:00] incredible amount of work and the best match analysis that I could find or other systematic review, which is sort of the next stage up, um, is something called a Cochrane review. And these are very well known in the medical community that the most highly respected collaboration of people who pulled together, these systematic reviews on all kinds of different topics.
And what they found was that with the vitamin supplements, they looked at, um, three of them had actually no effect on lifespan and the other two slightly increased your chance of death. And the, you know, it's not as though your vitamin E, which is one of the, one of the negative effecting ones is going to kill you immediately.
It wasn't, you know, doubling your chance of death or anything, but nonetheless, it really shows that the, um, you know, these things that we conventionally, they sound like they should be healthy. Don't the vitamins supplements. Richmond's a good, right. And yet, you know, if you actually dig down into the trial data, um, then they don't appear to have any effect or even have a slightly negative effect.
And I think that that's just, it's really, really difficult because I think that some of the more. Out there, sticking there, maybe NMN will turn out to be helpful. For example, there is some suggestive data in, in mice, and there's even starting to be some suggested data in humans. I think it was a study that came out this week about, uh, but, but, but [00:39:00] what you find with these studies quite often is they're very, very specific.
I think I might get this wrong. So don't quote me on this. But this study that came out this week showing a benefit of NMN was in older diabetic, maybe just women. And it was looking at their muscle function. Now it isn't to say that it doesn't have wider benefits. And obviously you have to start somewhere with science.
You have to start with these small studies, but, um, you know, what we haven't got is a trial, the trial I would like to see, which is a randomized control trial. We get a bunch of people who are age 60 or 65. We give them NMN supplements for a specified length of time. And we find out, you know, whether they, whether they get age-related diseases later, whether they die later, um, there is actually, I think recently an experiment came out in mice, which showed that doesn't work for mice.
It's, it's just such a naughty thing. And it's not to say none of these things are ever going to work. We just, haven't got the solid evidence that we've got for the interventions that I do talk about. And just to mention that, one of the thing you talked about with brushing your teeth, I think this is the other really fascinating thing about aging biology is that it does uncover some less conventional bits of health advice.
Um, obviously we, we all should brush our teeth for a variety of reasons. You know, you don't want to end up at the dentist with an enormous bill, but actually there's pretty good [00:40:00] evidence that having a good, good oral health,

good gum health can reduce your risk of heart disease and maybe even dementia.
And actually the reason behind that is that chronic inflammation process that I mentioned earlier. If your immune system is engaged in a constant low-level battle in your mouth with tooth decay, with gum disease, then that inflammation can drive the whole aging process. And that's how we think it causes an increased risk of heart disease.
There's even some suggestion that the bacteria that cause gum disease have been found in the brains of people who have dementia. And so it's suggested it's not conclusive. We don't know which way the causation runs yet, but it's good enough evidence for me to really concentrate on brushing my teeth. So that's quite an expansive answer.
There are these whole range of different things. I tried to really stick to the stuff, which is evidence-based, we've got solid, solid proof for, but actually, you know, as we get, as, as we all get older, we're going to have to make these decisions. You know, what level of evidence is sufficient for me? Do I need to see an absolute systematic review or when I'm 60, you know, will it, will I be slightly more or will I be slightly less conservative?
I should say because you know, my, my, my, you know, my clock is ticking and therefore, perhaps I'll be more interested in trying some of the more speculative speculative interventions, I guess.
[00:41:00] **Diana Hill:** Yeah. And that's why I wanted to interview you and why I really appreciate your approach because it is a measured one and you're not encouraging us to just go like have expensive urine. or even potentially some of these interventions may be harmful to us, right.
Because we don't have, we don't have the science to back them up, but there's one intervention. I am pretty sure is not harmful, but that's been talked about in the field of psychology, which is. Loving kindness, meditation. I can't see what the harm would be in that. and I see you a couple of papers this morning because I wanted to get your take on it.
Uh, because one of the things that that folks are talking about in the field of psychology, a lot, a very hot topic right now is compassion. And even though the researcher who has worked on telomers a lot in combination, with Dr. Blackburn. Elissa Epel has written a bit on using compassion based approaches as well as mindfulness approaches to, [00:42:00] help with telomere length.
So I'd love for you to chat with us a little bit about telomers, how they're a little more nuanced than we hear about, , and then also what you think about some of these, these interventions to increase our telomere length, like meditation or compassion, and are those papers just completely out there.
Andrew Steele: before we start. I'd just like to say I'm absolutely down with any podcast host who sends me references before we chat, because that's, that is my bread and butter. So that's really great. And I really enjoyed having a look at those two papers. Uh, it's a really fascinating area and it's, it's, it's this, as you say, nuanced, and it's complicated.

Um, so telomeres that's one of those common questions you get asked. If you've written a book about aging biology, people say, is that got something to do with telomeres? Because back in the 1990s, I think a lot of people thought that telomeres were going to be the key to aging. We're going to be able to sort out telomeres.
So are aging. So what are they that these little protective caps on the end of our DNA and every time our cells divide our cells have to copy that DNA cause it's in their nucleus, it's the instruction manual. So we need to make sure that both daughter cells have a copy of that DNA [00:43:00] instruction manual.
So what happens is, uh, uh, we've got little prep, proteins, or enzymes inside ourselves that copy that DNA. There's this bizarre mistake that evolution has made in creating those enzymes and that's that they can't make it quite all the way to the end of a string of DNA. They end up chopping a tiny bit of the molecule off every time a cell divides.
And I imagine that was in a really crucial bit of DNA. If it was starting to chop off some really important gene, that'd be incredibly bad news. Your cells would gradually be losing their function every time they divided. So what evolution has done rather than sorting out the obvious problem with this enzyme and making it that can copy all the way to the end.
Instead, it's added this huge repeated length, you know, tens of thousands of DNA letters often, which, um, they're effectively nonsense. It's a pattern of 60 and 80 letters repeated over and over and over again. And so if that gets chopped off, it doesn't really matter, you know, until it gets really what's called critically.
Sure. You can just carry on chopping off a little bit more of that telomere. None of the important DNA is affected, but you can see that, although this is a solution, it's a necessarily short-term solution. Because as your cells divide, they're gonna lose a bit more of that telomere. And [00:44:00] eventually the telomeres can become critically short.
And actually, this is what we think is behind the senescent cells that we talked about early. Some of them, those cells have divided too many times and become senescent the way the cell knows it's divided too many times is by looking at how long the telomeres are. And if they are below that sort of threshold length, then the cell goes right, that's it.
It's time to put on the brakes. So this, this really sounds like an obvious candidate for a cause of aging and Elizabeth Blackburn, who you mentioned, uh, she got the Nobel prize for discovering this whole mechanism behind how telomeres can be extended with an enzyme called telomerase. And so in the 1990s, it was thought that, you know, if we can just get, tell them into ourselves, extend those telomeres, maybe we can all live, if not forever, you know, a great deal longer in good health.
The problem is that, you know, if you think about adding telomerase to a cell that enables that cell to divide more times, and what is, what do we call a cell that can divide loads and loads of times? Well, as I mentioned earlier, we call that cell cancer. So it was found if you just put extra telomerase into the cells of mice, they basically get terrible, terrible cancer.

And so, you know, there's this, um, there's a total about face in the biology community. I think it's partly just because the [00:45:00] narrative is beautiful and cynical, isn't it? It's like scientists think they've discovered the fountain of youth that it turns out actually just to give you cancer. So there's this sort of cynical, cynical way of repackaging it.
What we found in more recent times, there's a bit more of a nuanced story. If you give that telomerase temporarily, if you just give someone longer telomeres without giving them extra telomerase. Uh, it seems at least in mice, I've had someone, but actually the experiments so far have been in mice. Then you do a, to be able to make them live longer in it and better health.
That's sort of a separate question though, to all this, um, all this stuff about what happens to our telomeres as we get older. So I said, they, they get shorter as you get older, there is definitely a what's called correlation. I think there's a relationship between how long your telomeres are and how old you are, but it's not as clear cut as is commonly portrayed.
So, you know, it's quite possible for someone who's 80 to have telomeres that are the same, that there's someone who's 30. Cause there's, it's just quite it's. You know, what we call insight is a noisy measurement. There's quite a lot of uncertainty on the length of someone's telling me it, depending on how old they are.
So you can sort of get a trend. And we know that people who are the same age, if one person has shorter telomeres and the other, the person with the shorter [00:46:00] telomeres is more likely to get ill and more likely to die sooner. But it's really not as clear cut as you know, your telomeres are a hundred basis shorter. Therefore I can tell you that you're going to die a week later. We just, you know, it's not as accurate as all that. And what we found is that not only are there biological things, you know, sort of classic molecules, things that you eat, et cetera, et cetera, that can have an effect on your telomere length.
There's also some increasingly good evidence, that lifestyle sort of broader lifestyle things. You know, things like stress, things like, uh, you know, effects on your psychology can change the length of your telomeres as well. And there's some really quite solid evidence that childhood stress can make your telomeres shorter.
And it's not at all implausible at having an awful stressful childhood could be something that caused you to go on to develop diseases, caused you to effectively aging a little bit faster. So that sounds like it's probably something in there. And therefore, you know, so that the first paper you sent me was a paper from 2009, uh, with Elizabeth Blackburn on the author list.
And they sort of, they speculate that perhaps therefore, these kinds of meditative practices that reduce stress, it seems plausible that those could go on to give you longer telomeres and maybe go on to [00:47:00] improve your health. And just from a, you know, a purely, not like not even a biological point of view, it does seem intuitive that having a less stressful life is going to be a more pleasant one. It's going to be, you know, potentially a healthier, longer one. Cause you know, being, being stressed is we know is not good for your health. However, the sort of

the later paper that you sent me, the 2019 one, which was looking at a specific study where they'd actually given that they had a control group of people who didn't meditate, they're actually annoyingly for the control.
A few of them did meditate anyway, which show makes it even more confusing. They had another group who had, um, the loving kindness meditation you talked about. And they had another group who were doing mindfulness meditation. And what they found was they followed these people for six weeks and they found that the people who did the love and kindness meditation had the longest telomeres at the end of the study.
Now, unfortunately, the thing that's really tricky about this is it's just not clear why the people in the control group had a measurably shorter telomere length after six weeks. So I've just told you that, you know, this is the sort of thing where it's sometimes, you know, really unfortunate 30 year old could have the same telomere length as a lucky 80 year old.
So how on earth, you know, these things don't change over the matter [00:48:00] of weeks, at least not necessarily detectable ways. So that sort of is a bit of a red flag. And so although the telomere length seem to be decreasing more slowly in the groups who are doing the meditation, it's just really not clear exactly what they were seeing.
Um, so I think the trouble is what you need in order to really, you know, prove something like this in a cast iron way is to have really, really solid data loads and loads of people. You need to follow them for a bit longer time. They're sort of suggestive information here, but I don't think the sort of gold standard of evidence actually, you know, trying this out in a randomized way, which they try to do.
I don't think they looked at it for long enough. They probably didn't look at enough people because the, because of the nature of telomere length and the final caveat in all of this is that because telomere, like this is so noisy because it's so uncertain exactly what it means. It's not necessarily clear if someone has longer telomeres, they'll definitely live longer.
So there's quite a few steps still to go to prove this stuff. But like you say, it's not obviously harmful. I don't think it's going to be, you know, it's certainly not going to be reducing the length of your life. And if you enjoy, you know, being mindful, if it calms you down, if it makes life, you know, we, I actually mentioned much, much earlier.
This isn't about lifestyle. It's about health span. It's [00:49:00] effectively about happy span, isn't it? And so if that's something that brings joy to your life, I certainly wouldn't try and talk you out of it because there's scientific evidence that it'll increase the length of your telomeres. Isn't fully robust.
Diana Hill: well, Andrew, that was fantastic. Wow. So you just did, you did so many things in that. Um, then that answer one was you gave us, uh, a little bit of an education on, on science and research and the need for multiple studies, with large groups of people replicated that sort of systematic review to come to draw any conclusions, you know, and then also your, your just capacity for me to throw two papers at you and for you to digest them so quickly and come back and in, in

real time, um, share your thoughts, which I think really demonstrates for you as a science writer, what you do in your work, because you take these complicated concepts and you, you both.
Simplify them so that a reader could understand them with things like story and metaphor and such a fantastic job of like, [00:50:00] at one point you talk about telomeres, like a brick later, that's laying bricks, but God himself in a corner and you can't keep, you know, laying the bricks when you're trying to replicate your DNA.
But you also do a really good job of not oversimplifying things. And so that's where I feel like we really need to like what science can we trust? Because these will be headlines that come out. And as consumers, we read that quick headline, like loving kindness, meditation will make you live longer from longer telomeres. And we just, we just eat it up because we want to eat it up. We want to believe this stuff. So we believe it, but it's really helpful to have, um, scientists like you be able to, uh, digest it for us and, and, and help clarify really what, what is, what is it that we can stand on here? So thank you for doing that.
, I'd love to talk more about, , some of the more, I think in the sort of later chapters of your, of your book and your work is some of the sort of bigger picture implications of this work. I think for us in the field of psychology, obviously we're, we're interested in what are the psychological implications.
If people [00:51:00] are now living these super long lives, what's the impact. You know, there's always people worried about overpopulation or, you know, things like that, but just what would be the impact of the course of our life, how it looks so different. If I was new, I was going to live healthily into 120 versus thinking I'm going to live until I'm 80.
, so maybe we can start there and then we can also talk more about sort of the impact of what you're hoping for this research, , in terms of, how we can change policy to support anti-aging research, but let's start with the psychological impact of, of living longer. I'm sure you've thought about that.
Andrew Steele: Huh. And, you know, with the caveat that I'm by no means a psychologist. I think that, you know, people, people think, I think when you start talking about anti aging research, people put it into this really, um, sort of special social ethical category, and they start asking all kinds of questions. They wouldn't ask you if you a cancer researcher, for example, you know, they wouldn't ask you, as you mentioned, just now about overpopulation.
They wouldn't say, Oh, you know, now you've come up with a way to cure cancer. Uh, is it gonna be the case that all those 80 year olds who are now living to 85 are going to cause this enormous overpopulation crisis and in the same way, you know, thinking about the psychology of what [00:52:00] happens as we lived longer and older and older ages, and you know, people ask me if I'm 300 years older, I'll be bored and you're like 300 Whoa, slow down.
Even if we, even if we sought this out, I'm not going to be 300 for another four, sorry, 270 years. So it's going to be something that I've got a bit of time to get used to. And my attitude to the psychology is that. I think it's going to be a lot

more mundane and prosaic than people imagine. Um, I think people imagine trying to sort of make sense of this enormous, you know, multisensory arc of life. But actually if I think about my own life, obviously I've got, I have got long-term goals, you know, I'd like to be healthy when I'm 80, I'd like to have a sufficient pension that I can live in a, you know, living relative comfort and so on. You know, I, I do put money aside and that kind of thing, but I don't have really sort of nitty-gritty detailed, um, plans for that time because I don't know what my interests will be.

I don't know what the world will look like. Um, and I think I actually live a lot of my life, you know, day to day, week to week, month to month with, you know, tasks that you've got to do to get to the next stage, know, maybe you're planning a holiday. Maybe that could be in a few months time. Maybe you've got an aspiration to, you know, change jobs or move house in the next few years. [00:53:00] But I don't think most of us sit back and consider the sort of multi-decade arc of our lives very often. Um, I just think that if I were to live to, even if I had to live to a thousand, obviously that's, you know, an incredibly, uh, that sounds almost Saifai then. I think I'd probably still live my life day to day, week to week, month to month, year to year.

And rarely what I sit back here and practice, perhaps at the age of 900, I might sit back and enjoy. So my achievements, at least I hope I've got some. Um, but I think it will be surprisingly, I think it'd be surprisingly unchanged. And I think about this as well, sort of even looking back in my own life. My, my memory has done a great job of pruning.

A lot of this sort of nonsense, the day-to-day stuff. I just don't need to remember if I try and think back, you know, even to quite significant events in my life. I try and imagine my, my first day at secondary school, I've got sort of flashes. Yeah. I can remember certain incidents and certain images. My first day at university, my first, my first day at work, all these things, even though they're increasingly, you know, near to me in time.

They haven't left this enormous imprint. I can't imagine them in detail. And I sort of imagined [00:54:00] if I get to the age of 300, I, you know, I probably won't remember what I did today. I probably don't remember what I had for breakfast, but, you know, I might remember some of the big, significant events in my life. And I really think that actually it's going to be far more mundane. You're not going to be bored in some completely wacky, psychological state. Of course, this might all fall down. We haven't got any 300 year olds to ask, but as I say, I just think it's going to be much less of a change than people sometimes.

Imagine

Diana Hill: Well, it also, you know, what I think about is just how I would have maybe changed some of the ways I went about things. Uh, when I was younger, if I felt like I had more time. You know, that this sort of like rush through to get your career going so that you can, especially, I think as women, uh, we feel this extreme pressure around at the same time.

That for me, when I'm launching my career, I'm also raising young children in the conflict of that. Right. But if I knew I had a lot more time, then I would do all sorts

of things. I'd like maybe take 10 years off to have children and then get my PhD when I'm, you know, 65 because I got time. [00:55:00] Right. So it may sort of changed the way that we view our lives are just sort of this prescriptive, a stage model of living, where you do your education and then you do your career and then you retire and then you die that we may have a little bit more flexibility and creativity around how we live our lives.
If we knew we had more health span.
Andrew Steele: definitely. And I think that's actually a really refreshing perspective because what a lot of people say is, uh, you know, um, isn't death, a great motivator because it means that you have to squeeze all this stuff and have to, you know, make your greatest achievements. And, you know, you know, that the clock's ticking and it's all going to be over by the age of 80.
That's what motivates people to, you know, get a job or, or, you know, write a great work of fiction or something. But I just don't think that rings true at all. I I've never, once in my life been motivated by my not even impending death, you know, the fact that I've got death coming in five decades or six decades time, that's never been, you know, why I've asked someone out on a date or why I've gone for a, you know, gone for a particular job or apply to a particular university. That just, isn't what drives me from day to day. And I think that's exactly right that, you know, if you did have a bit more time, I think it would give you more space to be creative. [00:56:00] And just talk specifically about sort of women and reproduction I'm at that age. Now, as I say, I'm in my I'm 35 at the moment.
So a lot of my friends are, you know, they've either had kids or they're imminently going to have kids or deciding whether or not they want kids because this is sort of final decision. If we really can slow down the aging process and we can do so globally throughout the body. And I really hope that we can concentrate a bit more and this is actually quite a neglected area and how to extend women's reproductive lifespan.
So I think it's genuinely a feminist issue because, you know, I can see that my male friends, if they, you know, if they haven't got partners that they're just not that worried they can, they can take their time, they can get around to it. And they, when they want to, but women, like you say, it's such a crunch time when it's, you know, doing a PhD or starting your career, um, it's really, really challenging for them.
And so I think if we could extend that window where women could have babies, we weren't gonna have kids, then I just think it will be transformational in terms of gender equality as well.
Diana Hill: Yeah. Well, I don't know if death is the great motivator. I think maybe impermanence can be a motivator of like, you know, what we have right now is going to be constantly changing. And so that can bring you to be [00:57:00] present in, in, in, in the here and now, knowing that everything is going to change, including your perception of aging and what you think aging is going to look like, because it's going to be different than what many of us expect.
And I think because it, because of that, and we haven't even tapped into this, this new science of bio gerontology and why it's so new and why it's not getting

enough respect, which I think is one of your big missions is get us some more respect over here. Um, the policies would benefit from changing to support. Um, some of this anti-aging research. Can you speak a little bit about that and some of your mission, it seems like this is some of your, your deeper values behind this work.

Andrew Steele: definitely. And that's the, that's the fundamental reason I wrote this book really, as, as you mentioned at the very top of, uh, of this chat, I switched into biology because I was a physicist and I sort of understood. I actually, I looked at a graph of humans, likelihood of death with time. And that ultimately is what caused me to change my career.

Not because I was terrified in my own mortality, because you look at [00:58:00] that graph and you think, well, you know, if human risk of death increases so rapidly at such a specific point in our lives, this is the aging process. It's responsible for so much suffering. And if we can intervene in that, that's something that we can do something about.

And I really think genuinely this is the way that I can make the biggest, biggest difference in the world by trying to transform this field. Um, so to give a couple of examples of policy change, the first thing we need to do, I think is just give aging research, more funding. And actually the U S is unusual in that it's got the national Institute of aging, which is this government body that specifically focuses on aging research.

Most countries in the world don't have one of those. So the fact, the very fact that that's sort of almost unique is a, is a problem in itself, but then we

Diana Hill: it the national Institute of Alzheimer's. At one point when I was reading some of yours at the Navy, it's more about, you know, one specific disease and not really, truly about aging that we're putting where we're putting our funding.

Andrew Steele: this is, yeah, there's this running joke in the, in sort of bio gerontology circles. Nia stands for national history on Alzheimer's and the reason is, uh, so if we look at their budget, their budget is about three and a half billion dollars a year. And the first thing to do is to compare that [00:59:00] to the scale of other things in, in the world that might be, uh, you know, affected by aging. And one great example of that is the $4 trillion a year that the us spends on healthcare. And you said just to say that again, three and a half billion versus 4 trillion. So that means less than a thousandth of what I spent on healthcare is spent on the national Institute of aging. And what that means is that, you know, obviously a huge amount of those healthcare costs are treatments and caring for people.

Who've got these chronic diseases of old age, and yet we spend this tiny, tiny fraction on trying to do something about that. And what that means is that, you know, even on a purely economic basis, you would surely want to spend more trying to reduce that enormous 4 trillion bill. Um, before you even get onto the sort of humanitarian sort of side of things I've been talking about mainly so far. And then as you said, the national Alzheimer's client comes because of that three and a half billion dollar budget. Over $2 billion goes to the, um, the neuroscience

division, which is the subset of the NIH that effectively works in Alzheimer's disease. So that means that, you know, over two thirds of the budget goes on dementia.
And then if [01:00:00] you drill down further and further some stuff on like social gerontology, you say how people adapt to becoming older and that kind of thing, all of which is really important work, but the actual aging biology division of that. Only gets about $300 million a year. So that's ballpark $1 per American.
It's this really, really tiny amount of money. And then sort of even worse. I can just carry on this, this Russian dollar of terrible things. It also that the Asian biology division mainly concentrates on understanding aging. And again, this is vitally important, crucial, crucial work, because without that understanding of what aging is, we wouldn't now be in a position where we can say, Oh, you know, there are these drugs that are these treatments we can do to slow it down.
But that means, you know, vastly less than one, 10000th of the cost of us healthcare is spent on actual trying to, you know, public funding for trying to develop treatments for aging. So there's just this massive imbalance between the enormous challenge of the social economic cost of growing older. And the amount of money we're spending on therapies that might be able to do something about it.
So I think the first thing that we need desperately is just more money. And what that means, the reason I wrote this book is because I want people to [01:01:00] be talking about this. I want to raise the profile of bio gerontology. You know, when we're, when it's safe to go back to pubs and bars and dinner parties, I want people to be chatting about this, you know, over their dinner.
I want scientists and doctors to be looking to devote their career, to doing something about this. Cause it's even neglected in scientific and medical circles. And I want policymakers, you know, people in Washington to be chatting about how we can get more money into not just researching the diseases of aging, but the causes, the hallmarks of aging as well.
Diana Hill: Fantastic. Well, so that, that brings us this back to, you know, I, well, first I just want to acknowledge you as a person, man. But, uh, I, you store all those statistics in your brain, whatever, whatever you're taking, we watch them. But I imagine some of this is also, um, you know, you think about this for, you know, in a global way, but you also do this for yourself, right?
And, um, you're a young whippersnapper at 30 something, 35. What, what are you doing on it on a daily basis to keep your ball dribbling down the fields, to the point that you can start taking some of these interventions that we hope will be coming out? [01:02:00] What, what does it look like for you in terms of your anti-aging practices?
Andrew Steele: a lot of us, as I said is sort of the quite basic stuff. I've, you know, I've tried to optimize my diet a bit, which is just a question of getting a nice varied diet. I've tried to reduce my meat consumption a bit, which is partly for environmental reasons as well, just cause it seems like having more plant protein in your diet is probably beneficial.

A lot of these things are quite suggestive. I think, you know, there, there are people who could come in and say, I'm going to blueberries are the new superfood or, you know, whatever miracle diet or combination of foods is going to make you, you know, the keto diet, whatever it is. I really don't think there's a huge amount of evidence for any one specific diets.
It's all about just. Having a variety, not eating too much of it. Um, I've again, I wasn't a complete couch potato before, but nonetheless, I've tried to incorporate a bit more running a bit more, um, a bit more, uh, cycling into my routine just to make sure that I do get those 30 minutes on average of exercise a day.
And another thing I've really tried to improve on is my strength training. And that's something I think is it's very easy to neglect. You know, when you visualize someone getting fit, you can visualize them, you know, running or biking or swimming or whatever it is, but actually your muscle mass declines quite substantially, as you get [01:03:00] older every decade after the age of about 30, I think your muscle mass declines by about 5% and your strength by about 10% every, every 10 years.
And what that means is that by the time you're 70 or 80, you've really lost quite a substantial amount of muscle, but actually a huge part of that decline is to some extent, reversible. In the sense that, you know, if you do do a bit of strength, training, resistance, training, lifting weights, it doesn't have to be lifting weights, but that kind of thing that really exerts your muscles rather than just getting your cardiovascular system going, you can substantially reverse those changes.
I'm not sure. I even saw our paper when I was writing the book about a resistance training program for nonagenarians. So people in their nineties, and they found that after I think it was a couple of months of this resistance training program, they substantially increased their strength. They increased their walking speed.
So it doesn't matter how old you are before you start, you know, you don't have to be in your thirties to start doing this stuff. Um, you know, however old you are, it's really, really important to start trying to do it. So, yeah, I just found it's reinforced the basic health advice. Um, I'm also trying to be a bit more aware of my body, and this is partly, this, this one's partly been motivated by the fact my wife's a doctor, but I think it's really [01:04:00] important to be aware of things about your body that you can't necessarily see or feel.
And a good example of that is measuring my heart rate and blood pressure. And it's not something I do every single day or religiously, but yeah, just occasionally you got a blood pressure cuff in the house. You can pop it on, sit down and find some way in a calm and quiet. Take a few deep breaths for a while just to get your heart into a nice, uh, you know, get everything nice and nice and relaxed, and then just take a blood pressure measurement.
And the reason is that high blood pressure is one of the most common problems and it really is behind so many different aspects of aging. Everything from, you know, heart disease to, you know, even dementia can be accelerated by having these tiny little blood vessels in your brain start to burst because of the high blood pressure.

But it's something that you can't feel until you have in, you know, until you have a heart attack or until he gets a major consequence of it. You don't really notice it because it's not something that gives you any symptoms. And so if you put a blood pressure cuff on and you start to notice, the readings are getting a bit high, if you find that it's happening consistently, the first thing to do is actually to double down on all the basic health advices, to get some exercises, to try and get your weight under control and that kind of thing.
But if you really can't get it below a sort of one 40 over 90 is typically thought of as [01:05:00] the threshold, if that keeps happening consistently, it's not just happening to you. You're a bit stressed out when you put the blood pressure cuff on, um, it's something that's really happening in no matter how calm you are, that it might be worth going and chatting to your doctor about it.
Because if you can get ahead of these health problems, it's exactly like, uh, the anti-ageing medication. You can potentially prevent all kinds of different diseases from coming up down the line.
Diana Hill: Yeah, I love that. Just keeping a monitor on yourself. We keep monitors on our cars. Like we, we have the gas, you know, monitor and other lights that show up for us, but we don't do as much of that for our own bodies. It seems sometimes like we know more about our cars than we do about our own, our own bodies.
I wear a. No. Um, we're not sponsored by aura ring, but I wear this ring and it's, it's been so fascinating for me just to see my own, um, you know, my baseline heart rate, some of the, just sort of basic information about sleep. But then when I got my vaccine to notice how much my aura ring changed, how much all those variables, my temperature, all sorts of things were off the charts.
So demonstrating that something really big just happened to my [01:06:00] body, right. And whether or not those are accurate measurements of what they're supposed to be measuring. They're just data to show that something big is happening. And you kind of want to know that if it does. So keeping some regular tabs on what's happening inside your body can be helpful.
Um, and so cool that you're married to a doctor. So the two of you must have really fun
Andrew Steele: all right. It's been very helpful for that. The poor woman has been subjected to so many hours of conversation when this book was being written. Like it's, it's, it's just so useful to have a live in, you know, biology, medical expert, because obviously, as I say, my background is as a physicist. And the other thing that's really, really useful is that I was a computational biologist.
See, and that's a pretty theoretical thing you spent basically most of your time sat at a desk analyzing data, but she's dealing with old people every day and, you know, That that, that that's part of it. The first thing is that no matter what ward she's rotating on, she's currently doing her sort of the training part, which is very extended thing in the UK is, you know, she's, she's, what's called a junior doctor, but I think most people are a junior doctor until their mid thirties.

So it's not as junior isn't necessarily sound, but I know what ward she's working on. Most of the [01:07:00] people she's seeing are old, and most of those people have this sort of collection of different chronic illnesses. And she's got to try and work out how to deal with them from a social point of view, you have to make sure that, you know, on average, an 80 year old is taking five different drugs and you got to make sure those drugs aren't interacting and causing sort of weird side effects with other, balancing them between all the different diseases.
And you're not pulling in different directions. And yes, I just found that really, really fascinating. I could, I could have some sort of pie in the sky theoretical idea of, well, why don't we just do this? And, you know, such and such would be cured and she'll be like, well, actually in practice, that's going to be incredibly, incredibly difficult.
So it's just really, really useful to have a, you know, a proper doctor who's, you know, seeing all this stuff, living all this stuff, um, it's just, sometimes it brings some of my ideas crashing back to earth.
Diana Hill: Yeah, she's she's in the real world, real world at the front lines of all of this and whatever baby you have. If you guys end up having children, that'll be. Went till they're 500. My goodness. There'll be a smart little cookie. Um, so, well, thank you so much, Andrew. It's just, it's such a treat to have you on.
We like barely scratched the surface of what you write about in your book. It is [01:08:00] a, um, it is a book you want to take your time with and it also, you don't have to understand all of it to get the gist of it and be inspired and motivated and excited about the changes that are are to come.
But also maybe change your mind a little bit about what aging is and, uh, how we can do a better job as a species and in, in, in funding, some of this research, as well as just taking care of our own bodies along the way. So thank you. Um, it's a pleasure to have you on, and we'll link to your book and, uh, some of the YouTube, videos that you have, which are fun.
And, uh, again, it's just, um, thank you for taking your time with
Andrew Steele: Thank you very much. Thank you for taking your time. Is there a pleasure? It's been a great shot. thank you for listening to psychologists off the clock. If you enjoy our podcast, you can help us out by leaving a review or contributing on Patrion.
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